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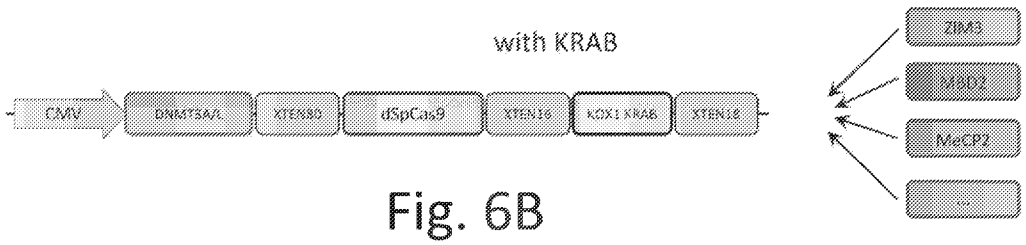

Fig. 6B

(57) Abstract: Disclosed herein are compositions and methods comprising epigenetic editors for epigenetic editing or cells, nucleic acids, and vectors comprising the same. Also disclosed are epigenetically modified chromosomes.

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COMPOSITIONS AND METHODS FOR EPIGENETIC EDITING

CROSS REFERENCE

[0001] This application claims the benefit of U.S. Provisional Application No. 63/129,283, filed December 22, 2020, and U.S. Provisional Application No. 62/280,452, filed November 17, 2021, which are each incorporated herein by reference in its entirety.

BACKGROUND

[0002] Genome editing has been considered a promising therapeutic approach for treatment of genetic disease for over a decade. However, manipulation on the DNA level remains risky given the potential for undesired double stranded breaks, heterogenous repair including large and small insertions and deletions at the intended site, and toxicity.

SUMMARY

[0003] Provided herein are compositions for epigenetic modification related to epigenetic editors and methods of using the same to generate epigenetic modification in target genomes, including those in host cells and organisms, without introducing changes to genomic sequences.

[0004] Described herein is an epigenetic editor comprising a fusion protein, wherein the fusion protein comprises (a) a first DNMT domain; (b) a DNA binding domain; (c) a first repressor domain; and (d) a second repressor domain. In some embodiments, the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene. In some embodiments, the repressor domain specifically binds to an epigenetic effector protein in a cell comprising a target gene and directs the epigenetic editor to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene.

[0005] In some embodiments, the fusion protein further comprises a second DNMT domain. In some embodiments, the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain. In some embodiments, the first DNMT domain is the DNMT3A domain. In some embodiments, the first DNMT domain is the DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT domain. In some embodiments, the human DNMT domain is a human DNMT3A domain. In some embodiments, the human DNMT domain is a human DNMT3L domain. In some embodiments, wherein the first DNMT domain is a mouse DNMT domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3A domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT

domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.

[0006] In some embodiments, the first DNMT domain is a catalytic portion of a DNMT domain. In some embodiments, the second DNMT domain is a catalytic portion of a DNMT domain. In some embodiments, the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.

[0007] In some embodiments, at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320, ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607, ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100, ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677, ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234,

ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2, ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1, RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1, ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2, and fragments thereof. In some embodiments, at least one of the repressor domains is selected from the group consisting of: SEQ ID NO: 67-595. In some embodiments, at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.

[0008] In some embodiments, one of the repressor domains is a KRAB domain. In some embodiments, the KRAB domain is a KOX1 KRAB domain.

[0009] In some embodiments, the DNA binding domain comprises a zinc finger motif. In some embodiments, the DNA binding domain comprises a zinc finger array. In some embodiments, the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide. In some embodiments, the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide. In some embodiments, the guide polynucleotide hybridizes with a target sequence. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9). In some embodiments, the dCas9 is a dSpCas9. In some embodiments, the dSpCas9 is defined as SEQ ID NO: 3. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a). In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).

[0010] In some embodiments, the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-dSpCas9-KOX1KRAB-the second repressor domain. In some embodiments, a linker connects the domains of the fusion protein. In some embodiments, the linker is an XTEN linker. In some embodiments, the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80. In some embodiments, the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-KOX1KRAB- XTEN18-the second repressor domain.

[0011] Also described herein is an epigenetic editor comprising a fusion protein, wherein the fusion protein comprises (a) a first DNMT domain; (b) a DNA binding domain; and (c) a repressor domain, wherein the repressor domain is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320, ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607,

ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100, ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677, ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234, ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2, ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1, RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1,

ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2 and fragments thereof.

[0012] In some embodiments, at least one of the repressor domains is selected from the group consisting of: SEQ ID NO: 67-595. In some embodiments, the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene. In some embodiments, the repressor domain specifically binds to an epigenetic effector protein in a cell comprising a target gene and directs the epigenetic editor to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene. In some embodiments, the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.

[0013] In some embodiments, the fusion protein further comprises a second DNMT domain. In some embodiments, the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain. In some embodiments, the first DNMT domain is the DNMT3A domain. In some embodiments, the first DNMT domain is the DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT domain. In some embodiments, the first human DNMT domain is a human DNMT3A domain. In some embodiments, the human DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3A domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is a catalytic portion of the DNMT domain. In some embodiments, the second DNMT domain is a catalytic portion of a DNMT domain. In some embodiments, the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.

[0014] In some embodiments, the DNA binding domain comprises a zinc finger motif. In some embodiments, the DNA binding domain comprises a zinc finger array. In some embodiments,

the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide. In some embodiments, the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide. In some embodiments, the guide polynucleotide hybridizes with a target sequence. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9). In some embodiments, the dCas9 is a dSpCas9. In some embodiments, the dSpCas9 is defined as SEQ ID NO: 3. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a). In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).

[0015] In some embodiments, the fusion protein domain comprises from N-terminus to C-terminus DNMT3A-DNMT3L-dSpCas9-the repressor domain. In some embodiments, a linker connects the domains of the fusion protein. In some embodiments, the linker is an XTEN linker. In some embodiments, the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80. In some embodiments, the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-the repressor domain.

[0016] Also described herein is an epigenetic editor comprising a fusion protein, wherein the fusion protein comprises (a) a demethylase domain; (b) a DNA binding domain; and (c) an activator domain. In some embodiments, there is increased expression of the target gene when contacted with the epigenetic editor of any of the preceding claims as compared to the target gene not contacted with the epigenetic editor.

[0017] Also described herein is an epigenetic editor comprising a fusion protein, wherein the fusion protein comprises (a) a DNA binding domain; (b) a repressor domain; (c) a first catalytic domain wherein the catalytic domain is selected from the group consisting of a DNMT3A catalytic domain and a DNMT3L catalytic domain; and (d) a second catalytic domain wherein the catalytic domain is selected from the group consisting of a DNMT3A catalytic domain and a DNMT3L catalytic domain, wherein the first catalytic domain has less than 380 amino acids, or wherein the second catalytic domain has less than 380 amino acids.

[0018] Also described herein is a method for modifying an epigenetic state of a target gene in a target chromosome, the method comprising contacting the target chromosome with an epigenetic editor, wherein the epigenetic editor comprises (a) a first DNMT domain; (b) a DNA binding domain; (c) a first repressor domain; and (d) a second repressor domain, and wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby modifying the epigenetic state of the target gene.

[0019] Also described herein is a method for modulating expression of a target gene in a target chromosome, the method comprising contacting the target chromosome with an epigenetic editor, wherein the epigenetic editor comprises (a) a first DNMT domain; (b) a DNA binding domain; (c) a first repressor domain; and a second repressor domain, and wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby modulating the epigenetic state of the target gene.

[0020] Also described herein is a method for treating a disease in a subject in need thereof, the method comprising administering to the subject an epigenetic editor, wherein the epigenetic editor comprises (a) a first DNMT domain; (b) a DNA binding domain; (c) a first repressor domain; and (d) a second repressor domain, wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby treating the disease, wherein the target gene is associated with disease, and wherein the site-specific epigenetic modification modulates expression of the target gene, thereby treating the disease.

[0021] In some embodiments, the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of the target sequence. In some embodiments, the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the target sequence. In some embodiments, the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of an expression regulatory sequence. In some embodiments, the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the expression regulatory sequence. In some embodiments, the site-specific epigenetic modification is within 1000 base pairs upstream or downstream of the expression regulatory sequence.

[0022] In some embodiments, the method comprises administering to the subject a cell comprising the epigenetic editor. In some embodiments, the cell is an allogeneic cell. In some embodiments, the cell is an autologous cell. In some embodiments, the epigenetic modification is within a coding region of the target gene. In some embodiments, the target gene comprises an allele associated with a disease.

[0023] In some embodiments, the fusion protein further comprises a second DNMT domain. In some embodiments, the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain. In some embodiments, the first DNMT domain is the DNMT3A domain. In some embodiments, the first

DNMT domain is the DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT domain. In some embodiments, the human DNMT domain is a human DNMT3A domain. In some embodiments, the human DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3A domain. In some embodiments, the mouse DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain. In some embodiments, the first DNMT domain is the mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain. In some embodiments, the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.

[0024] In some embodiments, the first DNMT domain is a catalytic portion of a DNMT domain. In some embodiments, the second DNMT domain is a catalytic portion of a DNMT domain. In some embodiments, the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.

[0025] In some embodiments, at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320, ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607, ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100,

ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677, ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234, ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2, ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1, RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1, ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2 and fragments thereof. In some embodiments, at least one of the repressor domains is selected from the group consisting

of: SEQ ID NO: 67-595. In some embodiments, at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.

[0026] In some embodiments, one of the repressor domains is a KRAB domain. In some embodiments, the KRAB domain is a KOX1 KRAB domain.

[0027] In some embodiments, the DNA binding domain comprises a zinc finger motif. In some embodiments, the DNA binding domain comprises a zinc finger array. In some embodiments, the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide. In some embodiments, the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide. In some embodiments, wherein the guide polynucleotide hybridizes with a target sequence. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9). In some embodiments, the dCas9 is a dSpCas9. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a). In some embodiments, the dSpCas9 is defined as SEQ ID NO: 3. In some embodiments, the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).

[0028] In some embodiments, the fusion protein comprises from N-terminus to C-terminus DNMT3A-DNMT3L-dSpCas9-KOX1KRAB-the second repressor domain. In some embodiments, a linker connects the domains of the fusion protein. In some embodiments, the linker is an XTEN linker. In some embodiments, the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80. In some embodiments, the fusion protein comprises from N-terminus to C-terminus DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-KOX1KRAB- XTEN18-the second repressor domain.

[0029] Also described herein is a composition for use in the treatment of a subject, the composition comprising a fusion protein, wherein the fusion protein comprises (a) a first DNMT domain; (b) a DNA binding domain; (c)a first repressor domain; and (d) a second repressor domain.

[0030] Additional aspects and advantages of the present disclosure will become readily apparent to those skilled in this art from the following detailed description, wherein only illustrative embodiments of the present disclosure are shown and described. As will be realized, the present disclosure is capable of other and different embodiments, and its several details are capable of modifications in various obvious respects, all without departing from the disclosure. Accordingly, the drawings and description are to be regarded as illustrative in nature, and not as restrictive.

INCORPORATION BY REFERENCE

[0031] All publications, patents, and patent applications mentioned in this specification are herein incorporated by reference to the same extent as if each individual publication, patent, or patent application was specifically and individually indicated to be incorporated by reference. To the extent publications and patents or patent applications incorporated by reference contradict the disclosure contained in the specification, the specification is intended to supersede and/or take precedence over any such contradictory material.

BRIEF DESCRIPTION OF THE DRAWINGS

[0032] The novel features of the invention are set forth with particularity in the appended claims. A better understanding of the features and advantages of the present invention will be obtained by reference to the following detailed description that sets forth illustrative embodiments, in which the principles of the invention are utilized, and the accompanying drawings ("FIGURE." or "FIGURES." herein), of which:

[0033] FIG. 1 is a schematic illustration of an example DNA methylation series plasmid containing a DNMT domain, XTEN80 linker, and a dSpCas9.

[0034] FIG. 2 shows a comparison of the ability of alternate mammalian DNMT effectors and effector fusions to reduce VIM expression in HEK293 cells.

[0035] FIG. 3A-B shows a comparison of the ability of alternate DNMT effectors and effector fusions to reduce VIM expression in HEK293 cells. FIG. 3A compares the ability of the mammalian effector fusions human DNMT3A catalytic domain-mouse DNMT3L catalytic domain and human DNMT3A catalytic domain-human DNMT3L catalytic domain to reduce VIM expression in HEK293 cells to that of plant effectors and effector fusions. FIG. 3B FIG. 3A compares the ability of the mammalian effector fusions human DNMT3A catalytic domain-mouse DNMT3L catalytic domain and human DNMT3A catalytic domain-human DNMT3L catalytic domain to reduce VIM expression in HEK293 cells to that of bacterial, fungal, and *Drosophila* effectors and effector fusions.

[0036] FIG. 4 is a schematic illustration of an example repressor series plasmid containing a dSpCas9, an XTEN80 linker, and a repressor domain.

[0037] FIG. 5 shows a comparison of the ability of alternate KRAB and non-KRAB repressors to effectively silence VIM expression in HEK293 cells.

[0038] FIG. 6A-B are schematic illustrations of the use of alternate KRAB and non-KRAB repressor domains. FIG. 6A is a schematic illustration of an OFF series plasmid containing a DNMT3A/3L domain; an XTEN80 linker, a dSpCas9, an XTEN16 linker, and an alternate

KRAB or non-KRAB repressor domain. FIG. 6B is a schematic illustration of an OFF series plasmid containing a DNMT3A/3L domain; an XTEN80 linker, a dSpCas9, an XTEN16 linker, a KOX1 KRAB domain, an XTEN18 linker, and an alternate KRAB or non-KRAB repressor domain.

[0039] FIG. 7A-7D show the ability of OFF series plasmids with various non-KRAB repressor domains to silence CD151 expression in KEH293 cells. FIG. 7A shows the results of plasmids that do not also contain a KOX1-KRAB domain; FIG 7B shows the results of plasmids that also contain a KOX1-KRAB domain. FIG. 7C shows additional results of plasmids that do not also contain a KOX1-KRAB domain; FIG. 7D shows additional results of plasmids that also contain a KOX1-KRAB domain.

DETAILED DESCRIPTION

[0040] While various embodiments of the disclosure have been shown and described herein, it will be obvious to those skilled in the art that such embodiments are provided by way of example only. Numerous variations, changes, and substitutions may occur to those skilled in the art without departing from the disclosure. It should be understood that various alternatives to the embodiments of the disclosure described herein may be employed.

[0041] The practice of the present invention will employ, unless otherwise indicated, conventional techniques of chemistry, biochemistry, molecular biology, microbiology and immunology, which are within the capabilities of a person of ordinary skill in the art. Such techniques are explained in the literature. See, for example, Sambrook, J., Fritsch, E.F., and Maniatis, T. (1989) Molecular Cloning: A Laboratory Manual, 2nd Edition, Cold Spring Harbor Laboratory Press; Ausubel, F.M. et al. (1995 and periodic supplements) Current Protocols in Molecular Biology, Ch. 9, 13 and 16, John Wiley & Sons; Roe, B., Crabtree, J., and Kahn, A. (1996) DNA Isolation and Sequencing: Essential Techniques, John Wiley & Sons; Polak, J.M., and McGee, J.O'D. (1990) In Situ Hybridization: Principles and Practice, Oxford University Press; Gait, M.J. (1984) Oligonucleotide Synthesis: A Practical Approach, IRL Press; and Lilley, D.M., and Dahlberg, J.E. (1992) Methods in Enzymology: DNA Structures Part A: Synthesis and Physical Analysis of DNA, Academic Press. Each of these general texts is herein incorporated by reference in its entirety.

[0042] Whenever the term “at least,” “greater than,” or “greater than or equal to” precedes the first numerical value in a series of two or more numerical values, the term “at least,” “greater than” or “greater than or equal to” applies to each of the numerical values in that series of

numerical values. For example, greater than or equal to 1, 2, or 3 is equivalent to greater than or equal to 1, greater than or equal to 2, or greater than or equal to 3.

[0043] Whenever the term "no more than," "less than," or "less than or equal to" precedes the first numerical value in a series of two or more numerical values, the term "no more than," "less than," or "less than or equal to" applies to each of the numerical values in that series of numerical values. For example, less than or equal to 3, 2, or 1 is equivalent to less than or equal to 3, less than or equal to 2, or less than or equal to 1.

[0044] Use of absolute or sequential terms, for example, "will," "will not," "shall," "shall not," "must," "must not," "first," "initially," "next," "subsequently," "before," "after," "lastly," and "finally," are not meant to limit scope of the present embodiments disclosed herein but as exemplary.

[0045] As used herein, the singular forms "a", "an" and "the" are intended to include the plural forms as well, unless the context clearly indicates otherwise. Furthermore, to the extent that the terms "including", "includes", "having", "has", "with", or variants thereof are used in either the detailed description and/or the claims, such terms are intended to be inclusive in a manner similar to the term "comprising."

[0046] As used herein, the terms, "clinic," "clinical setting," "laboratory" or "laboratory setting" refer to a hospital, a clinic, a pharmacy, a research institution, a pathology laboratory, a or other commercial business setting where trained personnel are employed to process and/or analyze biological and/or environmental samples. These terms are contrasted with point of care, a remote location, a home, a school, and otherwise non-business, non-institutional setting.

[0047] The terms "determining," "measuring," "evaluating," "assessing," "assaying," and "analyzing" are often used interchangeably herein to refer to forms of measurement. The terms include determining if an element is present or not (for example, detection). These terms can include quantitative, qualitative or quantitative and qualitative determinations. Assessing is relative or absolute. "Detecting the presence of" can include determining the amount of something present in addition to determining whether it is present or absent depending on the context.

[0048] The terms "subject," "patient", or "individual" are often used interchangeably herein. A "subject" may be a biological entity containing expressed genetic materials. The biological entity can be a plant, animal, or microorganism, including, for example, bacteria, viruses, fungi, and protozoa. The subject can be tissues, cells and their progeny of a biological entity obtained in vivo or cultured in vitro. The subject can be a mammal. The mammal can be a human. The subject may be diagnosed or suspected of being at high risk for a disease. In some cases, the

subject is not necessarily diagnosed or suspected of being at high risk for the disease. A subject may or may not have been exposed to a pathogen of interest as described herein, and may by symptomatic or symptomatic of a disease or condition associated with infection of or exposure to a pathogen as described herein. In some embodiments, a subject is suspected to have been exposed to a pathogen, e.g. a virus. In some embodiments, a subject has been exposed to an antigen or a protein representative or cross-reacts with antigens of a particular pathogen, e.g. a virus. In some embodiments, a subject has one or more symptoms that are indicative of a disease or condition associated with infection of or exposure to a pathogen as described herein. In some embodiments, the subject is currently infected by a pathogen, e.g. a virus described herein. In some embodiments, the subject is previously infected by a pathogen described herein. In some embodiments, a subject is a carrier of a virus described herein. In some embodiments, a subject is a carrier of fragments or remnants of a virus described herein. In some instances, a subject is carrier of adaptive immunity stemmed from previously or currently being infected by a virus described herein. In some embodiments, a subject is a carrier of adaptive immunity stemmed from previous or current exposure to a different virus or pathogen other than a virus or pathogen of interest.

[0049] The term "subject" encompasses mammals. Examples of mammals include, but are not limited to, any member of the mammalian class: humans, non-human primates such as chimpanzees, and other apes and monkey species; farm animals such as cattle, horses, sheep, goats, swine; domestic animals such as rabbits, dogs, and cats; laboratory animals including rodents, such as rats, mice and guinea pigs, and the like.

[0050] The term "about" or "approximately" means within an acceptable error range for the particular value as determined by one of ordinary skill in the art, which will depend in part on how the value is measured or determined, e.g., the limitations of the measurement system. For example, "about" can mean within 1 or more than 1 standard deviation, per the practice in the given value. Where particular values are described in the application and claims, unless otherwise stated the term "about" should be assumed to mean an acceptable error range for the particular value.

[0051] As used herein, the phrases "at least one", "one or more", and "and/or" are open-ended expressions that are both conjunctive and disjunctive in operation. For example, each of the expressions "at least one of A, B and C", "at least one of A, B, or C", "one or more of A, B, and C", "one or more of A, B, or C" and "A, B, and/or C" means A alone, B alone, C alone, A and B together, A and C together, B and C together, or A, B and C together.

[0052] The term "nucleic acid" as used herein refers to a polymer containing at least two nucleotides (i.e., deoxyribonucleotides or ribonucleotides) in either single- or double-stranded form and includes DNA and RNA. "Nucleotides" contain a sugar deoxyribose (DNA) or ribose (RNA), a base, and a phosphate group. Nucleotides are linked together through the phosphate groups. "Bases" include purines and pyrimidines, which further include natural compounds adenine, thymine, guanine, cytosine, uracil, inosine, and natural analogs, and synthetic derivatives of purines and pyrimidines, which include, but are not limited to, modifications which place new reactive groups such as, but not limited to, amines, alcohols, thiols, carboxylates, and alkylhalides. Nucleic acids include nucleic acids containing known nucleotide analogs or modified backbone residues or linkages, which are synthetic, naturally occurring, and non-naturally occurring, and which have similar binding properties as the reference nucleic acid. Examples of such analogs and/or modified residues include, without limitation, phosphorothioates, phosphoramidates, methyl phosphonates, chiral-methyl phosphonates, 2'-O-methyl ribonucleotides, and peptide-nucleic acids (PNAs).

[0053] The term "nucleic acid" includes any oligonucleotide or polynucleotide, with fragments containing up to 60 nucleotides generally termed oligonucleotides, and longer fragments termed polynucleotides. A deoxyribooligonucleotide consists of a 5-carbon sugar called deoxyribose joined covalently to phosphate at the 5' and 3' carbons of this sugar to form an alternating, unbranched polymer. DNA may be in the form of, e.g., antisense molecules, plasmid DNA, pre-condensed DNA, a PCR product, vectors, expression cassettes, chimeric sequences, chromosomal DNA, or derivatives and combinations of these groups. A ribooligonucleotide consists of a similar repeating structure where the 5-carbon sugar is ribose. Accordingly, the terms "polynucleotide" and "oligonucleotide" can refer to a polymer or oligomer of nucleotide or nucleoside monomers consisting of naturally-occurring bases, sugars and intersugar (backbone) linkages. The terms "polynucleotide" and "oligonucleotide" can also include polymers or oligomers comprising non-naturally occurring monomers, or portions thereof, which function similarly. Such modified or substituted oligonucleotides are often preferred over native forms because of properties such as, for example, enhanced cellular uptake, reduced immunogenicity, and increased stability in the presence of nucleases.

[0054] The "nucleic acid" described herein may include one or more nucleotide variants, including nonstandard nucleotide(s), non–natural nucleotide(s), nucleotide analog(s), and/or modified nucleotides. Examples of modified nucleotides include, but are not limited to diaminopurine, 5–fluorouracil, 5–bromouracil, 5–chlorouracil, 5–iodouracil, hypoxanthine, xantine, 4–acetylcytosine, 5–(carboxyhydroxylmethyl)uracil, 5–carboxymethylaminomethyl–2–

thiouridine, 5–carboxymethylaminomethyluracil, dihydrouracil, beta–D–galactosylqueosine, inosine, N6–isopentenyladenine, 1–methylguanine, 1–methylinosine, 2,2–dimethylguanine, 2–methyladenine, 2–methylguanine, 3–methylcytosine, 5–methylcytosine, N6–adenine, 7–methylguanine, 5–methylaminomethyluracil, 5–methoxyaminomethyl–2–thiouracil, beta–D–mannosylqueosine, 5'–methoxycarboxymethyluracil, 5–methoxyuracil, 2–methylthio–N6–isopentenyladenine, uracil–5–oxyacetic acid (v), wybutoxosine, pseudouracil, queosine, 2–thiocytosine, 5–methyl–2–thiouracil, 2–thiouracil, 4–thiouracil, 5–methyluracil, uracil–5–oxyacetic acid methylester, 5–methyl–2–thiouracil, 3–(3–amino– 3– N–2–carboxypropyl) uracil, (acp3)w, 2,6–diaminopurine and the like. In some cases, nucleotides may include modifications in their phosphate moieties, including modifications to a triphosphate moiety. Non–limiting examples of such modifications include phosphate chains of greater length (e.g., a phosphate chain having, 4, 5, 6, 7, 8, 9, 10 or more phosphate moieties) and modifications with thiol moieties (e.g., alpha–thiotriphosphate and beta–thiotriphosphates).

[0055] The nucleic acid described herein may be modified at the base moiety (e.g., at one or more atoms that typically are available to form a hydrogen bond with a complementary nucleotide and/or at one or more atoms that are not typically capable of forming a hydrogen bond with a complementary nucleotide), sugar moiety, or phosphate backbone. Backbone modifications can include, but are not limited to, a phosphorothioate, a phosphorodithioate, a phosphoroselenoate, a phosphorodiselenoate, a phosphoroanilothioate, a phosphoraniladate, a phosphoramidate, and a phosphorodiamidate linkage. A phosphorothioate linkage substitutes a sulfur atom for a non-bridging oxygen in the phosphate backbone and delay nuclease degradation of oligonucleotides. A phosphorodiamidate linkage (N3'→P5') allows prevents nuclease recognition and degradation. Backbone modifications can also include having peptide bonds instead of phosphorous in the backbone structure (e.g., N-(2-aminoethyl)-glycine units linked by peptide bonds in a peptide nucleic acid), or linking groups including carbamate, amides, and linear and cyclic hydrocarbon groups. Oligonucleotides with modified backbones are reviewed in Micklefield, Backbone modification of nucleic acids: synthesis, structure and therapeutic applications, Curr. Med. Chem., 8 (10): 1157-79, 2001 and Lyer et al., Modified oligonucleotides-synthesis, properties and applications, Curr. Opin. Mol. Ther., 1 (3): 344-358, 1999. Nucleic acid molecules described herein may contain a sugar moiety that comprises ribose or deoxyribose, as present in naturally occurring nucleotides, or a modified sugar moiety or sugar analog. The examples of modified sugar moieties include, but are not limited to, 2'-O-methyl, 2'-O-methoxyethyl, 2'-O-aminoethyl, 2'-Flouro, N3'→P5' phosphoramidate, 2'dimethylaminooxyethoxy, 2' 2′dimethylaminoethoxyethoxy, 2′-guanidinidium, 2′-O-

guanidinium ethyl, carbamate modified sugars, and bicyclic modified sugars. 2'-O-methyl or 2'-O-methoxyethyl modifications promote the A-form or RNA-like conformation in oligonucleotides, increase binding affinity to RNA, and have enhanced nuclease resistance. Modified sugar moieties can also include having an extra bridge bond (e.g., a methylene bridge joining the 2'-O and 4'-C atoms of the ribose in a locked nucleic acid) or sugar analog such as a morpholine ring (e.g., as in a phosphorodiamidate morpholino).

[0056] Unless otherwise indicated, a particular nucleic acid sequence also implicitly encompasses conservatively modified variants thereof (e.g., degenerate codon substitutions), alleles, orthologs, SNPs, and complementary sequences as well as the sequence explicitly indicated. Specifically, degenerate codon substitutions may be achieved by generating sequences in which the third position of one or more selected (or all) codons is substituted with mixed-base and/or deoxyinosine residues (Batzer et al., Nucleic Acid Res., 19:5081 (1991); Ohtsuka et al., J. Biol. Chem., 260:2605-2608 (1985); Rossolini et al., Mol. Cell. Probes, 8:91-98 (1994).

[0057] The present disclosure encompasses isolated or substantially purified nucleic acid molecules and compositions containing those molecules. As used herein, an "isolated" or "purified" DNA molecule or RNA molecule is a DNA molecule or RNA molecule that exists apart from its native environment. An isolated DNA molecule or RNA molecule may exist in a purified form or may exist in a non-native environment such as, for example, a transgenic host cell. For example, an "isolated" or "purified" nucleic acid molecule or biologically active portion thereof, is substantially free of other cellular material, or culture medium when produced by recombinant techniques, or substantially free of chemical precursors or other chemicals when chemically synthesized. In one embodiment, an "isolated" nucleic acid is free of sequences that naturally flank the nucleic acid (i.e., sequences located at the 5' and 3' ends of the nucleic acid) in the genomic DNA of the organism from which the nucleic acid is derived. For example, in some embodiments, the isolated nucleic acid molecule can contain less than about 5 kb, 4 kb, 3 kb, 2 kb, 1 kb, 0.5 kb, or 0.1 kb of nucleotide sequences that naturally flank the nucleic acid molecule in genomic DNA of the cell from which the nucleic acid is derived.

[0058] As used herein, the terms "protein," "polypeptide," and "peptide" are used interchangeably and refer to a polymer of amino acid residues linked via peptide bonds and which may be composed of two or more polypeptide chains. The terms "polypeptide," "protein," and "peptide" refer to a polymer of at least two amino acid monomers joined together through amide bonds. An amino acid may be the L–optical isomer or the D–optical isomer. More specifically, the terms "polypeptide," "protein," and "peptide" refer to a molecule composed of two or more amino acids in a specific order; for example, the order as determined by the base

sequence of nucleotides in the gene or RNA coding for the protein. Proteins are essential for the structure, function, and regulation of the body's cells, tissues, and organs, and each protein has unique functions. Examples are hormones, enzymes, antibodies, and any fragments thereof. In some cases, a protein can be a portion of the protein, for example, a domain, a subdomain, or a motif of the protein. In some cases, a protein can be a variant (or mutation) of the protein, wherein one or more amino acid residues are inserted into, deleted from, and/or substituted into the naturally occurring (or at least a known) amino acid sequence of the protein. A polypeptide can be a single linear polymer chain of amino acids bonded together by peptide bonds between the carboxyl and amino groups of adjacent amino acid residues. Polypeptides can be modified, for example, by the addition of carbohydrate, phosphorylation, etc. Proteins can comprise one or more polypeptides.

[0059] A protein or a variant thereof can be naturally occurring or recombinant. Methods for detection and/or measurement of polypeptides in biological material are well known in the art and include, but are not limited to, Western–blotting, flow cytometry, ELISAs, RIAs, and various proteomics techniques. An exemplary method to measure or detect a polypeptide is an immunoassay, such as an ELISA. This type of protein quantitation can be based on an antibody capable of capturing a specific antigen, and a second antibody capable of detecting the captured antigen. Exemplary assays for detection and/or measurement of polypeptides are described in Harlow, E. and Lane, D. Antibodies: A Laboratory Manual, (1988), Cold Spring Harbor Laboratory Press.

[0060] As used herein, the terms "fragment," or equivalent terms can refer to a portion of a protein that has less than the full length of the protein and optionally maintains the function of the protein. Further, when the portion of the protein is blasted against the protein, the portion of the protein sequence can align, for example, at least with 80% identity to a part of the protein sequence.

[0061] Any systems, methods, and platforms described herein are modular and not limited to sequential steps. Accordingly, terms such as "first" and "second" do not necessarily imply priority, order of importance, or order of acts.

[0062] The term "modulate" refers to a change in the quantity, degree or extent of a function. For example, the compositions for epigenetic modification disclosed herein may modulate the activity of a promoter sequence by binding to a motif within the promoter, thereby inducing, enhancing or suppressing transcription of a gene operatively linked to the promoter sequence. Alternatively, modulation may include inhibition of transcription of a gene wherein the epigenetic editor binds to the structural gene and blocks DNA dependent RNA polymerase from

reading through the gene, thus inhibiting transcription of the gene. The structural gene may be a normal cellular gene or an oncogene, for example. Alternatively, modulation may include inhibition of translation of a transcript. Thus, "modulation" of gene expression includes both gene activation and gene repression.

[0063] The term "Administering" and its grammatical equivalents as used herein can refer to providing one or more replication competent recombinant adenovirus or pharmaceutical compositions described herein to a subject or a patient. By way of example and without limitation, "administering" can be performed by intravenous (i.v.) injection, sub-cutaneous (s.c.) injection, intradermal (i.d.) injection, intraperitoneal (i.p.) injection, intramuscular (i.m.) injection, intravascular injection, infusion (inf.), oral routes (p.o.), topical (top.) administration, or rectal (p.r.) administration. One or more such routes can be employed. Parenteral administration can be, for example, by bolus injection or by gradual perfusion over time.

[0064] The terms "treat," "treating," or "treatment," and grammatical equivalents as used herein, can include alleviating, abating, or ameliorating at least one symptom of a disease or a condition, preventing additional symptoms, inhibiting the disease or the condition, e.g., arresting the development of the disease or the condition, relieving the disease or the condition, causing regression of the disease or the condition, relieving a condition caused by the disease or the condition, or stopping the symptoms of the disease or the condition either prophylactically and/or therapeutically. "Treating" may refer to administration of a vector, nucleic acid (e.g. mRNA), or LNP composition to a subject after the onset, or suspected onset, of a disease or condition. "Treating" includes the concepts of "alleviating," which refers to lessening the frequency of occurrence or recurrence, or the severity, of any symptoms or other ill effects related to a disease or condition and/or the side effects associated with the disease or condition. The term "treating" also encompasses the concept of "managing" which refers to reducing the severity of a particular disease or disorder in a patient or delaying its recurrence, e.g., lengthening the period of remission in a patient who had suffered from the disease. The term "treating" further encompasses the concept of "prevent," "preventing," and "prevention." It is appreciated that, although not precluded, treating a disorder or condition does not require that the disorder, condition, or symptoms associated therewith be completely eliminated. The term "treatment" as used herein covers any treatment of a disease in a mammal, particularly, a human, and includes: (a) preventing the disease from occurring in a subject which may be predisposed to the disease but has not yet been diagnosed as having it; (b) inhibiting the disease, i.e., arresting its development; or (c) relieving the disease, i.e., mitigating or ameliorating the

disease and/or its symptoms or conditions. The term "prophylaxis" is used herein to refer to a measure or measures taken for the prevention or partial prevention of a disease or condition.

[0065] By "treating or preventing a condition" is meant ameliorating any of the conditions or signs or symptoms associated with the disorder before or after it has occurred. For example, as compared with an equivalent untreated control, alleviating a symptom of a disorder may involve reduction or degree of prevention at least 3%, 5%, 10%, 20%, 40%, 50%, 60%, 80%, 90%, 95%, 98%, 99%, 99.5%, 99.9%, or 100% as measured by any standard technique. In some embodiments, alleviating a symptom of a disorder may involve reduction or degree of prevention by at least 2 fold, at least 3 fold, at least 4 fold, at least 5 fold, at least 10 fold, at least 20 fold, at least 25 fold, at least 30 fold, at least 40 fold, at least 50 fold, at least 60 fold, at least 70 fold, at least 80 fold, at least 90 fold, at least 100 fold, at least 200 fold, at least 300 fold, at least 400 fold, at least 500 fold, at least 600 fold, at least 700 fold, at least 800 fold, at least 900 fold, at least 1000 fold, at least 2000 fold, at least 3000 fold, at least 4000 fold, at least 5000 fold, at least 6000 fold, at least 7000 fold, at least 8000 fold, at least 9000 fold, or at least 10000 fold as compared with an equivalent untreated control.

[0066] The terms "pharmaceutical composition" and its grammatical equivalents as used herein can refer to a mixture or solution comprising a therapeutically effective amount of an active pharmaceutical ingredient together with one or more pharmaceutically acceptable excipients, carriers, and/or a therapeutic agent to be administered to a subject, e.g., a human in need thereof.

[0067] The term "pharmaceutically acceptable" and its grammatical equivalents as used herein can refer to an attribute of a material which is useful in preparing a pharmaceutical composition that is generally safe, non–toxic, and neither biologically nor otherwise undesirable and is acceptable for veterinary as well as human pharmaceutical use. "Pharmaceutically acceptable" can refer a material, such as a carrier or diluent, which does not abrogate the biological activity or properties of the compound, and is relatively nontoxic, i.e., the material may be administered to a subject without causing undesirable biological effects or interacting in a deleterious manner with any of the components of the pharmaceutical composition in which it is contained.

[0068] A "pharmaceutically acceptable excipient, carrier, or diluent" refers to an excipient, carrier, or diluent that can be administered to a subject, together with an agent, and which does not destroy the pharmacological activity thereof and is nontoxic when administered in doses sufficient to deliver a therapeutic amount of the agent.

[0069] A "pharmaceutically acceptable salt" may be an acid or base salt that is generally considered in the art to be suitable for use in contact with the tissues of human beings or animals without excessive toxicity, irritation, allergic response, or other problem or complication. Such

salts include mineral and organic acid salts of basic residues such as amines, as well as alkali or organic salts of acidic residues such as carboxylic acids. Specific pharmaceutical salts include, but are not limited to, salts of acids such as hydrochloric, phosphoric, hydrobromic, malic, glycolic, fumaric, sulfuric, sulfamic, sulfanilic, formic, toluenesulfonic, methanesulfonic, benzene sulfonic, ethane disulfonic, 2-hydroxyethyl sulfonic, nitric, benzoic, 2-acetoxybenzoic, citric, tartaric, lactic, stearic, salicylic, glutamic, ascorbic, pamoic, succinic, fumaric, maleic, propionic, hydroxymaleic, hydroiodic, phenylacetic, alkanoic such as acetic, HOOC-(CH2)n-COOH where n is 0-4, and the like. Similarly, pharmaceutically acceptable cations include, but are not limited to sodium, potassium, calcium, aluminum, lithium and ammonium. Those of ordinary skill in the art will recognize from this disclosure and the knowledge in the art that further pharmaceutically acceptable salts include those listed by Remington's Pharmaceutical Sciences, 17th ed., Mack Publishing Company, Easton, PA, p. 1418 (1985). In general, a pharmaceutically acceptable acid or base salt can be synthesized from a parent compound that contains a basic or acidic moiety by any conventional chemical method. Briefly, such salts can be prepared by reacting the free acid or base forms of these compounds with a stoichiometric amount of the appropriate base or acid in an appropriate solvent.

[0070] As used herein, the term “therapeutically effective amount” means an amount of an agent to be delivered (e.g., nucleic acid, drug, payload, composition, therapeutic agent, diagnostic agent, prophylactic agent, etc.) that is sufficient, when administered to a subject suffering from or susceptible to an infection, disease, disorder, and/or condition, to treat, improve symptoms of, diagnose, prevent, and/or delay the onset of the infection, disease, disorder, and/or condition.

[0071] The term "repressor domain" or “repression domain” are terms known in the art. Such domains typically refer to a part of a transcription repression protein which provides for the transcriptional repressive effect on a target gene, for example by participating in a reaction on the DNA or chromatin (e.g., methylation), by binding to an agent from within the nucleus to result in the repression of the transcription of the target gene or by inhibiting the recruitment of a protein in the natural transcriptional machinery that transcribes the target gene. Examples of repressor domains of this invention are provided through the specification.

[0072] The term "KRAB" or “KRAB domain” is a term known in the art. KRAB is also known as Krüppel associated box, a transcription repressor domain. A description of KRAB domains, including their function and use, may be found, for example, in Ecco, G., Imbeault, M., Trono, D., KRAB zinc finger proteins, Development 144, 2017 and Lambert SA, Jolma A, Campitelli LF, Das PK, Yin Y, Albu M, Chen X, Taipale J, Hughes TR, Weirauch MT, 2018, The human transcription factors, *Cell* **172**: 650–665, 10.1016/j.cell.2018.01.029, which are incorporated by

reference in their entirety. Examples of KRAB domains are also provided throughout the specification.

[0073] The term "DNMT" is a term known in the art. DNMT is also known as DNA methyltransferase. DNMT refers to an enzyme that catalyzes the transfer of a methyl group to DNA. Non-limiting examples of DNA methyltransferases include DNMT, DNMT3A, DNMT3B, DNMT3C and DNMT3L. In one preferred embodiment, a catalytic domain(s) of a DNMT is used in the invention.

[0074] The term "DNA binding domain" is a term known in the art. DNA binding domain typically refers to a part of a protein which binds to DNA in a nucleus. In one embodiment of this invention, a DNA-binding domain is a DNA binding region of a protein selected from a CRISPR Cas protein, a TAL protein, a zinc finger protein, a transcription repression protein, a transcription activation protein, or an variants thereon that bind DNA.

[0075] Ranges provided herein are understood to be shorthand for all of the values within the range. For example, a range of 1 to 50 is understood to include any number, combination of numbers, or sub-range from the group consisting of 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, or 50, as well as all intervening decimal values between the aforementioned integers such as, for example, 1.1, 1.2, 1.3, 1.4, 1.5, 1.6, 1.7, 1.8, and 1.9. With respect to sub-ranges, “nested sub-ranges” that extend from either end point of the range are specifically contemplated. For example, a nested sub-range of an exemplary range of 1 to 50 may comprise 1 to 10, 1 to 20, 1 to 30, and 1 to 40 in one direction, or 50 to 40, 50 to 30, 50 to 20, and 50 to 10 in the other direction.

[0076] The term “therapeutic agent” can refer to any agent that, when administered to a subject, has a therapeutic, diagnostic, and/or prophylactic effect and/or elicits a desired biological and/or pharmacological effect. Therapeutic agents can also be referred to as “actives” or “active agents.” Such agents include, but are not limited to, cytotoxins, radioactive ions, chemotherapeutic agents, small molecule drugs, proteins, and nucleic acids.

[0077] The term “ameliorate” as used herein can refer to decrease, suppress, attenuate, diminish, arrest, or stabilize the development or progression of a disease.

[0078] As used therein, “delaying” the development of a disease means to defer, hinder, slow, retard, stabilize, and/or postpone progression of the disease. This delay can be of varying lengths of time, depending on the history of the disease and/or individuals being treated. A method that “delays” or alleviates the development of a disease, or delays the onset of the disease, is a method that reduces probability of developing one or more symptoms of the disease in a given

time frame and/or reduces extent of the symptoms in a given time frame, when compared to not using the method. Such comparisons are typically based on clinical studies, using a number of subjects sufficient to give a statistically significant result.

[0079] "Development" or "progression" of a disease means initial manifestations and/or ensuing progression of the disease. Development of the disease can be detectable and assessed using standard clinical techniques as well known in the art. However, development also refers to progression that may be undetectable. For purpose of this disclosure, development or progression refers to the biological course of the symptoms. "Development" includes occurrence, recurrence, and onset.

[0080] As used herein, "onset" or "occurrence" of a disease includes initial onset and/or recurrence. Conventional methods, known to those of ordinary skill in the art of medicine, can be used to administer the isolated polypeptide or pharmaceutical composition to the subject, depending upon the type of disease to be treated or the site of the disease. This composition can also be administered via other conventional routes, e.g., administered orally, parenterally, by inhalation spray, topically, rectally, nasally, buccally, vaginally or via an implanted reservoir.

[0081] The term "parenteral" as used herein includes subcutaneous, intracutaneous, intravenous, intramuscular, intraarticular, intraarterial, intrasynovial, intrastemal, intrathecal, intralesional, and intracranial injection or infusion techniques. In addition, it can be administered to the subject via injectable depot routes of administration such as using 1-, 3-, or 6-month depot injectable or biodegradable materials and methods.

[0082] It will be understood that in addition to the specific proteins and nucleotides mentioned herein, the present invention also contemplates the use of variants, derivatives, homologues and fragments thereof. As used herein, a variant of any given sequence is a sequence in which the specific sequence of residues (whether amino acid or nucleic acid residues) has been modified in such a manner that the polypeptide or polynucleotide in question substantially retains at least one of its endogenous functions. A variant sequence can be obtained by addition, deletion, substitution, modification, replacement and/or variation of at least one residue present in the naturally-occurring protein. As used herein, a derivative of any given sequence as contemplated includes any substitution of, variation of, modification of, replacement of, deletion of and/or addition of one (or more) amino acid residues from or to the sequence providing that the resultant protein or polypeptide substantially retains at least one of its endogenous functions. Amino acid substitutions may be made, for example from 1, 2 or 3 to 10 or 20 substitutions provided that the modified sequence substantially retains the required activity or ability. Amino acid substitutions may include the use of non-naturally occurring analogues. Proteins used in the

present disclosure may also have deletions, insertions or substitutions of amino acid residues which do not affection function of the protein and result in a functionally equivalent protein. Deliberate amino acid substitutions may be made on the basis of similarity in polarity, charge, solubility, hydrophobicity, hydrophilicity and/or the amphipathic nature of the residues as long as the endogenous function is retained. For example, negatively charged amino acids include aspartic acid and glutamic acid; positively charged amino acids include lysine and arginine; and amino acids with uncharged polar head groups having similar hydrophilicity values include asparagine, glutamine, serine, threonine and tyrosine.

[0083] As used herein, a homologue of any herein contemplated protein or nucleic acid sequence includes sequences having a certain homology with the wild type amino acid and nucleic sequence. A homologous sequence may include a sequence, e.g. an amino acid sequence which may be at least 50%, 55%, 65%, 75%, 85% or 90% identical to the subject sequence. In particular embodiments, a homologous sequence may include an amino acid sequence at least 95% or 97% or 99% identical to the subject sequence.

[0084] Sequence identity may be measured using sequence analysis software (for example, Sequence Analysis Software Package of the Genetics Computer Group, University of Wisconsin Biotechnology Center, 1710 University Avenue, Madison, Wis. 53705, BLAST, BESTFIT, GAP, or PILEUP/PRETTYBOX programs). Such software matches identical or similar sequences by assigning degrees of homology to various substitutions, deletions, and/or other modifications. Conservative substitutions typically include substitutions within the following groups: glycine, alanine; valine, isoleucine, leucine; aspartic acid, glutamic acid, asparagine, glutamine; serine, threonine; lysine, arginine; and phenylalanine, tyrosine. In an exemplary approach to determining the degree of identity, a BLAST program may be used, with a probability score between e-3 and e-100 indicating a closely related sequence.

[0085] It will be understood that the numbering of the specific positions or residues in the respective sequences depends on the particular protein and numbering scheme used. Numbering might be different, e.g., in precursors of a mature protein and the mature protein itself, and differences in sequences from species to species may affect numbering. One of skill in the art will be able to identify the respective residue in any homologous protein and in the respective encoding nucleic acid by methods well known in the art, e.g., by sequence alignment and determination of homologous residues.

Nucleic acid binding domains

[0086] Epigenetic editors and epigenetic editing complexes described herein may comprise one or more nucleic acid binding protein domains, e.g. DNA binding domains, that may direct the epigenetic editor to a target gene associated with a certain condition.

[0087] As used herein, a target gene can comprise all nucleotide sequences of a gene of interest. For example, sequences or nucleotides of a target gene can include coding sequences and non-coding sequences. Sequence of a target gene can include exons or introns. Sequences of a target gene can include regulatory regions, including promoters, enhancers, terminators, 5' or 3' untranslated regions. In some embodiments, a sequence of a target gene comprises a remote enhancer sequence.

[0088] An epigenetic editor as described herein can comprise any polynucleotide binding domain. In some embodiments, the nucleic acid binding domain comprises one or more DNA binding proteins, for example, zinc finger proteins (ZFPs) or transcription activator like effectors (TALEs). In some embodiments, the nucleic acid binding domain comprises a polynucleotide guided DNA binding protein, for example, a nuclease inactive CRISPR-Cas protein guided by a guide RNA.

[0089] The nucleic acid binding domain of epigenetic editors described herein may be capable of recognizing and binding any gene of interest, for example, target genes associated with a disease or disorder. In some embodiments, the target gene associated with a disease or disorder contains a mutation as compared to a wild type gene. In some embodiments, the target gene associated with a disease or disorder contains a copy that harbors a mutation associated with the disease or disorder. In some embodiments, the target gene associated with a disease or disorder has one or both copies of wild type DNA sequences.

[0090] A DNA binding domain maybe modular and/or programmable. In some embodiments, the DNA binding domain comprises a zinc finger domain, a transcription activator like effector (TALE) domain, a meganuclease DNA binding domain or a polynucleotide guided nucleic acid binding domain. Examples of DNA binding domains can be found in US Patent No. 11,162,114, which is incorporated by refence in its entirety.

[0091] Transcription activator-like effectors (TALEs) can be engineered to bind practically any desired DNA sequence. Methods for programming TALEs are familiar to one skilled in the art. For example, such methods are described in Carroll et al, Genetics Society of America, 188 (4): 773-782, 2011; Miller et al., Nature Biotechnology 25 (7): 778-785, 2007; Christian et al, Genetics 186 (2): 757-61, 2008; Li et al, Nucleic Acids Res. 39 (1): 359-372, 2010; and Moscou et al, Science 326 (5959): 1501, 2009, each of which are incorporated herein by reference.

[0092] A DNA binding domain may be directed by a nucleic acid sequence, for example, a RNA sequence, to identify the target gene. In some embodiments, the DNA binding domain comprises a programmable nuclease. In some embodiments, the DNA binding domain comprises a programmable nuclease with reduced or abrogated nuclease activity. For example, a programmable nuclease may harbor one or two mutations in its catalytic domain that renders the nuclease inactive, but maintain DNA binding activity of the nuclease. In some embodiments, the DNA binding domain comprises a CRISPR-Cas protein domain. In some embodiments, the CRISPR-Cas protein domain lacks or has reduced nuclease activity.

[0093] In some embodiments, an epigenetic editor provided herein comprises a Cas protein, e.g. a Cas9 protein domain. The Cas9 domain may be any of the Cas9 domains or Cas9 proteins (e.g., nuclease inactive Cas9 or Cas9 nickase, or a Cas9 variant from any species) provided herein. In some embodiments, any of the Cas domains or Cas proteins provided herein may be fused with one or more any effector protein domain as described herein. In some embodiments, any of the Cas protein domains provided herein may be fused with two or more effector protein domains as described herein. Cas9 can refer to a polypeptide with at least about 50%, 60%, 70%, 80%, 90%, 100% sequence identity and/or sequence similarity to a wild type exemplary Cas9 polypeptide (e.g., from S. pyogenes). Cas9 can refer to the wild type or a modified form of the Cas9 protein that can comprise an amino acid change such as a deletion, insertion, substitution, variant, mutation, fusion, chimera, or any combination thereof.

[0094] Cas9 sequences and structures of variant Cas9 orthologs have been described in various species. Exemplary species that the Cas9 protein or other components can be from include, but are not limited to, Streptococcus pyogenes, Streptococcus thermophilus, Streptococcus sp., Staphylococcus aureus, Listeria innocua, Lactobacillus gasseri, Francisella novicida, Wolinella succinogenes, Sutterella wadsworthensis, Gamma proteobacterium, Neisseria meningitidis, Campylobacter jejuni, Pasteurella multocida, Fibrobacter succinogene, Rhodospirillum rubrum, Nocardiopsis dassonvillei, Streptomyces pristinaespiralis, Streptomyces viridochromogenes, Streptomyces viridochromogenes, Streptosporangium roseum, Alicyclobacillus acidocaldarius, Bacillus pseudomycoides, Bacillus selenitireducens, Exiguobacterium sibiricum, Lactobacillus delbrueckii, Lactobacillus salivarius, Lactobacillus buchneri, Treponema denticola, Microscilla marina,Burkholderiales bacterium, Polar omonas naphthalenivorans, Polar omonas sp., Crocosphaera watsonii, Cyanothece sp., Microcystis aeruginosa, Synechococcus sp., Acetohalobium arabaticum, Ammonifex degensii, Caldicelulosiruptor becscii, Candidatus Desulforudis, Clostridium botulinum, Clostridium difficile, Finegoldia magna, Natranaerobius thermophilus, Pelotomaculum thermopropionium, Acidithiobacillus caldus, Acidithiobacillus

ferrooxidans, Allochromatium vinosum, Marinobacter sp., Nitrosococcus halophilus, Nitrosococcus watsoni, Pseudoalteromonas haloplanktis, Ktedonobacter racemifer, Methanohalobium evestigatum, Anabaena variabilis, Nodularia spumigena, Nostoc sp., Arthrospira maxima, Arthrospira platensis, Arthrospira sp., Lyngbya sp., Microcoleus chthonoplastes, Oscillator ia sp., Petrotoga mobilis, Thermosipho africanus, Streptococcus pasteurianus, Neisseria cinerea, Campylobacter lari, Parvibaculum lavamentivorans, Coryne bacterium diphtheria, or Acaryochloris marina. In some embodiments, the Cas9 protein is from Streptococcus pyogenes. In some embodiments, the Cas9 protein may be from Streptococcus thermophilus. In some embodiments, the Cas9 protein is from Staphylococcus aureus.

[0095] Additional suitable Cas9 proteins, orthologs, variants, including nuclease inactive variants and sequences will be apparent to those of skill in the art based on this disclosure, and such Cas9 nucleases and sequences include Cas9 sequences from the organisms and loci disclosed in Chylinski et al., (2013) RNA Biology 10:5, 726-737; which are incorporated herein by reference.

[0096] In some embodiments, wild-type Cas9 corresponds to Cas9 from Streptococcus pyogenes (NCBI Reference Sequence: NC_002737.2 (SEQ ID NO.: 1); and Uniprot Reference Sequence: Q99ZW2 (SEQ ID NO.: 2).

[0097] An epigenetic editor may comprise a nuclease inactive Cas9 domain (dead Cas9 or dCas9). The dCas9 protein domain may comprise one, two, or more mutations as compared to a wild type Cas9 that abrogate its nuclease activity, but retains the DNA binding activity. For example, the DNA cleavage domain of Cas9 is known to include two subdomains, the HNH nuclease subdomain and the RuvC1 subdomain. The HNH subdomain cleaves the strand complementary to the gRNA, whereas the RuvC1 subdomain cleaves the non-complementary strand. Mutations within these subdomains can silence the nuclease activity of Cas9. For example, the mutations D10A and H840A completely inactivate the nuclease activity of S. pyogenes Cas9. In some embodiments, the dCas9 comprises at least one mutation in the HNH subdomain and the RuvC subdomain that reduces or abrogates nuclease activity. In some embodiments, the dCas9 only comprises a RuvC subdomain. In some embodiments, the dCas9 only comprises a HNR subdomain. It is to be understood that any mutation that inactivates the RuvC or the HNH domain may be included in a dCas9, e.g., insertion, deletion, or single or multiple amino acid substitution in the RuvC domain and/or the HNH domain.

[0098] In some embodiments, the dCas9 protein comprises a mutation at position D10 as numbered in the wild type Cas9 sequence as numbered in Uniprot Reference Sequence Q99ZW2.In some embodiments, the dCas9 protein comprises a mutation at position H840 as

numbered in Uniprot Reference Sequence: Q99ZW2. In some embodiments, the dCas9 protein comprises a D10A mutation as numbered in Uniprot Reference Sequence: Q99ZW2. In some embodiments, the dCas9 protein comprises a H840A mutation as numbered in Uniprot Reference Sequence: Q99ZW2. In some embodiments, the dCas9 protein comprises a D10A and a H840A mutation as numbered in Uniprot Reference Sequence: Q99ZW2. In some embodiments, a nuclease inactive Cas9 comprises the amino acid sequence of dCas9 (D10A and H840A) (SEQ ID NO.: 3).

[0099] Additional suitable mutations that inactivate Cas9 will be apparent to those of skill in the art based on this disclosure and knowledge in the field and are within the scope of this disclosure. Such additional exemplary suitable nuclease-inactive Cas9 domains include, but are not limited to, D839A, N863A, and/or K603R. Cas9, dCas9, or Cas9 variant also encompasses Cas9, dCas9, or Cas9 variants from any organism. Also appreciated is that dCas9, Cas9 nickase, or other appropriate Cas9 variants from any organisms may be used in accordance with the present disclosure.

[0100] In some embodiments, an epigenetic editor comprises a high fidelity Cas9 domain. For example, high fidelity Cas9 domains comprising one or more mutations that decrease electrostatic interactions between the Cas9 domain and the sugar-phosphate backbone of DNA may be incorporated in an epigenetic editor to confer increased target binding specificity as compared to a corresponding wild-type Cas9 domain. Without wishing to be bound by any particular theory, high fidelity Cas9 domains that have decreased electrostatic interactions with the sugar-phosphate backbone of DNA may have less off-target effects. In some embodiments, the Cas9 domain comprises one or more mutations that decreases the association between the Cas9 domain and the sugar-phosphate backbone of DNA. In some embodiments, a Cas9 domain comprises one or more mutations that decreases the association between the Cas9 domain and the sugar-phosphate backbone of DNA by at least 1%, at least 2%, at least 3%, at least 4%, at least 5%, at leastl0%, at least 15%, at least 20%, at least 25%, at least 30%, at least 35%, at least 40%, at least 45%, at least 50%, at least 55%, at least 60%, at least 65%, at least 70%, or more. In some embodiments, a high fidelity Cas9 domain comprises one or more of N497X, R661X, Q695X, and/or Q926X mutation as numbered in the wild type Cas9 amino acid sequence Uniprot Reference Sequence: Q99ZW2 or a corresponding amino acid in another Cas9, wherein X is any amino acid. In some embodiments, a high fidelity Cas9 domain comprises one or more of N497A, R661A, Q695A, and/or Q926A mutation of the amino acid sequence provided in the wild type Cas9 sequence, or a corresponding mutation as numbered in the wild type Cas9 amino acid sequence Uniprot Reference Sequence: Q99ZW2 or a corresponding amino acid in another

Cas9. It should be appreciated that any of the epigenetic editors provided herein, for example, any of the epigenetic activators or repressors provided herein, may be converted into high fidelity epigenetic editors by modifying the Cas9 domain as described. In preferred embodiments, the high fidelity Cas9 domain is a nuclease inactive Cas9 domain.

[0101] In some embodiments, a DNA binding domain in an epigenetic editor is a CRISPR protein that recognizes a protospacer adjacent motif (PAM) sequence in a target gene. A CRISPR protein may recognize a naturally occurring or canonical PAM sequence or may have altered PAM specificities. Cas9 domains that bind to non-canonical PAM sequences have been described in the art and would be apparent to the skilled artisan. For example, Cas9 domains that bind non-canonical PAM sequences have been described in Kleinstiver, B. P., et al., "Engineered CRISPR-Cas9 nucleases with altered PAM specificities" Nature 523, 481-485 (2015); and Kleinstiver, B. P., et ah, "Broadening the targeting range of Staphylococcus aureus CRISPR-Cas9 by modifying PAM recognition" Nature Biotechnology 33, 1293-1298 (2015); the entire contents of each are hereby incorporated by reference.

[0102] In some embodiments, the Cas9 domain is a Cas9 domain from S. pyogenes (SpCas9). In some embodiments, a SpCas9 recognizes a canonical NGG PAM sequence where the "N" in "NGG" is adenine (A), thymine (T), guanine (G), or cytosine (C), and the G is guanine. In some embodiments, an epigenetic editor or fusion protein provided herein contains a SpCas9 domain that is capable of binding a nucleotide sequence that does not contain a canonical (e.g., NGG) PAM sequence. In some embodiments, the SpCas9 domain, the nuclease inactive SpCas9 domain, or the SpCas9 nickase domain can bind to a nucleic acid sequence having a NGG, a NGA, or a NGCG PAM sequence. In some embodiments, the SpCas9 domain comprises one or more of a D1135X, a R1335X, and a T1337X mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein, wherein X is any amino acid. In some embodiments, the SpCas9 domain comprises one or more of a D1135E, R1335Q, and T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein. In some embodiments, the SpCas9 domain comprises one or more of a Dl 134V, a R1334Q, and a T1336R mutation as numbered in the wild type Cas9 amino acid sequence, or a corresponding mutation thereof. In some embodiments, the SpCas9 domain comprises a D1135V, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein. In some embodiments, the SpCas9 domain comprises one or more of a D1135X, a G1218X, a R1335X, and a T1337X mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein, wherein X is any

amino acid. In some embodiments, the SpCas9 domain comprises one or more of a D1135V, a G1218R, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein. In some embodiments, the SpCas9 domain comprises a D1135V, a G1218R, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein.

[0103] In some embodiments, the Cas9 domain is a modified SpCas9 domain having specificity for a 5'-NGCG-3' PAM sequence, where N is any one of nucleotides A, G, C, or T. In some embodiments, the modified SpCas9 domain having specificity for a 5'-NGCG-3' PAM sequence comprises a D1135V, a G1218R, a R1335E, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein (the "VRER" SpCas9). In some embodiments, the VRER SpCas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the SpCas9 may further comprise a D10A and a H840A mutation and is a nuclease inactive SpCas9. Amino acid sequence of an exemplary nuclease inactive VRER SpCas9 is provided in SEQ ID NO.: 4.

[0104] In some embodiments, the Cas9 domain is a modified SpCas9 domain having specificity for a 5'-NGAG-3' PAM sequence, where N is any one of nucleotides A, G, C, or T. In some embodiments, the modified SpCas9 domain having specificity for a 5'-NGAG-3' PAM sequence comprises a D1135E, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein (the "EQR" SpCas9). In some embodiments, the EQR SpCas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the SpCas9 may further comprise a D10A and a H840A mutation and is a nuclease inactive SpCas9.

[0105] Amino acid sequence of an exemplary nuclease inactive EQR SpCas9 is provided in SEQ ID NO.: 5.

[0106] In some embodiments, the Cas9 domain is a modified SpCas9 domain having specificity for a 5'-NGAN-3' or a 5-NGNG-3' PAM sequence, where N is any one of nucleotides A, G, C, or T. In some embodiments, the modified SpCas9 domain having specificity for a 5'-NGAN-3' or a 5-NGNG-3' PAM sequence comprises a D1135V, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein (the "VQR" SpCas9). In some embodiments, the VQR SpCas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the SpCas9 may further comprise a D10A and a H840A mutation and is a nuclease inactive SpCas9.

[0107] Amino acid sequence of an exemplary nuclease inactive VQR SpCas9 is provided in SEQ ID NO.: 6.

[0108] In some embodiments, the Cas9 domain is a modified SpCas9 domain having specificity for a 5'-NGN-3' PAM sequence, where N is any one of nucleotides A, G, C, or T. In some embodiments, the modified SpCas9 domain having specificity for a 5'-NGN-3' PAM sequence comprises a D1135L, a S1136W, a G1218K, a E1219Q, a R1335Q, a T1337R, a D1135V, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein (the "SpGCas9"). In some embodiments, the SpG Cas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the SpGCas9 may further comprise a D10A and a H840A mutation and is a nuclease inactive SpGCas9.

[0109] Amino acid sequence of an exemplary nuclease inactive SpG Cas9 is provided in SEQ ID NO.: 7.

[0110] In some embodiments, the Cas9 domain is a modified SpCas9 domain having specificity for a 5'-NRN-3' or a 5'-NYN-3' PAM sequence, where N is any one of nucleotides A, G, C, or T, where R is nucleotide A or G, and where Y is nucleotide C or T. In some embodiments, the modified SpCas9 domain having specificity for a 5'-NRN-3' or a 5'-NYN-3' PAM sequence comprises a A61R, a L1111R, a D1135L, a S1136W, a G1218K, a E1219Q, a N1317R, a A1322R, a R1333P, a R1335Q, and a T1337R mutation as numbered in the wild type SpCas9 amino acid sequence or a corresponding mutation in another SpCas9 protein (the "SpRYCas9"). In some embodiments, the SpRY Cas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the SpCas9 may further comprise a D10A and a H840A mutation and is a nuclease inactive SpRYCas9.

[0111] Amino acid sequence of an exemplary nuclease inactive SpRY Cas9 is provided in SEQ ID NO.: 8.

[0112] In some embodiments, the Cas9 domain is a Cas9 domain from Staphylococcus aureus (SaCas9). In some embodiments, the SaCas9 domain is a nuclease inactive SaCas9 (dSacas9). In some embodiments, the SaCas9 comprises a N579A mutation as numbered in the wild type SaCas9 sequence or a corresponding mutation in another SaCas9 protein. In some embodiments, the SaCas9 comprises a D10A mutation as numbered in the wild type SaCas9 sequence or a corresponding mutation in another SaCas9 protein. In some embodiments, the dSaCas9 comprises a D10A mutation and a N579A mutation as numbered in the wild type SaCas9 sequence or a corresponding mutation in another SaCas9 protein.

[0113] An exemplary wild type SaCas9 protein is provided in SEQ ID NO.: 9.

[0114] In some embodiments, the SaCas9 domain, the nuclease inactive SaCas9 domain, or the SaCas9 nickase domain can bind to a nucleic acid sequence having a non-canonical PAM. In some embodiments, the SaCas9 domain, the SaCas9d domain, or the SaCas9n domain can bind to a nucleic acid sequence having a NNGRRT PAM sequence, where N = A, T, C, or G, and R = A or G. In some embodiments, the SaCas9 domain comprises one or more of a E781K, a N967K, and a R1014H mutation as numbered in the wild type SaCas9 sequence or a corresponding mutation in another SaCas9 protein (the "KKH" SaCas9). In some embodiments, the SaCas9 domain comprises a E781K, a N967K, or a R1014H mutation as numbered in the wild type SaCas9 sequence or a corresponding mutation in another SaCas9 protein. In some embodiments, the SaCas9 domain, the SaCas9d domain, or the SaCas9n domain can bind to a nucleic acid sequence having a non-canonical PAM. In some embodiments, the SaCas9 domain or the nuclease inactive SaCas9d domain can bind to a nucleic acid sequence having a NNGRRT PAM sequence. In some embodiments, the SaCas9 domain comprises one or more of a E781K, a N967K, and a R1014H mutation, or one or more corresponding mutation in any of the amino acid sequences provided herein. In some embodiments, the SaCas9 domain comprises a E781K, a N967K, or a R1014H mutation, or corresponding mutations in any of the amino acid sequences provided herein. In some embodiments, the KKH SaCas9 further comprises one or more mutations that reduces or abolishes its nuclease activity. For example, the KKHSaCas9 may further comprise a D10A and a N579A mutation and is a nuclease inactive KKH SaCas9. Amino acid sequence of an exemplary nuclease inactive KKH dSaCas9 is provided in SEQ ID NO.: 10

[0115] In some embodiments, the Cas9 domain is a Cas9 domain from Neisseria meningitidis (NmeCas9). In some embodiments, the NmeCas9 domain is a nuclease inactive NmeCas9 (dNmeCas9). An NmeCas9 may have specificity for a 5'-NNNGATT-3' PAM, where N is any one of nucleotides A, G, C, or T. In some embodiments, the NmeCas9 comprises a D16A mutation, or a corresponding mutation in any of the amino acid sequences as numbered in the wild type NmeCas9 sequence. In some embodiments, the NmeCas9 comprises a H588A mutation as numbered in the wild type NmeCas9 sequence or a corresponding mutation in another NmeCas9 protein. In some embodiments, a dNmeCas9 comprises a D16A and a H588A mutation.

[0116] Amino acid sequence of an exemplary dNmeCas9 protein is provided in SEQ ID NO.: 11.

[0117] In some embodiments, the Cas9 domain is a Cas9 domain from Campylobacter jejuni (CjCas9). In some embodiments, the CjCas9 domain is a nuclease inactive CjCas9 (dCjCas9). A

Cj Cas9 may have specificity for a 5'-NNNVRYM-3' PAM, where N is any one of nucleotides A, G, C, or T, V is nucleotide A, C, or G, R is nucleotide A or G, Y is nucleotide C or T, and M is nucleotide A or C. In some embodiments, the CjCas9 comprises a D8A mutation, or a corresponding mutation in any of the amino acid sequences as numbered in the wild type CjCas9 sequence. In some embodiments, the CjCas9 comprises a H559A mutation as numbered in the wild type CjCas9 sequence or a corresponding mutation in another CjCas9 protein. In some embodiments, a dCjCas9 comprises a D16A and a H588A mutation.

[0118] Amino acid sequence of an exemplary dCjCas9 protein is provided in SEQ ID NO.: 12.

[0119] In some embodiments, the Cas9 domain is a Cas9 domain from Streptococcus thermophilus (StCas9). In some embodiments, the StCas9 is encoded by St CRISPR1 loci of the *Streptococcus thermophilus* (St1Cas9). In some embodiments, the St1Cas9 domain is a nuclease inactive St1Cas9 (dSt1Cas9). An St1Cas9 may have specificity for a 5'- NNAGAAW-3' PAM, where N is any one of nucleotides A, G, C, or T, and W is nucleotide A or T. In some embodiments, the St1Cas9 comprises a D10A mutation, or a corresponding mutation in any of the amino acid sequences as numbered in the wild type St1Cas9 sequence. In some embodiments, the St1Cas9 comprises a H600A mutation as numbered in the wild type St1Cas9 sequence or a corresponding mutation in another St1Cas9 protein. In some embodiments, a St1Cas9d comprises a D10A and a H600A mutation.

[0120] In some embodiments, the StCas9 is encoded by St CRISPR3 loci of the *Streptococcus thermophilus* (St3Cas9). In some embodiments, the St3Cas9 domain is a nuclease inactive St3Cas9 (dSt3Cas9). An St3Cas9 may have specificity for a 5'-NGGNG-3' PAM, where N is any one of nucleotides A, G, C, or T. In some embodiments, the St3Cas9 comprises a D10A mutation, or a corresponding mutation in any of the amino acid sequences as numbered in the wild type St3Cas9 sequence. In some embodiments, the St3Cas9 comprises a N870A mutation as numbered in the wild type St3Cas9 sequence or a corresponding mutation in another St3Cas9 protein. In some embodiments, a dSt3Cas9 comprises a D10A and a N870A mutation.

[0121] Amino acid sequence of an exemplary dSt1Cas9 protein is provided in SEQ ID NO.: 13.

[0122] Amino acid sequence of an exemplary dSt3Cas9 protein is provided in SEQ ID NO.: 14.

[0123] In some embodiments, the Cas9 domain of any of the fusion proteins provided herein comprises an amino acid sequence that is at least 60%, at least 65%, at least 70%, at least 75%, at least 80%, at least 85%, at least 90%, at least 95%, at least 96%, at least 97%, at least 98%, at least 99%, or at least 99.5% identical to any one of the Cas9 sequences provided herein.

[0124] In some embodiments, an epigenetic editor provided herein comprises a Cpf1 (or Cas12a) protein domain. For example, an epigenetic editor can comprise a nuclease inactive

Cpf1 protein or a variant thereof. The Cpf1 protein has a RuvC-like endonuclease domain that is similar to the RuvC domain of Cas9 but does not have a HNH endonuclease domain, and the N-terminal of Cpf1 does not have the alpha-helical recognition lobe of Cas9. In some embodiments, the Cpf1 comprises one or more mutations corresponding to D917A, E1006A, or D1255A as numbered in the Francisella novicida Cpf1 protein (FnCpf1). A FnCpf1 may have specificity for a 5'-TTN-3' PAM sequence, where N is any one of nucleotides A, T, G, or C. In some embodiments, the Cpf1 protein has reduced nuclease activity. In some embodiments, the nuclease activity of the Cpf1 protein is abolished (dCpf1). In some embodiments, the dCpf1 protein comprises mutations corresponding to D917A, E1006A, D1255A, D917A/E1006A, D917A/D1255A, E1006A/D1255A, or D917A/ E1006A/D1255A or a corresponding mutation in any of the Cpf1 amino acid sequences as numbered in the wild type FnCpf1 sequence provided herein. In some embodiments, the dCpf1 comprises a D917A mutation, or a corresponding mutation in any of the Cpf1 amino acid sequences as numbered in the wild type FnCpf1sequence.

[0125] In some embodiments, the Cpf1 protein comprises an amino acid sequence that is at least 85%, at least 90%, at least 91%, at least 92%, at least 93%, at least 94%, at least 95%, at least 96%, at least 97%, at least 98%, at least 99%, or at ease 99.5% identical to the FnCpf1 sequence provided herein. It should be appreciated that Cpf1 from other bacterial species may also be used in accordance with the present disclosure.

[0126] An exemplary wild type Francisella novicida Cpf1 amino acid sequence is provided in SEQ ID NO.: 15.

[0127] Amino acid sequence of an exemplary nuclease inactive FnCpf1 protein is provided in SEQ ID NO.: 16.

[0128] In some embodiments, the Cpf1 is a Cpf1 protein from *Lachnospiraceae bacterium* (LbCpf1). A LbCpf1 may have specificity for a 5'-TTTV-3' PAM sequence, where V is any one of nucleotides A, G, or C. In some embodiments, the LbCpf1 protein has reduced nuclease activity. In some embodiments, the nuclease activity of the LbCpf1 protein is abolished (dLbCpf1). In some embodiments, the dLbCpf1 protein comprises mutations corresponding to D832A or a corresponding mutation in any of the Cpf1 amino acid sequences as numbered in the wild type LbCpf1 sequence provided herein.

[0129] Amino acid sequence of an exemplary nuclease inactive dLbCpf1 protein is provided in SEQ ID NO.: 17.

[0130] In some embodiments, the Cpf1 is a Cpf1 protein from Acidaminococcus sp. (AsCpf1). A AsCpf1 may have specificity for a 5'-TTTV-3' PAM sequence, where V is any one of

nucleotides A, G, or C. In some embodiments, the AsCpf1 protein has reduced nuclease activity. In some embodiments, the nuclease activity of the AsCpf1 protein is abolished (dAsCpf1. In some embodiments, the dLbCpf1 protein comprises mutations corresponding to D908A or a corresponding mutation in any of the Cpf1 amino acid sequences as numbered in the wild type AsCpf1 sequence provided herein. In some embodiments, the dAsCpf1 or AsCpf1 further comprises mutations that improve targeting and editing efficiency. For example, an AsCpf1 may comprise mutations E174R, S542R, and K548R ("enAsCpf1") or corresponding mutations in any of the Cpf1 amino acid sequences as numbered in the wild type AsCpf1 sequence provided herein.

[0131] Amino acid sequence of an exemplary nuclease inactive AsCpf1 protein is provided in SEQ ID NO.: 18.

[0132] Amino acid sequence of an exemplary nuclease inactive enAsCpf1 protein is provided in SEQ ID NO.: 19.

[0133] In some embodiments, the dAsCpf1 or AsCpf1 protein further comprises mutations that improve fidelity of target recognition of the protein. For example, an AsCpf1 may comprise mutations E174R, N282A, S542R, and K548R ("HFAsCpf1") or corresponding mutations in any of the Cpf1 amino acid sequences as numbered in the wild type AsCpf1 sequence provided herein.

[0134] Amino acid sequence of an exemplary nuclease inactive HFAsCpf1 protein is provided in SEQ ID NO.: 20.

[0135] In some embodiments, the dAsCpf1 or AsCpf1 protein further comprises mutations that result in altered PAM specificity of the protein. In some embodiments, an AsCpf1 comprising mutations S542R, K548V, and N552R ("RVRAsCpf1") or corresponding mutations in any of the Cpf1 amino acid sequences as numbered in the wild type AsCpf1 sequence provided herein may have specificity for a 5'-TATV-3' PAM, where V is any one of nucleotides A, C, or G. In some embodiments, an AsCpf1 comprising mutations S542R and K607R ("RRAsCpf1") or corresponding mutations in any of the Cpf1 amino acid sequences as numbered in the wild type AsCpf1 sequence provided herein may have specificity for a 5'-TYCV-3' PAM, where Y is any one of nucleotides C or T and V is any one of nucleotide A, C, or G.

[0136] Amino acid sequence of an exemplary nuclease inactive RVRAsCpf1 protein is provided in SEQ ID NO.: 21.

[0137] Amino acid sequence of an exemplary nuclease inactive RRAsCpf1 protein is provided in SEQ ID NO.: 22.

[0138] In some embodiments, an epigenetic editor provided herein comprises a Cas protein domain other than Cas9. In some embodiments, the Cas9 protein comprises an inactivated nuclease domain. In some embodiments, an epigenetic editor comprises a Cas12a, a Cas12b, a Cas12c, a Cas12d, a Cas12e, a Cas12h, or a Cas12i domain. In some embodiments, the Cas9 protein is a RNA nuclease or an inactivated RNA nuclease. In some embodiments, an epigenetic editor comprises a Cas12g, a Cas13a, a Cas13b, a Cas13c, or a Cas13d domain. In some embodiments, an epigenetic editor comprises an Argonaut protein domain.

[0139] A CRISPR/Cas system or a Cas protein in an epigenetic editor system provided herein may comprise Class 1 or Class 2 Cas proteins. The Class 1 or Class 2 proteins used in an epigenetic editor may be inactivated in its nuclease activity. In some embodiments, an epigenetic editor comprises a Cas protein derived from a Type II, Type IIA, Type IIB, Type IIC, Type V, or Type VI Cas nuclease. In some embodiments, an epigenetic editor comprises a Cas protein derived from a Class 2 Cas nucleases derived from Cas1, Cas1B, Cas2, Cas3, Cas4, Cas5, Cas6, Cas7, Cas8, Cas10, Cas14a, Cas14b, Cas14c, CasX, CasY, CasPhi, C2c4, C2c8, C2c9, C2c10, Csy1, Csy2, Csy3, Cse1, Cse2, Csc1, Csc2, Csa5, Csn2, Csm2, Csm3, Csm4, Csm5, Csm6, Cmr1, Cmr3, Cmr4, Cmr5, Cmr6, Csb1, Csb2, Csb3, Csx17, Csx14, Csx10, Csx16, CsaX, Csx3, Csx1, Csx1S, Csf1, Csf2, CsO, Csf4, or homologues or modified versions thereof. In some embodiments, a Cas protein in an epigenetic editor is a nuclease inactivated Cas protein.

[0140] In some embodiments, the epigenetic editor comprises a CasX (Cas12e) protein. A CasX protein may have specificity for a 5'-TTCN-3' PAM sequence, where N is any one of nucleotides A, G, T, or C. In some embodiments, the CasX protein has reduced or abolished nuclease activity (dCasX), In some embodiments, the dCasX protein comprises one or more of E672X, E769X, D935X amino acid substitutions as compared to the CasX reference sequence provided below, where X is any amino acid other than the wild type amino acid. In some embodiments, the dCasX protein comprises one or more of E672A, E769A, D935A amino acid substitutions as compared to the CasX reference sequence provided below. In some embodiments, the CasX protein is a truncated CasX protein as compared to the wild type. In some embodiments, the CasX protein lacks a target strand loading domain (TSLD). CasX protein and sequences as described in US Patent No.s 10,570,415 and PCT application publication No.s WO2020023529, WO2020041456 are incorporated herein in the entirety.

[0141] An exemplary CasX amino acid sequence is provided in SEQ ID NO.: 23.

[0142] An exemplary dCasX amino acid sequence is provided in SEQ ID NO.: 24.

[0143] In some embodiments, the epigenetic editor comprises a CasY (Cas12d) protein. A CasY protein may have specificity for a 5'-TA-3' PAM sequence. In some embodiments, the CasY

protein has reduced or abolished nuclease activity (dCasY). In some embodiments, the dCasY protein comprises one or more of D828X, E914X, D1074X amino acid substitutions as compared to the CasY reference sequence provided below, where X is any amino acid other than the wild type amino acid. In some embodiments, the dCasY protein comprises one or more of D828A, E914A, D1074A amino acid substitutions as compared to the CasY reference sequence provided below. CasY protein and sequences as described in US Patent Application Publication No.s US20200255858 and US20190300908 are incorporated herein in the entirety.

[0144] An exemplary CasY amino acid sequence is provided in SEQ ID NO.: 25.

[0145] In some embodiments, the epigenetic editor comprises a Casφ (CasPhi) protein. A Casφ protein may have specificity for a 5'-TTN-3' PAM sequence, wherein N is any one of nucleotides A, T, G, or C. In some embodiments, the Casφ protein has reduced or abolished nuclease activity (dCasφ). In some embodiments, a dCasφ protein comprises a D394A mutation or a corresponding mutation in any of the Casφ amino acid sequences as numbered in the wild type Casφ sequence provided herein.

[0146] Cas φ protein and sequences as described in Pausch et al., *CRISPR-Cas φ from huge phages is a hypercompact genome editor*, Science 369, 333–337 (2020), which is incorporated herein in the entirety.

[0147] An exemplary wild type Casφ (CasPhi) amino acid sequence is provided in SEQ ID NO.: 26.

[0148] An exemplary dCasφ (dCasPhi) amino acid sequence is provided in SEQ ID NO.: 27.

[0149] In some embodiments, the epigenetic editor comprises a Cas12f1 (Cas14a) protein as in SEQ ID NO.: 28. In some embodiments, the epigenetic editor comprises a Cas12f2 (Cas14b) protein as in SEQ ID NO.: 29. In some embodiments, the epigenetic editor comprises a Cas12f3 (Cas14c) protein as in SEQ ID NO.: 30. In some embodiments, the epigenetic editor comprises a C2c8 protein as in SEQ ID NO.: 31.

[0150] In some embodiments, the Cas protein is a circular permutant Cas protein. For example, an epigenetic editor may comprise a circular permutant Cas9 as described in Oakes et al., Cell 176, 254-267 (2019), incorporated herein in its entirety. As used herein, the term "circular permutant" refers to a variant polypeptide (e.g., of a subject Cas protein) in which one section of the primary amino acid sequence has been moved to a different position within the primary amino acid sequence of the polypeptide, but where the local order of amino acids has not been changed, and where the three dimensional architecture of the protein is conserved. For example, a circular permutant of a wild type 1000 amino acid polypeptide may have an N-terminal residue of residue number 500 (relative to the wild type protein), where residues 1-499 of the wild type

protein are added the C-terminus. Such a circular permutant, relative to the wild type protein sequence would have, from N-terminus to C-terminus, amino acid numbers 500-1000 followed by 1-499, resulting in a circular permutant protein with amino acid 499 being the C-terminal residue. Thus, such an example circular permutant would have the same total number of amino acids as the wild type reference protein, and the amino acids would be in the same order locally in specific regions of the circular permutant, but the overall primary amino acid sequence is changed.

[0151] In some embodiments, an epigenetic editor comprises a circular permuted Cas protein, e.g. a circular permuted Cas9 protein. In some embodiments, the epigenetic editor comprises a fusion of a circular permuted Cas protein and an epigenetic effector domain, where the epigenetic effector domain is fused to the circular permuted Cas protein to a N-terminus or C-terminus that is different from that of wild type Cas protein.

[0152] In some embodiments, the circular permuted Cas protein comprises a N-terminal end of an N-terminal fragment of a wild type Cas protein fused to a C-terminus of a C-terminal fragment of the wild type Cas protein, hereby generating new N- and C-termini. Without wishing to be bound by any theory, the N-terminus and C-terminus of a wild type Cas protein may be locked in a small region, which may cause steric hinderance when the Cas protein is fused to an effect domain and reduced access to the target DNA sequence. In some embodiments, the epigenetic editor comprising a circular permutant Cas protein has reduced steric incompatibility as compared to an epigenetic editor comprising a wild type Cas protein counterpart. In some embodiments, the epigenetic editor comprising a circular permutant Cas protein has improved effectiveness as compared to an epigenetic editor comprising a wild type Cas protein counterpart. In some embodiments, the epigenetic editor comprising a circular permutant Cas protein has improved epigenetic editing accuracy as compared to an epigenetic editor comprising a wild type Cas protein counterpart. In some embodiments, the epigenetic editor comprising a circular permutant Cas protein has reduced off-target editing effect as compared to an epigenetic editor comprising a wild type Cas protein counterpart.

[0153] In some embodiments, the circular permutant Cas protein is a circular permutant Cas9 protein. In some embodiments, the circular permuted Cas9 protein includes an N-terminal fragment of a wild type Cas9 protein fused to the C-terminus of the Cas9 protein (e.g., in some cases via a linker, e.g., a cleavable linker), where the C-terminal amino acid of the N-terminal fragment (i.e., the C-terminus of the N-terminal fragment) includes an amino acid corresponding to amino acid 182D, 200P, 231G, 271Y, 311E, 1011G, 1017D, 1024K, 1029I, 1030G, 1032A, 1042I, 1245L, 1249P, 1250E, or 1283A of the wild type Cas9 protein sequence. In some cases, a

circular permuted Cas9 protein includes an N-terminal fragment of a wild type Cas9 protein fused to the C-terminus of a C terminal fragment the wild type Cas9 protein (e.g., in some cases via a linker, e.g., a cleavable linker), where the N-terminal fragment includes an amino acid sequence corresponding to amino acids 1-182, 1-200, 1-231, 1-271, 1-311, 1-1011, 1-1017, 1-1024, 1-1029, 1-1030, 1-1032, 1-1042, 1-1245, 1-1249, 1-1250, or 1-1283 of the wild type Cas9 protein. Additional circular permuted Cas9 proteins as described in US Patent Application No. US20190233847 is incorporated herein by reference in its entirety.

Guide polynucleotides

[0154] In some embodiments, an epigenetic editor comprises a guide polynucleotide (or guide nucleic acid). For example, an epigenetic editor with a DNA binding domain that includes a CRISPR-Cas protein may also include a guide nucleic acid that is capable of forming a complex with the CRISPR-Cas protein.

[0155] Methods of using guide nucleotide sequence-programmable DNA-binding protein, such as Cas9, for site-specific DNA targeting (e.g., to modify a genome) are known in the art. The guide RNA (gRNA) may guide the programmable DNA binding protein, e.g a Class 2 Cas protein such as a Cas9 to a target sequence on a target nucleic acid molecule, where the gRNA hybridizes with and the programmable DNA binding protein and generates modification at or near the target sequence. In some embodiments, the gRNA and an epigenetic editor fusion protein may form a ribonucleoprotein (RNP), e.g., a CRISPR/Cas complex.

[0156] A guide nucleotide sequence, e.g. a guide RNA sequence, may comprises two parts: 1) a nucleotide sequence that shares homology to a target nucleic acid (e.g., and directs binding of a guide nucleotide sequence-programmable DNA-binding protein to the target); and 2) a nucleotide sequence that binds a nucleic acid guided programmable DNA-binding protein, for example, a CRISPR-Cas protein. The nucleotide sequence in 1) may comprise a spacer sequence that hybridizes with a target sequence. The nucleotide sequence in 2) may be referred to as a scaffold sequence of a guide nucleic acid, a tracrRNA, or an activating region of a guide nucleic acid, and may comprise a stem-loop structure. The scaffold sequences of guide nucleic acids as described in Jinek et al., Science 337:816-821(2012), U.S. Patent Application Publication US20160208288, and U.S. Patent Application Publication US20160200779 are each incorporated herein by reference in its entirety.

A guide polynucleotide may be a single molecule or may comprise two separate molecules. For example, parts 1) and 2) as described above may be fused to form one single guide (e.g. a single guide RNA, or sgRNA), or may be two separate molecules. In some embodiments, a guide polynucleotide is a dual polynucleotides connected by a linker. In some embodiments, a guide

polynucleotide is a dual polynucleotides connected by a non-nucleic acid linker, for example, a peptide linker or a chemical linker.

[0157] Methods for selecting, designing, and validating gRNAs and targeting sequences (or spacer sequences) are described herein and known to those skilled in the art. Software tools can be used to optimize the gRNAs corresponding to a target nucleic acid sequence, e.g., to minimize total off-target activity across the genome. For example, DNA sequence searching algorithm can be used to identify a target sequence in crRNAs of a gRNA for use with Cas9. Exemplary gRNA design tools, including as described in Bae, et al., Cas-OFFinder: A fast and versatile algorithm that searches for potential off-target sites of Cas9 RNA-guided endonucleases. Bioinformatics 30, 1473-1475 (2014)), is herein incorporated in its entirety.

[0158] A guide polynucleotide may be of variant lengths. In some embodiments, the length of the spacer or targeting sequence depends on the CRISPR/Cas component of the epigenetic editor system and components used. For example, different Cas proteins from different bacterial species have varying optimal targeting sequence lengths. Accordingly, the spacer sequence may comprise 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, or more than 50 nucleotides in length. In some embodiments, the spacer comprised 18-24 nucleotides in length. In some embodiments, the spacer comprises 19-21 nucleotides in length. In some embodiments, the spacer sequence comprises 20 nucleotides in length. In some embodiments, a guide nucleic acid (e.g., guide RNA) is from 15- 100 nucleotides long and comprises a sequence of at least 10 contiguous nucleotides that is complementary to a target sequence. In some embodiments, the guide RNA is 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, or 50 nucleotides long. In some embodiments, the guide RNA comprises a sequence of 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, or 50 contiguous nucleotides that is complementary to a target sequence. In some embodiments, the target sequence is a DNA sequence. In some embodiments, the degree of complementarity between the targeting sequence of the gRNA and the target sequence on the target nucleic acid molecule is at least about 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 97%, 98%, 99%, or 100%. In some embodiments, the targeting sequence of the gRNA and the target sequence on the target nucleic acid molecule may be 100% complementary. In other embodiments, the targeting sequence of the gRNA and the target sequence on the target nucleic acid molecule may contain at least one mismatch. For example, the targeting sequence of the gRNA and the target sequence on the target nucleic acid molecule may contain 1, 2, 3, 4, 5, 6, 7, 8, 9, or 10 mismatches.

In some embodiments, the target sequence is a sequence in the genome of a mammal. In some embodiments, the target sequence is a sequence in the genome of a human. In some embodiments, the 3' end of the target sequence is immediately adjacent to a canonical PAM sequence (NGG). In some embodiments, the guide nucleic acid (e.g., guide RNA) is complementary to a sequence associated with a disease or disorder.

[0159] In some embodiments, a guide RNA is truncated. The truncation can comprise any number of nucleotide deletions. For example, the truncation can comprise 1, 2, 3, 4, 5, 10, 15, 20, 25, 30, 40, 50 or more nucleotides. In some embodiments, a guide polynucleotide comprises RNA. In some embodiments, a guide polynucleotide comprises DNA. In some embodiments, a guide polynucleotide comprises a mixture of DNA and RNA.

[0160] A guide polynucleotide may be modified. The modifications can comprise chemical alterations, synthetic modifications, nucleotide additions, and/or nucleotide subtractions. Modified nucleosides or nucleotides can be present in a gRNA. For example, a gRNA can comprise one or more non-naturally and/or naturally occurring components or configurations that are used instead of or in addition to the canonical A, G, C, and U residues. A modified RNA can include one or more of an alteration or a replacement, of one or both of the non-linking phosphate oxygens and/or of one or more of the linking phosphate oxygens in the phosphodiester backbone linkage, an alterations of the ribose sugar, e.g., of the 2' hydroxyl on the ribose sugar (an exemplary sugar modification), an alteration of the phosphate moiety, a modification or replacement of a naturally occurring nucleobase, replacement or modification of the ribose-phosphate backbone, a modification of the 3' end or 5' end of the oligonucleotide, or replacement of a terminal phosphate group or conjugation of a moiety, cap, or linker, or any combination thereof.

[0161] In some embodiments, the ribose group (or sugar) may be modified. In some embodiments, modified ribose group may control oligonucleotide binding affinity for complementary strands, duplex formation, or interaction with nucleases. Examples of chemical modifications to the ribose group include, but are not limited to, 2'-O-methyl (2'-OMe), 2'-fluoro (2'-F), 2'-deoxy, 2'-O-(2-methoxyethyl) (2'-MOE), 2'-NH2, 2'-O-Allyl, 2'-O-Ethylamine, 2'-O-Cyanoethyl, 2'-O-Acetalester, or a bicyclic nucleotide such as locked nucleic acid (LNA), 2'-(5-constrained ethyl (S-cEt)), constrained MOE, or 2'-0,4'-C-aminomethylene bridged nucleic acid (2',4'-BNANC). In some embodiments, 2'-O-methyl modification can increase binding affinity of oligonucleotides. In some embodiments, 2'-O-methyl modification can enhance nuclease stability of oligonucleotides. In some embodiments, 2'-fluoro modification can increase oligonucleotide binding affinity and nuclease stability.

[0162] In some embodiments, the phosphate group may be chemically modified. Examples of chemical modifications to the phosphate group includes, but are not limited to, a phosphorothioate (PS), phosphonoacetate (PACE), thiophosphonoacetate (thioPACE), amide, triazole, phosphonate, or phosphotriester modification. In some embodiments, PS linkage can refer to a bond where a sulfur is substituted for one nonbridging phosphate oxygen in a phosphodiester linkage, e.g., between nucleotides. An "s" may be used to depict a PS modification in gRNA sequences. In some embodiments, a gRNA or an sgRNA may comprise a phosphorothioate (PS) linkage at a 5' end or at a 3' end. In some embodiments, a gRNA or an sgRNA may comprise a phosphorothioate (PS) linkage at a 5' end. In some embodiments, a gRNA or an sgRNA may comprise a phosphorothioate (PS) linkage at a 3' end. In some embodiments, a gRNA or an sgRNA may comprise a phosphorothioate (PS) linkage at a 5' end and at a 3' end. In some embodiments, a gRNA or an sgRNA may comprise one, two, or three, or more than three phosphorothioate linkages at the 5' end or at the 3' end. In some embodiments, a gRNA or an sgRNA may comprise three phosphorothioate (PS) linkages at the 5' end or at the 3' end. In some embodiments, a gRNA or an sgRNA may comprise three phosphorothioate linkages at the 3' end. In some embodiments, a gRNA or an sgRNA may comprise two and no more than two (i.e., only two) contiguous phosphorothioate (PS) linkages at the 5' end or at the 3' end. In some embodiments, a gRNA or an sgRNA may comprise three contiguous phosphorothioate (PS) linkages at the 5' end or at the 3' end. In some embodiments, a gRNA or an sgRNA may comprise the sequence 5'- UsUsU-3' at the 3'end or at the 5' end, wherein U indicates a uridine and wherein s indicates a phosphorothioate (PS) linkage.

In some embodiments, the nucleobase may be chemically modified. Examples of chemical modifications to the nucleobase include, but are not limited to, 2-thiouridine, 4-thiouridine, N6-methyladenosine, pseudouridine, 2,6-diaminopurine, inosine, thymidine, 5-methylcytosine, 5-substituted pyrimidine, isoguanine, isocytosine, or halogenated aromatic groups.

Chemical modifications can be made at a part of a guide polynucleotide or the entire guide polynucleotide. In some embodiments, a total of 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, or 25 base pairs of a guide RNA are chemically modified. In some embodiments, a total of 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, or 50 base pairs of a guide RNA are chemically modified. In some embodiments, a total of 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118,

119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, or 150 base pairs of a guide RNA are chemically modified. Chemical modifications can be made in the protospacer region, the tracr RNA, the crRNA, the stem loop, or any combination thereof.

Zinc finger proteins

[0163] In some embodiments, an epigenetic editor described herein comprises a nucleic acid binding domain comprising a zinc finger domain.

[0164] Zinc finger proteins are DNA-binding proteins that contain one or more zinc fingers. In some embodiments, a zinc finger (ZF) comprises a relatively small polypeptide domain comprising approximately 30 amino acids. A zinc finger may comprise an α-helix adjacent an antiparallel β-sheet (known as a ββα-fold) which may co-ordinate with a zinc ion between four Cys and/or His residues, as described further below. In some embodiments, a ZF domain recognizes and binds to a nucleic acid triplet, or an overlapping quadruplet, in a double-stranded DNA target sequence. In certain embodiments, ZFs may also bind RNA and proteins.

[0165] As used herein, the term "zinc finger" (ZF) or "zinc finger motif" (ZF motif) refers to an individual "finger", which comprises a beta-beta-alpha (ββα)-protein fold stabilized by a zinc ion as described elsewhere herein. In some embodiments, each finger includes approximately 30 amino acids. In some embodiments, ZF proteins or ZF protein domains are protein motifs that contain multiple fingers or finger-like protrusions that make tandem contacts with their target molecule. For example, a ZF finger may bind a triplet or (overlapping) quadruplet nucleotide sequence. Accordingly, a tandem array of ZF fingers may be designed for ZF proteins that do not naturally exist to bind desired targets.

[0166] Zinc finger proteins are widespread in eukaryotic cells. An exemplary motif characterizing one class of these proteins (C2H2 class) is -Cys-(X)2-4-Cys-(X)12-His-(X)3-5His, where X is any amino acid. A single finger domain may be about 30 amino acids in length. In some embodiments, a single finger comprises an alpha helix containing the two invariant histidine residues co-ordinated through zinc with the two cysteines of a single beta turn.

[0167] In some embodiments, amino acid sequence of a zinc finger protein, e.g. a Zif268 protein may be altered by making amino acid substitutions at the helix positions (e.g., positions −1, 2, 3 and 6 of Zif268) on a zinc finger recognition helix. For example, modified zinc fingers with non-naturally occurring DNA recognition specificity may be generated by phage display and combinatorial libraries with randomized side-chains in either the first or middle finger of a Zif268 and then isolated with an altered Zif268 binding site in which the appropriate DNA sub-site was replaced by an altered DNA triplet.

[0168] In some embodiments, a zinc finger comprises a C2H2 finger. In some embodiments, a zinc finger protein comprises a ZF array that comprises sequential C2H2-ZFs each contacting three or more sequential bases. In some embodiments, Zinc finger protein structures, for example, zinc finger protein Zif268 and its variants bound to DNA show a semi-conserved pattern of interactions, in which typically three amino acids from the alpha-helix of the zinc finger contact three adjacent base pairs in the DNA. Accordingly, in embodiments, zinc finger DNA-binding domains function in a modular manner with a one-to-one interaction between a zinc finger and a three-base-pair tri-nucleotide sequence in a DNA sequence.

[0169] In some embodiments, an epigenetic editor comprises a zinc finger motif comprising of a sequence: N′--(Helix 1)- -(Helix 2)- -(Helix 3)- -(Helix 4)--(Helix 5)- -(Helix 6)- -C′, wherein the (Helix) is a-six contiguous amino acid residue peptide that forms a short alpha helix. In some embodiments, an epigenetic editor comprises a zinc finger motif comprising of a sequence: N′--(Helix 1)- -(Helix 2)- -(Helix 3)- -(Helix 4)--(Helix 5)-- -C′, wherein the (Helix) is a-six contiguous amino acid residue peptide that forms a short alpha helix.

[0170] In some embodiments, two or more zinc fingers are linked together in a tandem array to achieve specific recognition and binding of a contiguous DNA sequence. Zinc finger or zinc finger arrays in an epigenetic editor may be naturally occurring, or may be artificially engineered for desired DNA binding specificity. For example, DNA binding characteristics of individual zinc fingers may be engineered by randomizing the amino acids at the alpha-helical positions of the zinc fingers involved in DNA binding and using selection methodologies such as phage display to identify desired variants capable of binding to DNA target sites of interest.

[0171] Engineered zinc finger binding domain can have a novel binding specificity as compared to a naturally-occurring zinc finger protein. Zinc fingers with desired DNA binding specificity can be designed and selected via various approaches. For example, databases comprising triplet (or quadruplet) nucleotide sequences and individual zinc finger amino acid sequences, in which each triplet or quadruplet nucleotide sequence is associated with one or more amino acid sequences of zinc fingers which bind the particular triplet or quadruplet sequence may be used to design zinc finger arrays for specific DNA sequences. See, for example, U.S. Pat. Nos. 6,453,242, 6,534,261, and 8,772,453, incorporated by reference herein in their entirety. In some embodiments, a zinc finger array may be designed and selected from a library of zinc fingers, e.g., a randomized zinc finger library. In some embodiments, a zinc finger with novel DNA binding specific is generated by selection-based methods on combinatorial libraries. For example, a zinc finger can be selected with phage display which involves displaying zinc finger proteins on the surface of filamentous phage, followed by sequential rounds of affinity selection

with biotinylated target DNA to enrich for phage expressing proteins able to bind the specific target sequence. Bacterial-two-hybrid (B2H) system may also be used for selection of zinc fingers that bind specific target sites from randomized libraries. For example, a zinc finger binding site may be placed upstream of a weak promoter driving expression of two selectable markers in host cells, e.g. E. coli cells. A library of zinc fingers, fused to a fragment of the reporter protein, e.g. a yeast Gal11P protein, can be expressed in the cells and binding of a zinc finger to the target site recruits an RNA polymerase-Gal4 fusion, thus activating transcription and allowing survival of the cells on selective medium. Rational design and selection of zinc fingers as described in Maeder et al., 2008, Mol. Cell, 31:294-301; Joung et al., 2010, Nat. Methods, 7:91-92; Isalan et al., 2001, Nat. Biotechnol., 19:656-660, Rebar, et al., Science 263, 671–673 (1994), and Joung, et al. Proc Natl Acad Sci USA 97, 7382–7387 (2000), each of which incorporated herein by reference in its entirety.

[0172] In some embodiments, zinc fingers may be evolved and selected with a continuous evolution system (PACE) comprising a host cell, e.g. a E. coli cell, a "helper phagemid" present in all host cells and encoding all phage proteins except one phage protein (e.g. a g3p protein), an "accessory plasmid", present in all host cells, that expresses the g3p protein in response to an active library member; and a "selection phagemid" expressing the library of proteins or nucleic acids being evolved, which is replicated and packaged into secreted phage particles. Helper and accessory plasmids can be combined into a single plasmid. New host cells can only be infected by phage particles that contain g3p. Fit selection phagemids encode library members that induce g3p expression from the accessory plasmid can be packaged into phage particles that contain g3p. g3p containing phage particles can infect new cells, leading to further replication of the fit selection phagemids, while g3p-deficient phage particles are non-infectious, and therefore low-fitness selection phagemids cannot propagate. The selection system, in combination with a continuous flow of host cells through a lagoon that permits replication of the phagemid but not the host cells, may be used to rapidly select zinc fingers. PACE system as described in U.S. Patent No. 9,023,594 is incorporated by reference in its entirety.

[0173] A zinc finger DNA binding domain of an epigenetic editor may include one or multiple zinc fingers. For example, a zinc finger DNA binding domain may include 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12 or more zinc fingers. In some embodiments, a zinc finger DNA binding domain has at least three zinc fingers. In some embodiments, a zinc finger DNA binding domain has at least 4, 5, or 6 zinc fingers. In some embodiments, a zinc finger DNA binding domain has three zinc fingers. In some embodiments, a zinc finger DNA binding domain has at least two zinc fingers. In some embodiments, a zinc finger DNA binding domain has an array of two-finger units.

[0174] A zinc finger DNA binding domain of an epigenetic editor may be designed for optimized specificity. In some embodiments, a sequential selection strategy is used to design a multi-finger ZF domain. For example, in a multi-finger ZF domain, a first finger may be randomized and selected with phage display, a small pool of selected fingers may be carried into the next stage, in which the second finger is randomized and selected. The process may be repeated multiple times depending on the number of fingers in the ZF domain. In some embodiments, a parallel optimization is used to design a multi-finger ZF domain. For example, a master randomized library may be interrogated using a B2H system under low selection stringency to identify a variety of individual fingers capable of binding each 3 base pair sub-site of the target site. The three selected populations may then be randomly shuffled to generate a library of multi-finger proteins, which may subsequently be interrogated under high-stringency selection conditions to identify three-finger proteins targeted to a specific nine base pair site. In additional embodiments, a large number of low-stringency selections may be used to generate a master library of single fingers, from which multi-finger proteins, e.g., three finger ZF proteins may be selected. For example, a master library or an archive may include pre-selected zinc finger pools each containing a mixture of fingers targeted to a different three base pair subsite of DNA sequences at a defined position within a three finger ZF protein. In certain embodiments, a zinc finger archive comprises at least 192 finger pools (64 potential three bp target subsites for each position in a three-finger protein). In some embodiments, a zinc finger archive comprises at least a zinc finger pool comprises at least at least 10, 20, 30, 40, 50, 60, 70, 80, 90, 95, 100 or more different fingers. In some embodiments, a smaller library is created form the archive for interrogation with a reporting system, e.g., a bacterial two-hybrid selection system.

[0175] In some embodiments, a multiple-finger ZF domain, e.g., a three-finger ZF domain may be designed and selected using two complementary libraries. For example, a three-finger ZF domain may be designed with two pre-made zinc finger phage-display libraries, where the first library contains randomized DNA-binding amino acid positions in fingers 1 and 2, and a second library contains randomized DNA-binding amino acid positions in fingers 2 and 3. The two libraries are complementary because the first library contains randomizations in all the base-contacting positions of finger 1 and certain base-contacting positions of finger 2, whereas the second library contains randomizations in the remaining base-contacting positions of finger 2 and all the base-contacting positions of finger 3. Selections of "one-and-a-half" fingers from each master library may be carried out in parallel using DNA sequences in which five nucleotides have been fixed to a sequence of interest. Subsequently, zinc finger encoding

sequences may be amplified from the recovered phage using PCR, and sets of "one-and-a-half" fingers can be paired to yield recombinant three-finger DNA-binding domains.

[0176] In some embodiments, a multi-finger ZF domain may be designed depending on the context effects of adjacent fingers. In some embodiments, a multi-finger ZF domain is designed and without selection. For example, a three-finger ZF domain may be assembled using N-terminal and C-terminal fingers identified in other arrays containing a common middle finger, using libraries containing an archive of three-finger ZF arrays comprising pre-selected and/or tested three-finger arrays.

[0177] Software for designing and selecting ZF arrays, for example, ZiFit (http://bindr.gdcb.iastate.edu/ZiFiT/; http://www.zincfingers.org/software-tools.htm) are available and known to those skilled in the art.

[0178] Accordingly, a zinc finger DNA binding domain of an epigenetic editor may include one or multiple zinc fingers. For example, a zinc finger DNA binding domain may include 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12 or more zinc fingers. In some embodiments, a zinc finger DNA binding domain has at least three zinc fingers. In some embodiments, a zinc finger DNA binding domain has at least 4, 5, or 6 zinc fingers. In some embodiments, a zinc finger DNA binding domain has three zinc fingers. In some embodiments, a zinc finger DNA binding domain comprising at least three zinc fingers recognizes a target DNA sequence of 9 or 10 nucleotides. In some embodiments, a zinc finger DNA binding domain comprising at least four zinc fingers recognizes a target DNA sequence of 12 to 14 nucleotides. In some embodiments, a zinc finger DNA binding domain comprising at least six zinc fingers recognizes a target DNA sequence of 18 to 21 nucleotides.

[0179] In some embodiments, an epigenetic editor as disclosed herein comprises non-natural and suitably contain 3 or more zinc fingers. In some embodiments, an epigenetic editor comprises 4, 5, 6, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18 or more (e.g. up to approximately 30 or 32) zinc fingers motifs arranged adjacent one another in tandem, forming arrays of ZF motifs. In some embodiments, an epigenetic editor includes at least 3 ZF motifs, at least 4 ZF motifs, at least 5 ZF motifs, or at least 6 ZF motifs, at least 7 ZF motifs, at least 8 ZF motifs, at least 9 ZF motifs, at least 10 ZF motifs, at least 11 or at least 12 ZF motifs in the nucleic acid binding domain. In some embodiments, an epigenetic editor includes up to 6, 7, 8, 10, 11, 12, 16, 17, 18, 22, 23, 24, 28, 29, 30, 34, 35, 36, 40, 41, 42, 46, 47, 48, 54, 55, 56, 58, 59, or 60 ZF motifs in the nucleic acid binding domain.

[0180] In some embodiments, a zinc finger or zinc finger array targeting a specific DNA sequence is designed with a modular assembly approach. For example, two or more pre-selected zinc fingers may be fused in a tandem fashion.

[0181] In some embodiments, a zinc finger array comprises multiple zinc fingers fused via peptide bonds. In some embodiments, a zinc finger array comprises multiple zinc fingers, one or more of which connected by peptide linkers. For example, zinc fingers in a multiple finger array can be linked by peptide linkers of 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15 or more amino acids in length. In some embodiments, zinc fingers in a multiple finger array are linked by peptide linkers of 5 amino acids in length. In some embodiments, zinc fingers in a multiple finger array are linked by peptide linkers of 6 amino acids in length. In some embodiments, the two-finger units bind adjacent bases and are connected by a linker with the sequence TGSQKP (SEQ ID NO.: 1154). In some embodiments the two-finger units bind sequences that are separated by 1 or 2 nucleotides and the two-finger units are separated by a linker with the sequence TGGGGSQKP (SEQ ID NO.: 1155).

[0182] In some embodiments, ZF-containing proteins may contain ZF arrays of 2 or more ZF motifs, which may be directly adjacent one another (i.e. separated by a short (canonical) linker sequence), or may be separated by longer, flexible or structured polypeptide sequences. In some embodiments, directly adjacent fingers bind to contiguous nucleic acid sequences, i.e. to adjacent trinucleotides/triplets. In some embodiments, adjacent fingers cross-bind between each other's respective target triplets, which may help to strengthen or enhance the recognition of the target sequence, and leads to the binding of overlapping quadruplet sequences. In some embodiments, distant ZF domains within the same protein may recognize (or bind to) non-contiguous nucleic acid sequences or even to different molecules (e.g. protein rather than nucleic acid).

[0183] In some embodiments, an epigenetic editor comprises zinc fingers comprising more than 3-fingers. In some embodiments, an epigenetic editor comprises at least 6 zinc fingers in the DNA binding domain. In some embodiments, an epigenetic editor comprises 6 zinc fingers in the DNA binding domain that binds to a 18bp target sequence. In some embodiments, the 18bp target sequence is unique in the human genome. In some embodiments, an epigenetic editor comprises zinc fingers comprising at least 7, 8, 9, 10, 11, 12, 13, 14, 15 or more zinc fingers. In some embodiments, the strong affinity of three-finger proteins would allow subsets of the longer array to bind DNA and therefore decrease specificity. Without wishing to be bound by any theory, zinc finger proteins comprising multiple two-finger units or three-finger units joined by extended linkers may confer higher DNA binding specificity as compared to fewer fingers, or

an array with same number of fingers simply joined via peptide bonds. In some embodiments, an epigenetic editor comprises at least three two-finger units connected by peptide linkers, where each of the two finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least four two-finger units connected by peptide linkers, wherein each of the two finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least five two-finger units connected by peptide linkers, wherein each of the two finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least six, seven, eight, nine, ten, or more two-finger units connected by peptide linkers, wherein each of the two finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least two three-finger units connected by peptide linkers, where each of the three finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least three three-finger units connected by peptide linkers, where each of the three finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least four three-finger units connected by peptide linkers, wherein each of the three finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least five three-finger units connected by peptide linkers, wherein each of the three finger units binds a subsite in the target DNA sequence. In some embodiments, an epigenetic editor comprises at least six, seven, eight, nine, ten, or more three-finger units connected by peptide linkers, wherein each of the three finger units binds a subsite in the target DNA sequence.

[0184] In some embodiments, multiple zinc fingers, each recognizing three specific DNA nucleotides, or trinucleotide "subsites", are assembled to target specific DNA sequences in target genes. In some embodiments, such DNA subsites are contiguous sequences in a target gene. In some embodiments, one or more of the DNA subsites are separated by gaps in the target gene. for example, a multi-finger ZF may recognize DNA subsites that span a 1, 2, 3 or more base pairs of inter-subsite gaps between adjacent subsites. In some embodiments, zinc fingers in the multi-finger ZF are connect via peptide linkers. The peptide linkers may be of 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, or more amino acids in length. In some embodiments, a linker comprises 5 or more amino acids. In some embodiments, a linker comprises 7-17 amino acids. In some embodiments, the linker is a flexible linker. In some embodiments, the linker is a rigid linker, e.g., a linker comprising one or more Prolines.

[0185] Zinc finger arrays with sequence specific DNA binding activity may be fused to functional effector domains, e.g. epigenetic effector domains as described herein to confer

epigenetic modifications to DNA sequences, or associated histones in a target gene. In some embodiments, an epigenetic editor described herein comprises a zinc finger array having specificity for a target DNA sequence. In some embodiments a zinc finger array may have the sequence:

SRPGERPFQCRICMRNFSNNNNNNNHTRTHTGEKPFQCRICMRNFSNNNNNNNHLRTH[linker]FQCRICMRNFSNNNNNNNHTRTHTGEKPFQCRICMRNFSNNNNNNNHLRTH[linker]FQCRICMRNFSNNNNNNNHTRTHTGEKPFQCRICMRNFSNNNNNNNHLRTHLRGS (SEQ ID NO.: 1157).

Where NNNNNNN represents the amino acids of the zinc finger recognition helix, which confer DNA-binding specificity upon the zinc finger. And [linker] represents a linker sequence. In some embodiments the linker sequence may be TGSQKP (SEQ ID NO.: 1154). In some embodiments the linker sequence may be TGGGGSQKP (SEQ ID NO.: 1155). In some embodiments, the two linkers of the zinc finger array are the same. In some embodiments, the two linkers of the zinc finger array are different.

[0186] In some embodiments, the programmable DNA binding protein comprises an argonaute protein. One example of such a nucleic acid programmable DNA binding protein is an Argonaute protein from Natronobacterium gregoryi (NgAgo). NgAgo is a ssDNA-guided endonuclease. NgAgo binds 5' phosphorylated ssDNA of ~24 nucleotides (gDNA) to guide it to its target site and will make DNA double-strand breaks at the gDNA site. In contrast to Cas9, the NgAgo-gDNA system does not require a protospacer-adjacent motif (PAM). Using a nuclease inactive NgAgo (dNgAgo) can greatly expand the bases that may be targeted. The characterization and use of NgAgo have been described in Gao et al., Nat Biotechnol., 2016 Jul;34(7):768-73. PubMed PMID: 27136078; Swarts et al., Nature. 507(7491) (2014):258-61; and Swarts et al., Nucleic Acids Res. 43(10) (2015):5120-9, each of which is incorporated herein by reference.

[0187] In some embodiments, the nucleic acid binding domain comprises a virus derived RNA-binding domain guided by an RNA sequence to bind the target gene. In some embodiments, the nucleic acid binding domain comprises a K Homology (KH) domain, a MS2 coat protein domain, a PP7 coat protein domain, a SfMu Com coat protein domain, a sterile alpha motif, a telomerase Ku binding motif and Ku protein, a telomerase Sm7 binding motif and Sm7 protein, or any other RNA recognition motifs.

[0188] In some embodiments, the nucleic acid binding domain comprises an inactivated nuclease, for example, an inactivated meganuclease. Additional non-limiting examples of DNA binding domains include tetracycline-controlled repressor (tetR) DNA binding domain, leucine

zippers, helix-loophelix (HLH) domains, helix-turn-helix domains, zinc fingers, β-sheet motifs, steroid receptor motifs, bZIP domains homeodomains, and AT-hooks.

Effector domains

[0189] Epigenetic editors or epigenetic editing complexes provided herein may include one or more effector protein domains that modulate expression of a target gene. An effector domain can be used to contact a target polynucleotide sequence in a target gene to effect an epigenetic modification, for example, a change in methylation state of DNA nucleotides in the target gene. Accordingly, an epigenetic editor with one or more effector domains may provide the effect of modulating expression of a target gene without altering the DNA sequence of the target gene. For example, in some embodiments, an effector domain results in repression or silencing of expression of a target gene. In some embodiments, an effector domain results in activation or increased expression of a target gene.

[0190] In an aspect, the epigenetic modification described herein is sequence specific, or allele specific. For example, an epigenetic editor may specifically target a DNA sequence recognized by a DNA binding domain of the epigenetic editor. In some embodiments, the target DNA sequence is specific to one copy of a target gene. In some embodiments, the target gene sequence is specific to one allele of a target gene. Accordingly, the epigenetic modification and modulation of expression thereof may be specific to one copy or one allele of the target gene. For example, an epigenetic editor may repress or activate expression of a specific copy harboring a target sequence recognized by the DNA binding domain. In some embodiments, the epigenetic editor represses expression of a specific copy of a target gene, wherein the copy is associated with a disease or disorder. In some embodiments, the epigenetic editor represses expression of a specific copy of a target gene, wherein the copy harbors a mutation associated with a disease or disorder. In some embodiments, the epigenetic editor activates expression of a specific copy of a target gene. In some embodiments, the epigenetic editor activates expression of a specific copy of a target gene that is a wild type copy. The epigenetic modification mediated by an epigenetic editor may be in the vicinity of the target gene, or may be distal to the target gene. In some embodiments, an epigenetic editor may initiate a chemical modification, e.g, DNA methylation, in one or more nucleotides of the target gene. Such methylation may be initiated near the target sequence, and may subsequently spread to one or more nucleotides in the target gene distant from the target sequence.

[0191] An epigenetic effector may deposit a chemical modification at the chromatin at the position of a target gene. Non limiting examples of chemical modifications include methylation, demethylation, acetylation, deacetylation, phosphorylation, SUMOylation and/or ubiquitination

of the DNA or histone residues of the chromatin. In some embodiments, an epigenetic effector may make histone tail modifications. In some embodiments epigenetic effectors may add or remove active marks on histone tails. In some embodiments the active marks may include H3K4 methylation, H3K9 acetylation, H3K27 acetylation, H3K36 methylation, H3K79 methylation, H4K5 acetylation, H4K8 acetylation, H4K12 acetylation, H4K16 acetylation, and/or H4K20 methylation. In some embodiments epigenetic effectors may add or remove repressive marks on histone tails. In some embodiments these repressive marks may include H3K9 methylation and/or H3K27 methylation.

[0192] In some embodiments, an effector domain in an epigenetic editor alters a chemical modification state of a target gene harboring a target sequence. For example, an effector domain may alter a chemical modification state of a nucleotide in the target gene. In some embodiments, an effector domain of an epigenetic editor deposits a chemical modification at a nucleotide in the target gene. In some embodiments, an effector domain of an epigenetic editor deposits a chemical modification of a histone associated with the target gene. In some embodiments, an effector domain of an epigenetic editor removes a chemical modification at a nucleotide in the target gene. In some embodiments, an effector domain of an epigenetic editor removes a chemical modification of a histone associated with the target gene. In some embodiments, the chemical modification increases expression of the target gene. For example, the epigenetic editor may comprise an effector domain having histone acetyltransferase activity. In some embodiments, the chemical modification decreases expression of the target gene. For example, the epigenetic editor may comprise an effector domain having DNA methyltransferase activity.

[0193] The chemical modifications may be deposited or removed by the epigenetic editor in any region of a target gene. In some embodiments, the chemical modification is deposited or removed at a single nucleotide. In some embodiments, the chemical modification is deposited or removed at a single histone. In some embodiments, the chemical modification is deposited at more than 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides. In some embodiments, the chemical modification is removed from more than 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200,300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in a promoter region of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in a promoter region of the target gene. In some embodiments,

the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in a enhancer region of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in a enhancer region of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in a coding region of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in a coding region of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in an exon of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 , 1500, 2000, 2500, 3000or more nucleotides in an exon of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in an intron of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in an intron of the target gene. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in an insulator region of the target gene or chromosome. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in an insulator region of the target gene or chromosome. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in a nucleotide in a silencer region of the target gene or chromosome. In some embodiments, the effector domain of an epigenetic editor alters a chemical modification in at least 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more nucleotides in a silencer region of the target gene or chromosome. In some embodiments, the chemical modification is altered at a CTCF binding region of a target gene or chromosome. In some embodiments, the alteration of the chemical modification state is at or near a transcription initiation site (TSS). In some embodiments, the alteration of the chemical

modification state is 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, or 1000, 1500, 2000, 2500, 3000 nucleotides upstream of a TSS. In some embodiments, the alteration of the chemical modification state is 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 1000, 1500, 2000, 2500, 3000 nucleotides flanking a TSS. In some embodiments, the alteration of the chemical modification state is a DNA methylation state, for example, methylation of DNA near TSS by an epigenetic editor comprising an effector domain with DNA methyltransferase activity, thereby reducing or silencing expression of the target gene.

[0194] The epigenetic modification mediated by an epigenetic editor may be in the vicinity of the target gene, or may be distant to the target gene, or spread from an initial epigenetic modification initiated by the epigenetic editor at one or more nucleotides in a target sequence of the target gene. For example, an epigenetic editor may initiate a chemical modification, e.g, DNA methylation, in one or more nucleotides of the target gene. Such methylation may be initiated near the target sequence, and may subsequently spread to one or more nucleotides in the target gene distant from the target sequence. In some embodiments, the epigenetic editor places, deposits, or removes a modification at a single nucleotide in a target sequence in the target gene, which subsequently spreads to one or more nucleotides upstream or downstream of the single nucleotide. In some instances, additional proteins or transcription factors, for example, transcription repressors, methyltransferases, or transcription regulation scaffold proteins, are involved in the spreading of the chemical modification. In some instances, distant modification is solely mediated by the epigenetic editor. In some embodiments, the chemical modification mediated by an epigenetic editor is 50, 100, 150, 200, 250, 300, 350, 400, 450, or 500 nucleotides from the epigenetic editing target sequence. In some embodiments, the chemical modification mediated by an epigenetic editor is 50, 100, 150, 200, 250, 300, 350, 400, 450, or 500 nucleotides upstream of the epigenetic editing target sequence. In some embodiments, the chemical modification mediated by an epigenetic editor is 50, 100, 150, 200, 250, 300, 350, 400, 450, or 500 nucleotides downstream of the epigenetic editing target sequence. In some embodiments, the chemical modification mediated by an epigenetic editor is at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides from the epigenetic editing target sequence. In some embodiments, the chemical modification mediated by an epigenetic editor is at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides upstream of the epigenetic editing target sequence. In some embodiments, the chemical modification mediated

by an epigenetic editor is at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides downstream of the epigenetic editing target sequence.

[0195] Chemical modifications that may be deposited or removed from a target gene or chromosome region include, but are not limited to DNA or histone methylation, de-methylation, acetylation, deacetylation, phosphorylation, ubiquitination, or any combination thereof.

[0196] In some embodiments, the alteration of the chemical modification state is a DNA methylation state. For example, methylation can be introduced by an effector domain having DNA methyltransferase activity, or can be removed by an effector domain having DNA-demethylase activity. In some embodiments, alteration in methylation state mediated by an epigenetic effector is at a CpG dinucleotide sequence in the target gene or chromosome. In some embodiments, alteration in methylation state mediated by an epigenetic effector is at 1, 2, 3, 4, 5, 6, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, or 1000 CpG dinucleotide sequences in the target gene or chromosome. In some embodiments, the CpG dinucleotide sequences are methylated. In some embodiments, the CpG dinucleotide sequences are de-methylated. In some embodiments, CpG dinucleotide sequences methylated by the epigenetic editor are within target gene or chromosome regions known as CpG islands. In some embodiments, the CpG dinucleotide sequences methylated by the epigenetic editor are not in a CpG island. A CpG island generally refers to a nucleic acid sequence or chromosome region that comprises high frequency of CpG dinucleotides. For example, a CpG island may comprise at least 50% of GC content. In embodiments, a CpG island has a high of observed-to-expected CpG ratio, for example, an observed-to-expected CpG ratio of at least 60%. As used herein, observed-to-expected CpG ratio is determined by Number of CpG * (sequence length) / (Number of C * Number of G). In some embodiments, the CpG island has an observed-to-expected CpG ratio of at least 60%, 70%, 80%, 90% or more. In some embodiments, the CpG island is a sequence or region of at least 200 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 250 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 300 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 350 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 400 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 450 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 500 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 550 nucleotides. In some embodiments, the CpG island is a sequence or region of at least 550, at

least 600, at least 650, at least 700, at least 750, at least 800 or more nucleotides. In some embodiments, only 1, 2, 3, 4, 5, 6, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, or less than 50 CpG dinucleotides are methylated by the epigenetic editor. In some embodiments, CpG dinucleotide sequences de-methylated by the epigenetic editor are within target gene or chromosome regions known as CpG islands. In some embodiments, the CpG dinucleotide sequences de-methylated by the epigenetic editor are not in a CpG island. In some embodiments, only 1, 2, 3, 4, 5, 6, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 30, 40, or less than 50 CpG dinucleotides are de-methylated by the epigenetic editor. In some embodiments, sequence within about 3000 base pairs of the target sequence are methylated by the epigenetic editor. In some embodiments, sequences that is within about 3000, 2900, 2800, 2700, 2600, 2500, 2400, 2300, 2200, 2100, 2000, 1900, 1800, 1700, 1600, 1500, 1400, 1300, 1200, 1100, 1000, 900, 800, 700, 600, 500, 400, 300, 200, or 100 base pairs of the target sequence are methylated by the epigenetic editor.

[0197] In some embodiments, the alteration of chemical modification, e.g., methylation, is at a hypomethylated nucleic acid sequence. For example, the chemically modified sequence in the target gene or chromosome region may lack methyl groups on the 5 -methyl cytosine nucleotide (e.g., in CpG) as compared to a standard control. Hypomethylation may occur, for example, in aging cells or in cancer (e.g., early stages of neoplasia) relative to the younger cell or non-cancer cell, respectively. In some embodiments, the target polynucleotide sequence is within a CpG island. In some embodiments, the target gene is known to be associated with a disease or condition. In some embodiments, the target gene comprises a specific copy of disease related sequence. In some embodiments, the target gene harbors the target sequence which is related to a disease.

[0198] In some embodiments, the alteration of chemical modification, e.g., methylation, is at a hypermethylated nucleic acid sequence. In some embodiments, the chemical modification is within a CpG island.

[0199] Chromatin or DNA sequences chemically modified in the target gene may be within or near the target sequence recognized by an epigenetic editor. In some embodiments, DNA sequence within about 3000 base pairs of the target nucleic acid sequence is chemically modified, e.g., methylated, by the epigenetic editor. In some embodiments, DNA sequence within about 3000, 2900, 2800, 2700, 2600, 2500, 2400, 2300, 2200, 2100, 2000, 1900, 1800, 1700, 1600, 1500, 1400, 1300, 1200, 1100, 1000, 900, 800, 700, 600, 500, 400, 300, 200, or 100 base pairs of the target nucleic acid sequence is chemically modified by the epigenetic editor.

[0200] In some embodiments, chemical modification, e.g. methylation or demethylation, may be introduced by the epigenetic editor in a target gene where the modification isn't at a CpG dinucleotide. For example, the target gene sequence may be de-methylated at the C nucleotide of CpA, CpT, or CpC sequences. Without wishing to be bound by any theory, DNMT3A may be able to methylate nucleotides at non-CpG sites. In some embodiments, an epigenetic editor comprises a DNMT3A domain and effects methylation at CpG, CpA, CpT, and/or CpC sequences. In some embodiments, an epigenetic editor comprises a DNMT3A domain that lacks a regulatory subdomain and only maintains a catalytic domain. In some embodiments, the epigenetic editor comprising a DNMT3A with catalytic domain only effects methylation exclusively at CpG sequences. In some embodiments, an epigenetic editor comprises a DNMT3A domain comprises a mutation, e.g. a R836A mutation, has higher methylation activity at CpA, CpC, and/or CpT sequences as compared to an epigenetic editor comprising a wild type DNMT3A domain.

[0201] In some embodiments, the effector domain comprises a transcription related protein. For example, the effector domain may comprise a transcription factor, a transcription activator, or a transcription repressor. In some embodiments, the effector domain in an epigenetic editor recruits one or more transcription related proteins to a target gene that harbors a target sequence. For example, the effector domain may recruit a transcription factor, a transcription activator, or a transcription repressor to the target gene harboring the target sequence. In some embodiments, the transcription related proteins are endogenous. In some embodiments, the transcription related proteins are introduced together or sequentially with the epigenetic editor. In some embodiments, the transcription related protein is recruited to a region of the target gene in close proximity to the target sequence. In some embodiments, the transcription related protein is recruited to a region that is 100-200bp, 200-300bp, 300-400bp, 400-500bp, 500-600bp, 600-700bp, 700-800bp, 800-900bp, 900-1000bp or more 5' to the target sequence. In some embodiments, the transcription related protein is recruited to a region of the target gene in close proximity to the target sequence. In some embodiments, the transcription related protein is recruited to a region that is 100-200bp, 200-300bp, 300-400bp, 400-500bp, 500-600bp, 600-700bp, 700-800bp, 800-900bp, 900-1000bp or more 3' to the target sequence. In some embodiments, the effector domain comprises a protein that blocks or recruits one or more proteins that block access of a transcription factor to the target gene harboring the target sequence.

[0202] An effector domain alters a chemical modification state of DNA or histone residues associated with the DNA in a target gene. For example, an effector domain may deposit a

chemical modification, or remove a chemical modification, such as DNA methylation, histone tail methylation, or histone tail acetylation at DNA nucleotides in or histone residues bound to a target gene. In some embodiments, an effector domain may directly or indirectly mediate or induce a chemical modification, or remove a chemical modification, such as DNA methylation, histone tail methylation, or histone tail acetylation at DNA nucleotides in or histone residues bound to a target gene. For example, an effector domain may place, deposit, or remove an initial epigenetic modification, e.g., DNA methylation, at one or more nucleotides in a target sequence of the target gene, and the epigenetic modification state may then spread to nucleotides 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 or more base pairs upstream or downstream of the initial epigenetic modification sites. The chemical modification deposited at target gene DNA nucleotides or histone residues may be in close proximity to a target sequence (sequence recognized by a DNA binding portion of an epigenetic editor) in the target gene, or may be distant from the target sequence. In some embodiments, an effector domain alters a chemical modification state of a nucleotide or histone tail bound to a nucleotide within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, or 1000 nucleotides flanking the target sequence. As used herein, "flanking" refers to nucleotide positions 5' to the 5' end of and 3' to the 3' end of a particular sequence, e.g. a target sequence. In some embodiments, an effector domain mediates or induces a chemical modification change of a nucleotide or a histone tail bound to a nucleotide distant from a target sequence. Without wishing to be bound by any theory, an epigenetic editor effector domain may initiate a chemical modification, e.g, DNA methylation, in one or more nucleotides of the target gene. Such modification may be initiated near the target sequence, and may subsequently spread to one or more nucleotides in the target gene distant from the target sequence. In some instances, additional proteins or transcription factors, for example, transcription repressors, methyltransferases, or transcription regulation scaffold proteins, are involved in the spreading of the chemical modification. In some embodiments, an effector domain initiates alteration of a chemical modification state of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides from the target sequence in the target gene, either upstream or downstream of the target sequence. In certain embodiments, the chemical modification, e.g., methylation or demethylation, maybe initiated at less than 2, 3, 5, 10, 20, 30, 40, 50, or 100 nucleotides in the target gene and spreads to at least 100, 200, 300, 400, 500, 600,

700, 800, 900, 1000, 2000, or more nucleotides in the target gene. In some embodiments, the chemical modification spreads to nucleotides in the entire target gene. In some embodiments, the alteration in modification state is a DNA methylation state. In some embodiments, the alteration in modification state is a histone methylation state. In some embodiments, the alteration in modification state is a histone acetylation state.

[0203] In some embodiments, an effector domain makes an epigenetic modification at a target gene that increases or activates expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of DNA or histone residues associated with the DNA in a target gene harboring the target sequence, thereby increasing expression of the target gene. In some embodiments, the alteration in chemical modification state comprises removal of a methyl group form a DNA nucleotide in the target gene. In some embodiments, the alteration in chemical modification state comprises acetylation of a histone tail bound to a DNA nucleotide in the target gene. In some embodiments, the alteration in chemical modification state comprises methylation of a histone tail bound to a DNA nucleotide in the target gene, e.g., a H3K4me1 methylation. In some embodiments, the alteration in chemical modification state comprises removal of an acetyl group from histone tail bound to a DNA nucleotide in the target gene, e.g., a H3K9me2 methylation. An epigenetic editor may initiate a chemical modification, in one or more nucleotides of the target gene, near the target sequence, which may subsequently spread to one or more nucleotides in the target gene distant from the target sequence, thereby increasing or activating expression of the target gene. In some instances, distant modification is solely mediated by the epigenetic editor. In some instances, additional proteins or transcription factors, for example, transcription activators, are involved in the spreading of the chemical modification. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, or1000 nucleotides flanking a target sequence in a target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain initiates alteration of a chemical modification state of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides flanking the target sequence in the target gene, thereby increasing or activating expression of the target gene.

[0204] In some embodiments, an effector domain alters a chemical modification state, e.g., demethylation of a nucleotide, 100-200 nucleotides 5' to the target sequence in the target gene,

thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 200-300 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 300-400 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 400-500 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 500-600 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 600-700 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 700-800 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain initiates alteration of a chemical modification state of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 5' to the target sequence in the target gene, thereby increasing or activating expression of the target gene, thereby increasing expression of the target gene.

[0205] In some embodiments, an effector domain alters a chemical modification state, e.g., demethylation of a nucleotide, of a nucleotide 100-200 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 200-300 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 300-400 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 400-500 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 500-600 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 600-700 nucleotides 3' to the target

sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 700-800 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, the chemical modification state is a methylation state. In some embodiments, the effector domain of an epigenetic effector results in demethylation of one or more nucleotides in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain initiates alteration of a chemical modification state, e.g. DNA demethylation, of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 3' to the target sequence in the target gene, thereby increasing or activating expression of the target gene, thereby increasing expression of the target gene.

[0206] In some embodiments, an effector domain alters a histone modification state of a histone associated with or bound to the target gene. For example, an effector domain may deposit a modification on one or more lysine residues of histone tails of histones associated with the target gene. The histone amino acid residues modified may be within the vicinity of the target sequence within the target gene. In some embodiments, an effector domain alters a histone modification state 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 100-200 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 200-300 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 300-400 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 400-500 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 500-600 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 600-700 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state

700-800 nucleotides 5' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 100-200 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 200-300 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 300-400 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 400-500 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 500-600 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 600-700 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 700-800 nucleotides 3' to the target sequence in the target gene, thereby increasing expression of the target gene. In some embodiments, the histone modification state is a acetylation state. In some embodiments, the effector domain of an epigenetic effector results in acetylation of one or more histone tails of histones associated with the target gene, thereby increasing expression of the target gene. In some embodiments, the histone modification state is a methylation state. In some embodiments, the epigenetic effector results in H3K4 or H3K79 methylation (e.g. one or more of a H3K4me2, H3K4me3, and H3K79me3 methylation) at one or more histone tails associated with the target gene, thereby increasing expression of the target gene.

[0207] In some embodiments, an effector domain makes an epigenetic modification at a target gene that represses, decreases, or silences expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of DNA or histone residues associated with the DNA in a target gene harboring the target sequence, thereby reducing or silencing expression of the target gene. Epigenetic editors that decrease expression of a target gene may comprise multiple effector domains, resulting in multiple modifications to a target gene, for example, both DNA methylation and histone tail de-acetylation. In some embodiments, an effector domain alters a chemical modification state of DNA in the target gene or histone bound to the target gene near the target sequence, thereby decreasing expression of the target gene. In

some embodiments, an effector domain alters a chemical modification state of DNA in the target gene or histone bound to the target gene distant from the target sequence in the target gene, thereby decreasing expression of the target gene. In some embodiments, an effector domain mediates or induces a chemical modification state of DNA in the target gene or histone bound to the target gene that are distant from the target sequence in the target gene. For example, an epigenetic editor may initiate a chemical modification, e.g, DNA methylation, in one or more nucleotides of the target gene. Such modification may be initiated near the target sequence, and may subsequently spread to one or more nucleotides in the target gene distant from the target sequence, thereby decreasing expression of the target gene. In some instances, the distant modification is solely mediated by the epigenetic editor. In some instances, additional proteins or transcription factors, for example, transcription repressors, methyltransferases, or transcription regulation scaffold proteins, are involved in the spreading of the chemical modification. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state, e.g., DNA methylation, of one or more nucleotides in close proximity to the target gene, and the altered chemical modification state subsequently spreads to nucleotides 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene.

[0208] In some embodiments, an effector domain alters a chemical modification state, e.g., DNA methylation, of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the altered chemical modification state subsequently spreads to nucleotides 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene.

[0209] In some embodiments, an effector domain alters a chemical modification state of a nucleotide 100-200 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 200-300 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments,

an effector domain alters a chemical modification state of a nucleotide 300-400 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 400-500 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 500-600 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 600-700 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 700-800 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene.

[0210] In some embodiments, an effector domain alters a chemical modification state of a nucleotide 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain initiates alteration of a chemical modification state, e.g. DNA methylation, of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 3' to the target sequence in the target gene, thereby increasing or activating expression of the target gene, thereby increasing expression of the target gene.

[0211] In some embodiments, an effector domain alters a chemical modification state of a nucleotide 100-200 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 200-300 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 300-400 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 400-500 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 500-600 nucleotides 3' to the target sequence in the

target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 600-700 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a chemical modification state of a nucleotide 700-800 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the chemical modification state is a methylation state. In some embodiments, the effector domain of an epigenetic effector results in methylation of one or more nucleotides in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain initiates alteration of a chemical modification state, e.g. DNA methylation, of one or more nucleotides or one or more histone residues bound to one or more nucleotides within 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 200, 300, 400, 500 nucleotides flanking the target sequence, and the chemical modification state alteration spreads to one or more nucleotides at least 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000 or more nucleotides 5' to the target sequence in the target gene, thereby increasing or activating expression of the target gene, thereby increasing expression of the target gene.

[0212] In some embodiments, an effector domain alters a histone modification state of a histone associated with or bound to the target gene. For example, an effector domain may deposit a modification on one or more lysine residues of histone tails of histones associated with the target gene. The histone amino acid residues modified may be within the vicinity of the target sequence within the target gene. In some embodiments, an effector domain alters a histone modification state 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 100-200 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 200-300 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 300-400 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 400-500 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 500-600 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression

of the target gene. In some embodiments, an effector domain alters a histone modification state 600-700 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 700-800 nucleotides 5' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 50, 60, 70, 80, 90, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 or more nucleotides 5' or 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 100-200 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 200-300 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 300-400 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 400-500 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 500-600 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 600-700 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, an effector domain alters a histone modification state 700-800 nucleotides 3' to the target sequence in the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the histone modification state is a acetylation state. In some embodiments, the effector domain of an epigenetic effector results in de-acetylation of one or more histone tails of histones associated with the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the histone modification state is a methylation state. In some embodiments, the epigenetic effector results in a H3K9, H3K27 or H4K20 methylation (e.g. one or more of a H3K9me2, H3K9me3, H3K27me2, H3K27me3, and H4K20me3 methylation) at one or more histone tails associated with the target gene, thereby reducing or silencing expression of the target gene.

[0213] In an aspect, also provided herein is an epigenetically edited chromosome or an epigenetically edited genome or cell comprising the epigenetically edited chromosome, wherein one or more target nucleotides in the epigenetically edited chromosome comprises an epigenetic

modification mediated or induced by an epigenetic editor provided herein. For example, an epigenetically edited chromosome may comprise one or more methylated nucleotides as compared to a chromosome not contacted with an epigenetic editor. In some embodiments, the epigenetically edited chromosome comprises methylated CpGs. An epigenetically edited chromosome may comprise one or more types of epigenetic modifications as compared to an un-edited control chromosome of the same species, for example, epigenetic modifications to DNA nucleotides or histone tails of the chromosome. In some embodiments, an epigenetically edited chromosome comprises one or more methylated nucleotides as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more demethylated nucleotides as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more methylated histone tails as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more demethylated histone tails as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more acetylated histone tails as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more deacetylated histone tails as compared to a control chromosome not contacted with the epigenetic editor. In some embodiments, an epigenetically edited chromosome comprises one or more or any combination of epigenetic modifications, e.g, DNA methylation and histone deacetylation, DNA methylation and histone H3K9 methylation, DNA methylation and histone H3K4 demethylation, DNA demethylation and histone acetylation, DNA demethylation and histone H3K9 demethylation, DNA demethylation and histone H3K4 methylation, in any of the chromosome regions, e.g., chromosome regions as described herein, or any combination thereof. As used herein, a control chromosome may refer to the original epigenetic state, or unedited state, where a chromosome has not been contacted with an epigenetic editor as described herein. In some embodiments, a control chromosome may already bear epigenetic marks, e.g. DNA methylation, without being contacted with an epigenetic editor.

[0214] In some embodiments, all CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450,

500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0215] In some embodiments, all CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0216] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of

histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0217] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the

epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0218] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0219] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, all CpG dinucleotides within 1500bp flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 550, 500, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the

epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0220] In some embodiments, all CpG dinucleotides within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0221] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the

chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0222] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking

the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0223] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0224] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%,

75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, all CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0225] In some embodiments, all CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of

the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0226] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0227] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0228] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%,

15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0229] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.In some embodiments, all CpG dinucleotides within 500bps flanking a transcription start site of a gene in

the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0230] In some embodiments, all CpG dinucleotides within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0231] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the

chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0232] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not

contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0233] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0234] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0235] In some embodiments, all CpG dinucleotides within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90 or more CpG dinucleotides within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 200bps flanking a

transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0236] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0237] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the

original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0238] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the

chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0239] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a transcription start site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a transcription start site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0240] In some embodiments, all CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the

epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0241] In some embodiments, all CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0242] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%,

6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0243] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some

embodiments, one single histone octamer bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0244] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0245] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35,

40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0246] In some embodiments, all CpG dinucleotides within 1500bp flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 550, 500, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0247] In some embodiments, all CpG dinucleotides within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0248] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic

editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0249] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0250] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0251] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or

the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0252] In some embodiments, all CpG dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0253] In some embodiments, all CpG dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG

dinucleotides within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0254] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0255] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some

embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0256] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the

epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0257] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0258] In some embodiments, all CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited

chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0259] In some embodiments, all CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0260] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic

editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0261] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a

promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0262] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0263] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the

original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0264] In some embodiments, all CpG dinucleotides within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90 or more CpG dinucleotides within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0265] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2,

3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0266] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones

bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0267] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0268] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the

chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0269] In some embodiments, all CpG dinucleotides within 2000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding

sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0270] In some embodiments, all CpG dinucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0271] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator

sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0272] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a

comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0273] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0274] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0275] In some embodiments, all CpG dinucleotides within 1500bp flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 550, 500, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%,

30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0276] In some embodiments, all CpG dinucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0277] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or

CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0278] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the

original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0279] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene

in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0280] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0281] In some embodiments, all CpG dinucleotides within 1000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a

gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0282] In some embodiments, all CpG dinucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0283] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15,

16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0284] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a enhancer

sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0285] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some

embodiments, one single histone octamer bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0286] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0287] In some embodiments, all CpG dinucleotides within 500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically

edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a enhancer sequence, an isolator sequence, or a CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0288] In some embodiments, all CpG dinucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0289] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15,

16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0290] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator

sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0291] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single

histone octamer bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0292] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a enhancer sequence, isolator sequence, or CTCF binding sequence of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0293] In some embodiments, all CpG dinucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90 or more CpG dinucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are demethylated

as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0294] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In

some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0295] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0296] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18,

19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0297] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a enhancer sequence, isolator

sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a enhancer sequence, isolator sequence, or CTCF binding site of a gene in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0298] In some embodiments, an epigenetically modified chromosome results from contacting a chromosome with an epigenetic editor as described herein. For example, an epigenetic editor may target a target sequence in a target gene in the chromosome and alter an epigenetic modification state of one or more nucleotides or one or more histone tails in the chromosome. The epigenetic modification placed or removed by the epigenetic editor may be in close proximity to the target sequence, or may be 50, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000, 2500, 3000 or more base pairs upstream or downstream of such target sequence. in some embodiments, the epigenetic editor initiates an epigenetic modification, e.g. DNA methylation, at one or more nucleotides in close proximity to the target sequence. The initial epigenetic modification may spread to nucleotides or histones upstream or downstream of the target sequence, for example, 50, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, 1500, 1600, 1700, 1800, 1900, 2000, 2500, 3000 or more base pairs upstream or downstream of such target sequence.

[0299] In some embodiments, all CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is

methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0300] In some embodiments, all CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0301] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the

chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0302] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0303] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0304] In some embodiments, all histone tails of histones bound to DNA nucleotides within 2000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120 or more histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the

chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 2000bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0305] In some embodiments, all CpG dinucleotides within 1500bp flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 550, 500, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0306] In some embodiments, all CpG dinucleotides within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700 or more CpG dinucleotides within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within

1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0307] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0308] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some

embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0309] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the

epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0310] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90 or more histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1500bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0311] In some embodiments, all CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited

chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0312] In some embodiments, all CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 350, 400, 450, 500 or more CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0313] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic

editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0314] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a target sequence in the epigenetically

edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0315] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0316] In some embodiments, all histone tails of histones bound to DNA nucleotides within 1000bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 55, 60 or more histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted

with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 1000bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0317] In some embodiments, all CpG dinucleotides within 500bps flanking a promoter sequence of a gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0318] In some embodiments, all CpG dinucleotides within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30, 35, 40, 45, 50, 60, 70, 80, 90, 100, 150, 200 or more CpG dinucleotides within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as

compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0319] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0320] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0321] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone

tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0322] In some embodiments, all histone tails of histones bound to DNA nucleotides within 500bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25, 30 or more histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 500bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0323] In some embodiments, all CpG dinucleotides within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 25,

30, 35, 40, 45, 50, 60, 70, 80, 90 or more CpG dinucleotides within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of CpG dinucleotides within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single CpG dinucleotide within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the gene or the gene in a comparable cell not contacted with the epigenetic editor.

[0324] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is methylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0325] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is demethylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, the histone is histone H3 and methylation is at Lysine 9, marking the target gene in the epigenetically edited chromosome for repressed expression. In some embodiments, the histone is histone H3 and methylation is at Lysine 4, marking the target gene in the epigenetically edited chromosome for increased expression.

[0326] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs

within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is acetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0327] In some embodiments, all histone tails of histones bound to DNA nucleotides within 200bps flanking a promoter sequence of a target gene in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20 or more histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, at least 5%, 6%, 7%, 8%, 9%, 10%, 15%, 20%, 25%, 30%, 35%, 40%, 45%, 50%, 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, 96%, 97%, 98%, or 99% of histone tails of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell are deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone tail of histones bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor. In some embodiments, one single histone octamer bound to DNAs within 200bps flanking a target sequence in the epigenetically edited chromosome in a cell is deacetylated as compared to the original state of the chromosome or the chromosome in a comparable cell not contacted with the epigenetic editor.

[0328] In some embodiments, the effector domain comprises a histone methyltransferase domain. For example, repression (or silencing) may result from repressive chromatin markers, methylation of DNA, methylation of histone residues (e.g., H3K9, H3K27), or deacetylation of histone residues.) on chromatin containing a target nucleic acid sequence. Without intending to

be bound by any theory, the method can be used to change epigenetic state by, for example, closing chromatin via methylation or introducing repressive chromatin markers on chromatin containing the target nuclei acid sequence (e.g., gene).

[0329] Specific epigenetic imprints direct gene transcription or gene silencing. For example, DNA methylation, histone modification, repressor proteins binding to silencer regions, and other transcriptional activities alter gene expression without changing the underlying DNA sequence. Thus, the transcriptional regulation allows for expression of specific genes in a particular manner, while repressing other genes. In certain instances, cell fate or function can be controlled, either for initial differentiation (e.g., during the organism's development) or to reprogram a cell or cell type (e.g., during disease such as cancer, chronic inflammation, auto-immune disease, illnesses related to various microbiomes of an organism, etc.). Histone modifications play a structural and biochemical role in gene transcription, in one avenue by formation or disruption of the nucleosome structure that binds to the histone and prevents gene transcription. Histones are basic proteins that are commonly found in the nucleus of eukaryotic cells, ranging from multicellular organisms including humans to unicellular organisms represented by fungi (mold and yeast) and ionically bind to genomic DNA. Histones usually consist of five components (H1, H2A, H2B, H3 and H4) and are highly similar across biological species. In the case of histone H4, for example, budding yeast histone H4 (full-length 102 amino acid sequence) and human histone H4 (full-length 102 amino acid sequence) are identical in 92% of the amino acid sequences and differ only in 8 residues. Among the natural proteins assumed to be present in several tens of thousands of organisms, histones are known to be proteins most highly preserved among eukaryotic species. Genomic DNA is folded with histones by ordered binding, and a complex of the both forms a basic structural unit called a nucleosome. In addition, aggregation of the nucleosomes forms a chromosomal chromatin structure. Histones are subject to modifications, such as acetylation, methylation, phosphorylation, ubiquitination, SUMOylation and the like, at their N-terminal ends called histone tails, and maintain or specifically convert the chromatin structure, thereby controlling responses such as gene expression, DNA replication, DNA repair and the like, which occur on chromosomal DNA. Post-translational modification of histones is an epigenetic regulatory mechanism, and is considered essential for the genetic regulation of eukaryotic cells. Recent studies have revealed that chromatin remodeling factors such as SWI/SNF, RSC, NURF, NRD and the like, which encourage DNA access to transcription factors by modifying the nucleosome structure, histone acetyltransferases (HATs) that regulate the acetylation state of histones, and histone deacetylases (HDACs), act as important regulators. DNA methylation occurs primarily at CpG

sites (shorthand for "C-phosphate-G-" or "cytosine-phosphate-guanine"). Highly methylated areas of DNA tend to be less transcriptionally active than lesser methylated sites. Many mammalian genes have promoter regions near or including CpG islands (regions with a high frequency of CpG sites).

[0330] In particular, the unstructured N-termini of histones may be modified by at least one of acetylation, methylation, ubiquitylation, phosphorylation, sumoylation, ribosylation, citrullination O-GlcNAcylation, or crotonylation. For example, acetylation of K14 and K9 lysines of histone H3 by histone acetyltransferase enzymes may be linked to transcriptional competence in humans. Lysine acetylation may directly or indirectly create binding sites for chromatin-modifying enzymes that regulate transcriptional activation. For example, histone acetyltransferases (HATs) utilize acetyl-CoA as a cofactor and catalyze the transfer of an acetyl group to the epsilon amino group of the lysine side chains. This neutralizes the lysine's positive charge and weakens the interactions between histones and DNA, thus opening the chromosomes for transcription factors to bind and initiate transcription. Likewise, histone methylation of lysine 9 of histone H3 may be associated with heterochromatin, or transcriptionally silent chromatin. Particular DNA methylation patterns may be established and modified by at least one or more, two or more, three or more, four or more, or five or more independent DNA methyltransferases, including DNMT1, DNMT3A, and DNMT3B.

[0331] In some embodiments, the effector domain comprises a histone methyltransferase domain. In some embodiments, the effector domain comprises a DOT1L domain, a SET domain, a SUV39H1 domain, a G9a/EHMT2 protein domain, a EZH1 domain, a EZH2 domain, a SETDB1 domain, or any combination thereof. In some embodiments, the effector domain comprises a histone-lysine-N-methyltransferase SETDB1 domain.

[0332] In some embodiments, the effector domain comprises a DNA methyltransferase domain or a Histone methyltransferase domain. DNA methyltransferase domains may mediate methylation at DNA nucleotides, for example at any of an A, T, G or C nucleotide. In some embodiments, the methylated nucleotide is a N6-methyladenosine (m6A). In some embodiments, the methylated nucleotide is a 5-methylcytosine (5mC). In some embodiments, the methylation is at a CG (or CpG) dinucleotide sequence. In some embodiments, the methylation is at a CHG or CHH sequence, where H is any one of A, T, or C.

[0333] In some embodiments, the effector domain comprises a DNA methyltransferase DNMT domain that catalyzes transfer of a methyl group to cytosine, thereby repressing expression of the target gene through the recruitment of repressive regulatory proteins. In some embodiments, the effector domain comprises a DNA methyltransferase (DNMT) family protein domain. In

some embodiments, the effector domain comprises a DNMT1 domain. In some embodiments, the effector domain comprises a TRDMT1 domain. In some embodiments, the effector domain comprises a DNMT3 domain. In some embodiments, the effector domain comprises a DNMT3A domain. In some embodiments, the effector domain comprises a DNMT3B domain. In some embodiments, the effector domain comprises a DNMT3C domain. In some embodiments, the effector domain comprises a DNMT3L domain. In some embodiments, the effector domain comprises a fusion of DNMT3A-DNMT3L domain.

[0334] Exemplary methyltransferase that may be part of an epigenetic effector domain are provided in Table 1 below.

Table 1. Exemplary methyltransferase sequences that may be used in epigenetic effector domains

Protein Name	Species	Target	Protein Sequence
DNMT1	Human	5mC	SEQ ID NO.: 32
DNMT3A	Human	5mC	SEQ ID NO.: 33
DNMT3B	Human	5mC	SEQ ID NO.: 35
DNMT3C	Mouse	5mC	SEQ ID NO.: 36
DNMT3L	Human	5mC	SEQ ID NO.: 37
DNMT3L	Mouse	5mC	SEQ ID NO.: 39
TRDMT1 (DNMT2)	Human	tRNA 5mC	SEQ ID NO.: 41
M.MpeI	Mycoplasma penetrans	5mC	SEQ ID NO.: 42
M.SssI	Spiroplasma monobiae	5mC	SEQ ID NO.: 43
M.HpaII	Haemophilus parainfluenzae	5mC (CCGG)	SEQ ID NO.: 44
M.AluI	Arthrobacter luteus	5mC (AGCT)	SEQ ID NO.: 45
M.HaeIII	Haemophilus aegyptius	5mC (GGCC)	SEQ ID NO.: 46
M.HhaI	Haemophilus haemolyticus	5mC (GCGC)	SEQ ID NO.: 47
M.MspI	Moraxella	5mC (CCGG)	SEQ ID NO.: 48
Masc1	Ascobolus	5mC	SEQ ID NO.: 49
MET1	Arabidopsis	5mC	SEQ ID NO.: 50
Masc2	Ascobolus	5mC	SEQ ID NO.: 51
Dim-2	Neurospora	5mC	SEQ ID NO.: 52
dDnmt2	Drosophila	5mC	SEQ ID NO.: 53
Pmt1	S. Pombe	5mC	SEQ ID NO.: 54
DRM1	Arabidopsis	5mC	SEQ ID NO.: 55
DRM2	Arabidopsis	5mC	SEQ ID NO.: 56
CMT1	Arabidopsis	5mC	SEQ ID NO.: 57
CMT2	Arabidopsis	5mC	SEQ ID NO.: 58

Protein Name	Species	Target	Protein Sequence
CMT3	Arabidopsis	5mC	SEQ ID NO.: 59
Rid	Neurospora	5mC	SEQ ID NO.: 60
hsdM gene	bacteria (E.coli, strain 12)	m6A	SEQ ID NO.: 61
hsdS gene	bacteria (E.coli, strain 12)	m6A	SEQ ID NO.: 62
M.TaqI	bacteria; Thermus aquaticus	m6A	SEQ ID NO.: 63
M.EcoDam	E. coli	m6A	SEQ ID NO.: 64
M.CcrMI	Caulobacter crescentus	m6A	SEQ ID NO.: 65
CamA	Clostridioides difficile	m6A	SEQ ID NO.: 66

[0335] In some embodiments, the effector domain recruits one or more protein domains that repress expression of the target gene. In some embodiments, the effector domain interacts with a scaffold protein domain that recruits one or more protein domains that repress expression of the target gene. For example, the effector domain may recruit or interact with a scaffold protein domain that recruits a PRMT protein, a HDAC protein, a SETDB1 protein, or a NuRD protein domain. In some embodiments, the effector domain comprises a Krüppel associated box (KRAB) repression domain; a Repressor Element Silencing Transcription Factor (REST) repression domain, KRAB-associated protein 1 (KAP1) domain, a MAD domain, a FKHR (forkhead in rhabdosarcoma gene) repressor domain, aEGR-1 (early growth response gene product-1) repressor domain, a ets2 repressor factor repressor domain (ERD), a MAD smSIN3 interaction domain (SID), a WRPW motif of the hairy-related basic helix-loop-helix (bHLH) repressor proteins; an HP1 alpha chromo-shadow repression domain, or any combination thereof. In some embodiments, the effector domain comprises a KRAB domain. In some embodiments, the effector domain comprises a Tripartite motif containing 28 (TRIM28, TIF1-beta, or KAP1) protein.

[0336] In some embodiments, an effector domain comprises a protein domain that represses expression of the target gene. For example, the effector domain may comprise a functional domain derived from a zinc finger repressor protein. In some embodiments, the effector domain comprises a functional repression domain derived from a KOX1/ZNF10 domain, a KOX8/ZNF708 domain, a ZNF43 domain, a ZNF184 domain, a ZNF91 KRAB domain, a HPF4 domain, a HTF10 domain or a HTF34 domain or any combination thereof. In some embodiments, the effector domain comprises a functional repression domain derived from a ZIM3 protein domain, a ZNF436 domain, a ZNF257 domain, a ZNF675 domain, a ZNF490 domain, a ZNF320 domain, a ZNF331 domain, a ZNF816 domain, a ZNF680 domain, a ZNF41 domain, a ZNF189 domain, a ZNF528 domain, a ZNF543 domain, a ZNF554

domain, a ZNF140 domain, a ZNF610 domain, a ZNF264 domain, a ZNF350 domain, a ZNF8 domain, a ZNF582 domain, a ZNF30 domain, a ZNF324 domain, a ZNF98 domain, a ZNF669 domain, a ZNF677 domain, a ZNF596 domain, a ZNF214 domain, a ZNF37A domain, a ZNF34 domain, a ZNF250 domain, a ZNF547 domain, a ZNF273 domain, a ZNF354A domain, a ZFP82 domain, a ZNF224 domain, a ZNF33A domain, a ZNF45 domain, a ZNF175 domain, a ZNF595 domain, a ZNF184 domain, a ZNF419 domain, a ZFP28-1 domain, a ZFP28-2 domain, a ZNF18 domain, a ZNF213 domain, a ZNF394 domain, a ZFP1 domain, a ZFP14 domain, a ZNF416 domain, a ZNF557 domain, a ZNF566 domain, a ZNF729 domain, a ZIM2 domain, a ZNF254 domain, a ZNF764 domain, a ZNF785 domain or any combination thereof. In some embodiments, the domain is a ZIM3 domain, a ZNF554 domain, a ZNF264 domain, a ZNF324 domain, a ZNF354A domain, a ZNF189 domain, a ZNF543 domain, a ZFP82 domain, a ZNF669 domain, or a ZNF582 domain or any combination thereof. In some embodiments, the domain is a ZIM3 domain, a ZNF554 domain, a ZNF264 domain, a ZNF324 domain, or a ZNF354A domain or any combination thereof. In some embodiments, the domain is a ZIM3 domain.

[0337] In some embodiments, an effector domain can be an alternate KRAB domain (e.g.,). Alternatively or in addition to, an effector domain can be a non-KRAB domain (e.g.)

[0338] In some embodiments, the protein fusion construct can have 1 effector domain, 2 effector domains, 3 effector domains, 4 effector domains, 5 effector domains, 6 effector domains, 7 effector domains, 8 effector domains, 9 effector domains, or 10 effector domains.

[0339] Sequences of exemplary functional domains that may reduce or silence target gene expression are provided in Table 2 below. Further examples of repressors and repressor domains can be found in PCT/US2021/030643 and Tycko *et al.* (Tycko J, DelRosso N, Hess GT, Aradhana, Banerjee A, Mukund A, Van MV, Ego BK, Yao D, Spees K, Suzuki P, Marinov GK, Kundaje A, Bassik MC, Bintu L. High-Throughput Discovery and Characterization of Human Transcriptional Effectors. Cell. 2020 Dec 23;183(7):2020-2035.e16. doi: 10.1016/j.cell.2020.11.024. Epub 2020 Dec 15. PMID: 33326746; PMCID: PMC8178797.), which are incorporated here by reference to it entirety.

Table 2. Exemplary effector domains that may reduce or silence gene expression

Protein	**Protein Sequence**
ZIM3	SEQ ID NO.: 67
ZNF436	SEQ ID NO.: 68
ZNF257	SEQ ID NO.: 69
ZNF675	SEQ ID NO.: 70

Protein	Protein Sequence
ZNF490	SEQ ID NO.: 71
ZNF320	SEQ ID NO.: 72
ZNF331	SEQ ID NO.: 73
ZNF816	SEQ ID NO.: 74
ZNF680	SEQ ID NO.: 75
ZNF41	SEQ ID NO.: 76
ZNF189	SEQ ID NO.: 77
ZNF528	SEQ ID NO.: 78
ZNF543	SEQ ID NO.: 79
ZNF554	SEQ ID NO.: 80
ZNF140	SEQ ID NO.: 81
ZNF610	SEQ ID NO.: 82
ZNF264	SEQ ID NO.: 83
ZNF350	SEQ ID NO.: 84
ZNF8	SEQ ID NO.: 85
ZNF582	SEQ ID NO.: 86
ZNF30	SEQ ID NO.: 87
ZNF324	SEQ ID NO.: 88
ZNF98	SEQ ID NO.: 89
ZNF669	SEQ ID NO.: 90
ZNF677	SEQ ID NO.: 91
ZNF596	SEQ ID NO.: 92
ZNF214	SEQ ID NO.: 93
ZNF37A	SEQ ID NO.: 94
ZNF34	SEQ ID NO.: 95
ZNF250	SEQ ID NO.: 96
ZNF547	SEQ ID NO.: 97
ZNF273	SEQ ID NO.: 98
ZNF354A	SEQ ID NO.: 99
ZFP82	SEQ ID NO.: 100
ZNF224	SEQ ID NO.: 101
ZNF33A	SEQ ID NO.: 102
ZNF45	SEQ ID NO.: 103
ZNF175	SEQ ID NO.: 104
ZNF595	SEQ ID NO.: 105
ZNF184	SEQ ID NO.: 106

Protein	Protein Sequence
ZNF419	SEQ ID NO.: 107
ZFP28-1	SEQ ID NO.: 108
ZFP28-2	SEQ ID NO.: 109
ZNF18	SEQ ID NO.: 110
ZNF213	SEQ ID NO.: 111
ZNF394	SEQ ID NO.: 112
ZFP1	SEQ ID NO.: 113
ZFP14	SEQ ID NO.: 114
ZNF416	SEQ ID NO.: 115
ZNF557	SEQ ID NO.: 116
ZNF566	SEQ ID NO.: 117
ZNF729	SEQ ID NO.: 118
ZIM2	SEQ ID NO.: 119
ZNF254	SEQ ID NO.: 120
ZNF764	SEQ ID NO.: 121
ZNF785	SEQ ID NO.: 122
ZNF10 (KOX1)	SEQ ID NO.: 123
CBX5 (chromoshadow domain)	SEQ ID NO.: 124
RYBP (YAF2_RYBP component of PRC1)	SEQ ID NO.: 125
YAF2 (YAF2_RYBP component of PRC1)	SEQ ID NO.: 126
MGA (component of PRC1.6)	SEQ ID NO.: 127
CBX1 (chromoshadow)	SEQ ID NO.: 128
SCMH1 (SAM_1/SPM)	SEQ ID NO.: 129
MPP8 (Chromodomain)	SEQ ID NO.: 130
SUMO3 (Rad60-SLD)	SEQ ID NO.: 131
HERC2 (Cyt-b5)	SEQ ID NO.: 132
BIN1 (SH3_9)	SEQ ID NO.: 133
PCGF2 (RING finger protein domain)	SEQ ID NO.: 134
TOX (HMG box)	SEQ ID NO.: 135
FOXA1 (HNF3A C-terminal domain)	SEQ ID NO.: 136
FOXA2 (HNF3B C-terminal domain)	SEQ ID NO.: 137
IRF2BP1 (IRF-2BP1_2 N-terminal domain)	SEQ ID NO.: 138
IRF2BP2 (IRF-2BP1_2 N-terminal domain)	SEQ ID NO.: 139
IRF2BPL IRF-2BP1_2 N-terminal domain	SEQ ID NO.: 140
HOXA13 (homeodomain)	SEQ ID NO.: 141
HOXB13 (homeodomain)	SEQ ID NO.: 142

Protein	Protein Sequence
HOXC13 (homeodomain)	SEQ ID NO.: 143
HOXA11 (homeodomain)	SEQ ID NO.: 144
HOXC11 (homeodomain)	SEQ ID NO.: 145
HOXC10 (homeodomain)	SEQ ID NO.: 146
HOXA10 (homeodomain)	SEQ ID NO.: 147
HOXB9 (homeodomain)	SEQ ID NO.: 148
HOXA9 (homeodomain)	SEQ ID NO.: 149

[0340] Sequences of additional exemplary functional domains that may reduce or silence target gene expression are provided in Table 3 below.

Table 3. Exemplary effector domains that may reduce or silence gene expression

Gene name	Extended Domain sequence
ZFP28_HUMAN	SEQ ID NO.: 150
ZN334_HUMAN	SEQ ID NO.: 151
ZN568_HUMAN	SEQ ID NO.: 152
ZN37A_HUMAN	SEQ ID NO.: 153
ZN181_HUMAN	SEQ ID NO.: 154
ZN510_HUMAN	SEQ ID NO.: 155
ZN862_HUMAN	SEQ ID NO.: 156
ZN140_HUMAN	SEQ ID NO.: 157
ZN208_HUMAN	SEQ ID NO.: 158
ZN248_HUMAN	SEQ ID NO.: 159
ZN571_HUMAN	SEQ ID NO.: 160
ZN699_HUMAN	SEQ ID NO.: 161
ZN726_HUMAN	SEQ ID NO.: 162
ZIK1_HUMAN	SEQ ID NO.: 163
ZNF2_HUMAN	SEQ ID NO.: 164
Z705F_HUMAN	SEQ ID NO.: 165
ZNF14_HUMAN	SEQ ID NO.: 166
ZN471_HUMAN	SEQ ID NO.: 167
ZN624_HUMAN	SEQ ID NO.: 168
ZNF84_HUMAN	SEQ ID NO.: 169
ZNF7_HUMAN	SEQ ID NO.: 170
ZN891_HUMAN	SEQ ID NO.: 171
ZN337_HUMAN	SEQ ID NO.: 172
Z705G_HUMAN	SEQ ID NO.: 173

Gene name	Extended Domain sequence
ZN529_HUMAN	SEQ ID NO.: 174
ZN729_HUMAN	SEQ ID NO.: 175
ZN419_HUMAN	SEQ ID NO.: 176
Z705A_HUMAN	SEQ ID NO.: 177
ZNF45_HUMAN	SEQ ID NO.: 178
ZN302_HUMAN	SEQ ID NO.: 179
ZN486_HUMAN	SEQ ID NO.: 180
ZN621_HUMAN	SEQ ID NO.: 181
ZN688_HUMAN	SEQ ID NO.: 182
ZN33A_HUMAN	SEQ ID NO.: 183
ZN554_HUMAN	SEQ ID NO.: 184
ZN878_HUMAN	SEQ ID NO.: 185
ZN772_HUMAN	SEQ ID NO.: 186
ZN224_HUMAN	SEQ ID NO.: 187
ZN184_HUMAN	SEQ ID NO.: 188
ZN544_HUMAN	SEQ ID NO.: 189
ZNF57_HUMAN	SEQ ID NO.: 190
ZN283_HUMAN	SEQ ID NO.: 191
ZN549_HUMAN	SEQ ID NO.: 192
ZN211_HUMAN	SEQ ID NO.: 193
ZN615_HUMAN	SEQ ID NO.: 194
ZN253_HUMAN	SEQ ID NO.: 195
ZN226_HUMAN	SEQ ID NO.: 196
ZN730_HUMAN	SEQ ID NO.: 197
Z585A_HUMAN	SEQ ID NO.: 198
ZN732_HUMAN	SEQ ID NO.: 199
ZN681_HUMAN	SEQ ID NO.: 200
ZN667_HUMAN	SEQ ID NO.: 201
ZN649_HUMAN	SEQ ID NO.: 202
ZN470_HUMAN	SEQ ID NO.: 203
ZN484_HUMAN	SEQ ID NO.: 204
ZN431_HUMAN	SEQ ID NO.: 205
ZN382_HUMAN	SEQ ID NO.: 206
ZN254_HUMAN	SEQ ID NO.: 207
ZN124_HUMAN	SEQ ID NO.: 208
ZN607_HUMAN	SEQ ID NO.: 209

Gene name	Extended Domain sequence
ZN317_HUMAN	SEQ ID NO.: 210
ZN620_HUMAN	SEQ ID NO.: 211
ZN141_HUMAN	SEQ ID NO.: 212
ZN584_HUMAN	SEQ ID NO.: 213
ZN540_HUMAN	SEQ ID NO.: 214
ZN75D_HUMAN	SEQ ID NO.: 215
ZN555_HUMAN	SEQ ID NO.: 216
ZN658_HUMAN	SEQ ID NO.: 217
ZN684_HUMAN	SEQ ID NO.: 218
RBAK_HUMAN	SEQ ID NO.: 219
ZN829_HUMAN	SEQ ID NO.: 220
ZN582_HUMAN	SEQ ID NO.: 221
ZN112_HUMAN	SEQ ID NO.: 222
ZN716_HUMAN	SEQ ID NO.: 223
HKR1_HUMAN	SEQ ID NO.: 224
ZN350_HUMAN	SEQ ID NO.: 225
ZN480_HUMAN	SEQ ID NO.: 226
ZN416_HUMAN	SEQ ID NO.: 227
ZNF92_HUMAN	SEQ ID NO.: 228
ZN100_HUMAN	SEQ ID NO.: 229
ZN736_HUMAN	SEQ ID NO.: 230
ZNF74_HUMAN	SEQ ID NO.: 231
CBX1_HUMAN	SEQ ID NO.: 232
ZN443_HUMAN	SEQ ID NO.: 233
ZN195_HUMAN	SEQ ID NO.: 234
ZN530_HUMAN	SEQ ID NO.: 235
ZN782_HUMAN	SEQ ID NO.: 236
ZN791_HUMAN	SEQ ID NO.: 237
ZN331_HUMAN	SEQ ID NO.: 238
Z354C_HUMAN	SEQ ID NO.: 239
ZN157_HUMAN	SEQ ID NO.: 240
ZN727_HUMAN	SEQ ID NO.: 241
ZN550_HUMAN	SEQ ID NO.: 242
ZN793_HUMAN	SEQ ID NO.: 243
ZN235_HUMAN	SEQ ID NO.: 244
ZNF8_HUMAN	SEQ ID NO.: 245

Gene name	Extended Domain sequence
ZN724_HUMAN	SEQ ID NO.: 246
ZN573_HUMAN	SEQ ID NO.: 247
ZN577_HUMAN	SEQ ID NO.: 248
ZN789_HUMAN	SEQ ID NO.: 249
ZN718_HUMAN	SEQ ID NO.: 250
ZN300_HUMAN	SEQ ID NO.: 251
ZN383_HUMAN	SEQ ID NO.: 252
ZN429_HUMAN	SEQ ID NO.: 253
ZN677_HUMAN	SEQ ID NO.: 254
ZN850_HUMAN	SEQ ID NO.: 255
ZN454_HUMAN	SEQ ID NO.: 256
ZN257_HUMAN	SEQ ID NO.: 257
ZN264_HUMAN	SEQ ID NO.: 258
ZFP82_HUMAN	SEQ ID NO.: 259
ZFP14_HUMAN	SEQ ID NO.: 260
ZN485_HUMAN	SEQ ID NO.: 261
ZN737_HUMAN	SEQ ID NO.: 262
ZNF44_HUMAN	SEQ ID NO.: 263
ZN596_HUMAN	SEQ ID NO.: 264
ZN565_HUMAN	SEQ ID NO.: 265
ZN543_HUMAN	SEQ ID NO.: 266
ZFP69_HUMAN	SEQ ID NO.: 267
SUMO1_HUMAN	SEQ ID NO.: 268
ZNF12_HUMAN	SEQ ID NO.: 269
ZN169_HUMAN	SEQ ID NO.: 270
ZN433_HUMAN	SEQ ID NO.: 271
SUMO3_HUMAN	SEQ ID NO.: 272
ZNF98_HUMAN	SEQ ID NO.: 273
ZN175_HUMAN	SEQ ID NO.: 274
ZN347_HUMAN	SEQ ID NO.: 275
ZNF25_HUMAN	SEQ ID NO.: 276
ZN519_HUMAN	SEQ ID NO.: 277
Z585B_HUMAN	SEQ ID NO.: 278
ZIM3_HUMAN	SEQ ID NO.: 279
ZN517_HUMAN	SEQ ID NO.: 280
ZN846_HUMAN	SEQ ID NO.: 281

Gene name	Extended Domain sequence
ZN230_HUMAN	SEQ ID NO.: 282
ZNF66_HUMAN	SEQ ID NO.: 283
ZFP1_HUMAN	SEQ ID NO.: 284
ZN713_HUMAN	SEQ ID NO.: 285
ZN816_HUMAN	SEQ ID NO.: 286
ZN426_HUMAN	SEQ ID NO.: 287
ZN674_HUMAN	SEQ ID NO.: 288
ZN627_HUMAN	SEQ ID NO.: 289
ZNF20_HUMAN	SEQ ID NO.: 290
Z587B_HUMAN	SEQ ID NO.: 291
ZN316_HUMAN	SEQ ID NO.: 292
ZN233_HUMAN	SEQ ID NO.: 293
ZN611_HUMAN	SEQ ID NO.: 294
ZN556_HUMAN	SEQ ID NO.: 295
ZN234_HUMAN	SEQ ID NO.: 296
ZN560_HUMAN	SEQ ID NO.: 297
ZNF77_HUMAN	SEQ ID NO.: 298
ZN682_HUMAN	SEQ ID NO.: 299
ZN614_HUMAN	SEQ ID NO.: 300
ZN785_HUMAN	SEQ ID NO.: 301
ZN445_HUMAN	SEQ ID NO.: 302
ZFP30_HUMAN	SEQ ID NO.: 303
ZN225_HUMAN	SEQ ID NO.: 304
ZN551_HUMAN	SEQ ID NO.: 305
ZN610_HUMAN	SEQ ID NO.: 306
ZN528_HUMAN	SEQ ID NO.: 307
ZN284_HUMAN	SEQ ID NO.: 308
ZN418_HUMAN	SEQ ID NO.: 309
MPP8_HUMAN	SEQ ID NO.: 310
ZN490_HUMAN	SEQ ID NO.: 311
ZN805_HUMAN	SEQ ID NO.: 312
Z780B_HUMAN	SEQ ID NO.: 313
ZN763_HUMAN	SEQ ID NO.: 314
ZN285_HUMAN	SEQ ID NO.: 315
ZNF85_HUMAN	SEQ ID NO.: 316
ZN223_HUMAN	SEQ ID NO.: 317

Gene name	Extended Domain sequence
ZNF90_HUMAN	SEQ ID NO.: 318
ZN557_HUMAN	SEQ ID NO.: 319
ZN425_HUMAN	SEQ ID NO.: 320
ZN229_HUMAN	SEQ ID NO.: 321
ZN606_HUMAN	SEQ ID NO.: 322
ZN155_HUMAN	SEQ ID NO.: 323
ZN222_HUMAN	SEQ ID NO.: 324
ZN442_HUMAN	SEQ ID NO.: 325
ZNF91_HUMAN	SEQ ID NO.: 326
ZN135_HUMAN	SEQ ID NO.: 327
ZN778_HUMAN	SEQ ID NO.: 328
RYBP_HUMAN	SEQ ID NO.: 329
ZN534_HUMAN	SEQ ID NO.: 330
ZN586_HUMAN	SEQ ID NO.: 331
ZN567_HUMAN	SEQ ID NO.: 332
ZN440_HUMAN	SEQ ID NO.: 333
ZN583_HUMAN	SEQ ID NO.: 334
ZN441_HUMAN	SEQ ID NO.: 335
ZNF43_HUMAN	SEQ ID NO.: 336
CBX5_HUMAN	SEQ ID NO.: 337
ZN589_HUMAN	SEQ ID NO.: 338
ZNF10_HUMAN	SEQ ID NO.: 339
ZN563_HUMAN	SEQ ID NO.: 340
ZN561_HUMAN	SEQ ID NO.: 341
ZN136_HUMAN	SEQ ID NO.: 342
ZN630_HUMAN	SEQ ID NO.: 343
ZN527_HUMAN	SEQ ID NO.: 344
ZN333_HUMAN	SEQ ID NO.: 345
Z324B_HUMAN	SEQ ID NO.: 346
ZN786_HUMAN	SEQ ID NO.: 347
ZN709_HUMAN	SEQ ID NO.: 348
ZN792_HUMAN	SEQ ID NO.: 349
ZN599_HUMAN	SEQ ID NO.: 350
ZN613_HUMAN	SEQ ID NO.: 351
ZF69B_HUMAN	SEQ ID NO.: 352
ZN799_HUMAN	SEQ ID NO.: 353

Gene name	Extended Domain sequence
ZN569_HUMAN	SEQ ID NO.: 354
ZN564_HUMAN	SEQ ID NO.: 355
ZN546_HUMAN	SEQ ID NO.: 356
ZFP92_HUMAN	SEQ ID NO.: 357
YAF2_HUMAN	SEQ ID NO.: 358
ZN723_HUMAN	SEQ ID NO.: 359
ZNF34_HUMAN	SEQ ID NO.: 360
ZN439_HUMAN	SEQ ID NO.: 361
ZFP57_HUMAN	SEQ ID NO.: 362
ZNF19_HUMAN	SEQ ID NO.: 363
ZN404_HUMAN	SEQ ID NO.: 364
ZN274_HUMAN	SEQ ID NO.: 365
CBX3_HUMAN	SEQ ID NO.: 366
ZNF30_HUMAN	SEQ ID NO.: 367
ZN250_HUMAN	SEQ ID NO.: 368
ZN570_HUMAN	SEQ ID NO.: 369
ZN675_HUMAN	SEQ ID NO.: 370
ZN695_HUMAN	SEQ ID NO.: 371
ZN548_HUMAN	SEQ ID NO.: 372
ZN132_HUMAN	SEQ ID NO.: 373
ZN738_HUMAN	SEQ ID NO.: 374
ZN420_HUMAN	SEQ ID NO.: 375
ZN626_HUMAN	SEQ ID NO.: 376
ZN559_HUMAN	SEQ ID NO.: 377
ZN460_HUMAN	SEQ ID NO.: 378
ZN268_HUMAN	SEQ ID NO.: 379
ZN304_HUMAN	SEQ ID NO.: 380
ZIM2_HUMAN	SEQ ID NO.: 381
ZN605_HUMAN	SEQ ID NO.: 382
ZN844_HUMAN	SEQ ID NO.: 383
SUMO5_HUMAN	SEQ ID NO.: 384
ZN101_HUMAN	SEQ ID NO.: 385
ZN783_HUMAN	SEQ ID NO.: 386
ZN417_HUMAN	SEQ ID NO.: 387
ZN182_HUMAN	SEQ ID NO.: 388
ZN823_HUMAN	SEQ ID NO.: 389

Gene name	Extended Domain sequence
ZN177_HUMAN	SEQ ID NO.: 390
ZN197_HUMAN	SEQ ID NO.: 391
ZN717_HUMAN	SEQ ID NO.: 392
ZN669_HUMAN	SEQ ID NO.: 393
ZN256_HUMAN	SEQ ID NO.: 394
ZN251_HUMAN	SEQ ID NO.: 395
CBX4_HUMAN	SEQ ID NO.: 396
PCGF2_HUMAN	SEQ ID NO.: 397
CDY2_HUMAN	SEQ ID NO.: 398
CDYL2_HUMAN	SEQ ID NO.: 399
HERC2_HUMAN	SEQ ID NO.: 400
ZN562_HUMAN	SEQ ID NO.: 401
ZN461_HUMAN	SEQ ID NO.: 402
Z324A_HUMAN	SEQ ID NO.: 403
ZN766_HUMAN	SEQ ID NO.: 404
ID2_HUMAN	SEQ ID NO.: 405
TOX_HUMAN	SEQ ID NO.: 406
ZN274_HUMAN	SEQ ID NO.: 407
SCMH1_HUMAN	SEQ ID NO.: 408
ZN214_HUMAN	SEQ ID NO.: 409
CBX7_HUMAN	SEQ ID NO.: 410
ID1_HUMAN	SEQ ID NO.: 411
CREM_HUMAN	SEQ ID NO.: 412
SCX_HUMAN	SEQ ID NO.: 413
ASCL1_HUMAN	SEQ ID NO.: 414
ZN764_HUMAN	SEQ ID NO.: 415
SCML2_HUMAN	SEQ ID NO.: 416
TWST1_HUMAN	SEQ ID NO.: 417
CREB1_HUMAN	SEQ ID NO.: 418
TERF1_HUMAN	SEQ ID NO.: 419
ID3_HUMAN	SEQ ID NO.: 420
CBX8_HUMAN	SEQ ID NO.: 421
CBX4_HUMAN	SEQ ID NO.: 422
GSX1_HUMAN	SEQ ID NO.: 423
NKX22_HUMAN	SEQ ID NO.: 424
ATF1_HUMAN	SEQ ID NO.: 425

Gene name	Extended Domain sequence
TWST2_HUMAN	SEQ ID NO.: 426
ZNF17_HUMAN	SEQ ID NO.: 427
TOX3_HUMAN	SEQ ID NO.: 428
TOX4_HUMAN	SEQ ID NO.: 429
ZMYM3_HUMAN	SEQ ID NO.: 430
I2BP1_HUMAN	SEQ ID NO.: 431
RHXF1_HUMAN	SEQ ID NO.: 432
SSX2_HUMAN	SEQ ID NO.: 433
I2BPL_HUMAN	SEQ ID NO.: 434
ZN680_HUMAN	SEQ ID NO.: 435
CBX1_HUMAN	SEQ ID NO.: 436
TRI68_HUMAN	SEQ ID NO.: 437
HXA13_HUMAN	SEQ ID NO.: 438
PHC3_HUMAN	SEQ ID NO.: 439
TCF24_HUMAN	SEQ ID NO.: 440
CBX3_HUMAN	SEQ ID NO.: 441
HXB13_HUMAN	SEQ ID NO.: 442
HEY1_HUMAN	SEQ ID NO.: 443
PHC2_HUMAN	SEQ ID NO.: 444
ZNF81_HUMAN	SEQ ID NO.: 445
FIGLA_HUMAN	SEQ ID NO.: 446
SAM11_HUMAN	SEQ ID NO.: 447
KMT2B_HUMAN	SEQ ID NO.: 448
HEY2_HUMAN	SEQ ID NO.: 449
JDP2_HUMAN	SEQ ID NO.: 450
HXC13_HUMAN	SEQ ID NO.: 451
ASCL4_HUMAN	SEQ ID NO.: 452
HHEX_HUMAN	SEQ ID NO.: 453
HERC2_HUMAN	SEQ ID NO.: 454
GSX2_HUMAN	SEQ ID NO.: 455
BIN1_HUMAN	SEQ ID NO.: 456
ETV7_HUMAN	SEQ ID NO.: 457
ASCL3_HUMAN	SEQ ID NO.: 458
PHC1_HUMAN	SEQ ID NO.: 459
OTP_HUMAN	SEQ ID NO.: 460
I2BP2_HUMAN	SEQ ID NO.: 461

Gene name	Extended Domain sequence
VGLL2_HUMAN	SEQ ID NO.: 462
HXA11_HUMAN	SEQ ID NO.: 463
PDLI4_HUMAN	SEQ ID NO.: 464
ASCL2_HUMAN	SEQ ID NO.: 465
CDX4_HUMAN	SEQ ID NO.: 466
ZN860_HUMAN	SEQ ID NO.: 467
LMBL4_HUMAN	SEQ ID NO.: 468
PDIP3_HUMAN	SEQ ID NO.: 469
NKX25_HUMAN	SEQ ID NO.: 470
CEBPB_HUMAN	SEQ ID NO.: 471
ISL1_HUMAN	SEQ ID NO.: 472
CDX2_HUMAN	SEQ ID NO.: 473
PROP1_HUMAN	SEQ ID NO.: 474
SIN3B_HUMAN	SEQ ID NO.: 475
SMBT1_HUMAN	SEQ ID NO.: 476
HXC11_HUMAN	SEQ ID NO.: 477
HXC10_HUMAN	SEQ ID NO.: 478
PRS6A_HUMAN	SEQ ID NO.: 479
VSX1_HUMAN	SEQ ID NO.: 480
NKX23_HUMAN	SEQ ID NO.: 481
MTG16_HUMAN	SEQ ID NO.: 482
HMX3_HUMAN	SEQ ID NO.: 483
HMX1_HUMAN	SEQ ID NO.: 484
KIF22_HUMAN	SEQ ID NO.: 485
CSTF2_HUMAN	SEQ ID NO.: 486
CEBPE_HUMAN	SEQ ID NO.: 487
DLX2_HUMAN	SEQ ID NO.: 488
ZMYM3_HUMAN	SEQ ID NO.: 489
PPARG_HUMAN	SEQ ID NO.: 490
PRIC1_HUMAN	SEQ ID NO.: 491
UNC4_HUMAN	SEQ ID NO.: 492
BARX2_HUMAN	SEQ ID NO.: 493
ALX3_HUMAN	SEQ ID NO.: 494
TCF15_HUMAN	SEQ ID NO.: 495
TERA_HUMAN	SEQ ID NO.: 496
VSX2_HUMAN	SEQ ID NO.: 497

Gene name	Extended Domain sequence
HXD12_HUMAN	SEQ ID NO.: 498
CDX1_HUMAN	SEQ ID NO.: 499
TCF23_HUMAN	SEQ ID NO.: 500
ALX1_HUMAN	SEQ ID NO.: 501
HXA10_HUMAN	SEQ ID NO.: 502
RX_HUMAN	SEQ ID NO.: 503
CXXC5_HUMAN	SEQ ID NO.: 504
SCML1_HUMAN	SEQ ID NO.: 505
NFIL3_HUMAN	SEQ ID NO.: 506
DLX6_HUMAN	SEQ ID NO.: 507
MTG8_HUMAN	SEQ ID NO.: 508
CBX8_HUMAN	SEQ ID NO.: 509
CEBPD_HUMAN	SEQ ID NO.: 510
SEC13_HUMAN	SEQ ID NO.: 511
FIP1_HUMAN	SEQ ID NO.: 512
ALX4_HUMAN	SEQ ID NO.: 513
LHX3_HUMAN	SEQ ID NO.: 514
PRIC2_HUMAN	SEQ ID NO.: 515
MAGI3_HUMAN	SEQ ID NO.: 516
NELL1_HUMAN	SEQ ID NO.: 517
PRRX1_HUMAN	SEQ ID NO.: 518
MTG8R_HUMAN	SEQ ID NO.: 519
RAX2_HUMAN	SEQ ID NO.: 520
DLX3_HUMAN	SEQ ID NO.: 521
DLX1_HUMAN	SEQ ID NO.: 522
NKX26_HUMAN	SEQ ID NO.: 523
NAB1_HUMAN	SEQ ID NO.: 524
SAMD7_HUMAN	SEQ ID NO.: 525
PITX3_HUMAN	SEQ ID NO.: 526
WDR5_HUMAN	SEQ ID NO.: 527
MEOX2_HUMAN	SEQ ID NO.: 528
NAB2_HUMAN	SEQ ID NO.: 529
DHX8_HUMAN	SEQ ID NO.: 530
FOXA2_HUMAN	SEQ ID NO.: 531
CBX6_HUMAN	SEQ ID NO.: 532
EMX2_HUMAN	SEQ ID NO.: 533

Gene name	Extended Domain sequence
CPSF6_HUMAN	SEQ ID NO.: 534
HXC12_HUMAN	SEQ ID NO.: 535
KDM4B_HUMAN	SEQ ID NO.: 536
LMBL3_HUMAN	SEQ ID NO.: 537
PHX2A_HUMAN	SEQ ID NO.: 538
EMX1_HUMAN	SEQ ID NO.: 539
NC2B_HUMAN	SEQ ID NO.: 540
DLX4_HUMAN	SEQ ID NO.: 541
SRY_HUMAN	SEQ ID NO.: 542
ZN777_HUMAN	SEQ ID NO.: 543
NELL1_HUMAN	SEQ ID NO.: 544
ZN398_HUMAN	SEQ ID NO.: 545
GATA3_HUMAN	SEQ ID NO.: 546
BSH_HUMAN	SEQ ID NO.: 547
SF3B4_HUMAN	SEQ ID NO.: 548
TEAD1_HUMAN	SEQ ID NO.: 549
TEAD3_HUMAN	SEQ ID NO.: 550
RGAP1_HUMAN	SEQ ID NO.: 551
PHF1_HUMAN	SEQ ID NO.: 552
FOXA1_HUMAN	SEQ ID NO.: 553
GATA2_HUMAN	SEQ ID NO.: 554
FOXO3_HUMAN	SEQ ID NO.: 555
ZN212_HUMAN	SEQ ID NO.: 556
IRX4_HUMAN	SEQ ID NO.: 557
ZBED6_HUMAN	SEQ ID NO.: 558
LHX4_HUMAN	SEQ ID NO.: 559
SIN3A_HUMAN	SEQ ID NO.: 560
RBBP7_HUMAN	SEQ ID NO.: 561
NKX61_HUMAN	SEQ ID NO.: 562
TRI68_HUMAN	SEQ ID NO.: 563
R51A1_HUMAN	SEQ ID NO.: 564
MB3L1_HUMAN	SEQ ID NO.: 565
DLX5_HUMAN	SEQ ID NO.: 566
NOTC1_HUMAN	SEQ ID NO.: 567
TERF2_HUMAN	SEQ ID NO.: 568
ZN282_HUMAN	SEQ ID NO.: 569

Gene name	Extended Domain sequence
RGS12_HUMAN	SEQ ID NO.: 570
ZN840_HUMAN	SEQ ID NO.: 571
SPI2B_HUMAN	SEQ ID NO.: 572
PAX7_HUMAN	SEQ ID NO.: 573
NKX62_HUMAN	SEQ ID NO.: 574
ASXL2_HUMAN	SEQ ID NO.: 575
FOXO1_HUMAN	SEQ ID NO.: 576
GATA3_HUMAN	SEQ ID NO.: 577
GATA1_HUMAN	SEQ ID NO.: 578
ZMYM5_HUMAN	SEQ ID NO.: 579
ZN783_HUMAN	SEQ ID NO.: 580
SPI2B_HUMAN	SEQ ID NO.: 581
LRP1_HUMAN	SEQ ID NO.: 582
MIXL1_HUMAN	SEQ ID NO.: 583
SGT1_HUMAN	SEQ ID NO.: 584
LMCD1_HUMAN	SEQ ID NO.: 585
CEBPA_HUMAN	SEQ ID NO.: 586
GATA2_HUMAN	SEQ ID NO.: 587
SOX14_HUMAN	SEQ ID NO.: 588
WTIP_HUMAN	SEQ ID NO.: 589
PRP19_HUMAN	SEQ ID NO.: 590
CBX6_HUMAN	SEQ ID NO.: 591
NKX11_HUMAN	SEQ ID NO.: 592
RBBP4_HUMAN	SEQ ID NO.: 593
DMRT2_HUMAN	SEQ ID NO.: 594
SMCA2_HUMAN	SEQ ID NO.: 595

[0341] In some embodiments, an effector domain comprises a functional domain that represses or silences gene expression, and the functional domain is a part of a larger protein, e.g., a zinc finger repressor protein. Functional domains that are capable of modulating gene expression, e.g., repress or increase gene expression can be identified from the larger protein with known methods and methods provided herein. For example, functional effector domains that can reduce or silence target gene expression may be identified based on sequences of repressor or activator proteins. Amino acid sequences of proteins having the function of modulating gene expression may be obtained from available genome browsers, such as UCSD genome browser or Ensembl genome browser. For example, a full length 573 amino acid sequence of the ZNF10 protein is provided in SEQ ID NO.: 596.

[0342] Protein annotation databases such as UniProt or Pfam can be used to identify functional domains within the full protein sequence. Using these tools, the repression domain can be identified within the ZNF10 protein sequence. In some instances, various functional domains identified from a larger protein may be tested. Databases may differ in the specific boundary domains. For example, in some embodiments, a repression domain derived from ZNF10 includes amino acids 14-85 of the above referenced ZNF10 sequence. In some embodiments, a repression domain derived from ZNF10 consists of amino acids 14-85 of the above referenced ZNF10 sequence. In some embodiments, a repression domain derived from ZNF10 includes amino acids 13-54 of the above referenced ZNF10 sequence. In some embodiments, a repression domain derived from ZNF10 consists of amino acids 13-54 of the above referenced ZNF10 sequence. As a starting point, the largest sequence, encompassing all regions identified by different databases, may be tested for gene expression modulation activity, for example, a region of the ZN10 protein comprising amino acids 13-85 is tested as a starting point. In further embodiments, the starting point region may be truncated by 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50 or more amino acids at the N-terminus or C-terminus and various truncations may be tested to identify the minimal functional unit.

[0343] In some embodiments, the effector domain comprises a histone deacetylase protein domain. In some embodiments, the effector domain comprises a HDAC family protein domain, for example, a HDAC1, HDAC3, HDAC5, HDAC7, or HDAC9 protein domain. In some embodiments, the effector domain removes the acetyl group. In some embodiments, the effector domain comprises a nucleosome remodeling domain. In some embodiments, the effector domain comprises a nucleosome remodeling and deacetylase complex (NURD), which removes acetyl groups from histones.

[0344] In some embodiments, the effector domain comprises a Tripartite motif containing 28 (TRIM28, TIF1-beta, or KAP1) protein. In some embodiments, the effector domain comprises one or more KAP1 protein. The KAP1 protein in an epigenetic editor may form a complex with one or more other effector domains of the epigenetic editor or one or more proteins involved in modulation of gene expression in a cellular environment. For example, KAP1 may be recruited by a KRAB domain of a transcriptional repressor. In some embodiments, KAP1 interacts with or recruits a histone deacetylase protein, a histone-lysine methyltransferase protein (e.g. depositing methyl groups on lysine 9 [K9] of a histone H3 tail [H3K9]), a chromatin remodeling protein, and/or a heterochromatin protein. In some embodiments, a KAP1 protein interacts with or recruits one or more protein complexes that reduces or silences gene expression. In some

embodiments, a KAP1 protein interacts with or recruits a heterochromatin protein 1 (HP1) protein (e.g. via a chromoshadow domain of the HP1 protein) , a SETDB1 protein, a HDAC protein, and/or a NuRD protein complex component. In some embodiments, a KAP1 protein recruits a CHD3 subunit of the nucleosome remodeling and deacetylation (NuRD) complex, thereby decreasing or silencing expression of a target gene. In some embodiments, a KAP1 protein recruits a SETDB1 protein (e.g. to a promoter region of a target gene), thereby decreasing or silencing expression of the target gene via H3K9 methylation associated with, e.g. the promoter region of the target gene. In some embodiments, recruitment of the SETDB1 protein results in heterochromatinization of a chromosome region harboring the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, a KAP1 protein interacts with or recruits a HP1 protein, thereby decreasing or silencing expression of a target gene via reduced acetylation of H3K9 or H3K14 on histone tails associated with the target gene. Recruitment of SETDB1 induces heterochromatinization. In some embodiments, a KAP1 protein interacts with or recruits a ZFP90 protein (e.g. isoform 2 of ZFP90), and/or a FOXP3 protein.

[0345] Amino acid sequence of an exemplary KAP1 protein is provided in SEQ ID NO.: 597.

[0346] In some embodiments, the effector domain comprises a protein domain that interacts with or is recruited by one or more DNA epigenetic marks. For example, the effector domain may comprise a methyl CpG binding protein 2 (MECP2) protein that interacts with methylated DNA nucleotides in the target gene. In some embodiments, the MECP2 protein interacts with methylated DNA nucleotides in a CpG island of the target gene. In some embodiments, the MECP2 protein interacts with methylated DNA nucleotides not in a CpG island of the target gene. In some embodiments, the MECP2 protein in an epigenetic editor results in condensed chromatin structure, thereby reducing or silencing expression of the target gene. In some embodiments, the MECP2 protein in an epigenetic editor interacts with a histone deacetylase (e.g. HDAC), thereby repressing or silencing expression of the target gene. In some embodiments, the MECP2 protein in an epigenetic editor blocks access of a transcription factor or transcriptional activator to the target gene, thereby repressing or silencing expression of the target gene.

[0347] Amino acid sequence of an exemplary MECP2 protein is provided in SEQ ID NO.: 598.

[0348] In some embodiments, an effector domain comprises a chromoshadow domain, a ubiquitin-2 like Rad60 SUMO-like (Rad60-SLD/SUMO) domain, a chromatin organization modifier domain (Chromo) domain, a Yaf2/RYBP C-terminal binding motif domain (YAF2_RYBP), a CBX family C-terminal motif domain (CBX7_C), a Zinc finger C3HC4 type

(RING finger) domain (zf-C3HC4_2), a Cytochrome b5 domain (Cyt-b5), a helix-loop-helix domain (HLH), a high mobility group box domain (HMG-box), a Sterile alpha motif domain (SAM_1), basic leucine zipper domain (bZIP_1), a Myb_DNA-binding domain, a Homeodomain, a MYM-type Zinc finger with FCS sequence domain (zf-FCS), a interferon regulatory factor 2-binding protein zinc finger domain (IRF-2BP1_2), a SSX repression domain (SSXRD), a B-box-type zinc finger domain (zf-B_box), a sterile alpha motif domain (SAM_2), a CXXC zinc finger domain (zf-CXXC), a regulator of chromosome condensation 1 domain (RCC1), a SRC homology 3 domain (SH3_9), a sterile alpha motif/Pointed domain (SAM_PNT), a Vestigial/Tondu family domain (Vg_Tdu), a LIM domain, a RNA recognition motif domain (RRM_1), a basic leucine zipper domain (bZIP_2), a paired amphipathic helix domain (PAH), a proteasomal ATPase OB C-terminal domain (Prot_ATP_ID_OB), a nervy homology 2 domain (NHR2), a helix-hairpin-helix motif domain (HHH_3), a hinge domain of cleavage stimulation factor subunit 2 (CSTF2_hinge), a PPAR gamma N-terminal region domain (PPARgamma_N), a CDC48 N-terminal domain (CDC48_2), a WD40 repeat domain (WD40), a Fip1 motif domain (Fip1), a PDZ domain (PDZ_6), a Von Willebrand factor type C domain (VWC), a NAB conserved region 1 domain (NCD1), a S1 RNA-binding domain (S1), a HNF3 C-terminal domain (HNF_C), a Tudor domain (Tudor_2), a histone-like transcription factor (CBF/NF-Y) and archaeal histone domain (CBFD_NFYB_HMF), a Zinc finger protein domain (DUF3669), a EGF-like domain (cEGF), a GATA zinc finger domain (GATA), a TEA/ATTS domain (TEA), a phorbol esters/diacylglycerol binding domain (C1-1), polycomb-like MTF2 factor 2 domain (Mtf2_C), a transactivation domain of FOXO protein family (FOXO-TAD), a Homeobox KN domain (Homeobox_KN), a BED zinc finger domain (zf-BED), a zinc finger of C3HC4-type RING domain (zf-C3HC4_4), a RAD51 interacting motif domain (RAD51_interact), a p55-binding region of a Methyl-CpG-binding domain protein MBD (MBDa), Notch domain, a Raf-like Ras-binding domain (RBD), a Spin/Ssty family domain (Spin-Ssty), a PHD finger domain (PHD_3), a Low-density lipoprotein receptor domain class A (Ldl_recept_a), a CS domain, a DM DNA binding domain, or a QLQ domain. In some embodiments, the effector domain is a protein domain comprising a YAF2_RYBP domain, or homeodomain or any combination thereof. In some embodiments, the homeodomain of the YAF2_RYBP domain is a PRD domain, a NKL domain, a HOXL domain, or a LIM domain. In some embodiments, the effector domain comprises a protein domain selected from a group consisting of SUMO3 domain, Chromo domain from M phase phosphoprotein 8 (MPP8), chromoshadow domain from Chromobox 1 (CBX1), and SAM_1/SPM domain from Scm Polycomb Group Protein Homolog 1 (SCMH1). In some embodiments, the effector domain

comprises a HNF3 C-terminal domain (HNF_C). In some embodiments, the HNF_C domain is from FOXA1 or FOXA2. In some embodiments, the HNF_C domain comprises an EH1 (engrailed homology 1) motif. In some embodiments, the effector domain comprises an interferon regulatory factor 2-binding protein zinc finger domain (IRF-2BP1_2).In some embodiments, the effector domain comprises a Cyt-b5 domain from DNA repair factor HERC2 E3 ligase. In some embodiments, the effector domain comprises a variant SH3 domain (SH3_9) from Bridging Integrator 1 (BIN1). In some embodiments, the effector domain is HMG-box domain from transcription factor TOX or zf-C3HC4_2 RING finger domain from the polycomb component PCGF2. In some embodiment, the effector domain comprises a Chromodomain-helicase-DNA-binding protein 3 (CHD3). In some embodiments, the effector domain comprises a ZNF783 domain. In some embodiments, the effector domain comprises a YAF2_RYBP domain. In some embodiment, the YAF2_RYBP domain comprises a 32 amino acid Yaf2/RYBP C-terminal binding motif domain (32 AA RYBP).

[0349] In some embodiments, an effector domain makes an epigenetic modification at a target gene that activates expression of the target gene. In some embodiments, an effector domain modifies the chemical modification of DNA or histone residues associated with the DNA at a target gene harboring the target sequence, thereby activating or increasing expression of the target gene. In some embodiments, the effector domain comprises a DNA demethylase, a DNA dioxygenase, a DNA hydroxylase, or a histone demethylase domain.

[0350] In some embodiments, the effector domain comprises a DNA demethylase domain that removes a methyl group from DNA nucleotides, thereby increasing or activating expression of the target gene.

[0351] In some embodiments, the effector domain comprises a TET (ten-eleven translocation methylcytosine dioxygenase) family protein domain that demethylates cytosine in methylated form and thereby increases expression of a target gene. In some embodiments, the effector domain comprises a TET1, TET2, or TET3 protein domain or any combination thereof. In some embodiments, the effector domain comprises a TET1 domain. In some embodiments, the effector domain comprises a KDM family protein domain that demethylates lysines in DNA-associated histones, thereby increasing expression of the target gene.

[0352] Exemplary demethylase domains that may be part of an epigenetic effector domain are provided in Table 4 below.

Table 4. Exemplary demethylase sequences that may be used in epigenetic effector domains

Protein	**Species**	**Protein Sequence**
TET1	Human	SEQ ID NO.: 599

Protein	Species	Protein Sequence
TET2	Human	SEQ ID NO.: 600
TET3	Human	SEQ ID NO.: 601
TDG	Human	SEQ ID NO.: 602
ROS1	Arabidopsis	SEQ ID NO.: 603
DME	Arabidopsis	SEQ ID NO.: 604
DML2	Arabidopsis	SEQ ID NO.: 605
DML3	Arabidopsis	SEQ ID NO.: 606

[0353] The effector domain may activate expression of the target gene. In some embodiments, the effector domain comprises a protein domain that recruits one or more transcription activator domains. In some embodiments, the effector domain comprises a protein domain that recruits one or more transcription factors. In some embodiments, the effector domain comprises a transcription activator or a transcription factor domain. In some embodiments, the effector domain comprises a Herpes Simplex Virus Protein 16 (VP16) activation domain. In some embodiments, the effector domain comprises an activation domain comprising a tandem repeat of multiple VP16 activation domains. In some embodiments, the effector domain comprises four tandem copies of VP16, a VP64 activation domain. In some embodiments, the effector domain comprises a p65 activation domain of NFκB; an Epstein-Barr virus R transactivator (Rta) activation domain. In some embodiments, the effector domain comprises a fusion of multiple activators, e.g., a tripartite activator of the VP64, the p65, and the Rta activation domains, (a VPR activation domain).

[0354] In some embodiments, an effector domain comprises a transactivation domain of FOXO protein family (FOXO-TAD), a LMSTEN motif domain (LMSTEN), a Transducer of regulated CREB activity C terminus domain (TORC_C), a QLQ domain, a Nuclear receptor coactivator domain (Nuc_rec_co-act), an Autophagy receptor zinc finger-C2H2 domain (Zn-C2H2-12), an Anaphase-promoting complex subunit 16 (ANAPC16), a Dpy-30 domain, a ANC1 homology domain (AHD), a Signal transducer and activator of transcription 2 C terminal (STAT2_C), a I-kappa-kinase-beta NEMO binding domain (IKKbetaNEMObind), a Early growth response N-terminal domain (DUF3446), a TFIIE beta subunit core domain (TFIIE_beta), a N-terminal domain of DPF2/REQ (Requiem_N), a LNR domain (Notch), a Atypical Arm repeat (Arm_3), a Protein kinase C terminal domain (PKinase_C), WW domain, a SH3 domain (SH3_1), a Myb-like DNA-binding domain, a WD domain G-beta repeat (WD40), a PHD-finger (PHD), a RNA recognition motif domain (RRM_1), a GATA zinc finger domain (GATA), a Vps4 C terminal oligomerization domain (Vps4_C), or in any combination thereof. In some embodiments, the

effector domain comprises a KRAB domain that activates expression of a target gene. For example the KRAB domain may be a ZNF473 KRAB domain, a ZFP28 KRAB domain, a ZNF496 KRAB domain, or a ZNF597 KRAB domain or any combination thereof. In some embodiments, the KRAB domain comprises a 41-amino-acid ZNF473 KRAB domain (41 AA ZNF473). In some embodiments, the effector domain comprises a FOXO-TAD domain, a LMSTEN domain, or a TORC_C domain. In some embodiment, the protein domain comprises a RNA polymerase II transcription mediator complex subunit 9 (Med9), TFIIE beta subunit core domain (TFIIEβ), nuclear receptor coactivator 3 domain (NCOA3), transactivation domain of FOXO protein family (FOXO-TAD), LMSTEN motif domain, early growth response N-terminal domain (DUF3446), QLQ domain, or Dpy-30 motif domain or any combination thereof. In some embodiment, the effector domain comprises a ZNF473 KRAB domain or a Med9 domain.

[0355] Exemplary domains that can activate or increase target gene expression are provided in Table 5 below.

Table 5. Exemplary protein domains that may be used in epigenetic effector domains to increase target gene expression

Protein	**Species**	**Protein Sequence**
VP16	Herpes simplex virus type 1 (strain 17)	SEQ ID NO.: 607
VP64	Herpes simplex virus type 1	SEQ ID NO.: 608
VP160	Herpes simplex virus type 1	SEQ ID NO.: 609
HIF1alpha	Human	SEQ ID NO.: 610
CITED2	Human	SEQ ID NO.: 611
Stat3	Human	SEQ ID NO.: 612
p65	Human	SEQ ID NO.: 613
p53	Human	SEQ ID NO.: 614
ZNF473	Human	SEQ ID NO.: 615
FOXO1	Human	SEQ ID NO.: 616
Myb	Human	SEQ ID NO.: 617
CRTC1	Human	SEQ ID NO.: 618
Med9	Human	SEQ ID NO.: 619
EGR3	Human	SEQ ID NO.: 620
SMARCA2	Human	SEQ ID NO.: 621
Dpy-30	Human	SEQ ID NO.: 622
NCOA3	Human	SEQ ID NO.: 623
ZFP28	Human	SEQ ID NO.: 624
ZNF496	Human	SEQ ID NO.: 625

Protein	Species	Protein Sequence
ZNF597	Human	SEQ ID NO.: 626
HSF1	Human	SEQ ID NO.: 627
RTA	Epstein-barr virus (strain B95-8)	SEQ ID NO.: 628

[0356] Additional exemplary domains that can activate or increase target gene expression are provided in Table 6 below.

Table 6. Exemplary protein domains that may be used in epigenetic effector domains to increase target gene expression

Gene name	Extended Domain sequence
ABL1_HUMAN	SEQ ID NO.: 629
AF9_HUMAN	SEQ ID NO.: 630
ANM2_HUMAN	SEQ ID NO.: 631
APBB1_HUMAN	SEQ ID NO.: 632
APC16_HUMAN	SEQ ID NO.: 633
BTK_HUMAN	SEQ ID NO.: 634
CACO1_HUMAN	SEQ ID NO.: 635
CRTC2_HUMAN	SEQ ID NO.: 636
CRTC3_HUMAN	SEQ ID NO.: 637
CXXC1_HUMAN	SEQ ID NO.: 638
DPF1_HUMAN	SEQ ID NO.: 639
DPY30_HUMAN	SEQ ID NO.: 640
EGR3_HUMAN	SEQ ID NO.: 641
ENL_HUMAN	SEQ ID NO.: 642
FIGN_HUMAN	SEQ ID NO.: 643
FOXO1_HUMAN	SEQ ID NO.: 644
FOXO3_HUMAN	SEQ ID NO.: 645
IKKA_HUMAN	SEQ ID NO.: 646
IMA5_HUMAN	SEQ ID NO.: 647
ITCH_HUMAN	SEQ ID NO.: 648
KIBRA_HUMAN	SEQ ID NO.: 649
KPCI_HUMAN	SEQ ID NO.: 650
KS6B2_HUMAN	SEQ ID NO.: 651
MTA3_HUMAN	SEQ ID NO.: 652
MYB_HUMAN	SEQ ID NO.: 653
MYBA_HUMAN	SEQ ID NO.: 654
NCOA2_HUMAN	SEQ ID NO.: 655

NCOA3_HUMAN	SEQ ID NO.: 656
NOTC1_HUMAN	SEQ ID NO.: 657
NOTC1_HUMAN	SEQ ID NO.: 658
NOTC2_HUMAN	SEQ ID NO.: 659
PRP19_HUMAN	SEQ ID NO.: 660
PYGO1_HUMAN	SEQ ID NO.: 661
PYGO2_HUMAN	SEQ ID NO.: 662
SAV1_HUMAN	SEQ ID NO.: 663
SMCA2_HUMAN	SEQ ID NO.: 664
SMRC2_HUMAN	SEQ ID NO.: 665
STAT2_HUMAN	SEQ ID NO.: 666
T2EB_HUMAN	SEQ ID NO.: 667
U2AF4_HUMAN	SEQ ID NO.: 668
WBP4_HUMAN	SEQ ID NO.: 669
WWP1_HUMAN	SEQ ID NO.: 670
WWP2_HUMAN	SEQ ID NO.: 671
WWTR1_HUMAN	SEQ ID NO.: 672
ZFP28_HUMAN	SEQ ID NO.: 673
ZN473_HUMAN	SEQ ID NO.: 674
ZN496_HUMAN	SEQ ID NO.: 675
ZN597_HUMAN	SEQ ID NO.: 676

[0357] In some embodiments, an effector domain regulates acetylation of a histone associated with the target gene. In some embodiments, the effector domain comprises a histone acetyltransferase domain. In some embodiments, the effector domain comprises a protein domain that interacts with a histone acetyltransferase domain to effect histone acetylation. In some embodiments, the effector domain comprises a histone acetyltransferase 1 (HAT1) domain. In some embodiments, the effector domain comprises a histone acetyltransferase (HAT) core domain of the human E1A-associated protein p300. In some embodiments, the effector domain comprises a CBP/p300 histone acetyltransferase or a catalytic domain thereof. In some embodiments, the effector domain comprises a CREBBP, GCN4, GCN5, SAGA, SALSA, HAP2, HAP3, HAP4, PCAF, KMT2A, or any combination thereof.

[0358] Sequences of exemplary histone acetyltransferase domains are provided below:

Exemplary p300 amino acid sequence: SEQ ID NO.: 677.

[0359] Exemplary CREBBP amino acid sequence: SEQ ID NO.: 678.

[0360] In some embodiments, an epigenetic editor described herein alters chemical modification of a target gene that harbors the target sequence. For example, an epigenetic editor comprising a methyltransferase domain can methylate the DNA or histone residues of the target gene, at nucleotides (or histones) near the target sequence, or within 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000, 1500, 2000, 2500, 3000 base pairs flanking the target sequence, thereby repress or silent expression of the target gene. An epigenetic editor comprising a DNA or histone demethylase can remove the methylation of the DNA or histone residues associated with or bound to the target gene, thereby activating or increasing expression of the target gene.

[0361] Chemical modifications mediated by an epigenetic editor may be near a target sequence of a target gene. For example, such modifications may occur within 50, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 base pairs flanking the target sequence. In some embodiments, the chemical modification occurs within 50, 100, 200, 300, 400, 500, 600, 700, 800, 900, 1000 base pairs upstream of the 5' end of the target sequence.

Epigenetic Editors

[0362] Described herein are epigenetic editors for epigenetic modification and expression regulation of target genes. As used herein, an epigenetic editor can be any agent that binds a target polynucleotide and has epigenetic modulation activity. In some embodiments, the epigenetic editor binds the polynucleotide at a specific sequence using a DNA binding domain. In some embodiments, the epigenetic editor binds the polynucleotide at a specific sequence using a nucleic acid guided DNA binding protein. In some embodiments, the epigenetic editor comprises an effector domain capable of modulating epigenetic state of a nucleic acid sequence at or adjacent to the target polynucleotide. In some embodiments, the epigenetic editor is capable of depositing an epigenetic editing mark on a chromatin region, a nucleic acid sequence, or a histone amino acid residue, at or adjacent to the target polynucleotide. For example, the epigenetic editor can be capable of methylating, demethylating, acetylating, deacetylating, ubiquitinating or deubiquitinating a chromatin region, a nucleic acid sequence, or a histone amino acid residue, at or adjacent to the target polynucleotide. In some embodiments, the epigenetic editor is capable of recruiting one or more proteins or complexes involved in transcription regulation, for example, a transcription factor, a transcription activator, a transcription repressor, or an insulator to a chromatin region, a nucleic acid sequence, or a histone amino acid residue, at or adjacent to the target polynucleotide.

[0363] Epigenetic editors provided herein can comprise one or more effector domains as described. In some embodiments, an epigenetic editor comprises multiple effector domains. In some embodiments, an epigenetic editor comprises one effector domain. In some embodiments,

the epigenetic editor comprises at least 2, 3, 4, 5, 6, 7, 8, 9, 10 or more effector domains. In some embodiments, the epigenetic editor comprises at least 2 effector domains, e.g., two repressor domains. In some embodiments, the epigenetic editor comprises at least 2 effector domains. In some embodiments, the epigenetic editor comprises two or more effector domains. In some embodiments, the two or more effector domains function synergistically to result in enhanced modulation of a target gene. For example, an epigenetic editor may comprise two effector domains, one of which induces histone deacetylation and the other results in DNA methylation of the target gene.

[0364] In some embodiments, an epigenetic editor comprises a DNA methylation domain and a histone deacetylation domain. In some embodiments, an epigenetic editor comprises a DNA methylation domain and a repression domain that recruits additional DNA methylation, histone methylation, or histone deacetylation proteins. In some embodiments, an epigenetic editor comprises a DNA methylation domain and a scaffold protein that recruits additional DNA methylation, histone methylation, or histone deacetylation proteins. In some embodiments, an epigenetic editor comprises a DNA methylation domain, a histone deacetylation domain, and a scaffold protein that recruits additional DNA methylation, histone methylation, or histone deacetylation proteins. In some embodiments, an epigenetic editor comprises two or more DNA methylation domains, a histone deacetylation domain, and a scaffold protein that recruits additional DNA methylation, histone methylation, or histone deacetylation proteins. In some embodiments, an epigenetic editor comprises two or more DNA methylation domains, two or more histone deacetylation domains, and/or two or more scaffold proteins that recruits additional DNA methylation, histone methylation, or histone deacetylation proteins. In some embodiments, the epigenetic editor comprises a KRAB domain and a DNMT3 domain, both of which may synergistically effect enhanced reduction or silencing of expression of a target gene, as compared to an epigenetic effector having only one of the two repressor domains. In some embodiments, the epigenetic editor comprises a KRAB domain, a Dnmt3A domain, and a Dnmt3L domain. In some embodiments, the epigenetic editor comprises the configuration of a DNA binding domain flanked by a KRAB domain and a Dnmt3A-Dnmt3L fusion protein domain. In some embodiments, the epigenetic editor comprises the following configuration: N-[KRAB]-[DNA binding domain]-[Dnmt3A-Dnmt3L]-C, where "]-[" is any nuclear localization signal, any tag sequence, or any linker as provided herein.

[0365] In some embodiments, an epigenetic editor comprises a DNA demethylation domain and a histone acetylation domain. In some embodiments, an epigenetic editor comprises a DNA demethylation domain and an activation domain that recruits additional DNA demethylation or

histone acetylation proteins. In some embodiments, an epigenetic editor comprises a DNA demethylation domain, a histone acetylation domain, and a scaffold protein that recruits additional DNA demethylation or histone acetylation proteins. In some embodiments, an epigenetic editor comprises two or more DNA demethylation domains, two or more histone acetylation domains, and/or two or more scaffold proteins that recruits additional DNA demethylation or histone deacetylation proteins.

[0366] In some embodiments, an epigenetic editor may comprise a VP64 activation domain, a p65 activation domain, and a Rta activation domains (together, a VPR activation domain), all of which synergistically effect enhanced activation of expression of a target gene, as compared to an epigenetic effector having only one of the three activation domains.

[0367] An effector domain of an epigenetic editor can be linked to another effector domain via direct fusion, or via any linker as described herein. An effector domain and a DNA binding domain of the epigenetic editor can also be linked via direct fusion or any linker as described herein.

[0368] In some embodiments, the two or more effector domains are identical. In some embodiments, the two or more effector domains belong to the same protein family. In some embodiments, the two or more effector domains are different proteins involved in the same transcriptional machinery or regulatory mechanism.

[0369] Multiple epigenetic editors, e.g. epigenetic editor fusion proteins or complexes may be used to effect activation or repression of a target gene or multiple target genes. For example, an epigenetic editor fusion protein comprising a DNA binding domain (e.g. dCas9 domain) and a methylation domain may be co-delivered with two or more guide RNAs, each targeting a different target DNA sequence. The two or more target DNA sequences may be in the same target gene, or may be in different target genes. The two or more target DNA sequences recognized by the DNA-binding domain may be overlapping or non-overlapping. The target sites for two of the DNA-binding domains may be separated by, for example, about 100 base pairs, about 200 base pairs, about 300 base pairs, about 400 base pairs, about 500 base pairs, about 600 or more base pairs. In addition, when targeting double-stranded DNA, such as an endogenous genome, the DNA-binding domains of the artificial transcription factors may target the same or different strands (one or more to positive strand and/or one or more to negative strand). Further, the same or different DNA-binding domains may be used in the epigenetic editors described herein.

Linkers

[0370] Epigenetic editors provided herein may comprise one or more linkers that connect one or more components of the epigenetic editors. A linker may be a covalent bond or a polymeric linker with many atoms in length. A linker may be a peptide linker or a non-peptide linker.

[0371] In certain embodiments, linkers may be used to link any of the peptides or peptide domains of the epigenetic editor. The linker may be as simple as a covalent bond, or it may be a polymeric linker many atoms in length. In certain embodiments, the linker is a polypeptide or based on amino acids. In other embodiments, the linker is not peptide-like. In certain embodiments, the linker is a covalent bond (e.g., a carbon-carbon bond, disulfide bond, carbon-heteroatom bond, etc.). In certain embodiments, the linker is a carbon-nitrogen bond of an amide linkage. In certain embodiments, the linker is a cyclic or acyclic, substituted or unsubstituted, branched or unbranched aliphatic or heteroaliphatic linker. In certain embodiments, the linker is polymeric (e.g., polyethylene, polyethylene glycol, polyamide, polyester, etc.). In certain embodiments, the linker comprises a monomer, dimer, or polymer of aminoalkanoic acid. In certain embodiments, the linker comprises an aminoalkanoic acid (e.g., glycine, ethanoic acid, alanine, beta-alanine, 3-aminopropanoic acid, 4-aminobutanoic acid, 5-pentanoic acid, etc.). In certain embodiments, the linker comprises a monomer, dimer, or polymer of aminohexanoic acid (Ahx). In certain embodiments, the linker is based on a carbocyclic moiety (e.g., cyclopentane, cyclohexane). In other embodiments, the linker comprises a polyethylene glycol moiety (PEG). In other embodiments, the linker comprises amino acids. In certain embodiments, the linker comprises a peptide. In certain embodiments, the linker comprises an aryl or heteroaryl moiety. In certain embodiments, the linker is based on a phenyl ring. The linker may include functionalized moieties to facilitate attachment of a nucleophile (e.g., thiol, amino) from the peptide to the linker. Any electrophile may be used as part of the linker. Exemplary electrophiles include, but are not limited to, activated esters, activated amides, Michael acceptors, alkyl halides, aryl halides, acyl halides, and isothiocyanates.

[0372] In some embodiments, the linker is a non-peptide linker. For example, the linker may be a carbon bond, a disulfide bond, or carbon-heteroatom bond. In certain embodiments, the linker is a carbon-nitrogen bond of an amide linkage. In certain embodiments, the linker is a cyclic or acyclic, substituted or unsubstituted, branched or unbranched aliphatic or heteroaliphatic linker.

[0373] In certain embodiments, the linker is polymeric (e.g. , polyethylene, polyethylene glycol, polyamide, polyester, etc.). In certain embodiments, the linker comprises a monomer, dimer, or polymer of aminoalkanoic acid. In certain embodiments, the linker comprises an aminoalkanoic acid (e.g. , glycine, ethanoic acid, alanine, beta-alanine, 3-aminopropanoic acid, 4-

aminobutanoic acid, 5-pentanoic acid, etc.). In certain embodiments, the linker comprises a monomer, dimer, or polymer of aminohexanoic acid (Ahx). In certain embodiments, the linker is based on a carbocyclic moiety (e.g. , cyclopentane, cyclohexane). In other embodiments, the linker comprises a polyethylene glycol moiety (PEG). In other embodiments, the linker comprises amino acids. In certain embodiments, the linker comprises a peptide. In certain embodiments, the linker comprises an aryl or heteroaryl moiety. In certain embodiments, the linker is based on a phenyl ring. The linker may include functionalized moieties to facilitate attachment of a nucleophile (e.g. , thiol, amino) from the peptide to the linker. Any electrophile may be used as part of the linker. Exemplary electrophiles include, but are not limited to, activated esters, activated amides, alkyl halides, aryl halides, acyl halides, and isothiocyanates.

[0374] In some embodiments, one or more linkers of an epigenetic editor provided herein is a peptide linker. For example, a zinc finger array and a repressor domain may be connected by a peptide linker, forming a zinc finger–repressor fusion protein. A peptide linker can be any length applicable to the epigenetic editor fusion proteins described herein. In some embodiments, the linker can comprise a peptide between 1 and 200 amino acids. In some embodiments, a DNA binding domain, e.g., a zinc finger array and an effector domain are fused via a linker that comprises from 1 to 5, 1 to 10, 1 to 20, 1 to 30, 1 to 40, 1 to 50, 1 to 60, 1 to 80, 1 to 100, 1 to 150, 1 to 200, 5 to 10, 5 to 20, 5 to 30, 5 to 40, 5 to 60, 5 to 80, 5 to 100, 5 to 150, 5 to 200, 10 to 20, 10 to 30, 10 to 40, 10 to 50, 10 to 60, 10 to 80, 10 to 100, 10 to 150, 10 to 200, 20 to 30, 20 to 40, 20 to 50, 20 to 60, 20 to 80, 20 to 100, 20 to 150, 20 to 200, 30 to 40, 30 to 50, 30 to 60, 30 to 80, 30 to 100, 30 to 150, 30 to 200, 40 to 50, 40 to 60, 40 to 80, 40 to 100, 40 to 150, 40 to 200, 50 to 60 50 to 80, 50 to 100, 50 to 150, 50 to 200, 60 to 80, 60 to 100, 60 to 150, 60 to 200, 80 to 100, 80 to 150, 80 to 200, 100 to 150, 100 to 200, or 150 to 200 amino acids in length. Longer or shorter linkers are also contemplated. In some embodiments, the peptide linker is 4, 16, 32, or 104 amino acids in length. In some embodiments, the peptide linker is a flexible linker. In some embodiments, the peptide linker is a rigid linker.

[0375] In some embodiments, the peptide linker comprises the amino acid sequence of SEQ ID NO.: 679-683

[0376] In some embodiments, the peptide linker is a XTEN linker. In some embodiments, the peptide linker comprises the amino acid sequence SEQ ID NO.: 684. In some embodiments, the linker is 24 amino acids in length. In some embodiments, the linker comprises the amino acid sequence SEQ ID NO.: 685. In some embodiments, the linker is 40 amino acids in length. In some embodiments, the linker comprises the amino acid sequence SEQ ID NO.: 686. In some

embodiments, the linker is 64 amino acids in length. In some embodiments, the linker comprises the amino acid sequence SEQ ID NO.: 687.

[0377] In some embodiments, the linker is 92 amino acids in length. In some embodiments, the linker comprises the amino acid sequence SEQ ID NO.: 688.

[0378] Various linker lengths and flexibilities between a effector domain (e.g. , a repressor domain) and a DNA binding protein (e.g., a Cas9 domain), between a effector domain and a second effector domain, or between any two components of an epigenetic editor can be employed (e.g., ranging from very flexible linkers of the form (GGGGS)n, (GGGGS)n, and (G)n to more rigid linkers of the form (EAAAK)n, (SGGS)n, and (XP)n) in order to achieve the optimal length for effector domain activity for the specific application. In some embodiments, n is any integer between 3 and 30. In some embodiments, n is 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, or 15. In some embodiments, the linker comprises a (GGS)n motif, wherein n is 1, 3, or 7.

[0379] In some embodiments, a linker in an epigenetic editor comprises a nuclear localization signal, for example, of peptide sequence SEQ ID NO.: 689-694. In some embodiments, a linker in an epigenetic editor comprises a cleavable peptide, e.g., a T2A peptide, a p2A peptide, or a furin/p2A peptide. In some embodiments, a linker in an epigenetic editor comprises an expression tag, e.g. a detectable tag such as a green fluorescence protein.

[0380] In some embodiments, a linker comprises a nucleic acid. For example, one or more linkers of an epigenetic editor may include a nucleic acid that is capable of binding to, interacting with, associating with, or forming a complex with a polypeptide. In some embodiments, the nucleic acid linker may be a RNA linker capable of binding to and/or interacting with a RNA binding protein domain, e.g. a phase derived RNA binding domain. In some embodiments, the nucleic acid linker may be fused to a guide polynucleotide capable of binding to a Cas protein of an epigenetic editor. In some embodiments, the nucleic acid linker comprises a K homology (KH) domain binding sequence, a MS2 coat protein binding sequence, a PP7 coat protein binding sequence, a SfMu COM coat protein binding sequence, a telomerase Ku binding motif binding sequence, a sm7 protein binding sequence, or other RNA recognition motif binding sequence thereof.

[0381] In some embodiments, a linker comprises an affinity domain that specifically binds a component of an epigenetic effector. For example, an epigenetic effector may comprise a programmable DNA binding domain, a linker comprising an affinity domain having specific binding affinity to an epigenetic effector domain. The affinity domain may comprise an antibody, a single chain antibody, a nanobody, and antigen binding sequence, an antibody, a

nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a KAP1 antibody which binds to a KAP1 protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a KRAB antibody which binds to a KRAB protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a DNMT1 antibody which binds to a DNMT1 protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a DNMT3A antibody which binds to a DNMT3A protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a DNMT3L antibody which binds to a DNMT3L protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a ZIM3 antibody which binds to a ZIM3 protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a TET1 antibody which binds to a TET1 protein. In some embodiments, an epigenetic effector domain comprises a programmable DNA binding domain and a VP16 or VP64 antibody which binds to a VP16 or VP64 protein.

[0382] In some embodiments, a linker comprises a repeat peptide array. In some embodiments, a linker comprises an epitope tag, for example, a SunTag. In some embodiments, an epigenetic editor comprises one or more peptide arrays comprising multiple copies of an epitope tag that can link multiple effector domains attached to or fused to peptide recognizing the epitope tag. For example, a epitope tag array can link a DNA binding domain and multiple effector domains or multiple copies of effector domains fused to or attached to antibody sequences recognizing the epitope tag. In some embodiments, an epigenetic editor comprises at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 or more epitope tag repeats that link at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 or more effector domains or copies of effector domains. In some embodiments, an epigenetic editor comprises multiple epitope tag repeats that link multiple effector domains and detectable expression tag domains, e.g. GFPs. In some embodiments, the repeat peptide array comprises gene control non-depressible 4 (GCN4) peptide sequences. In some embodiments, the repeat peptide arrays are further linked by linking peptide sequences of 15 to 50 amino acids. Repeat peptide arrays as described in US patent application No. US20170219596 and US patent No. 10,612, 044 are incorporated herein by reference in its entirety.

Nuclear localization signals

[0383] In some embodiments, the epigenetic editors provided herein comprise one or more nuclear targeting sequences. For example, a zinc finger – repressor fusion protein described herein may further comprise one or more nuclear targeting sequences, for example, a nuclear localization sequence (NLS). In some embodiments, the fusion protein comprises multiple NLSs. In some embodiments, the fusion protein comprises a NLS at the N-terminus or the C-terminus of the fusion protein. In some embodiments, the fusion protein comprises a NLS at both the N-terminus and the C-terminus. In some embodiments, the NLS is embedded in the middle of the fusion protein. In some embodiments, a NLS comprises an amino acid sequence that facilitates the importation of a protein, that comprises an NLS, into the cell nucleus. In some embodiments, the NLS is fused to the N-terminus of the fusion protein. In some embodiments, the NLS is fused to the C- terminus of the fusion protein. In some embodiments, the NLS is fused to the N-terminus of the nucleic acid binding protein, e.g. the Cas9 or zinc finger array. In some embodiments, the NLS is fused to the C-terminus of the nucleic acid binding protein. In some embodiments, the NLS is fused to the N-terminus of a effector domain, e.g., a repressor domain. In some embodiments, the NLS is fused to the C-terminus of a effector domain, e.g., a repressor domain. In some embodiments, the NLS is fused to the fusion protein via one or more linkers. In some embodiments, the NLS is fused to the fusion protein without a linker. In some embodiments, the NLS comprises an amino acid sequence of any one of the NLS sequences provided or referenced herein. In some embodiments, a NLS comprises the amino acid sequence SEQ ID NO.: 687 or SEQ ID NO.: 692. Additional nuclear localization sequences are known in the art and would be apparent to the skilled artisan.

Tags

[0384] Epigenetic editors provided herein may comprise one or more additional sequences domains, tags, for tracking, detection, and localization of the editors. In some embodiments, an epigenetic editor comprises one or more detectable tags. In some embodiments, the epigenetic editor comprises 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 or more detectable tags. Each of the detectable tags may be same or different.

[0385] For example, an epigenetic editor fusion protein may comprise cytoplasmic localization sequences, export sequences, such as nuclear export sequences, or other localization sequences, as well as sequence tags that are useful for solubilization, purification, or detection of the fusion proteins. Suitable protein tags provided herein include, but are not limited to, biotin carboxylase carrier protein (BCCP) tags, myc-tags, calmodulin-tags, FLAG-tags, hemagglutinin (HA)-tags, polyhistidine tags, also referred to as histidine tags or His-tags, maltose binding protein (MBP)-tags, nus-tags, glutathione-S-transferase (GST)-tags, green fluorescent protein (GFP)-tags,

thioredoxin-tags, S-tags, Softags (e.g. , Softag 1, Softag 3), strep-tags , biotin ligase tags, FlAsH tags, V5 tags, and SBP-tags. Additional suitable sequences will be apparent to those of skill in the art.

[0386] In some embodiments, an epigenetic editor comprises from 1 to 2 detectable tags. In aspects, the fusion protein comprises 1 detectable tag. In aspects, the fusion protein comprises 2 detectable tags. In aspects, the fusion protein comprises 3 detectable tags. In aspects, the fusion protein comprises 4 detectable tags. In aspects, the fusion protein comprises 5 detectable tags.

Epigenetic editor structure

[0387] The multiple components of epigenetic editors described herein may be in any order. In some embodiments, an epigenetic editor comprises the structure: N']-[D1]-[D2]-[C', wherein any one of D1 and D2 is a DNA binding domain or an effector domain.

[0388] In some embodiments, an epigenetic editor comprises the structure: N']-[D1]-[D2]-[D3]-[C', wherein any one of D1, D2, and D3 is a DNA binding domain, or an effector domain. In some embodiments, D1 is a DNA binding domain. In some embodiments, D2 is a DNA binding domain. In some embodiments, D3 is a DNA binding domain. In some embodiments, D1 is the only DNA binding domain. In some embodiments, D2 is the only DNA binding domain. In some embodiments, D3 is the only DNA binding domain.

[0389] In some embodiments, an epigenetic editor comprises the structure: N']-[D1]-[D2]-[D3]-[D4]-[C', wherein any one of D1, D2, D3, and D4 is a DNA binding domain, or an effector domain. In some embodiments, D1 is a DNA binding domain. In some embodiments, D2 is a DNA binding domain. In some embodiments, D3 is a DNA binding domain. In some embodiments, D4 is a DNA binding domain. In some embodiments, D1 is the only DNA binding domain. In some embodiments, D2 is the only DNA binding domain. In some embodiments, D3 is the only DNA binding domain. In some embodiments, D4 is the only DNA binding domain.

[0390] In some embodiments, an epigenetic editor comprises the structure: N']-[D1]-[D2]-[D3]-[D4]-[D5]-[C', wherein any one of D1, D2, D3, D4, and D5 is a DNA binding domain, or an effector domain. In some embodiments, D1 is a DNA binding domain. In some embodiments, D2 is a DNA binding domain. In some embodiments, D3 is a DNA binding domain. In some embodiments, D4 is a DNA binding domain. In some embodiments, D5 is a DNA binding domain. In some embodiments, D1 is the only DNA binding domain. In some embodiments, D2 is the only DNA binding domain. In some embodiments, D3 is the only DNA binding domain. In some embodiments, D4 is the only DNA binding domain. In some embodiments, D5 is the only DNA binding domain.

[0391] In some embodiments, the epigenetic editor comprises at least one effector domain that is a DNMT domain. In some embodiments, the epigenetic editor comprises at least one effector domain that is a KRAB domain. In some embodiments, the epigenetic effector comprises at least one effector domain that is a fusion of a DNMT3A-DNMT3L domain.

[0392] In some embodiments, the epigenetic editor comprises at least one effector domain that is a TET1 domain. In some embodiments, the epigenetic editor comprises at least one effector domain that is a VP16 domain. In some embodiments, the epigenetic editor comprises at least one effector domain that is a VP64 domain. In some embodiments, the epigenetic editor comprises at least one effector domain that is a RTA domain.

[0393] Components of an epigenetic editor may be structured in different configurations. For example, the DNA binding domain may be at the C terminus, the N terminus, or in between two or more epigenetic effector domains or additional domains. In some embodiments, the DNA binding domain is at the C terminus of the epigenetic editor. In some embodiments, the DNA binding domain is at the N terminus of the epigenetic editor. In some embodiments, the DNA binding domain is linked to one or more nuclear localization signals. In some embodiments, the DNA binding domain is linked to two or more nuclear localization signals. In some embodiments, the DNA binding domain is flanked by an epigenetic effector domain or an additional domain on both termini. In some embodiments, the epigenetic editor comprises the configuration of N']-[epigenetic effector domain 1]-[DNA binding domain]-[epigenetic effector domain 2]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[epigenetic effector domain 1]-[DNA binding domain]-[epigenetic effector domain 2]-[epigenetic effector domain 3]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[epigenetic effector domain 1]- [epigenetic effector domain 2]- [DNA binding domain]- [epigenetic effector domain 3]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[epigenetic effector domain 1]- [epigenetic effector domain 2]- [DNA binding domain]- [epigenetic effector domain 3]- [epigenetic effector domain 4]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[KRAB]-[DNA binding domain]-[Dnmt3A]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[KRAB]-[DNA binding domain]-[Dnmt3A]-[Dnmt3L]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[SETDB1]-[DNA binding domain]-[Dnmt3A]-[Dnmt3L]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[SETDB1]-[DNA binding domain]-[Dnmt3A]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[KRAB]-[DNA binding

domain]-[Dnmt3A-Dnmt3L]-[C', wherein Dnmt3A and Dnmt3L are directly fused via a peptide bond.

[0394] In some embodiments, the epigenetic editor comprises the configuration of N']-[Dnmt3A]-[DNA binding domain]-[KRAB]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[Dnmt3A]- [Dnmt3L]- [DNA binding domain]-[KRAB]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']- [Dnmt3A-Dnmt3L]- [DNA binding domain]- [KRAB]- [C', wherein Dnmt3A and Dnmt3L are directly fused via a peptide bond. In some embodiments, the epigenetic editor comprises the configuration of N']-[Dnmt3A]-[DNA binding domain]-[SETDB1]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']-[Dnmt3A]- [Dnmt3L]- [DNA binding domain]-[SETDB1]-[C'. In some embodiments, the epigenetic editor comprises the configuration of N']- [Dnmt3A-Dnmt3L]- [DNA binding domain]- [SETDB1]- [C', wherein Dnmt3A and Dnmt3L are directly fused via a peptide bond. In some embodiments, a connecting structure "]-["in any one of the epigenetic editor structures is a linker, e.g., a peptide linker. In some embodiments, a connecting structure "]-[" in any one of the epigenetic editor structures is a detectable tag. In some embodiments, a connecting structure "]-[" in any one of the epigenetic editor structures is a peptide bond. In some embodiments, a connecting structure "]-[" in any one of the epigenetic editor structures is a nuclear localization signal. In some embodiments, a connecting structure "]-[" in any one of the epigenetic editor structures is a promoter or a regulatory sequence. In an epigenetic editor structure, the multiple connecting structures "]-[" may be same or may each be a different linker, tag, NLS, or peptide bond.

[0395] The DNA binding domain (DBD) of an epigenetic editor may comprise any one of the DNA binding domains described herein or known to those skilled in the art. In some embodiments, the DBD comprises one or more zinc finger arrays. In some embodiments, the DBD comprises a TALE DNA binding domain. In some embodiments, the DBD is a RNA guided programmable DNA binding domain, e.g. a CRISPR-Cas protein domain. Suitable Cas proteins has been provided herein, including nuclease inactive Cas proteins for the purpose of epigenetic editing without causing target DNA strand breaks. A Cas protein in an epigenetic editor may be a nuclease inactive Cas9 (dCas9), a SaCas9d, a SpCas9d, a dCas9 with modified PAM specificity, a high-fidelity dCas9, a nuclease inactive Cpf1 (dCpf1), a dCpf1 with modified PAM specificity, a high-fidelity dCpf1, a dCas12e, a dCasY, or any other Cas protein as described herein.

[0396] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD) and an effector domain that represses or silences expression of a target gene. In some

embodiments, the epigenetic editor comprises the configuration of N']-[repression domain]-[DBD]-[-C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[DBD]-[repression domain]-[-C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0397] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD) and a DNA methyltransferase domain that deposits one or more methylation marks at a target gene, thereby repressing or silencing expression of the target gene. In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA methyltransferase domain]-[DBD]-[-C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[DBD]-[DNA methyltransferase domain]-[-C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0398] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD), a DNA methyltransferase domain, and an effector domain that represses or silences expression of a target gene. In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA methyltransferase domain]-[DBD]-[repression domain]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[repression domain]-[DBD]-[DNA methyltransferase domain]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0399] In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA methyltransferase domain]-[repression domain]– [DBD]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[repression domain]-[DNA methyltransferase domain]– [DBD]- [C', wherein the connecting structure]-[is any one

of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0400] The repression domain in an epigenetic editor may comprise any one of the expression repression proteins known to those skilled in the art and as described herein, or any homologs or combination thereof. In some embodiments, the repression domain comprises a histone deacetylase domain. In some embodiments, the repression domain interacts with a scaffold protein domain that recruits one or more protein domains that repress expression of the target gene. For example, the repression domain may recruit or interact with a scaffold protein domain that recruits a PRMT protein, a HDAC protein, a SETDB1 protein, or a NuRD protein domain. In some embodiments, the repression domain interacts with epigenetically marked DNA nucleotides in a target gene thereby repressing or silencing expression of the target gene. In some embodiments, the repression domain comprises a MECP2 domain. In some embodiments, the repression domain comprises a KAP1 domain. In some embodiments, the repression domain comprises any one of the domains of Table 2 or Table 3, or any combination or homologs thereof.

[0401] The DNA methyltransferase domain in an epigenetic editor may comprise any one of the DNA methyltransferase proteins known to those skilled in the art and as described herein, or any homologs or combination thereof. In some embodiments, the effector domain comprises a DNMT3 domain. In some embodiments, the DNA methyltransferase domain comprises a DNMT3A domain. In some embodiments, the DNA methyltransferase domain comprises a DNMT3B domain. In some embodiments, the DNA methyltransferase domain comprises a DNMT3C domain. In some embodiments, the DNA methyltransferase domain comprises a DNMT3L domain. In some embodiments, the DNA methyltransferase domain comprises a fusion of DNMT3A-DNMT3L domain. As described herein, the DNMT3A-DNMT3L fusion domain may be in either order, e.g., N-DNMT3A-DNMT3L-C, or N-DNMT3L-DNMT3A-C. In some embodiments, the DNA methyltransferase domain comprises any one of the domains of Table 1, or any combination or homologs thereof.

[0402] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD) and an effector domain that increases expression of a target gene. In some embodiments, the epigenetic editor comprises the configuration of N']-[activation domain]-[DBD]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[DBD]-[activation domain]-[C', wherein the connecting structure]-[is any one of the linkers as

described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0403] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD) and a DNA demethylation domain that removes one or more methylation marks at a target gene, thereby increasing expression of the target gene. In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA demethylase domain]-[DBD]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[DBD]-[DNA demethylase domain]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0404] In some embodiments, an epigenetic editor comprises a DNA binding domain (DBD), a DNA demethylase domain, and an activation effector domain that increases expression of a target gene. In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA demethylase domain]-[DBD]-[activation domain]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[activation domain]-[DBD]-[DNA demethylase domain]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0405] In some embodiments, the epigenetic editor comprises the configuration of N']-[DNA demethylase domain]-[activation domain]– [DBD]-[C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence. In some embodiments, the epigenetic editor comprises the configuration of N']-[activation domain]-[DNA demethylase domain]– [DBD]- [C', wherein the connecting structure]-[is any one of the linkers as described herein, a detectable tag, an affinity domain, a peptide bond, a nuclear localization signal, a promoter, and/or a regulatory sequence.

[0406] The activation domain in an epigenetic editor may comprise any one of the expression activation proteins known to those skilled in the art and as described herein, or any homologs or combination thereof. In some embodiments, the activation domain comprises a histone acetyltransferase domain. In some embodiments, the activation domain interacts with a scaffold

protein domain that recruits one or more protein domains that activate expression of the target gene. For example, the activation domain may recruit or interact with a scaffold protein domain that recruits one or more transcription factors or activators. In some embodiments, the activation domain comprises a Herpes Simplex Virus Protein 16 (VP16) activation domain. In some embodiments, the activation domain comprises an activation domain comprising a tandem repeat of multiple VP16 activation domains. In some embodiments, the activation domain comprises four tandem copies of VP16, a VP64 activation domain. In some embodiments, the activation domain comprises eight tandem copies of VP16, a VP128 activation domain. In some embodiments, the activation domain comprises ten tandem copies of VP16, a VP160 activation domain. In some embodiments, the activation domain comprises p65 activation domain of NFκB. In some embodiments, the activation domain comprises an Epstein-Barr virus R transactivator (Rta) activation domain. In some embodiments, the activation domain comprises a fusion of multiple activators, e.g., a tripartite activator of the VP64, the p65, and the Rta activation domains, (a VPR activation domain). In some embodiments, the activation domain comprises any one of the domains of Table 5 or Table 6, or any homologs or combination thereof.

[0407] The DNA demethylation domain in an epigenetic editor may comprise any one of the DNA demethylation proteins known to those skilled in the art and as described herein, or any homologs or combination thereof. In some embodiments, the DNA demethylation domain comprises a TET family protein domain. In some embodiments, the DNA demethylation domain comprises a TET1, TET2, or TET3 protein domain. In some embodiments, the DNA demethylation domain comprises a TET1 protein domain. In some embodiments, the DNA demethylation domain comprises any one of the domains of Table 4, or any homologs or combination thereof.

[0408] In some embodiments, an epigenetic editor that can reduce or silence expression of a target gene comprises a Dnmt3A-Dnmt3L fusion protein domain. In some embodiments, the epigenetic editor further comprises a repression scaffold or recruiting protein domain, for example, a KRAB domain, a KAP1 domain, or a MECP2 domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and an additional repression domain that reduces or silences expression of a target gene. The repression domain in an epigenetic editor may comprise any one of the expression repression proteins known to those skilled in the art and as described herein, or any homologs or combination thereof. In some embodiments, the repression domain comprises a histone deacetylase domain. In some embodiments, the repression domain interacts with a scaffold protein domain that recruits one or

more protein domains that repress expression of the target gene. For example, the repression domain may recruit or interact with a scaffold protein domain that recruits a PRMT protein, a HDAC protein, a SETDB1 protein, or a NuRD protein domain. In some embodiments, the repression domain interacts with epigenetically marked DNA nucleotides in a target gene thereby represses or silences expression of the target gene. In some embodiments, the repression domain comprises a MECP2 domain. In some embodiments, the repression domain comprises a KAP1 domain. In some embodiments, the repression domain comprises any one of the domains of Table 2 or Table 3, or any combination or homologs thereof.

[0409] In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and a KAP1 domain. In some embodiments, the epigenetic editor comprises the following configuration: N]-[Dnmt3A-3L]-[KAP1]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[KAP1]-[Dnmt3A-3L]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[Dnmt3A-3L]-[KAP1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[KAP1]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [KAP1]-[DBD]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [Dnmt3A-3L]-[DBD]-[KAP1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein.

[0410] In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and a MECP2 domain. In some embodiments, the epigenetic editor comprises the following configuration: N]-[Dnmt3A-3L]-[MECP2]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[MECP2]-[Dnmt3A-3L]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [DBD]-[Dnmt3A-3L]-[MECP2]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [DBD]-[MECP2]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor

comprises the following configuration: N]- [MECP2]-[DBD]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [Dnmt3A-3L]-[DBD]-[MECP2]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein.

[0411] In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and a heterochromatin protein 1 (HP1) domain. In some embodiments, the epigenetic editor comprises the following configuration: N]-[Dnmt3A-3L]-[HP1]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[HP1]-[Dnmt3A-3L]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[Dnmt3A-3L]-[HP1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[HP1]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [HP1]-[DBD]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [Dnmt3A-3L]-[DBD]-[HP1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein.

[0412] In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and a SETDB1 domain. In some embodiments, the epigenetic editor comprises the following configuration: N]-[Dnmt3A-3L]-[SETDB1]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[SETDB1]-[Dnmt3A-3L]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[Dnmt3A-3L]-[SETDB1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[SETDB1]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [SETDB1]-[DBD]-[Dnmt3A-3L]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [Dnmt3A-3L]-[DBD]-

[SETDB1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein.

[0413] In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnmt3L fusion domain and a SETDB1 domain, a KAP1, domain, a KRAB domain, and/or a MECP2 domain, in any order and combination thereof.

[0414] In some embodiments, the epigenetic editor that reduces or silences expression of a target gene comprises a DBD and an affinity domain that specifically binds to a repression domain. For example, the epigenetic editor may comprise a DBD and a repression domain antibody. In some embodiments, the epigenetic editor comprises a DBD and a KAP1 affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a KRAB affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a SETDB1 affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a MECP2 affinity domain. In some embodiments, the epigenetic editor comprises a DNA methyltransferase and a repression domain binding affinity domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnm3L fusion and a repression domain binding affinity domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnm3L fusion and KAP1 affinity domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnm3L fusion and KRAB affinity domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnm3L fusion and SETDB1 affinity domain. In some embodiments, the epigenetic editor comprises a Dnmt3A-Dnm3L fusion and MECP2 affinity domain. As used herein, an affinity domain may be an antibody, a single chain antibody, a nanobody, and antigen binding sequence, an antibody, a nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof.

[0415] In some embodiments, the epigenetic editor that reduces or silences expression of a target gene comprises a DBD and an affinity domain that specifically binds to a DNA methyltransferase domain. For example, the epigenetic editor may comprise a DBD and a DNA methyltransferase antibody. In some embodiments, the epigenetic editor comprises a DBD and a Dnmt3A affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a Dnmt3L affinity domain. In some embodiments, the epigenetic editor comprises a repression domain and a DNA methyltransferase binding affinity domain. In some embodiments, the epigenetic editor comprises a repression domain and a Dnmt3A binding affinity domain. In some embodiments, the epigenetic editor comprises a repression domain and Dnmt3L affinity

domain. In some embodiments, the epigenetic editor comprises one or more of a KAP1, a KRAB and a MECP2 domain, and a Dnmt3A binding affinity domain. In some embodiments, the epigenetic editor comprises one or more of a KAP1 domain, and a Dnmt3A binding affinity domain. In some embodiments, the epigenetic editor comprises one or more of a KAP1, a KRAB and a MECP2 domain, and a Dnmt3L binding affinity domain. In some embodiments, the epigenetic editor comprises one or more of a KAP1 domain, and a Dnmt3L binding affinity domain. The affinity domain may be an antibody, a single chain antibody, a nanobody, and antigen binding sequence, an antibody, a nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof.

[0416] In some embodiments, the epigenetic editor that reduces or silences expression of a target gene comprises a DBD and a first affinity domain that specifically binds to a DNA methyltransferase domain and a second affinity domain that specifically binds to a repression domain. For example, the epigenetic editor may comprise a DBD and a DNA methyltransferase antibody and a repression domain antibody. In some embodiments, the epigenetic editor comprises a DBD, a KAP1 affinity domain and a Dnmt3A affinity domain. In some embodiments, the epigenetic editor comprises a DBD, a KAP1 affinity domain and a Dnmt3L affinity domain. In some embodiments, the epigenetic editor comprises a DBD, a MECP2 affinity domain and a Dnmt3A affinity domain. In some embodiments, the epigenetic editor comprises a DBD, a MECP2 affinity domain and a Dnmt3L affinity domain. In some embodiments, the epigenetic editor comprises a DBD, a KRAB affinity domain and a Dnmt3A affinity domain. In some embodiments, the epigenetic editor comprises a DBD, a KRAB affinity domain and a Dnmt3L affinity domain. The affinity domain may be an antibody, a single chain antibody, a nanobody, and antigen binding sequence, an antibody, a nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof.

[0417] In some embodiments, an epigenetic editor that can increase expression of a target gene comprises a TET1 protein domain. In some embodiments, the epigenetic editor further comprises a activation protein domain, for example, a VP16 domain, a VP64 domain, a p65 domain or a Rta domain. In some embodiments, the epigenetic editor comprises a VP64-p65-Rta activation domains (a VPR activation domain) and a TET1 domain. In some embodiments, the epigenetic editor comprises the following configuration: N]-[TET1]-[VPR]-[DBD]-[C, wherein

the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[VPR]-[TET1]-[DBD]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]-[DBD]-[TET1]-[VPR]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [DBD]-[VPR]-[TET1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [VPR]-[DBD]-[TET1]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein. In some embodiments, the epigenetic editor comprises the following configuration: N]- [TET1]-[DBD]-[VPR]-[C, wherein the connecting structure]-[may be any one of the linkers as provided herein, for example, a peptide linker, an array of epitope tags, or a scaffold nucleic acid (e.g. a RNA that recognizes a MS2 domain fused to the DBD, the TET, or the VPR domain).

[0418] In some embodiments, the epigenetic editor that increases expression of a target gene comprises a DBD and an affinity domain that specifically binds to an activation domain. For example, the epigenetic editor may comprise a DBD and an activation domain antibody. In some embodiments, the epigenetic editor comprises a DBD and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a VP16 affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a p65 affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a Rta affinity domain. In some embodiments, the epigenetic editor comprises a DNA demethylase and an activation domain binding affinity domain. In some embodiments, the epigenetic editor comprises a activation domain and a demethylase affinity domain. In some embodiments, the epigenetic editor comprises a DBD and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a VP16 domain and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a VP64 domain and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a Rta domain and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a p65 domain and a TET1 affinity domain. In some embodiments, the epigenetic editor comprises a VPR activation domain and a TET1 affinity domain. The affinity domain may be an antibody, a single chain antibody, a nanobody, and antigen binding sequence, an antibody, a nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL

domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof.

Additional domains

[0419] An epigenetic editor system may further comprise an additional heterologous portion or domain (e.g., polynucleotide binding domain such as an RNA or DNA binding protein) that is capable of interacting with, associating with, or capable of forming a complex with a portion or segment (e.g., a polynucleotide motif) of a guide polynucleotide. In some embodiments, the additional heterologous portion or domain (e.g., polynucleotide binding domain such as an RNA or DNA binding protein) can be fused or linked to the DNA binding domain or an effector domain. In some embodiments, the additional heterologous portion may be capable of binding to, interacting with, associating with, or forming a complex with a polypeptide. In some embodiments, the additional heterologous portion may be capable of binding to, interacting with, associating with, or forming a complex with a polynucleotide. In some embodiments, the additional heterologous portion may be capable of binding to a guide polynucleotide. In some embodiments, the additional heterologous portion may be capable of binding to a polypeptide linker. In some embodiments, the additional heterologous portion may be capable of binding to a polynucleotide linker. The additional heterologous portion may be a protein domain. In some embodiments, the additional heterologous portion may be a K Homology (KH) domain, a MS2 coat protein domain, a PP7 coat protein domain, a SfMu Com coat protein domain, a sterile alpha motif, a telomerase Ku binding motif and Ku protein, a telomerase Sm7 binding motif and Sm7 protein, or any other RNA recognition motif.

Target Sequences

[0420] As used herein, a "target polynucleotide sequence" may be a nucleic acid sequence present in a gene of interest. The target sequence may be in a genome of, or expressed in, a cell. In an aspect, epigenetic editors provided herein are used to bind target polynucleotide sequences and effect epigenetic modifications and/or transcription modulation of the target gene. For example, a target sequence may be recognized by a zinc finger array of an epigenetic editor, or may hybridize with a guide RNA sequence complexed with a nuclease inactive CRISPR protein of an epigenetic editor. In embodiments where the epigenetic editor comprises a gRNA-dCas-effector domain complex, the gRNA is designed to have complementarity to the target sequence (or identity to the opposing strand of the target sequence, e.g. the protospacer sequence). In some embodiments, the gRNA comprises a spacer sequence is 100% identical to a protospacer sequence in the target sequence. In some embodiments, the gRNA sequence comprises a spacer

sequence that is about 95%, 90%, 85%, or 80% identical to a protospacer sequence in the target sequence.

[0421] In some embodiments, the target sequence is an endogenous sequence of an endogenous gene of a host cell. In some embodiments, the target sequence is an exogenous sequence.

[0422] The target sequence may be any region of the polynucleotide (e.g., DNA sequence) suitable for epigenetic editing. For example, the target polynucleotide sequence may be any part of a target gene. In some embodiments, the target polynucleotide sequence is part of a transcriptional regulatory sequence. In some embodiment, the target polynucleotide sequence is part of a promoter, enhancer or silencer. In some embodiments, the target polynucleotide sequence is part of a promoter. In some embodiments, the target polynucleotide sequence is part of an enhancer. In some embodiments, the target polynucleotide sequence is part of a silencer. In some embodiments, the target polynucleotide sequence is within about 3000, 2900, 2800, 2700, 2600, 2500, 2400, 2300, 2200, 2100, 2000, 1900, 1800, 1700, 1600, 1500, 1400, 1300, 1200, 1100, 1000, 900, 800, 700, 600, 500, 400, 300, 200, or 100 base pairs (bp) flanking a transcription start site. In some embodiments, the target polynucleotide sequence is within about 1000, 900, 800, 700, 600, 500, 400, 300, 200, or 100 base pairs (bp) flanking a transcription start site. In some embodiments, the target polynucleotide sequence is within about 500, 400, 300, 200, or 100 base pairs (bp) flanking a transcription start site.

[0423] In some embodiments, the target polynucleotide sequence is within about 100 base pairs (bp) flanking a transcription start site.

[0424] In some embodiments, the target polynucleotide sequence is a hypomethylated nucleic acid sequence. In some embodiments, the target polynucleotide sequence is a hypermethylated nucleic acid sequence. In some embodiments, the target polynucleotide sequence is at, near, or within a promoter sequence. In some embodiments, the target polynucleotide sequence is at, near, or within a promoter sequence. In aspects, the target polynucleotide sequence is adjacent to a CpG island. In aspects, the target polynucleotide sequence is known to be associated with a disease or condition.

Modulation of Expression of Target gene

[0425] In some embodiments, the disclosure provides epigenetic editor systems, compositions and methods for epigenetic modifications at a target polynucleotide in a target gene encoding a protein. In some embodiments, the epigenetic editor results in epigenetic modification, e.g. DNA methylation, in a coding region of the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the epigenetic editor results in epigenetic modification, e.g. DNA methylation, in a regulatory sequence such as a promoter or enhancer of the target

gene, thereby reducing or silencing expression of the target gene. In some embodiments, the epigenetic editor results in transcription repression or recruits a transcription repressor to a coding region of the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the epigenetic editor recruits a transcription repressor to a regulatory sequence such as a promoter or enhancer of the target gene, thereby reducing or silencing expression of the target gene. In some embodiments, the epigenetic editor results in epigenetic modification, e.g. DNA demethylation, in a coding region of the target gene, thereby increasing expression of the target gene. In some embodiments, the epigenetic editor results in epigenetic modification, e.g. DNA demethylation, in a regulatory sequence such as a promoter or enhancer of the target gene, thereby increasing expression of the target gene. In some embodiments, the epigenetic editor results in transcription activation or recruits a transcription activator to a coding region of the target gene, thereby increasing expression of the target gene. In some embodiments, the epigenetic editor recruits a transcription activator to a regulatory sequence such as a promoter or enhancer of the target gene, thereby increasing expression of the target gene.

[0426] In some embodiments, the target gene and/or the protein encoded are associated with a disease, disorder, or pathogenic condition.

[0427] Epigenetic modifications effected by the epigenetic editors described herein are sequence specific. In some embodiments, the modification is at a specific site of the target polynucleotide. In some embodiments, the modification is at a specific allele of the target gene. Accordingly, the epigenetic modification may result in modulated expression, for example, reduced or increased expression, of one copy of a target gene harboring a specific allele, and not the other copy of the target gene. In some embodiments, the specific allele is associated with a disease, condition, or disorder.

[0428] Epigenetic modification may be made at any target genes of a genome of interest, for example, a prokaryote genome, a plant genome, mammalian or human genome. The target gene can be of or derived from any organism and genome thereof. For example, the target gene can be a prokaryotic gene, a eukaryotic gene, an animal gene, a plant gene, a mouse gene, a rat gene, a rabbit gene, a fish gene, an avian gene, a monkey gene, or a human gene. In some embodiments, the target gene is a reporter gene the expression of which can be readily tracked and monitored. Reporter genes and reporter systems include, for example, sequences encoding green fluorescence proteins, red fluorescence proteins, enhanced yellow or enhanced cyan proteins, or luciferase proteins. In some embodiments, the target gene encodes a selectable marker, for example, a beta-galactosidase, a Chloramphenicol acetyltransferase, or a antibiotic resistance

marker. In some embodiments, the target gene is associated with, or harbors one or more mutations that are associated with a disease, condition, or disorder. Non-limiting exemplary target genes include HBB, HBA, hMSH2, HMLH1, growth factors GM-SCF, VEGF, EPO, Erb-B2, and hGH. .

[0429] Target genes also include plant genes for which repression or activation leads to an improvement in plant characteristics, such as improved crop production, disease or herbicide resistance. For example, repression of expression of the FAD2-1 gene results in an advantageous increase in oleic acid and decrease in linoleic and linoleic acids.

[0430] In some embodiments, an epigenetic editor provided herein effects an epigenetic modification in a gene that harbors a target sequence. In some embodiments, the epigenetic editor modulates expression of a protein encoded by the gene. In some embodiments, the epigenetic editor reduces the level of a protein encoded by the gene. In some embodiments, the epigenetic editor increases the level of a protein encoded by the gene.

[0431] To generate epigenetic edits at a target gene, a target gene polynucleotide may be contacted with the epigenetic editing compositions disclosed herein comprising a target DNA binding domain, an epigenetic effector domain, e.g. an epigenetic repressor domain, wherein the DNA binding domain directs the epigenetic effector domain to a target polynucleotide sequence in the target gene, resulting in the epigenetic modification, e.g., a methylation state modification. In some embodiments, the epigenetic editor effects an alteration in the methylation state of a target DNA sequence in the target gene. In some embodiments, the epigenetic editor effects an alteration in the methylation state of a specific allele in the target gene. In some embodiments, the epigenetic editor effects an alteration in the methylation state of a histone protein associated with the target gene.

[0432] In some embodiments, the epigenetic modification reduces transcription of the target gene harboring the target sequence. In some embodiments, the epigenetic modification abolishes transcription of the target gene harboring the target sequence. In some embodiments, the epigenetic modification reduces transcription of a copy of the target gene harboring a specific allele recognized by the epigenetic editor. In some embodiments, the epigenetic modification abolishes transcription of a copy of the target gene harboring a specific allele recognized by the epigenetic editor. In some embodiments, the epigenetic editor reduces the level of a protein encoded by the target gene. In some embodiments, the epigenetic editor eliminates expression of a protein encoded by the target gene. In some embodiments, the epigenetic editor reduces the level of a protein encoded by a copy of the target gene harboring a specific allele recognized by the epigenetic editor. In some embodiments, the epigenetic editor eliminates expression of a

protein encoded by a copy of the target gene harboring a specific allele recognized by the epigenetic editor.

[0433] In some embodiments, the epigenetic modification increases transcription of the target gene harboring the target sequence. In some embodiments, the epigenetic modification increases transcription of a copy of the target gene harboring a specific allele recognized by the epigenetic editor. In some embodiments, the epigenetic editor increases the level of a protein encoded by the target gene. In some embodiments, the epigenetic editor increases the level of a protein encoded by a copy of the target gene harboring a specific allele recognized by the epigenetic editor.

[0434] The target gene may be epigenetically modified in vitro, ex vivo, or in vivo. Accordingly, epigenetic modification of the target gene may modulate expression of a target gene, or an allele thereof, in a cell ex vivo or in a subject in vivo. In some embodiments, the target polynucleotide sequence is the gene locus in the genomic DNA of a cell. In some embodiments, the cell is a cultured cell. In some embodiments, the cell is in vitro. In some embodiments, the cell is ex vivo. In some embodiments, the cell is in vivo. For example, an epigenetic editor, e.g. a fusion protein comprising a zinc finger array and an effector domain, or a sgRNA complexed with a Cas protein-effector domain fusion, may be expressed in a cell where modulated expression of a target gene is desired to thereby allow contact of the target gene with the epigenetic editor described herein. In some embodiments, the cell is from a mammal. In some embodiments, the mammal is a human. In some embodiments, the mammal is a rodent. In some embodiments, the rodent is a mouse. In some embodiments, the rodent is a rat.

[0435] In some embodiments, the epigenetic editors described herein reduces expression of a target gene by at least about 20%, at least about 30%, at least about 40%, at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, at least about 95%, at least about 99%, or more, as measured by transcription of the target gene in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject. In some embodiments, the epigenetic editors described herein reduces expression of a copy of target gene by at least about 20%, at least about 30%, at least about 40%, at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, at least about 95%, at least about 99%, or more, as measured by transcription of the copy of the target gene in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject. In some embodiments, the copy of the target gene harbors a specific sequence or allele recognized by the epigenetic editor. In some embodiments, the epigenetically modified copy encodes a functional protein. Accordingly, in some embodiments, an epigenetic editor composition disclosed herein

reduces or abolishes expression and/or function of protein encoded by a target gene, by reducing or abolishing expression of a functional protein encoded by the target gene. For example, the methods and composition disclosed herein may reduce expression and/or function of a protein encoded by the target gene by at least 3-fold, at least 4-fold, at least 5-fold, at least 6-fold, at least 7-fold, at least 8-fold, at least 9-fold, at least 10-fold, at least 11-fold, at least 12-fold, at least 13-fold, at least 14-fold, at least 15-fold, at least 20-fold, at least 25-fold, at least 30-fold, at least 35-fold, at least 40-fold, at least 45-fold, at least 50-fold, at least 60-fold, at least 70-fold, at least 80-fold, at least 90-fold, or at least 100 fold in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject.

[0436] In some embodiments, the epigenetic editors described herein increases expression of a target gene by at least about 20%, at least about 30%, at least about 40%, at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, at least about 95%, at least about 99%, at least 100%, at least 110%, at least 120%, at least 130%, at least 140%, at least 150%, at least 160%, at least 170%, at least 180%, at least 190%, at least 200%, at least 250%, at least 300%, at least 350%, at least 400%, at least 450%, at least 500% or more, as measured by transcription of the target gene in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject. In some embodiments, the epigenetic editors described herein increases expression of a copy of target gene by at least about 20%, at least about 30%, at least about 40%, at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, at least about 95%, at least about 99%, at least 100%, at least 110%, at least 120%, at least 130%, at least 140%, at least 150%, at least 160%, at least 170%, at least 180%, at least 190%, at least 200%, at least 250%, at least 300%, at least 350%, at least 400%, at least 450%, at least 500% or more, as measured by transcription of the copy of the target gene in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject. In some embodiments, the copy of the target gene harbors a specific sequence or allele recognized by the epigenetic editor. In some embodiments, the epigenetically modified copy encodes a functional protein. Accordingly, in some embodiments, an epigenetic editor composition disclosed herein increases expression and/or function of protein encoded by a target gene, by increasing expression of a functional protein encoded by the target gene. For example, the methods and composition disclosed herein may increase expression and/or function of a protein encoded by the target gene by at least 3-fold, at least 4-fold, at least 5-fold, at least 6-fold, at least 7-fold, at least 8-fold, at least 9-fold, at least 10-fold, at least 11-fold, at least 12-fold, at least 13-fold, at least 14-fold, at least 15-fold, at least 20-fold, at least 25-fold, at least 30-fold, at least 35-fold, at least 40-fold, at least 45-fold, at least 50-fold, at least 60-fold, at least

70-fold, at least 80-fold, at least 90-fold, or at least 100 fold in a cell, a tissue, or a subject as compared to a control cell, control tissue, or a control subject.

[0437] Methods for determining the expression level of a gene, for example the target of an epigenetic editor, are known in the art. For example, transcript level of a gene may be determined by reverse transcription PCR, quantitative RT-PCR, droplet digital PCR (ddPCR), Northern blot, RNA sequencing, DNA sequencing (e.g., sequencing of complementary deoxyribonucleic acid (cDNA) obtained from RNA); next generation (Next-Gen) sequencing, nanopore sequencing, pyrosequencing, or Nanostring sequencing. Protein level expressed from a gene may be determined by western blotting, enzyme linked immuno-absorbance assays, mass-spectrometry, immunohistochemistry, or flow cytometry analysis. Gene expression product levels may be normalized to an internal standard such as total messenger ribonucleic acid (mRNA) or the expression level of a particular gene, e.g., a house keeping gene.

[0438] In some embodiments, the effect of an epigenetic editor in modulating target gene expression may be examined using a reporter system. For example, an epigenetic editor may be designed to target a reporter gene encoding a reporter protein, e.g. a fluorescent protein. Expression of the reporter gene in such a model system may be monitored by, e.g., flow cytometry, fluorescence-activated cell sorting (FACS), or fluorescence microscopy. In some embodiments, a population of cells may be transfected with a vector which harbors a reporter gene. The vector may be constructed such that the reporter gene is expressed when the vector transfects a cell. Suitable reporter genes include genes encoding fluorescent proteins, for example green, yellow, cherry, cyan or orange fluorescent proteins. The population of cells carrying the reporter system may be transfected with DNA, mRNA, or vectors encoding the epigenetic editor targeting the reporter gene. The level of expression of the reporter gene may be quantified using a suitable technique, such as FACS.

[0439] Epigenetic editors described herein may be expressed in a host cell transiently, or may be integrated in a genome of the host cell. Both transiently expressed and integrated epigenetic editors can effect stable epigenetic modifications. For example, after introduction of an epigenetic editor comprising a DNA binding domain specific for a target gene and an epigenetic repression domain to a host cell, the target gene in the host cell may be stably or permanently repressed. In some embodiments, expression of the target gene is reduced for at least 1 week, at least 2 weeks, at least 3 weeks, at least 4 weeks, at least 5 weeks, at least 6 weeks, at least 7 weeks, at least 2 months, at least 3 months, at least 5 months, at least 6 months, at least 1 year, at least 2 years, or for the entire lifetime of the cell or the subject carrying the cell, as compared to the level of expression in the absence of the epigenetic editor. In some embodiments, expression

of the target gene is silenced for at least 1 week, at least 2 weeks, at least 3 weeks, at least 4 weeks, at least 5 weeks, at least 6 weeks, at least 7 weeks, at least 2 months, at least 3 months, at least 5 months, at least 6 months, at least 1 year, at least 2 years, or for the entire lifetime of the cell or the subject carrying the cell as compared to the level of expression in the absence of the epigenetic editor. In some embodiments, after introduction of an epigenetic editor comprising a DNA binding domain specific for a target gene and an epigenetic activation domain to a host cell, the target gene in the host cell is stably or permanently activated. In some embodiments, expression of the target gene is increased for at least 1 week, at least 2 weeks, at least 3 weeks, at least 4 weeks, at least 5 weeks, at least 6 weeks, at least 7 weeks, at least 2 months, at least 3 months, at least 5 months, at least 6 months, at least 1 year, at least 2 years, or for the entire lifetime of the cell or the subject carrying the cell as compared to the level of expression in the absence of the epigenetic editor.

[0440] The epigenetic modification described herein may be inherited by the progeny of host cells that are contacted or introduced with an epigenetic editor. For example, in some embodiments, after introduction of an epigenetic editor comprising a DNA binding domain specific for a target gene and an epigenetic repression domain to a stem cell, e.g., a hematopoietic stem cell, expression of the target gene is also repressed in cells differentiated from the stem cell compared to cells differentiated from a control stem cell in the absence of the epigenetic editor. In some embodiments, expression of the target gene is silenced in cells differentiated from the stem cell. In some embodiments, after introduction of an epigenetic editor comprising a DNA binding domain specific for a target gene and an epigenetic activation domain to a stem cell, e.g., a hematopoietic stem cell, expression of the target gene is also increased in cells differentiated from the stem cell compared to cells differentiated from a control stem cell in the absence of the epigenetic editor.

[0441] Modulation of target gene expression can be assayed by determining any parameter that is indirectly or directly affected by the expression of the target gene. Such parameters include, e.g., changes in RNA or protein levels; changes in protein activity; changes in product levels; changes in downstream gene expression; changes in transcription or activity of reporter genes such as, for example, luciferase, CAT, beta-galactosidase, or GFP; changes in signal transduction; changes in phosphorylation and dephosphorylation; changes in receptor-ligand interactions; changes in concentrations of second messengers such as, for example, cGMP, cAMP, IP3, and Ca2+; changes in cell growth, changes in neovascularization, and/or changes in any functional effect of gene expression. Measurements can be made in vitro, in vivo, and/or ex vivo. Such functional effects can be measured by conventional methods, e.g., measurement of

RNA or protein levels, measurement of RNA stability, and/or identification of downstream or reporter gene expression. Readout can be by way of, for example, chemiluminescence, fluorescence, colorimetric reactions, antibody binding, inducible markers, ligand binding assays; changes in intracellular second messengers such as cGMP and inositol triphosphate (IP3); changes in intracellular calcium levels; cytokine release, and the like.

[0442] To determine the level of gene expression modulation by a ZFP, cells contacted with ZFPs are compared to control cells, e.g., without the zinc finger protein or with a non-specific ZFP, to examine the extent of inhibition or activation. Control samples are assigned a relative gene expression activity value of 100%. Modulation/inhibition of gene expression is achieved when the gene expression activity value relative to the control is about 80%, preferably 50% (i.e., 0.5× the activity of the control), more preferably 25%, more preferably 5-0%. Modulation/activation of gene expression is achieved when the gene expression activity value relative to the control is 110%, more preferably 150% (i.e., 1.5× the activity of the control), more preferably 200-500%, more preferably 1000-2000% or more.

Delivery

[0443] In an aspect, provided herein is a composition for gene expression modulation comprising the epigenetic editor as provided herein that generates epigenetic modifications at target genes. The epigenetic editor, or nucleic acid encoding the epigenetic editor or components thereof (e.g. nucleic acids encoding an epigenetic editor fusion protein comprising a zinc finger – repressor fusion, a Cas9-repressor fusion, and or nucleic acids encoding one or more guide RNAs) may be introduced to a cell via various ways known in the art. For example, in some embodiments, the epigenetic editor is delivered to a host cell or integrated into the genome of the host cell, or for transient expression in the host cell.

[0444] In some embodiments, the nucleic acid encoding the epigenetic editor or components thereof is operatively linked to a promoter and/or a regulatory sequence. The term "operably linked," as used herein, means that the nucleotide sequence of interest is linked to regulatory sequence(s) in a manner that allows for expression of the nucleotide sequence. The term "regulatory sequence," as used herein, includes, but is not limited to promoters, enhancers and other expression control elements. Such regulatory sequences are well known in the art and are described, for example, in Goeddel; Gene Expression Technology: Methods in Enzymology 185, Academic Press, San Diego, CA (1990).

[0445] In some embodiments, the composition further comprises a vector that comprises the nucleic acid sequence encoding an epigenetic editor protein. In some embodiments, the vector may be an expression vector. In some embodiments, the vector is a plasmid or a viral vector.

The term "vector," as used herein, refers to a nucleic acid molecule capable of transporting another nucleic acid to which it has been linked. In some examples, a vector is an expression vector that is capable of directing the expression of nucleic acids to which they are operatively linked. Examples of expression vectors include, but are not limited to, plasmid vectors, viral vectors based on vaccinia virus, poliovirus, adenovirus, adeno-associated virus, SV40, herpes simplex virus, human immunodeficiency virus, retrovirus (e.g., Murine Leukemia Virus, spleen necrosis virus, and vectors derived from retroviruses such as Rous Sarcoma Virus, Harvey Sarcoma Virus, avian leukosis virus, a lentivirus, human immunodeficiency virus, myeloproliferative sarcoma virus, and mammary tumor virus) and other recombinant vectors.

[0446] Non-viral delivery systems include but are not limited to DNA transfection methods. Here, transfection includes a process using a non-viral vector to deliver a gene to a target cell. Typical transfection methods include electroporation, DNA biolistics, lipid-mediated transfection, compacted DNA-mediated transfection, liposomes, immunoliposomes, lipofection, cationic agent-mediated transfection, cationic facial amphiphiles (CFAs).

[0447] In some embodiments, the epigenetic editor is delivered to a host cell for transient expression, e.g., via a transient expression vector. Transient expression of a epigenetic editor may result in prolonged or permanent epigenetic modification of the target gene. For example, the epigenetic modification may be stable for at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12 weeks, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12 months or more after introduction of the epigenetic editor into the host cell. The epigenetic modification may be maintained after one or more mitotic events of the host cell. The epigenetic modification may be maintained after one or more meiotic events of the host cell. In some embodiments, the epigenetic modification is maintained across generations in offspring generated or derived from the host cell.

[0448] In some embodiments, a nucleic acid sequence encoding an epigenetic editor or components thereof is a DNA, an RNA or mRNA, or a modified nucleic acid sequence. For example, a mRNA sequence encoding an epigenetic editor fusion protein may be chemically modified, or may comprise a 5'Cap, or one or more 3' modifications.

[0449] Nucleic acids encoding epigenetic editors can be delivered directly to cells as naked DNA or RNA, for instance by means of transfection or electroporation, or can be conjugated to molecules (e.g., N-acetylgalactosamine) promoting uptake by the target cells. Nucleic acid vectors, such as the vectors can also be used. In particular embodiments, a polynucleotide, e.g. a mRNA encoding an epigenetic editor or a functional component thereof may be co-electroporated with a combination of multiple guide RNAs as described herein.

[0450] Nucleic acid vectors can comprise one or more sequences encoding a domain of a fusion protein or an epigenetic editor as described herein. A vector can also comprise a sequence encoding a signal peptide (e.g., for nuclear localization, nucleolar localization, or mitochondrial localization), associated with (e.g., inserted into or fused to) a sequence coding for a protein. As one example, a nucleic acid vectors can include a Cas9 coding sequence that includes one or more nuclear localization sequences (e.g., a nuclear localization sequence from SV40), and one or more effector domains such as repression domains.

[0451] In particular embodiments, a fusion protein, a protein domain, or a whole or a part of epigenetic editor components is encoded by a polynucleotide present in a viral vector (e.g., adeno-associated virus (AAV), AAV3, AAV3b, AAV4, AAV5, AAV6, AAV7, AAV8, AAV9, AAVrh8, AAV10, and variants thereof), or a suitable capsid protein of any viral vector. Thus, in some aspects, the disclosure relates to the viral delivery of a fusion protein. Examples of viral vectors include retroviral vectors (e.g. Maloney murine leukemia virus, MML-V), adenoviral vectors (e.g. AD100), lentiviral vectors (HIV and FIV-based vectors), herpesvirus vectors (e.g. HSV-2).

[0452] In some embodiments, an epigenetic editor protein is encoded by a polynucleotide present in an adeno-associated virus (AAV) vector. In some embodiments, the epigenetic editor protein comprises a zinc finger array in the DNA binding domain. Without wishing to be bound by any theory, epigenetic editors using zinc finger array instead of larger DNA binding domains such as Cas protein domains can be conveniently packed in viral vectors, e.g. AAV vector, given the small size of zinc fingers. In some embodiments, the polynucleotide encoding the epigenetic editor is of length of about 1000 bp, 1.1 kilobases (kb), 1.2 kb, 1.3 kb, 1.4 kb, 1.5 kb, 1.6 kb, 1.7 kb, 1.8 kb, 1.9 kb, 2.0 kb, 2.1 kb, 2.2 kb, 2.3 kb, 2.4 kb, 2.5 kb, 2.6 kb, 2.7 kb, 2.8 kb, 2.9 kb, 3.0 kb, 3.1 kb, 3.2 kb, 3.3 kb, 3.4 kb, 3.5 kb, 3.6 kb, 3.7 kb, 3.8 kb, 3.9 kb, 4.0 kb, or less. In some embodiments, The polynucleotide encoding the epigenetic editor is of length of about 2.0 kb, 2.1 kb, 2.2 kb, 2.3 kb, 2.4 kb, 2.5 kb, 2.6 kb, 2.7 kb, 2.8 kb, 2.9 kb, 3.0 kb, 3.1 kb, 3.2 kb, 3.3 kb, 3.4 kb, 3.5 kb, 3.6 kb, 3.7 kb, 3.8 kb, 3.9 kb, 4.0 kb, 4.1kb, 4.2kb, 4.3kb, 4.4kb, 4.5kb, 4.6kb, 4.7kb, 4,8 kb, 4.9kb, 5kb or less.

[0453] Any AAV serotype, e.g., human AAV serotype, can be used including, but not limited to, AAV serotype 1 (AAV1), AAV serotype 2 (AAV2), AAV serotype 3 (AAV3), AAV serotype 4 (AAV4), AAV serotype 5 (AAV5), AAV serotype 6 (AAV6), AAV serotype 7 (AAV7), AAV serotype 8 (AAV8), AAV serotype 9 (AAV9), AAV serotype 10 (AAV10), AAV serotype 11 (AAV11), AAV serotype 11 (AAV11), a variant thereof, or a shuffled variant thereof (e.g., a chimeric variant thereof). In some embodiments, an AAV variant has at least 90%, e.g., 90%,

91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV. An AAV1 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV1. An AAV2 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV2. An AAV3 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV3. An AAV4 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV4. An AAV5 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV5. An AAV6 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV6. An AAV7 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV7. An AAV8 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV8. An AAV9 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV9. An AAV10 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV10. An AAV11 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV11. An AAV12 variant can have at least 90%, e.g., 90%, 91%, 92%, 93%, 94%, 95%, 96%, 97%, 98%, 99% or more amino acid sequence identity to a wild-type AAV12.

[0454] In some instances, one or more regions of at least two different AAV serotype viruses are shuffled and reassembled to generate an AAV chimera virus. For example, a chimeric AAV can comprise inverted terminal repeats (ITRs) that are of a heterologous serotype compared to the serotype of the capsid. The resulting chimeric AAV virus can have a different antigenic reactivity or recognition, compared to its parental serotypes. In some embodiments, a chimeric variant of an AAV includes amino acid sequences from 2, 3, 4, 5, or more different AAV serotypes.

[0455] Descriptions of AAV variants and methods for generating thereof are found, e.g., in Weitzman and Linden. Chapter 1-Adeno-Associated Virus Biology in Adeno-Associated Virus: Methods and Protocols Methods in Molecular Biology, vol. 807. Snyder and Moullier, eds., Springer, 2011; Potter et al., Molecular Therapy—Methods & Clinical Development, 2014, 1,

14034; Bartel et al., Gene Therapy, 2012, 19, 694-700; Ward and Walsh, Virology, 2009, 386(2):237-248; and Li et al., Mol Ther, 2008, 16(7):1252-1260, each incorporated herein by reference in its entirety. AAV virions (e.g., viral vectors or viral particle) described herein can be transduced into cells to introduce the epigenetic editor or any component thereof into the cell. An epigenetic editor can be packaged into an AAV viral vector according to any method known to those skilled in the art. Examples of useful methods are described in McClure et al., J Vis Exp, 2001, 57:3378.

[0456] A nucleic acid vector described herein can also include any suitable number of regulatory/control elements, e.g., promoters, enhancers, introns, polyadenylation signals, Kozak consensus sequences, or internal ribosome entry sites (IRES). These elements are well known in the art.

[0457] Nucleic acid vectors according to this disclosure include recombinant viral vectors. Exemplary viral vectors are set forth herein above. Other viral vectors known in the art can also be used. In addition, viral particles can be used to deliver genome editing system components in nucleic acid and/or peptide form. For example, "empty" viral particles can be assembled to contain any suitable cargo. Viral vectors and viral particles can also be engineered to incorporate targeting ligands to alter target tissue specificity.

[0458] In addition to viral vectors, non-viral vectors can be used to deliver nucleic acids encoding genome editing systems according to the present disclosure. One important category of non-viral nucleic acid vectors are nanoparticles, which can be organic or inorganic. Nanoparticles are well known in the art. Any suitable nanoparticle design can be used to deliver genome editing system components or nucleic acids encoding such components. For instance, organic (e.g. lipid and/or polymer) nanoparticles can be suitable for use as delivery vehicles in certain embodiments of this disclosure.

Method of treatment

[0459] Also provided herein are methods for treating or preventing a condition in a subject in need thereof, the method comprising administering to the subject the epigenetic editor composition as described herein, wherein the epigenetic editor complex or protein effects an epigenetic modification of a target polynucleotide in a target gene associated with a disease, condition or disorder in a subject and modulates expression of the target, thereby treating or preventing the disease, condition or disorder.

[0460] Epigenetic modifications effected by the epigenetic editors described herein are sequence specific. In some embodiments, the modification is at a specific site of the target polynucleotide. In some embodiments, the modification is at a specific allele of the target gene. Accordingly, the

epigenetic modification may result in modulated expression, for example, reduced or increased expression, of one copy of a target gene harboring a specific allele, and not the other copy of the target gene. In some embodiments, the specific allele is associated with a disease, condition, or disorder.

[0461] In some embodiments, the epigenetic editor reduces expression of a target gene associated with a disease, condition or disorder.

[0462] Epigenetic editors described herein may be administered to a subject in need thereof, in a therapeutically effective amount, to treat a disease, condition or disorder.

[0463] In another aspect, provided herein is a method for treating or preventing a condition in a subject in need thereof, the method comprising administering to the subject the epigenetic editing complex, vectors, nucleic acids, proteins, or compositions as provided herein, wherein the nucleic acid binding domain of the epigenetic editor directs the effector domain to generate an epigenetic modification in a target polynucleotide sequence in a cell of the subject, thereby modulating expression of the target gene and treating or preventing the condition.

[0464] In some embodiments, the modification reduces expression of a functional protein encoded by the target gene in the subject.

[0465] A patient who is being treated for a condition, a disease or a disorder is one who a medical practitioner has diagnosed as having such a condition. Diagnosis may be by any suitable means. Diagnosis and monitoring may involve, for example, detecting the presence of diseased, dying or dead cells in a biological sample (e.g., tissue biopsy, blood test, or urine test), detecting the presence of plaques, detecting the level of a surrogate marker in a biological sample, or detecting symptoms associated with a condition. A patient in whom the development of a condition is being prevented may or may not have received such a diagnosis. One in the art will understand that these patients may have been subjected to the same standard tests as described above or may have been identified, without examination, as one at high risk due to the presence of one or more risk factors (e.g., family history or genetic predisposition).

[0466] A subject may have a disease, a symptom of the disease, or a predisposition toward the disease, with the purpose to cure, heal, alleviate, relieve, alter, remedy, ameliorate, improve, or affect the disease, the symptom of the disease, or the predisposition toward the disease. In some embodiments, the subject has hypercholesterolemia. In some embodiments, the subject has atherosclerotic vascular disease. In some embodiments, the subject has hypertriglyceridemia. In some embodiments, the subject has diabetes. In some embodiments, the subject is a mammal. In some embodiments, the subject is a non-human primate. In some embodiments, the subject is human. Alleviating a disease includes delaying the development or progression of the disease,

or reducing disease severity. Alleviating the disease does not necessarily require curative results.

[0467] As used therein, "delaying" the development of a disease means to defer, hinder, slow, retard, stabilize, and/or postpone progression of the disease. This delay can be of varying lengths of time, depending on the history of the disease and/or individuals being treated. A method that "delays" or alleviates the development of a disease, or delays the onset of the disease, is a method that reduces probability of developing one or more symptoms of the disease in a given time frame and/or reduces extent of the symptoms in a given time frame, when compared to not using the method. Such comparisons are typically based on clinical studies, using a number of subjects sufficient to give a statistically significant result.

[0468] "Development" or "progression" of a disease means initial manifestations and/or ensuing progression of the disease. Development of the disease can be detectable and assessed using standard clinical techniques as well known in the art. However, development also refers to progression that may be undetectable. For purpose of this disclosure, development or progression refers to the biological course of the symptoms. "Development" includes occurrence, recurrence, and onset.

[0469] As used herein "onset" or "occurrence" of a disease includes initial onset and/or recurrence. Conventional methods, known to those of ordinary skill in the art of medicine, can be used to administer the isolated polypeptide or pharmaceutical composition to the subject, depending upon the type of disease to be treated or the site of the disease. This composition can also be administered via other conventional routes, e.g., administered orally, parenterally, by inhalation spray, topically, rectally, nasally, buccally, vaginally or via an implanted reservoir.

[0470] The therapeutic methods of the disclosure may be carried out on subjects displaying pathology resulting from a disease or a condition, subjects suspected of displaying pathology resulting from a disease or a condition, and subjects at risk of displaying pathology resulting from a disease or a condition. For example, subjects that have a genetic predisposition to a disease or a condition can be treated prophylactically. Subjects exhibiting symptoms associated with a condition, a disease or a disorder may be treated to decrease the symptoms or to slow down or prevent further progression of the symptoms. The physical changes associated with the increasing severity of a disease or a condition are shown herein to be progressive. Thus, in embodiments of the disclosure, subjects exhibiting mild signs of the pathology associated with a condition or a disease may be treated to improve the symptoms and/or prevent further progression of the symptoms.

[0471] The dosage and frequency (single or multiple doses) administered to a mammal can vary depending upon a variety of factors, for example, whether the mammal suffers from another disease, and its route of administration; size, age, sex, health, body weight, body mass index, and diet of the recipient; nature and extent of symptoms of the disease being treated, kind of concurrent treatment, complications from the disease being treated or other health-related problems. Adjustment and manipulation of established dosages (e.g., frequency and duration) are well within the ability of those skilled in the art. The treatment, such as those disclosed herein, can be administered to the subject on a daily, twice daily, biweekly, monthly or any applicable basis that is therapeutically effective. In embodiments, the treatment is only on an as-needed basis, e.g., upon appearance of signs or symptoms of a condition or a disease.

[0472] Toxicity and therapeutic efficacy of the compositions of the disclosure can be determined by standard pharmaceutical procedures in cell cultures or experimental animals, e.g., for determining the LD50 (the dose lethal to 50% of the population) and the ED50 (the dose therapeutically effective in 50% of the population). The dose ratio between toxic and therapeutic effects (the ratio LD50/ED50) is the therapeutic index. Agents that exhibit high therapeutic indices are preferred. The dosage of agents lies preferably within a range of circulating concentrations that include the ED50 with little or no toxicity. While agents that exhibit toxic side effects may be used, care should be taken to design a delivery system that targets such agents to the site of affected tissue in order to minimize potential damage to uninfected cells and, thereby, reduce side effects.

[0473] The skilled artisan will appreciate that certain factors may influence the dosage and frequency of administration required to effectively treat a subject, including but not limited to the severity of the disease or disorder, previous treatments, the general characteristics of the subject including health, sex, weight and/or age of the subject, and other diseases present. Moreover, treatment of a subject with a therapeutically effective amount of the compositions can include a single treatment or, preferably, can include a series of treatments. It will also be appreciated that the effective dosage of the composition of the disclosure used for treatment may increase or decrease over the course of a particular treatment. Changes in dosage may result and become apparent from the results of diagnostic assays as described herein. The therapeutically-effective dosage will generally be dependent on the patient's status at the time of administration. The precise amount can be determined by routine experimentation but may ultimately lie with the judgment of the clinician, for example, by monitoring the patient for signs of disease and adjusting the treatment accordingly.

[0474] Frequency of administration may be determined and adjusted over the course of therapy, and is generally, but not necessarily, based on treatment and/or suppression and/or amelioration and/or delay of a disease. Alternatively, sustained continuous release formulations of a polypeptide or a polynucleotide may be appropriate. Various formulations and devices for achieving sustained release are known in the art. In some embodiments, dosage is daily, every other day, every three days, every four days, every five days, or every six days. In some embodiments, dosing frequency is once every week, every 2 weeks, every 4 weeks, every 5 weeks, every 6 weeks, every 7 weeks, every 8 weeks, every 9 weeks, or every 10 weeks; or once every month, every 2 months, or every 3 months, or longer. The progress of this therapy is easily monitored by conventional techniques and assays.

[0475] The dosing regimen (including a composition disclosed herein) can vary over time. In some embodiments, for an adult subject of normal weight, doses ranging from about 0.01 to 1000 mg/kg may be administered. In some embodiments, the dose is between 1 to 200 mg. The particular dosage regimen, i.e., dose, timing and repetition, will depend on the particular subject and that subject's medical history, as well as the properties of the polypeptide or the polynucleotide (such as the half-life of the polypeptide or the polynucleotide, and other considerations well known in the art).

[0476] For the purpose of the present disclosure, the appropriate therapeutic dosage of a composition as described herein will depend on the specific agent (or compositions thereof) employed, the formulation and route of administration, the type and severity of the disease, whether the polypeptide or the polynucleotide is administered for preventive or therapeutic purposes, previous therapy, the subject's clinical history and response to the antagonist, and the discretion of the attending physician. Typically, the clinician will administer a polypeptide until a dosage is reached that achieves the desired result.

[0477] Administration of one or more compositions can be continuous or intermittent, depending, for example, upon the recipient's physiological condition, whether the purpose of the administration is therapeutic or prophylactic, and other factors known to skilled practitioners. The administration of a composition may be essentially continuous over a preselected period of time or may be in a series of spaced dose, e.g., either before, during, or after developing a disease.

[0478] The methods and compositions of the disclosure described herein including embodiments thereof can be administered with one or more additional therapeutic regimens or agents or treatments, which can be co-administered to the mammal. By “co-administering” is meant administering one or more additional therapeutic regimens or agents or treatments and the

composition of the disclosure sufficiently close in time to enhance the effect of one or more additional therapeutic agents, or vice versa. In this regard, the composition of the disclosure described herein can be administered simultaneously with one or more additional therapeutic regimens or agents or treatments, at a different time, or on an entirely different therapeutic schedule (e.g., the first treatment can be daily, while the additional treatment is weekly). For example, in embodiments, the secondary therapeutic regimens or agents or treatments are administered simultaneously, prior to, or subsequent to the composition of the disclosure.

Pharmaceutical compositions

[0479] In some aspects, provided herein, is a pharmaceutical composition for epigenetic modification comprising an epigenetic editor or epigenetic editor complex described herein, or one or more nucleic acid sequences encoding components of the epigenetic editor complex, e.g., nucleic acids encoding an epigenetic editor fusion protein and/or a guide RNA, and a pharmaceutically acceptable carrier. The composition for epigenetic modification described herein can be formulated into pharmaceutical compositions. Pharmaceutical compositions are formulated in a conventional manner using one or more pharmaceutically acceptable inactive ingredients that facilitate processing of the active compounds into preparations that can be used pharmaceutically. Suitable formulations for use in the present disclosure and methods of delivery are generally well known in the art. Proper formulation is dependent upon the route of administration chosen. A summary of pharmaceutical compositions described herein can be found, for example, in Remington: The Science and Practice of Pharmacy, Nineteenth Ed (Easton, Pa.: Mack Publishing Company, 1995); Hoover, John E., Remington's Pharmaceutical Sciences, Mack Publishing Co., Easton, Pennsylvania 1975; Liberman, H.A. and Lachman, L., Eds., Pharmaceutical Dosage Forms, Marcel Decker, New York, N.Y., 1980; and Pharmaceutical Dosage Forms and Drug Delivery Systems, Seventh Ed. (Lippincott Williams & Wilkins1999), herein incorporated by reference for such disclosure.

[0480] A pharmaceutical composition can be a mixture of an epigenetic editor or nucleic acids encoding same as described herein and one or more other chemical components (i.e., pharmaceutically acceptable ingredients), such as carriers, excipients, binders, filling agents, suspending agents, flavoring agents, sweetening agents, disintegrating agents, dispersing agents, surfactants, lubricants, colorants, diluents, solubilizers, moistening agents, plasticizers, stabilizers, penetration enhancers, wetting agents, anti–foaming agents, antioxidants, preservatives, or one or more combination thereof. The pharmaceutical composition facilitates administration of the epigenetic editor, for example, a nucleic acid encoding a zinc finger-

epigenetic effector fusion protein or a Cas9-epigenetic effector fusion protein and a gRNA or sgRNA described herein to an organism or a subject in need thereof.

[0481] The pharmaceutical compositions of the present disclosure can be administered to a subject using any suitable methods known in the art. The pharmaceutical compositions described herein can be administered to the subject in a variety of ways, including parenterally, intravenously, intradermally, intramuscularly, colonically, rectally, or intraperitoneally. In some embodiments, the pharmaceutical compositions can be administered by intraperitoneal injection, intramuscular injection, subcutaneous injection, or intravenous injection of the subject. In some embodiments, the pharmaceutical compositions can be administered parenterally, intravenously, intramuscularly, or orally.

[0482] For administration by inhalation, the adenovirus described herein can be formulated for use as an aerosol, a mist, or a powder. For buccal or sublingual administration, the pharmaceutical compositions may be formulated in the form of tablets, lozenges, or gels formulated in a conventional manner. In some embodiments, the adenovirus described herein can be prepared as transdermal dosage forms. In some embodiments, the adenovirus described herein can be formulated into a pharmaceutical composition suitable for intramuscular, subcutaneous, or intravenous injection. In some embodiments, the adenovirus described herein can be administered topically and can be formulated into a variety of topically administrable compositions, such as solutions, suspensions, lotions, gels, pastes, medicated sticks, balms, creams, or ointments. In some embodiments, the adenovirus described herein can be formulated in rectal compositions such as enemas, rectal gels, rectal foams, rectal aerosols, suppositories, jelly suppositories, or retention enemas. In some embodiments, the adenovirus described herein can be formulated for oral administration such as a tablet, a capsule, or liquid in the form of aqueous suspensions or solutions selected from the group including, but not limited to, aqueous oral dispersions, emulsions, solutions, elixirs, gels, and syrups.

[0483] In some embodiments, the pharmaceutical composition for epigenetic modification comprising an epigenetic editor described herein or nucleic acid sequences encoding the same further comprises a therapeutic agent. The additional therapeutic agent may modulate different aspects of the disease, disorder, or condition being treated and provide a greater overall benefit than administration of either the replication competent recombinant adenovirus or the therapeutic agent alone. Therapeutic agents include, but are not limited to, a chemotherapeutic agent, a radiotherapeutic agent, a hormonal therapeutic agent, and/or an immunotherapeutic agent. In some embodiments, the therapeutic agent may be a radiotherapeutic agent. In some embodiments, the therapeutic agent may be a hormonal therapeutic agent. In some

embodiments, the therapeutic agent may be an immunotherapeutic agent. In some embodiments, the therapeutic agent is a chemotherapeutic agent. Preparation and dosing schedules for additional therapeutic agents can be used according to manufacturers' instructions or as determined empirically by a skilled practitioner. For example, preparation and dosing schedules for chemotherapy are also described in The Chemotherapy Source Book, 4th Edition, 2008, M. C. Perry, Editor, Lippincott, Williams & Wilkins, Philadelphia, PA.

[0484] The subjects that can be treated with epigenetic modification compositions can be any subject with a disease or a condition. For example, the subject may be a eukaryotic subject, such as an animal. In some embodiments, the subject is a mammal, e.g., human. In some embodiments, the subject is a human. In some embodiments, the subject is a non–human animal. In some embodiments, the subject is a fetus, an embryo, or a child. In some embodiments, the subject is a non–human primate such as chimpanzee, and other apes and monkey species; farm animals such as cattle, horses, sheep, goats, pigs; domestic animals such as rabbits, dogs, and cats; laboratory animals including rodents, such as rats, mice, and guinea pigs, and the like.

[0485] In some embodiments, the subject is prenatal (e.g., a fetus), a child (e.g., a neonate, an infant, a toddler, a preadolescent), an adolescent, a pubescent, or an adult (e.g., an early adult, a middle-aged adult, a senior citizen). The human subject can be between about 0 month and about 120 years old, or older. The human subject can be between about 0 and about 12 months old; for example, about 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, or 12 months old. The human subject can be between about 0 and 12 years old; for example, between about 0 and 30 days old; between about 1 month and 12 months old; between about 1 year and 3 years old; between about 4 years and 5 years old; between about 4 years and 12 years old; about 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, or 12 years old. The human subject can be between about 13 years and 19 years old; for example, about 13, 14, 15, 16, 17, 18, or 19 years old. The human subject can be between about 20 and about 39 years old; for example, about 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, or 39 years old. The human subject can be between about 40 to about 59 years old; for example, about 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, or 59 years old. The human subject can be greater than 59 years old; for example, about 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, or 120 years old. The human subjects can include male subjects and/or female subjects.

[0486] In another aspect, provided herein is a lipid nanoparticle (LNP) comprising the composition as provided herein. As used herein, a "lipid nanoparticle (LNP) composition" or a

"nanoparticle composition" is a composition comprising one or more described lipids. LNP compositions are typically sized on the order of micrometers or smaller and may include a lipid bilayer. Nanoparticle compositions encompass lipid nanoparticles (LNPs), liposomes (e.g., lipid vesicles), and lipoplexes. In some embodiments, a LNP refers to any particle that has a diameter of less than 1000 nm, 500 nm, 250 nm, 200 nm, 150 nm, 100 nm, 75 nm, 50 nm, or 25 nm. In some embodiments, a nanoparticle may range in size from 1-1000 nm, 1-500 nm, 1-250 nm, 25-200 nm, 25-100 nm, 35-75 nm, or 25-60 nm.

[0487] In some embodiments, an LNP may be made from cationic, anionic, or neutral lipids. In some embodiments, an LNP may comprise neutral lipids, such as the fusogenic phospholipid 1,2-Dioleoyl-sn-glycero-3-phosphoethanolamine (DOPE) or the membrane component cholesterol, as helper lipids to enhance transfection activity and nanoparticle stability. In some embodiments, an LNP may comprise hydrophobic lipids, hydrophilic lipids, or both hydrophobic and hydrophilic lipids. Any lipid or combination of lipids that are known in the art can be used to produce an LNP. Examples of lipids used to produce LNPs include, but are not limited to DOTMA (N[1-(2,3-dioleyloxy)propyl]-N,N,N-trimethylammonium chloride), DOSPA (N,N-dimethyl-N-([2-sperminecarboxamido]ethyl)-2,3-bis(dioleyloxy)-1-propaniminium pentahydrochloride), DOTAP (1,2-Dioleoyl-3-trimethylammonium propane), DMRIE (N-(2-hydroxyethyl)- N,N-dimethyl-2,3-bis(tetradecyloxy-1-propanaminiumbromide), DC-cholesterol (3β-[N-(N',N'-dimethylaminoethane)-carbamoyl]cholesterol), DOTAP-cholesterol, GAP-DMORIE-DPyPE, and GL67A-DOPE-DMPE (,2-Bis(dimethylphosphino)ethane)-polyethylene glycol (PEG). Examples of cationic lipids include, but are not limited to, 98N12-5, C12-200, DLin-KC2-DMA (KC2), DLin-MC3 -DMA (MC3), XTC, MD1, and 7C1. Examples of neutral lipids include, but are not limited to, DPSC, DPPC (Dipalmitoylphosphatidylcholine), POPC (1-palmitoyl-2-oleoyl-sn-glycero-3-phosphocholine), DOPE, and SM (sphingomyelin). Examples of PEG-modified lipids include, but are not limited to, PEG-DMG (Dimyristoyl glycerol), PEG-CerC14, and PEG-CerC20. In some embodiments, the lipids may be combined in any number of molar ratios to produce a LNP. In some embodiments, the polynucleotide may be combined with lipid(s) in a wide range of molar ratios to produce an LNP.

[0488] Also disclosed herein, in certain embodiments, are kits and articles of manufacture for use with one or more methods described herein. Such kits include a carrier, package, or container that is compartmentalized to receive one or more containers such as vials, tubes, and the like, each of the container(s) comprising one of the separate elements to be used in a method described herein. Suitable containers include, for example, bottles, vials, syringes, and test

tubes. In one embodiment, the containers are formed from a variety of materials such as glass or plastic.

[0489] The articles of manufacture provided herein contain packaging materials. Examples of pharmaceutical packaging materials include, but are not limited to, blister packs, bottles, tubes, bags, containers, and any packaging material suitable for a selected formulation and intended mode of administration and treatment.

[0490] For example, the container(s) include the composition of the disclosure, and optionally in addition with therapeutic regimens or agents disclosed herein. Such kits optionally include an identifying description or label or instructions relating to its use in the methods described herein.

[0491] A kit typically includes labels listing contents and/or instructions for use, and package inserts with instructions for use. A set of instructions will also typically be included.

[0492] In embodiments, a label is on or associated with the container. In one embodiment, a label is on a container when letters, numbers or other characters forming the label are attached, molded or etched into the container itself; a label is associated with a container when it is present within a receptacle or carrier that also holds the container, e.g., as a package insert. In one embodiment, a label is used to indicate that the contents are to be used for a specific therapeutic application. The label also indicates directions for use of the contents, such as in the methods described herein.

EXAMPLES

[0493] The following examples are included for illustrative purposes only and are not intended to limit the scope of the disclosure.

[0494] Example 1: Zinc finger design

[0495] Zinc finger binding sites were selected based on the availability of zinc finger modules, their location and orientation in the target gene of interest. For example, in a sequence comprising the EF1alpha promoter driving expression of GFP, an exemplary sequence contains the 3' 200 base pairs of the EF1alpha promoter, the 23 base pairs between the promoter and the GFP start codon and the 5' 177 base pairs of the GFP coding sequence. Exemplary binding sites for 6-finger zinc finger proteins are in "Target Site Table" and are shown in bold, or in italics when the binding site overlaps with another binding site in SEQ ID NO.: 695, shown below:

GTACGTCGTCTT**TAGGTTGGGGGGAGGGGTT**TTATGCGATGGAGTTTCCCCACAC
TGA**GTGGGTGGAGACTGAAGTT**AGGCCAGCTTGGCACTTGATGTAATTCTCCTTG
GAATTTGCCCTTTTTGAGTTTGGATCTTGGTTCATTCTCAAGCCTCAGACAGTGGTTC
AAAGTTTTTTTCTTCCATTTCAGGTGTCGTGACGCTAGCGCTACCGGTCGCCACCAT

GGTGAGCAAGGGCGCCGAGCTG**TTCACCGGCATCGTGCCCA**TCCTGATCGAGCTG AATGGCGATGTGAATGGCCACAAGTTCAGCGTGAGC**GGC*GAGGGCGAGGGCGAT***G *CC*ACCTACGGCAAGCTGACCCTGAAG**TTCATC*TGCACCACCGGC***AAGCTGC*CTGTGC CCTGGCCC

Table 7: Target Site Table

Target Site	Sequence
GFP-1	SEQ ID NO.: 696
GFP-2	SEQ ID NO.: 697
GFP-3	SEQ ID NO.: 698
GFP-4	SEQ ID NO.: 699
GFP-5	SEQ ID NO.: 700
GFP-6	SEQ ID NO.: 701
GFP-7	SEQ ID NO.: 702

Zinc finger sequences were designed for binding of the above described target site. Exemplary Zinc finger sequences are as follows:

SRPGERPFQCRICMRNFS[F1]HTRTHTGEKPFQCRICMRNFS[F2]HLRTH[linker1]FQCRIC MRNFS[F3]HTRTHTGEKPFQCRICMRNFS[F4]HLRTH[linker2]FQCRICMRNFS[F5]HTRT HTGEKPFQCRICMRNFS[F6]HLRTHLRGS (SEQ ID NO.: 703)

Where zinc finger proteins for a given target site have the following linkers:

Table 8: Linkers for a Given Target Site

Target Site	Sequence	Linker 1	Linker 2
GFP-1	SEQ ID NO.: 696	SEQ ID NO.: 704	SEQ ID NO.: 705
GFP-2	SEQ ID NO.: 697	SEQ ID NO.: 705	SEQ ID NO.: 704
GFP-3	SEQ ID NO.: 698	SEQ ID NO.: 704	SEQ ID NO.: 705
GFP-4	SEQ ID NO.: 699	SEQ ID NO.: 704	SEQ ID NO.: 704
GFP-5	SEQ ID NO.: 700	SEQ ID NO.: 704	SEQ ID NO.: 704
GFP-6	SEQ ID NO.: 701	SEQ ID NO.: 704	SEQ ID NO.: 704
GFP-7	SEQ ID NO.: 702	SEQ ID NO.: 704	SEQ ID NO.: 705

and where recognition helices for a given target site may be selected from the following SEQ ID NO.: 716-961:

Table 9: Recognition Helices for a Given Target Site

Target Site	Zinc Finger Protein Name	F1	F2	F3	F4	F5	F6
GFP-1	GFP1-ZF1	HKSSLTR	RTEHLAR	QSAHLKR	RTEHLAR	HKSSLTR	RPESLAP
	GFP1-ZF2	HKSSLTR	RTEHLAR	TSAHLAR	RREHLVR	HKSSLTR	RPESLAP
	GFP1-ZF3	IKAILTR	RREHLVR	QSAHLKR	RTEHLAR	HKSSLTR	RPESLAP

Target Site	Zinc Finger Protein Name	F1	F2	F3	F4	F5	F6
	GFP1-ZF4	IKAILTR	RREHLVR	TSAHLAR	RREHLVR	HKSSLTR	RPESLAP
GFP-2	GFP2-ZF1	TSTLLNR	QQTNLTR	DEANLRR	QSAHLKR	IPNKLAR	RREVLEN
	GFP2-ZF2	TSTLLNR	QQTNLTR	DEANLRR	QSAHLKR	EAHHLSR	RKDALHV
	GFP2-ZF3	TSTLLNR	QQTNLTR	DRGNLTR	QGGHLKR	IPNKLAR	RREVLEN
	GFP2-ZF4	TSTLLNR	QQTNLTR	DRGNLTR	QGGHLKR	EAHHLSR	RKDALHV
	GFP2-ZF5	HKSSLTR	QTNNLGR	DEANLRR	QSAHLKR	IPNKLAR	RREVLEN
	GFP2-ZF6	HKSSLTR	QTNNLGR	DEANLRR	QSAHLKR	EAHHLSR	RKDALHV
	GFP2-ZF7	HKSSLTR	QTNNLGR	DRGNLTR	QGGHLKR	IPNKLAR	RREVLEN
	GFP2-ZF8	HKSSLTR	QTNNLGR	DRGNLTR	QGGHLKR	EAHHLSR	RKDALHV
GFP-3	GFP3-ZF1	QQTNLTR	IRHHLKR	DSSVLRR	LSTNLTR	QSTTLKR	RSDHLSL
	GFP3-ZF2	QQTNLTR	IRHHLKR	DGSTLNR	VRHNLTR	QSTTLKR	RSDHLSL
	GFP3-ZF3	RKPNLLR	EAHHLSR	DSSVLRR	LSTNLTR	QSTTLKR	RSDHLSL
	GFP3-ZF4	RKPNLLR	EAHHLSR	DGSTLNR	VRHNLTR	QSTTLKR	RSDHLSL
GFP-4	GFP4-ZF1	VRHNLTR	ESGHLKR	RQDNLGR	KNHSLNN	RQDNLGR	KNHSLNN
GFP-5	GFP5-ZF1	DSSVLRR	LSTNLTR	LKEHLTR	RVDNLPR	LKEHLTR	RVDNLPR
	GFP5-ZF2	DSSVLRR	LSTNLTR	LKEHLTR	RVDNLPR	SPSKLVR	RQDNLGR
	GFP5-ZF3	DSSVLRR	LSTNLTR	SPSKLVR	RQDNLGR	LKEHLTR	RVDNLPR
	GFP5-ZF4	DSSVLRR	LSTNLTR	SPSKLVR	RQDNLGR	SPSKLVR	RQDNLGR
	GFP5-ZF5	DGSTLNR	VRHNLTR	LKEHLTR	RVDNLPR	LKEHLTR	RVDNLPR
	GFP5-ZF6	DGSTLNR	VRHNLTR	LKEHLTR	RVDNLPR	SPSKLVR	RQDNLGR
	GFP5-ZF7	DGSTLNR	VRHNLTR	SPSKLVR	RQDNLGR	LKEHLTR	RVDNLPR
	GFP5-ZF8	DGSTLNR	VRHNLTR	SPSKLVR	RQDNLGR	SPSKLVR	RQDNLGR
GFP-6	GFP6-ZF1	RKPNLLR	VRHNLTR	DKAQLGR	EAHHLSR	RQSRLQR	KGDHLRR
	GFP6-ZF2	RKPNLLR	VRHNLTR	DKAQLGR	EAHHLSR	EAHHLSR	DPSNLRR
	GFP6-ZF3	RKPNLLR	VRHNLTR	QSTTLKR	VDHHLRR	RQSRLQR	KGDHLRR
	GFP6-ZF4	RKPNLLR	VRHNLTR	QSTTLKR	VDHHLRR	EAHHLSR	DPSNLRR
	GFP6-ZF5	QQTNLTR	VGSNLTR	DKAQLGR	EAHHLSR	RQSRLQR	KGDHLRR
	GFP6-ZF6	QQTNLTR	VGSNLTR	DKAQLGR	EAHHLSR	EAHHLSR	DPSNLRR
	GFP6-ZF7	QQTNLTR	VGSNLTR	QSTTLKR	VDHHLRR	RQSRLQR	KGDHLRR
	GFP6-ZF8	QQTNLTR	VGSNLTR	QSTTLKR	VDHHLRR	EAHHLSR	DPSNLRR
GFP-7	GFP7-ZF1	QSTTLKR	VDHHLRR	EAHHLSR	DPSNLRR	QRSDLTR	QGGTLRR
	GFP7-ZF2	QSTTLKR	VDHHLRR	EAHHLSR	DPSNLRR	TKQILGR	QSTTLKR
	GFP7-ZF3	QSTTLKR	VDHHLRR	RQSRLQR	DSSVLRR	QRSDLTR	QGGTLRR
	GFP7-ZF4	QSTTLKR	VDHHLRR	RQSRLQR	DSSVLRR	TKQILGR	QSTTLKR
	GFP7-ZF5	DKAQLGR	EAHHLSR	EAHHLSR	DPSNLRR	QRSDLTR	QGGTLRR
	GFP7-ZF6	DKAQLGR	EAHHLSR	EAHHLSR	DPSNLRR	TKQILGR	QSTTLKR

Target Site	Zinc Finger Protein Name	F1	F2	F3	F4	F5	F6
	GFP7-ZF7	DKAQLGR	EAHHLSR	RQSRLQR	DSSVLRR	QRSDLTR	QGGTLRR
	GFP7-ZF8	DKAQLGR	EAHHLSR	RQSRLQR	DSSVLRR	TKQILGR	QSTTLKR

[0496] Example 2: epigenetic editor sequences.

[0497] Amino acid sequences of exemplary epigenetic editors are provided below.

Exemplary fusion protein DNMT3A-3L-ZF-KRAB (SEQ ID NO.: 978) where zinc finger is GFP1-ZF1:

MAPKKKRK*MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQVDRYIASE VCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDLVIGGSPCNDLSIVNPARKGLYEG TGRLFFEFYRLLHDARPKEGDDRPFFWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAA HRARYFWGNLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGKDQHFPV FMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLGRSWSVPVIRHLFAPLKEYFACV* **SSGNSNANSRGPSFSSGLVPLSLRGSHMGPMEIYKTVSAWKRQPVRVLSLFRNIDK VLKSLGFLESGSGSGGGTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDR CPGWYMFQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVTL QDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSKLDAPKVDLL VKNCLLPLREYFKYFSQNSLPL**SGGGGSGGGGSVGIHGVPSRPGERPFQCRICMRNFS HKSSLTRHTRTHTGEKPFQCRICMRNFSRTEHLARHLRTHTGSQKPFQCRICMRNFSQS AHLKRHTRTHTGEKPFQCRICMRNFSRTEHLARHLRTHTGGGGSQKPFQCRICMRNFSH KSSLTRHTRTHTGEKPFQCRICMRNFSRPESLAPHLRTHLRGSGGGSMDAKSLTAWSRT LVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLGYQLTKPDVILRLEKGE EPWLVEREIHQETHPDSETAFEIKSSV(italics: DNMT3A; Bold: DNMT3L; underline: KRAB)

[0498] Example 3: guide RNA design.

[0499] Cas9 protospacers are chosen based on homology from sequences that perfectly match or nearly perfectly match spacer sequences in target DNA sequences and predicted by the MIT Specificity Score (calculated by http://crispor.tefor.net/).

[0500] gRNA protospacer sequences that would permit epigenetic editors containing a Streptococcus pyogenes Cas9, or another Cas that can use the NGG PAM, to recognize the protospacer sequences identified throughout the target gene. gRNAs containing spacers of 20nts and a total length of 100nts are synthesized. gRNAs are co-transfected with mRNA encoding the Cas9 epigenetic editor fusion protein into primary human hepatocytes via MessengerMax

reagent (Lipofectamine). After transfection, genomic DNA from the hepatocytes is harvested, and transcript expression level of the target gene was assessed by qRT-PCR.

[0501] Example 4: epigenetic editor mediated repression of target gene

[0502] Candidate zinc fingers are screened as described using ZiFit (http://bindr.gdcb.iastate.edu/ZiFiT/). Human K562 cells are cultured in RPMI 1640 medium (Gibco) with 10% HI-FBS (Gibco), 1% Glutamax (Gibco) and 1% Pen/Strep (Gibco), and are transfected with plasmids encoding various KRAB-ZF-Dnmt3A-Dnmt3L fusion proteins by nucleofecting 1×10^6 dividing cells with 10 μg of DNA in 100 μl of Kit V solution (Lonza) using program T-016 on the Nucleofector 2b Device (Lonza). Nucleofected cells are incubated in 6-well plates at 37° C for 4 days following nucleofection. Genomic DNA and total RNA are harvested 4 days post-transfection. Genomic DNA is used for methylation analysis. Total RNA is extracted and the expression of the target gene and two reference genes (ATP5b and RPL38) are monitored using real-time RT-qPCR.

[0503] Methylation state determination: Bisulfite DNA sequencing of the target gene locus from these transfected cell populations are performed as follows. Genomic DNA is isolated from transfected cells using the Qiagen Blood Mini kit. 200-1000 ng of genomic DNA is bisulfite treated using either the EZ DNA Methylation Kit (Zymo), EZ DNA Methylation-Lightning Kit (Zymo), or Cells-to-CpG Bisulfite Conversion Kit (Applied Biosystems) following recommended protocols. PCR amplification of Bis-DNA is performed using Pyromark PCR kit (Qiagen). Illumina adapters and barcodes are added by PCR with Phusion High-Fidelity PCR enzyme (NEB) and amplicons were sequenced on an Illumina MiSeq system. Total RNA is isolated from the same cells with the PureLink RNA mini kit (Ambion) according to manufacturer's instructions. Reverse transcription is performed with the Superscriptlll RT kit (Invitrogen) and Taqman assays were run on an Applied Biosystems 7500Fast Real Time PCR machine.

[0504] Testing Repression Domains: To test the functionality of candidate repression domains, the domain is fused to a DNA-binding domain for testing in human cells. The effector domain, identified and extracted from the full protein sequence may be fused to the N-terminal or C-terminal end of any DNA-binding domain, using a variety of linkers. For example, a repressor domain may be fused to Cas9. This fusion protein is then co-delivered into cells, along with a gRNA, using standard cell culture techniques. This may include plasmid transfection or electroporation, mRNA transfection or electroporation, or viral transduction. Initial testing of effector domains can easily be performed in reporter cell lines in which a fluorescent marker has been integrated to enable easy FACS-based readout. Alternatively, endogenous genes can be

targeted. Genes encoding cell surface markers can be easily quantified by flow cytometry and expression of any gene target can be quantified by standard molecular biology techniques such as RT-qPCR, ddPCR, Western blot, etc. To test candidate repression domains, decreased expression of the target gene is quantified by these methods. Truncations and mutations can be introduced into the effector domain to generate multiple variants for testing.

[0505] Testing Activation Domains: To test the functionality of candidate activation domains, the domain is fused to a DNA-binding domain for testing in human cells. The effector domain, identified and extracted from the full protein sequence may be fused to the N-terminal or C-terminal end of any DNA-binding domain, using a variety of linkers. For example, an activation domain may be fused to Cas9. This fusion protein is then co-delivered into cells, along with a gRNA, using standard cell culture techniques. This may include plasmid transfection or electroporation, mRNA transfection or electroporation, or viral transduction. Initial testing of effector domains can easily be performed in reporter cell lines in which a fluorescent marker has been integrated to enable easy FACS-based readout. Alternatively, endogenous genes can be targeted. Genes encoding cell surface markers can be easily quantified by flow cytometry and expression of any gene target can be quantified by standard molecular biology techniques such as RT-qPCR, ddPCR, Western blot, etc. To test candidate activation domains, increased expression of the target gene is quantified by these methods. Truncations and mutations can be introduced into the effector domain to generate multiple variants for testing.

[0506] Testing DNA methyltransferase domains: To test the functionality of candidate DNA methyltransferase domains, the domain is fused to a DNA-binding domain for testing in human cells. The effector domain, identified and extracted from the full protein sequence may be fused to the N-terminal or C-terminal end of any DNA-binding domain, using a variety of linkers. For example, a DNA methyltransferase domain may be fused to Cas9. This fusion protein is then co-delivered into cells, along with a gRNA, using standard cell culture techniques. This may include plasmid transfection or electroporation, mRNA transfection or electroporation, or viral transduction. Because DNA methylation is expected to reduce target gene expression, this may be assayed by standard techniques such as RT-qPCR, staining for cell surface marker and quantifying by flow cytometry, ddPCR and Western blotting. Additionally, direct readout of DNA methylation is obtained through bisulfite sequencing. In this method, bisulfite treatment of DNA converts cytosine residues to uracil but leaves 5-methylcytosine residues unaffected. Standard Sanger sequencing or next-generation sequencing can then be performed to determine the rate of methylation at CpG dinucleotides.

Testing DNA demethylation domains: To test the functionality of candidate domains for removing DNA methylation, the domain is fused to a DNA-binding domain for testing in human cells. The effector domain, identified and extracted from the full protein sequence may be fused to the N-terminal or C-terminal end of any DNA-binding domain, using a variety of linkers. For example, a domain may be fused to Cas9. This fusion protein is then co-delivered into cells, along with a gRNA, using standard cell culture techniques. This may include plasmid transfection or electroporation, mRNA transfection or electroporation, or viral transduction. Because removal of DNA methylation marks at CpG dinucleotides is expected to increase target gene expression, this may be assayed by standard techniques such as RT-qPCR, staining for cell surface marker and quantifying by flow cytometry, ddPCR and Western blotting. Additionally, direct readout of DNA methylation is obtained through bisulfite sequencing. In this method, bisulfite treatment of DNA converts cytosine residues to uracil but leaves 5-methylcytosine residues unaffected. Standard Sanger sequencing or next-generation sequencing can then be performed to determine the rate of methylation at CpG dinucleotides.

[0507] Example 5: alternate DNMT effectors and effector fusions.

[0508] GripTite293 cells were seeded in 96-well plates and transfected with 25 ng of a gRNA-expressing plasmid (targeting VIM), 50 ng of an Effector-DBD fusion plasmid, and 5ng of a Puromycin resistance plasmid using Mirus TransIT transfection reagent. VIM-targeting gRNAs used can be found in SEQ ID NO.: 962-969. Effector-DBD fusions can be found in SEQ ID NO.: 1112-1153.

[0509] At day 1 post transfection, cells were cultured with Puromycin to select for positively transfected cells. At day 6 or day 7 post transfection, cells were analyzed for VIM expression via FACS (FIG. 2).

[0510] When human-human and human-mouse fusions were tested against plant DNMT effectors and effector fusions, the mammalian fusions exhibited greateer VIM silencing (FIG. 3A); similar results were found when the mammalian fusions were compared to DNMT effectors and effector fusions from bacteria, fungi, and *Drosophila* (FIG. 3B).

[0511] Example 6: alternate KRAB and non-KRAB repressors.

[0512] GripTite293 cells were seeded in 96-well plates and transfected with 25 ng of a gRNA-expressing plasmid (targeting VIM), 50 ng of a DBD-Effector fusion plasmid, and 5ng of a Puromycin resistance plasmid using Mirus TransIT transfection reagent. VIM-targeting gRNAs used can be found in SEQ ID NO.: 962-969. DBD-Effector fusions can be found in SEQ ID NO.: 1022-1111.

[0513] At day 1 post transfection, cells were cultured with Puromycin to select for positively transfected cells. At day 6 post transfection, cells were analyzed for VIM expression via FACS (FIG 5). Many alternate KRAB and non-KRAB repressors effectively silenced VIM expression.

[0514] Example 7: gene repression.

[0515] GripTite293 cells were seeded in 96-well plates and transfected with 25 ng of a gRNA-expressing plasmid (either single gRNA or 4x (quad) gRNA plasmid targeting CD151 or CLTA), 50 ng of a DBD-Effector fusion plasmid, and 5ng of a Puromycin resistance plasmid using Mirus TransIT transfection reagent. CD151-targeting gRNAs used can be found in SEQ ID NO.: 970-977. DBD-Effector fusion plasmids used can be found in SEQ ID NO.: 979-1021.

[0516] At day 1 post transfection, cells were cultured with Puromycin to select for positively transfected cells. At day 6 post transfection, cells were analyzed for CD151 or CLTA expression via FACS. FIG 6-7 show that many of the alternate KRAB combination effectively silence CD151.

[0517] While preferred embodiments of the present disclosure have been shown and described herein, it will be obvious to those skilled in the art that such embodiments are provided by way of example only. Numerous variations, changes, and substitutions will now occur to those skilled in the art without departing from the disclosure. It should be understood that various alternatives to the embodiments of the disclosure described herein may be employed in practicing the disclosure. It is intended that the following claims define the scope of the disclosure and that methods and structures within the scope of these claims and their equivalents be covered thereby.

Other Embodiments

[0518] From the foregoing description, it will be apparent that variations and modifications may be made to the disclosure described herein to adopt it to various usages and conditions. Such embodiments are also within the scope of the following claims.

[0519] The recitation of a listing of elements in any definition of a variable herein includes definitions of that variable as any single element or combination (or subcombination) of listed elements. The recitation of an embodiment herein includes that embodiment as any single embodiment, any portion of the embodiment, or in combination with any other embodiments or any portion thereof.

[0520] As is set forth herein, it will be appreciated that the disclosure comprises specific embodiments and examples of base editing systems to effect a nucleobase alteration in a gene and methods of using same for treatment of disease including compositions that comprise such base editing systems, designs and modifications thereto; and specific examples and

embodiments describing the synthesis, manufacture, use, and efficacy of the foregoing individually and in combination including as pharmaceutical compositions for treating disease and for in vivo and in vitro delivery of active agents to mammalian cells under described conditions.

[0521] While specific examples and numerous embodiments have been provided to illustrate aspects and combinations of aspects of the foregoing, it should be appreciated and understood that any aspect, or combination thereof, of an exemplary or disclosed embodiment may be excluded therefrom to constitute another embodiment without limitation and that it is contemplated that any such embodiment can constitute a separate and independent claim. Similarly, it should be appreciated and understood that any aspect or combination of aspects of one or more embodiments may also be included or combined with any aspect or combination of aspects of one or more embodiments and that it is contemplated herein that all such combinations thereof fall within the scope of this disclosure and can be presented as separate and independent claims without limitation. Accordingly, it should be appreciated that any feature presented in one claim may be included in another claim; any feature presented in one claim may be removed from the claim to constitute a claim without that feature; and any feature presented in one claim may be combined with any feature in another claim, each of which is contemplated herein. The following enumerated clauses are further illustrative examples of aspects and combination of aspects of the foregoing embodiments and examples:

1. A method of modifying an epigenetic state of a target gene in a target chromosome, the method comprising contacting the target chromosome with an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby modifying the epigenetic state of the target gene.
2. A method of modulating expression of a target gene in a target chromosome in a cell, the method comprising contacting the target gene with an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene, thereby modulating expression of the target gene.

3. A method of treating a disease in a subject in need thereof, the method comprising administering to the subject an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene in the subject and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene, wherein the target gene is associated with disease and wherein the site-specific epigenetic modification modulates expression of the target gene, thereby treating the disease.
4. The method of any one of the preceding claims, wherein the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of the target sequence.
5. The method of claim 4, wherein the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the target sequence.
6. The method of any one of the preceding claims, wherein the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of an expression regulatory sequence.
7. The method of claim 6, wherein the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the expression regulatory sequence.
8. The method of claim 7, wherein the site-specific epigenetic modification is within 1000 base pairs upstream or downstream of the expression regulatory sequence.
9. A method of modifying an epigenetic state of a target gene in a target chromosome, the method comprising contacting the target gene with an epigenetic editor, wherein the epigenetic editor comprises a DNA biding domain and an epigenetic effector domain, wherein the DNA biding domain binds to a target sequence in the target chromosome, and wherein the epigenetic effector domain results in an epigenetic modification in at least 10% of all nucleotides or all histone tails bound with nucleotides within 200 base pairs upstream or downstream of the target sequence in the target genome.
10. A method of modulating expression of a target gene in a target chromosome in a cell, the method comprising contacting the target gene with an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in the target chromosome, and wherein the epigenetic effector domain results in an epigenetic modification in at least 10% of all nucleotides or all histone tails bound with nucleotides within 200 base pairs upstream or downstream of the target sequence in a target genome in the cell.

11. A method of treating a disease in a subject in need thereof, the method comprising administering to the subject an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene in the subject, wherein the epigenetic effector domain results in an epigenetic modification in at least 10% of all nucleotides or at least 10% of all histone tails bound with nucleotides within 200 base pairs upstream or downstream of the target sequence in a target genome in the subject, wherein the target gene is associated with the disease and wherein the epigenetic modification modulates expression of the target gene, thereby treating the disease.
12. The method of any one of claims 9-11, wherein the epigenetic effector domain results in the epigenetic modification in at least 20% of all nucleotides within 200 base pairs upstream or downstream of the target sequence.
13. The method of any one of claims 9-11, wherein the epigenetic effector domain results in the epigenetic modification in at least 50% of all nucleotides within 200 base pairs upstream or downstream of the target sequence.
14. The method of any one of claims 9-11, wherein the epigenetic effector domain results in the epigenetic modification in at least 10% of all nucleotides within 500 base pairs upstream or downstream of the target sequence.
15. The method of any one of claims 9-11, wherein the epigenetic effector domain results in the epigenetic modification in at least 20% of all nucleotides within 500 base pairs upstream or downstream of the target sequence.
16. A method of modifying an epigenetic state of a target gene in a target chromosome, the method comprising contacting the target gene with an epigenetic editor, wherein the epigenetic editor comprises a DNA biding domain and an epigenetic effector domain, wherein the DNA biding domain binds to a target sequence in the target chromosome, and wherein the epigenetic effector domain results in an epigenetic modification in at least 10% of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence in the target genome.
17. A method of modulating expression of a target gene in a target chromosome in a cell, the method comprising contacting the target gene with an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in the target chromosome, and wherein the epigenetic effector domain results in an epigenetic modification in at least 10%

of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence in a target genome in the cell.

18. A method of treating a disease in a subject in need thereof, the method comprising administering to the subject an epigenetic editor, wherein the epigenetic editor comprises a DNA binding domain and an epigenetic effector domain, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene in the subject, wherein the epigenetic effector domain results in an epigenetic modification in at least 10% of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence in a target genome in the subject, wherein the target gene is associated with disease and wherein the epigenetic modification modulates expression of the target gene, thereby treating the disease.
19. The method of any one of claims 16-18, wherein the epigenetic effector domain results in the epigenetic modification in at least 20% of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence.
20. The method of any one of claims 16-18, wherein the epigenetic effector domain results in the epigenetic modification in at least 50% of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence.
21. The method of any one of claims 16-18, wherein the epigenetic effector domain results in the epigenetic modification in at least 10% of all CpG dinucleotides within 500 base pairs upstream or downstream of the target sequence.
22. The method of any one of claims 16-18, wherein the epigenetic effector domain results in the epigenetic modification in at least 20% of all CpG dinucleotides within 500 base pairs upstream or downstream of the target sequence.
23. The method of any one of claims 16-18, wherein the epigenetic effector domain results in the epigenetic modification in at least 80% of all CpG dinucleotides within 200 base pairs upstream or downstream of the target sequence.
24. The method of any one of claims 9-14, wherein the epigenetic effector domain results in the epigenetic modification in at least 50% of all nucleotides within 500 base pairs upstream or downstream of an expression regulatory sequence.
25. The method of any one of claims 3-8 or 11-24, comprising administering to the subject a cell comprising the epigenetic editor.
26. The method of claim 25, wherein the cell is an allogeneic cell.
27. The method of claim 25, wherein the cell is an autologous cell.

28. The method of any one of claims 6-8 or 15-27, wherein the expression regulatory sequence comprises a promoter.
29. The method of any one of claims 6-8 or 15-27, wherein the expression regulatory sequence comprises a transcription initiation start site.
30. The method of any one of claims 6-8 or 15-27, wherein the expression regulatory sequence comprises an enhancer.
31. The method of any one of the preceding claims, wherein the epigenetic modification is within a coding region of the target gene.
32. The method of any one of the preceding claims, wherein the target gene comprises an allele associated with a disease.
33. The method of any one of the preceding claims, wherein the target gene comprises two heterozygotic copies.
34. The method of claim 33, wherein the target gene is heterozygous at an allele.
35. The method of claim 33 or 34, wherein the epigenetic modification is at one of the two heterozygotic copies and not the other.
36. The method of claim 34, wherein the epigenetic modification is at the heterozygotic allele.
37. The method of any one of the preceding claims, wherein the DNA binding domain comprises a zinc finger motif.
38. The method of any one of the preceding claims, wherein the DNA binding domain comprises a zinc finger array.
39. The method of claim 38, wherein the zinc finger array comprises at least six zinc fingers.
40. The method of claim 39, wherein the zinc finger array comprises at least three subsets of zinc fingers each comprising at least two zinc fingers.
41. The method of any one of claims 1-36, wherein the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide.
42. The method of claim 41, wherein the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide.
43. The method of claim 41, wherein the guide polynucleotide hybridizes with the target sequence.
44. The method of claim 41, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9).
45. The method of claim 41, wherein the CRISRP-Cas protein comprises a nuclease inactive Cas12a (dCas12a) or a nuclease inactive CasX (dCasX).

46. The method of any one of the preceding claims, wherein the epigenetic effector domain results in reduced or silenced expression of the target gene as compared to a control cell not contacted with the epigenetic editor.
47. The method of claim 46, wherein the epigenetic effector domain specifically reduces or silences expression from one of the heterozygotic copies of the target gene as compared to a control gene in a cell not contacted with the epigenetic editor.
48. The method of claim 46 or 47, wherein the site-specific epigenetic modification or the epigenetic modification comprises DNA methylation.
49. The method of claim 48, wherein the site-specific epigenetic modification or the epigenetic modification is in a CpG dinucleotide.
50. The method of claim 48, wherein the CpG dinucleotide is in a CpG island.
51. The method of claim 48, wherein the CpG dinucleotide is not in a CpG island.
52. The method of claim 46 or 47, wherein the site-specific epigenetic modification or the epigenetic modification comprises de-acetylation of the histone bound to the target gene.
53. The method of claim 46 or 47, wherein the site-specific epigenetic modification or the epigenetic modification comprises methylation of the histone bound to the target gene, optionally wherein the methylation of the histone is H3K9 methylation.
54. The method of claim 46 or 47, wherein the site-specific epigenetic modification comprises demethylation of the histone bound to the target gene, optionally wherein the demethylation of the histone is H3K4 demethylation.
55. The method of any one of claims 46-54, wherein the epigenetic effector domain comprises a DNA methyltransferase domain.
56. The method of claim 55, wherein the epigenetic effector domain comprises a Dnmt1 domain, a Dnmt3A domain, a Dnmt3L domain, or a Dnmt3B domain.
57. The method of claim 56, wherein the epigenetic effector domain comprises a Dnmt3A-Dnmt3L fusion protein.
58. The method of any one of claims 46-55, wherein the epigenetic effector domain comprises transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof.
59. The method of any one of claims 46-55, wherein the epigenetic effector domain recruits a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof to the target gene.
60. The method of claim 58 or 59, wherein the epigenetic effector domain comprises a KRAB domain, a KAP1 domain, a MECP2 domain, a chromoshadow domain, or a HP1 domain.

61. The method of any one of claims 58-59, wherein the epigenetic effector domain comprises a protein from Table 2 or Table 3.
62. The method of any one of claims 46-61, wherein the epigenetic editor further comprises a second epigenetic effector domain that results in reduced or silenced expression of the target gene.
63. The method of claim 62, wherein the second epigenetic effector domain comprises a DNA methyltransferase domain.
64. The method of claim 62, wherein the second epigenetic effector domain comprises a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof.
65. The method of claim 62, wherein the second epigenetic effector domain recruits a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof to the target gene.
66. The method of claim 62, wherein the second epigenetic effector domain comprises a KRAB domain, a KAP1 domain, a HP1 domain, a Dnmt3A domain, a Dnmt3L domain, or any combination thereof.
67. The method of claim 62, wherein the second epigenetic effector domain comprises a protein of Table 2 or Table 3.
68. The method of any one of claims 62-67, wherein the epigenetic effector domain and the second epigenetic effector domain synergistically reduces or silences expression of the target gene.
69. The method of any one of claims 46-68, wherein the epigenetic editor comprises a DNA methyltransferase domain and a repression domain that reduces or silences expression of the target gene.
70. The method of any one of claims 46-68, wherein the epigenetic editor comprises a DNA methyltransferase domain and a repression scaffold protein domain that recruits transcription repressor proteins to the target gene.
71. The method of any one of claims 46-68, wherein the epigenetic editor comprises a DNA methyltransferase domain and a histone deacetylase domain.
72. The method of claim 71, wherein the epigenetic editor further comprises a KRAB domain, a KAP1 domain, a HP1 domain, a chromoshadow domain, or a MECP2 domain.
73. The method of any one of claim 46-72, wherein the epigenetic editor comprises from N terminus to C terminus: (i) a Dnmt3A-Dnmt3L fusion protein domain, (ii) the DNA binding domain, and (iii) a KRAB domain, a KAP1 domain, a HP1 domain, or a MECP2 domain.

74. The method of any one of claim 46-72, wherein the epigenetic editor comprises from N terminus to C terminus the (i) a KRAB domain, a KAP1 domain, a HP1 domain, or a MECP2 domain, (ii) the DNA binding domain, and (iii) Dnmt3A-Dnmt3L fusion protein domain.
75. The method of claim 73 or 74, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3A-Dnmt3L.
76. The method of claim 73 or 74, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3L-Dnmt3A.
77. The method of any one of claims 46-76, wherein the epigenetic editor reduces expression of the target gene by at least 50% as compared to a wild-type expression level.
78. The method of any one of claims 46-77, wherein the reduction in expression of the target gene is maintained for at least 1 week, 4 weeks, 6 months, or 1 year.
79. The method of any one of claims 46-78, wherein the reduction in expression of the target gene is maintained in offspring cells derived from a cell comprising the target gene.
80. The method of any one of claims 1-45, wherein the epigenetic editor comprises an epigenetic effector domain that increases expression of the target gene as compared to a control gene in a cell not contacted with the epigenetic editor.
81. The method of claim 80, wherein the site-specific epigenetic modification or the epigenetic modification comprises DNA demethylation.
82. The method of claim 80 or 81, wherein the site-specific epigenetic modification or the epigenetic modification is in a CpG dinucleotide.
83. The method of claim 82, wherein the CpG dinucleotide is in a CpG island.
84. The method of claim 82, wherein the CpG dinucleotide is not in a CpG island.
85. The method of claim 83, wherein the site-specific epigenetic modification or the epigenetic modification comprises acetylation of the histone bound to the target gene.
86. The method of claim 80, wherein the site-specific epigenetic modification or the epigenetic modification comprises methylation of the histone bound to the target gene, optionally wherein the methylation of the histone is H3K4 methylation.
87. The method of claim 80, wherein the site-specific epigenetic modification comprises demethylation of the histone bound to the target gene, optionally wherein the demethylation of the histone is H3K9 demethylation.
88. The method of any one of claims 80-87, wherein the epigenetic effector domain comprises a DNA demethylase domain.
89. The method of claim 88, wherein the DNA demethylase domain comprises a TET family protein domain.

90. The method of claim 89, wherein the DNA demethylase domain comprises a TET1 protein.

91. The method of claim 88, wherein the epigenetic effector domain comprises a histone acetylase domain.

92. The method of any one of claims 80-87, wherein the epigenetic effector domain comprises a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof.

93. The method of any one of claims 80-87, wherein the epigenetic effector domain recruits a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof to the target gene.

94. The method of claim 92 or 93, wherein the epigenetic effector domain comprises a VP16 domain, a VP64 domain, a p65 domain, or a RTA domain.

95. The method of any one of claims 80-87, wherein the epigenetic effector domain comprises a protein from Table 5 or Table 6.

96. The method of any one of claims 80-95, wherein the epigenetic editor further comprises a second epigenetic effector domain that increases expression of the target gene.

97. The method of claim 96, wherein the second epigenetic effector domain comprises a DNA demethylase domain.

98. The method of claim 96, wherein the second epigenetic effector domain comprises a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof.

99. The method of claim 96, wherein the second epigenetic effector domain recruits a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof.

100. The method of claim 98 or 99, wherein the second epigenetic effector domain comprises a TET1 domain, a VP16 domain, a VP64 domain, a p65 domain, a RTA domain, or any combination thereof.

101. The method of claim 96, wherein the second epigenetic effector domain comprises a protein form Table 5 or Table 6.

102. The method of any one of claims 80-101, wherein the epigenetic editor comprises a DNA demethylase domain and a fusion of a VP64 domain, a p65 domain, and a RTA domain.

103. The method of any one of claim 80-102, wherein the epigenetic editor increases expression of the target gene by at least 50% as compared to a wild-type expression level.

104. The method of claim 80-103, wherein the increase in expression of the target gene expression is maintained for at least 1 week, 4 weeks, 6 months, or 1 year.

105. The method of any one of claims 80-104, wherein the increase in expression of the target gene is maintained in offspring cells derived from a cell comprising the target gene.

106. The method of any one of the preceding claims, wherein the epigenetic editor further comprises a second DNA binding domain that binds to a second target sequence in a second target gene, and wherein the DNA binding domain directs the epigenetic effector domain to effect an epigenetic modification in the second target gene or a histone bound to the second target gene.

107. The method of any one of claims 41-106, wherein the epigenetic editor further comprises a second guide polynucleotide that binds to the DNA binding domain and hybridizes with a second target sequence in a second target gene and directs the epigenetic editor to effect an epigenetic modification in the second target gene or a histone bound to the second target gene.

108. The method of claim 106 or 107, wherein the second target gene is the same as the target gene.

109. The method of claim 108, wherein the second target sequence overlaps with the target sequence.

110. The method of claim 108, wherein the second target sequence is within 1000 base pairs upstream or downstream of the target sequence.

111. The method of claim 108, wherein the second target sequence is within 500 base pairs upstream or downstream of the target sequence.

112. The method of claim 106 or 107, wherein the second target gene is different from the target gene.

113. The method of claim 112, wherein the target gene and the second target gene are associated with in a same metabolic pathway or function.

114. The method of claim 112, wherein the target gene and the second target gene are associated with a same disease or condition.

115. The method of any one of the preceding claims, wherein the epigenetic editor further comprises a linker.

116. The method of claim 115, wherein the linker is a peptide linker.

117. The method of claim 116, wherein the linker comprises an XTEN linker.

118. The method of any one of the preceding claims, wherein the contacting is ex vivo.

119. The method of any one of claims 1-114, wherein the contacting is in vivo in a subject.

120. The method of claim 119, wherein the subject is a human.

121. An epigenetically modified chromosome comprising a gene of interest (GOI), wherein at least 10% of all nucleotides or at least 10% of all histone tails bound with nucleotides within 200 base pairs upstream or downstream of an expression regulatory sequence of the GOI comprise an epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

122. The epigenetically modified chromosome of claim 121, wherein at least 20% of all nucleotides within 200 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

123. The epigenetically modified chromosome of claim 121, wherein at least 50%of all nucleotides within 200 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest

124. The epigenetically modified chromosome of claim 121, wherein at least 10% of all nucleotides within 500 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

125. The epigenetically modified chromosome of claim 115, wherein the at least 20% of all nucleotides within 500 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

126. An epigenetically modified chromosome comprising a gene of interest (GOI), wherein at least 10% of all CpG dinucleotides within 200 base pairs upstream or downstream of an expression regulatory sequence of the GOI comprise an epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

127. The epigenetically modified chromosome of claim 126, wherein at least 20% of all CpG dinucleotides within 200 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

128. The epigenetically modified chromosome of claim 126, wherein at least 50% of all CpG dinucleotides within 200 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

129. The epigenetically modified chromosome of claim 126, wherein at least 10% of all CpG dinucleotides within 500 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

130. The epigenetically modified chromosome of claim 126, wherein at least 20% of all CpG dinucleotides within 500 base pairs upstream or downstream of the expression regulatory sequence comprise the epigenetic modification as compared to an unmodified control chromosome comprising the gene of interest.

131. The epigenetically modified chromosome of any one of claims 126-130, wherein the CpG dinucleotides comprising the epigenetic modification are in a CpG island.

132. the epigenetically modified chromosome of any one of claims 126-130, wherein the CpG dinucleotides comprising the epigenetic modification are not in a CpG island.

133. The epigenetically modified chromosome of any one of claims 121-132, wherein the expression regulatory sequence comprises a promoter.

134. The epigenetically modified chromosome of any one of claims 121-132, wherein the expression regulatory sequence comprises a transcription start site.

135. The epigenetically modified chromosome of any one of claims 121-132, wherein the expression regulatory sequence comprises an enhancer.

136. The epigenetically modified chromosome of any one of claims 121-135, wherein the epigenetic modification is within a coding region of the GOI.

137. The epigenetically modified chromosome of any one of claims 121-136, wherein the target gene comprises an allele associated with a disease.

138. The epigenetically modified chromosome of any one of claims 121-136, wherein the target gene comprises two heterozygotic copies.

139. The epigenetically modified chromosome of any one of claims 121-137, wherein the target gene is heterozygous at an allele.

140. The epigenetically modified chromosome of claim 139, wherein the epigenetic modification is at one of the two heterozygotic copies and not the other.

141. The epigenetically modified chromosome of claim 140, wherein the epigenetic modification is at the heterozygotic allele.

142. The epigenetically modified chromosome of any one of claims 121-140, wherein the epigenetically modified chromosome is in a cell.

143. The epigenetically modified chromosome of claim 141, wherein the epigenetic modification results in reduced or silenced expression of the GOI as compared to the GOI in an unmodified control chromosome in a control cell.

144. The epigenetically modified chromosome of claim 143, wherein the epigenetic modification comprises DNA methylation.

145. The epigenetically modified chromosome of claim 143, wherein the epigenetic modification comprises de-acetylation of the histone tails.

146. The epigenetically modified chromosome of claim 143, wherein the site-specific epigenetic modification or the epigenetic modification comprises methylation of the histone bound to the target gene, optionally wherein the methylation of the histone is H3K9 methylation.

147. The epigenetically modified chromosome of claim 143, wherein the site-specific epigenetic modification comprises demethylation of the histone bound to the target gene, optionally wherein the demethylation of the histone is H3K4 demethylation.

148. The epigenetically modified chromosome of any one of claims 143-147, wherein the expression of the GOI is reduced by at least 50% as compared to a wild-type expression level.

149. The epigenetically modified chromosome of claim, wherein the reduction in expression of the GOI is maintained for at least 1 week, 4 weeks, 6 months, or 1 year.

150. The epigenetically modified chromosome any one of claims 143-149, wherein the reduction in expression of the GOI is maintained in offspring cells derived from the cell.

151. The epigenetically modified chromosome of claim 141, wherein the epigenetic modification results in increased expression of the GOI as compared to the GOI in an unmodified control chromosome in a control cell.

152. The epigenetically modified chromosome of claim 151, wherein the epigenetic modification comprises DNA demethylation.

153. The epigenetically modified chromosome of claim 151, wherein the epigenetic modification comprises acetylation of the histone tails.

154. The epigenetically modified chromosome of claim 151, wherein the epigenetic modification comprises methylation of the histone tails, optionally wherein the methylation of the histone is H3K4 methylation.

155. The epigenetically modified chromosome of claim 151, wherein the epigenetic modification comprises demethylation of the histone tails, optionally wherein the demethylation of the histone is H3K9 demethylation.

156. The epigenetically modified chromosome any one of claims 151-155, wherein the expression of the GOI is increased by at least 50% as compared to a wild-type expression level.

157. The epigenetically modified chromosome any one of claims 151-156, wherein the increase in expression of the GOI is maintained for at least 1 week, 4 weeks, 6 months, or 1 year.

158. The epigenetically modified chromosome of any one of claims 151-157, wherein the increase in expression of the GOI is maintained in offspring cells derived from the cell.

159. A cell comprising the epigenetically modified chromosome of any one of claims 121-158.

160. The cell of claim 159, wherein the cell is a non-dividing cell.

161. The cell of claim 159, wherein the cell is a primary cell.

162. The cell of claim 159, wherein the cell is a mammalian cell.

163. The cell of claim 159, wherein the cell is a human cell.

164. The epigenetically modified chromosome of any one of claims 121-158, wherein the epigenetically modified chromosome is in a subject.

165. The epigenetically modified chromosome of claim 164, wherein the subject is a human.

166. An epigenetic editor that comprises a DNA binding domain, a DNA methylation regulatory protein, and an affinity domain, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene, wherein the affinity domain specifically binds to an epigenetic effector protein in a cell comprising the target gene and directs the epigenetic effector protein to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene when contacted with the target chromosome.

167. An epigenetic editor that comprises a DNA binding domain, an epigenetic effector protein, and an affinity domain, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene, wherein the affinity domain specifically binds to a DNA methylation regulatory protein in a cell comprising the target gene and directs the DNA methylation regulatory protein to the target gene to effect an epigenetic modification in a nucleotide in the target gene.

168. The epigenetic editor of claim 166 or 167, wherein the DNA methylation regulatory protein comprises a DNA methyltransferase domain.

169. The epigenetic editor of claim 168, wherein the DNA methyltransferase domain comprises a Dnmt1 domain, a Dnmt3A domain, a Dnmt3L domain, or a Dnmt3B domain.

170. The epigenetic editor of claim 168, wherein the DNA methyltransferase domain comprises a Dnmt3A-Dnmt3L fusion.

171. The epigenetic editor of any one of claims 166-170, wherein the epigenetic effector protein results in decreased or silenced expression of the target gene as compared to the target gene not contacted with the epigenetic editor.

172. The epigenetic editor of any one of claims 166-171, wherein the epigenetic effector protein comprises a histone deacetylase.

173. The epigenetic editor of any one of claims 166-171, wherein the epigenetic effector protein comprises a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof.

174. The epigenetic editor of any one of claims 166-171, wherein the epigenetic effector protein recruits a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof in the cell to the target gene.

175. The epigenetic editor of any one of claims 166-171, wherein the epigenetic effector protein comprises a KRAB protein, a KAP1 protein, a MECP2 protein, or a HP1 protein.

176. The epigenetic editor of any one of claims 166-171, wherein the epigenetic effector protein comprises a protein from Table 2 or Table 3.

177. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor comprises a Dnmt3A-Dnm3L fusion protein domain and the affinity domain that specifically binds to KAP1.

178. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor comprises a Dnmt3A-Dnm3L fusion protein domain and the affinity domain that specifically binds to KRAB.

179. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor comprises a Dnmt3A-Dnm3L fusion protein domain and the affinity domain that specifically binds to MECP2.

180. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor comprises a Dnmt3A-Dnm3L fusion protein domain and the affinity domain that specifically binds to HP1.

181. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor comprises a Dnmt3A-Dnm3L fusion protein domain and the affinity domain that specifically binds to a chromoshadow domain.

182. The epigenetic editor of any one of claims 177-181, wherein the epigenetic editor comprises from N terminus to C terminus: (i) the Dnmt3A-Dnmt3L fusion protein domain, (ii) the DNA binding domain, and (iii) the affinity domain.

183. The epigenetic editor of any one of claims 177-181, wherein the epigenetic editor comprises from N terminus to C terminus (i) the affinity domain, (ii) the DNA binding domain, and (iii) the Dnmt3A-Dnmt3L fusion protein domain.

184. The epigenetic editor of any one of claims 177-183, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3A-Dnmt3L.

185. The epigenetic editor of any one of claims 177-183, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3L-Dnmt3A.

186. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a histone deacetylase domain and the affinity domain specifically binds to a Dnmt3A domain.

187. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a histone deacetylase domain and the affinity domain specifically binds to a Dnmt3L domain.

188. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a histone deacetylase domain and the affinity domain specifically binds to a Dnmt3B domain.

189. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a histone deacetylase domain and the affinity domain specifically binds to a Dnmt1 domain.

190. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a KAP1 domain and the affinity domain that specifically binds to a Dnmt3A domain, a Dnmt3L domain, a Dnmt3B domain, or a Dnmt1 domain.

191. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a KRAB domain and the affinity domain that specifically binds to a Dnmt3A domain, a Dnmt3L domain, a Dnmt3B domain, or a Dnmt1 domain.

192. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a MECP2 domain and the affinity domain that specifically binds to a Dnmt3A domain, a Dnmt3L domain, a Dnmt3B domain, or a Dnmt1 domain.

193. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a HP1 domain and the affinity domain that specifically binds to a Dnmt3A domain, a Dnmt3L domain, a Dnmt3B domain, or a Dnmt1 domain.

194. The epigenetic editor of any one of claims 167-175, wherein the epigenetic effector protein comprises a chromoshadow domain and an affinity domain that specifically binds to a Dnmt3A domain, a Dnmt3L domain, a Dnmt3B domain, or a Dnmt1 domain.

195. The epigenetic editor of any one of claims 167-175, wherein the epigenetic editor comprises from N terminus to C terminus: (i) a KAP1 domain, a KRAB domain, a HP1 domain, a MECP2 domain, or a chromoshadow domain, (ii) the DNA binding domain, and (iii) the affinity domain.

196. The epigenetic editor of any one of claims 167-175, wherein the epigenetic editor comprises from N terminus to C terminus (i) the affinity domain, (ii) the DNA binding domain, and (iii) (i) a KAP1 domain, a KRAB domain, a HP1 domain, a MECP2 domain, or a chromoshadow domain.

197. The epigenetic editor of any one of claims 166 or 168-175, wherein the epigenetic editor further comprises a second affinity domain that specifically binds to a second epigenetic effector protein in the cell, wherein the second epigenetic effector protein results in reduced or silenced expression of the target gene.

198. The epigenetic editor of claim 197, wherein the second effector protein comprises a DNA methyltransferase domain.

199. The epigenetic editor of claim 197, wherein the second epigenetic effector protein comprises a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof.

200. The epigenetic editor of claim 197, wherein the second epigenetic effector protein recruits a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof to the target gene.

201. The epigenetic editor of claim 197, wherein the second epigenetic effector protein comprises a KRAB domain, a KAP1 domain, a HP1 domain, a Dnmt3A domain, a Dnmt3L domain, a chromoshadow domain, or any combination thereof.

202. The epigenetic editor of claim 197, wherein the second epigenetic effector domain comprises a protein of Table 2 or Table 3.

203. The epigenetic editor of claim 166 or 167, wherein the DNA methylation regulatory protein comprises a DNA demethylase domain.

204. The epigenetic editor of claim 203, wherein the DNA demethylase domain comprise a TET family protein.

205. The epigenetic editor of claim 204, wherein the DNA demethylase domain comprise TET1.

206. The epigenetic editor of any one of claims 203-205, wherein the epigenetic effector protein results in increased expression of the target gene as compared to the target gene not contacted with the epigenetic editor.

207. The epigenetic editor of any one of claims 203-206, wherein the epigenetic effector protein comprises a histone acetyltransferase.

208. The epigenetic editor of any one of claims 203-206, wherein the epigenetic effector protein recruits a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof to the target gene.

209. The epigenetic editor of any one of claims 203-206, wherein the epigenetic effector protein comprises a VP16 domain, a VP64 domain, a p65 domain, or a RTA domain.

210. The epigenetic editor of any one of claims 203-206, wherein the epigenetic effector protein comprises a protein from Table 5 or Table 6.

211. The epigenetic editor of any one of claims 203-210, wherein the epigenetic editor further comprises a second affinity domain that specifically binds to a second epigenetic effector protein that increases expression of the target gene.

212. The epigenetic editor of claim 211, wherein the second epigenetic effector protein comprises a DNA demethylase domain.

213. The epigenetic editor of claim 211, wherein the second epigenetic effector protein comprises a histone acetyltransferase domain.

214. The epigenetic editor of claim 211, wherein the second epigenetic effector protein recruits a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetylase, or any combination thereof.

215. The epigenetic editor of claim 211, wherein the second epigenetic effector protein comprises a TET1 domain, a VP16 domain, a VP64 domain, a p65 domain, a RTA domain, or any combination thereof.

216. The epigenetic editor of claim 211, wherein the second epigenetic effector protein comprises a protein form Table 5 or Table 6.

217. The epigenetic editor of any one of claims 166-216, wherein the affinity domain comprises a single chain antibody, a nanobody, an antigen binding sequence, an antibody, a nanobody, a functional antibody fragment, a single chain variable fragment (scFv), an Fab, a single-domain antibody (sdAb), a VH domain, a VL domain, a VNAR domain, a VHH domain, a bispecific antibody, a diabody, or a functional fragment or a combination thereof.

218. An epigenetic editor that comprises a DNA binding domain, a DNA methyltransferase domain, and an epigenetic effector domain, wherein the epigenetic effector domain is a KAP1 domain, a HP1 domain, a chromoshadow domain, or a MECP2 domain.

219. An epigenetic editor that comprises a DNA binding domain, a DNA methyltransferase domain selected from Table 1, and an epigenetic effector domain selected from Table 2 or Table 3.

220. An epigenetic editor that comprises a DNA binding domain, a DNA demethylase domain selected from Table 4, and an epigenetic effector domain selected from Table 5 or Table 6.

221. The epigenetic editor of any one of claims 218 or 220, wherein the DNA methyltransferase domain comprises a Dnmt1 domain, a Dnmt3A domain, a Dnmt3L domain, or a Dnmt3B domain.

222. The epigenetic editor of any one of claim 218 or 220, wherein the DNA methyltransferase domain comprises a Dnmt3A-Dnmt3L fusion.

223. The epigenetic editor of claim 222, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3A-Dnmt3L.

224. The epigenetic editor of claim 222, wherein the Dnmt3A-Dnmt3L fusion protein domain comprises from N terminus to C terminus: Dnmt3L-Dnmt3A.

225. The epigenetic editor of any one of claims 222-224, comprising from N terminus to C terminus (i) the Dnmt3A-Dnmt3L fusion protein domain, (ii) the DNA binding domain, and (iii) epigenetic effector domain.

226. The epigenetic editor of any one of claims 222-225, comprising from N terminus to C terminus (i) the epigenetic effector domain, (ii) the DNA binding domain, and (iii) Dnmt3A-Dnmt3L fusion protein domain.

227. The epigenetic editor of any one of claims 218-226, wherein the DNA binding domain binds to a target sequence in a target gene and directs the epigenetic effector domain to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene when contacted with the target gene.

228. The method of any one of claims 227, wherein the epigenetic effector domain results in reduced or silenced expression of the target gene as compared to the target gene not contacted with the epigenetic editor.

229. The method of any one of claims 227, wherein the epigenetic effector domain results in increased expression of the target gene as compared to the target gene not contacted with the epigenetic editor.

230. The epigenetic editor of any one of claims 166-229, wherein the epigenetic modification is within a coding region of the target gene.

231. The epigenetic editor of any one of claims 166-229, wherein the epigenetic modification is in an expression regulatory sequence of the target gene.

232. The epigenetic editor of any one of claim 166-229, wherein the epigenetic modification is within 3000 base pairs upstream or downstream of an expression regulatory sequence of the target gene.

233. The epigenetic editor of claim 231 or 232, wherein the expression regulatory sequence comprises a promoter.

234. The epigenetic editor of claim 231 or 232, wherein the expression regulatory sequence comprises a transcription initiation start site.

235. The epigenetic editor of claim 231 or 232, wherein the expression regulatory sequence comprises an enhancer.

236. The method of any one of claims 219 or 221-235, wherein the epigenetic editor further comprises a second epigenetic effector domain that results in reduced or silenced expression of the target gene.

237. The method of claim 236, wherein the second epigenetic effector domain comprises or recruits a transcription repressor, a DNA methyltransferase, a histone methyltransferase, a histone demethylase, a histone deacetylase, or any combination thereof.

238. The method of claim 236, wherein the second epigenetic effector domain comprises a protein of Table 2 or Table 3.

239. The method of any one of claims 232-238, wherein the epigenetic effector domain and the second epigenetic effector domain synergistically reduces or silences expression of the target gene.

240. The method of any one of claims 218 or 220-234, wherein the epigenetic editor further comprises a second epigenetic effector domain that results in increased expression of the target gene.

241. The method of claim 240, wherein the second epigenetic effector domain comprises a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetyltransferase, or any combination thereof.

242. The method of claim 240, wherein the second epigenetic effector domain recruits a transcription activator, a DNA demethylase, a histone methyltransferase, a histone demethylase, a histone acetyltransferase, or any combination thereof to the target gene.

243. The method of claim 240, wherein the second epigenetic effector domain comprises a protein of table 5 or Table 6.

244. The method of any one of claims 241-243, wherein the epigenetic effector domain and the second epigenetic effector domain synergistically reduces or silences expression of the target gene.

245. The epigenetic editor of any one of claims 166-244, wherein the target gene comprises an allele associated with a disease.

246. The epigenetic editor of any one of claims 166-244, wherein the target gene comprises two heterozygotic copies and wherein the DNA binding domain binds to one of the two heterozygotic copies and not the other.

247. The epigenetic editor of any one of claims 166-244, wherein the target gene is heterozygous at an allele.

248. The epigenetic editor of any one of claims 166-247, wherein the DNA binding domain comprises a zinc finger motif.

249. The epigenetic editor of any one of claims 166-248, wherein the DNA binding domain comprises a zinc finger array.

250. The epigenetic editor of claim 249, wherein the zinc finger array comprises at least six zinc fingers.

251. The epigenetic editor of claim 249, wherein the zinc finger array comprises at least three subsets of zinc fingers each comprising at least two zinc fingers.

252. The epigenetic editor of any one of claims 166-247, wherein the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide.

253. The epigenetic editor of claim 252, wherein the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide.

254. The epigenetic editor of claim 252, wherein the guide polynucleotide hybridizes with the target sequence.

255. The epigenetic editor of claim 253 or 254, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9).

256. The epigenetic editor of claim 253 or 254, wherein the CRISRP-Cas protein comprises a nuclease inactive Cas12a (dCas12a).

257. The epigenetic editor of claim 237 or 238, wherein the CRISRP-Cas protein comprises a nuclease inactive CasX (dCasX).

258. The epigenetic editor of any one of claims 248-257, wherein the epigenetic editor further comprises a second DNA binding domain that binds to a second target sequence in a second

target gene, and wherein the second DNA binding domain directs the epigenetic effector domain to effect an epigenetic modification in the second target gene or a histone bound to the second target gene.

259. The epigenetic editor of claim 258, wherein the second DNA binding domain comprises a zinc finger array.

260. The epigenetic editor of claim 259, wherein the zinc finger array comprises at least six zinc fingers.

261. The epigenetic editor of claim 259, wherein the zinc finger array comprises at least three subsets of zinc fingers each comprising at least two zinc fingers.

262. The epigenetic editor of claim 258, wherein the second DNA binding domain comprises a second nucleic acid guided DNA binding domain bound to a second guide polynucleotide.

263. The epigenetic editor of claim 262, wherein the second guide polynucleotide hybridizes with the second target sequence in the second target gene.

264. The method of any one of claims 258-263, wherein the second target gene is the same as the target gene.

265. The method of claim 264, wherein the second target sequence overlaps with the target sequence.

266. The method of claim 264 or 265, wherein the second target sequence is within 1000 base pairs flanking the target sequence.

267. The method of claim 264 or 265, wherein the second target sequence is within 500 base pairs flanking the target sequence.

268. The method of any one of claims 258-263, wherein the second target gene is different from the target gene.

269. The method of claim 268, wherein the target gene and the second target gene are associated with in a same metabolic pathway or function.

270. The method of claim 268, wherein the target gene and the second target gene are associated with a same disease or condition.

271. The epigenetic editor of any one of claims 166-270, wherein the epigenetic editor further comprises a linker.

272. The epigenetic editor of claim 271, wherein the linker is a peptide linker, thereby forming a fusion protein.

273. A nucleic acid encoding the fusion protein of claim 272.

274. A set of nucleic acids comprising a first nucleic acid encoding a first part and a second nucleic acid encoding a second part of the fusion protein of claim 272, wherein the first part and the second part comprise the fusion protein of claim 272 when combined.

275. The set of nucleic acids of claim 274, wherein the first nucleic acid further encodes a N terminal part of an intein and wherein the second nucleic acid further comprises a C terminal part of the intein.

276. A vector comprising the nucleic acid of claim 273.

277. A set of vectors comprising a first vector comprising the first nucleic acid of claim 274 and a second vector comprising the second nucleic acid of claim 274.

278. The vector of claim 276, wherein the vector is a virus vector.

279. The vector of claim 278, wherein the vector is a lentivirus vector, an adenovirus vector, a herpes virus vector, or an adeno-associated virus (AAV) vector.

280. The vector of claim 279, wherein the vector is an AAV1, AAV2, AAV3, AAV4, AAV5, AAV6, AAV7, AAV8, AAV9, or AAV10 vector.

281. The set of vectors of claim 277, wherein the first vector and the second vector are virus vectors.

282. The set of vectors of claim 277, wherein the first vector and the second vector are lentivirus vectors, adenovirus vectors, herpes virus vectors, or adeno-associated virus (AAV) vectors.

283. The vector of claim 279, wherein the first vector or the second vector is an AAV1, AAV2, AAV3, AAV4, AAV5, AAV6, AAV7, AAV8, AAV9, or AAV10 vector.

284. A cell comprising the epigenetic editor of any one of claims 161-272, the nucleic acid of claim 273, the set of nucleic acids of claim 274 or 275, the vector of any one of claims 276 or 278-280, or the set of vectors of any one of claims 277 or 281-283.

285. The cell of any one of claims 159-163 or 284, wherein the cell is a primary cell.

286. The cell of any one of claims 159-163 or 284, wherein the cell is a non-dividing cell.

287. The cell of any one of claims 159-163 or 284, wherein the cell is a stem cell.

288. The cell of any one of claims 159-163 or 284-287, wherein the cell is a mammalian cell.

289. The cell of claim 288, wherein the cell is a human cell.

290. The cell of any one of claims 285-289, wherein the cell is ex vivo or in vivo.

291. A composition comprising the epigenetic editor of any one of claims 161-272, the nucleic acid of claim 273, the set of nucleic acids of claim 274 or 275, the vector of any one of claims 276 or 278-280, the set of vectors of any one of claims 277 or 281-283, or the cell of any one of claims 284-290.

292. The composition of claim 291, further comprising a pharmaceutically acceptable carrier.

293. An Epigenetic Editor comprising:

a DNA binding domain capable of binding to a target sequence in a target chromosome and directing the Epigenetic Editor to repress or silence expression of a target gene;

one or more effector domains selected from the group consisting of a DNA methyltransferase domain and an effector domain that recruits a DNA methyltransferase; and

one or more effector domains selected from the group consisting of a histone methyltransferase domain that reduces transcription at the target gene, a histone demethylase domain that reduces transcription at the target gene, a histone deacetylase domain, an effector domain that recruits a histone methyltransferase that reduces transcription at the target gene, an effector domain that recruits a histone demethylase that reduces transcription at the target gene and an effector domain that recruits a histone deacetylase.

294. The Epigenetic Editor of claim 293, wherein the Epigenetic Editor further comprises one or more effector domains selected from the group consisting of a transcription repressor domain and an effector domain that recruits a transcriptional repressor.

295. The Epigenetic Editor of claim 294, wherein the transcriptional repressor domain or the effector domain that recruits a transcriptional repressor is not an effector domain from claims 293 (c).

296. The Epigenetic Editor of claims 293-295, wherein the effector domain from (c) is a KRAB repression domain.

297. The Epigenetic Editor of claim 296, wherein the KRAB repression domain is a KOX1/ZNF10 domain or a ZIM3 domain.

298. An Epigenetic Editor comprising:

a DNA binding domain capable of binding to a target sequence in a target chromosome and directing the Epigenetic Editor to increase expression of a target gene;

one or more effector domains selected from the group consisting of a DNA demethylase domain and an effector domain that recruits a DNA demethylase; and

one or more effector domains selected from the group consisting of a histone methyltransferase domain that increases transcription at the target gene, a histone demethylase domain that increases transcription at the target gene, a histone acetylase domain, an effector domain that recruits a histone methyltransferase that increases transcription at the target gene, an effector domain that recruits a histone demethylase that increases transcription at the target gene and an effector domain that recruits a histone acetylase.

299. The Epigenetic Editor of claim 298, wherein the Epigenetic Editor further comprises one or more effector domains selected from the group consisting of a transcription activation domain and an effector domain that recruits a transcription activator.

300. The Epigenic Editor of claim 299, wherein the selected effector domain is not an effector domain from claim 298 (c).

301. The Epigenetic Editor of claim 300, wherein the selected effector domain is a VP16 domain, a VP64 domain, a p65 domain, or ab RTA domain.

302. The Epigenetic Editor of claims 293-301, wherein the Epigenetic Editor is a polypeptide.

Sequence Tables

SEQ ID NO	Description	Sequence
1	S. pyogenes WT Cas9 NT Sequence	ATGGATAAGAAATACTCAATAGGCTTAGATATCGGCACAAATAGCGTCG GATGGGCGGTGATCACTGATGAATATAAGGTTCCGTCTAAAAAGTTCAA GGTTCTGGGAAATACAGACCGCCACAGTATCAAAAAAAATCTTATAGGG GCTCTTTTATTTGACAGTGGAGAGACAGCGGAAGCGACTCGTCTCAAAC GGACAGCTCGTAGAAGGTATACACGTCGGAAGAATCGTATTTGTTATCT ACAGGAGATTTTTTCAAATGAGATGGCGAAAGTAGATGATAGTTTCTTT CATCGACTTGAAGAGTCTTTTTTGGTGGAAGAAGACAAGAAGCATGAAC GTCATCCTATTTTTGGAAATATAGTAGATGAAGTTGCTTATCATGAGAA ATATCCAACTATCTATCATCTGCGAAAAAAATTGGTAGATTCTACTGAT AAAGCGGATTTGCGCTTAATCTATTTGGCCTTAGCGCATATGATTAAGTT TCGTGGTCATTTTTTGATTGAGGGAGATTTAAATCCTGATAATAGTGATG TGGACAAACTATTTATCCAGTTGGTACAAACCTACAATCAATTATTTGA AGAAAACCCTATTAACGCAAGTGGAGTAGATGCTAAAGCGATTCTTTCT GCACGATTGAGTAAATCAAGACGATTAGAAAATCTCATTGCTCAGCTCC CCGGTGAGAAGAAAAATGGCTTATTTGGGAATCTCATTGCTTTGTCATT GGGTTTGACCCCTAATTTTAAATCAAATTTTGATTTGGCAGAAGATGCTA AATTACAGCTTTCAAAAGATACTTACGATGATGATTTAGATAATTTATTG GCGCAAATTGGAGATCAATATGCTGATTTGTTTTTGGCAGCTAAGAATTT ATCAGATGCTATTTTACTTTCAGATATCCTAAGAGTAAATACTGAAATA ACTAAGGCTCCCCTATCAGCTTCAATGATTAAACGCTACGATGAACATC ATCAAGACTTGACTCTTTTAAAAGCTTTAGTTCGACAACAACTTCCAGA AAAGTATAAAGAAATCTTTTTTGATCAATCAAAAAACGGATATGCAGGT TATATTGATGGGGGAGCTAGCCAAGAAGAATTTTATAAATTTATCAAAC CAATTTTAGAAAAAATGGATGGTACTGAGGAATTATTGGTGAAACTAAA TCGTGAAGATTTGCTGCGCAAGCAACGGACCTTTGACAACGGCTCTATT CCCCATCAAATTCACTTGGGTGAGCTGCATGCTATTTTGAGAAGACAAG AAGACTTTTATCCATTTTTAAAAGACAATCGTGAGAAGATTGAAAAAAT CTTGACTTTTCGAATTCCTTATTATGTTGGTCCATTGGCGCGTGGCAATA GTCGTTTTGCATGGATGACTCGGAAGTCTGAAGAAACAATTACCCCATG GAATTTTGAAGAAGTTGTCGATAAAGGTGCTTCAGCTCAATCATTTATTG AACGCATGACAAACTTTGATAAAAATCTTCCAAATGAAAAAGTACTACC AAAACATAGTTTGCTTTATGAGTATTTTACGGTTTATAACGAATTGACAA AGGTCAAATATGTTACTGAAGGAATGCGAAAACCAGCATTTCTTTCAGG TGAACAGAAGAAAGCCATTGTTGATTTACTCTTCAAAACAAATCGAAAA GTAACCGTTAAGCAATTAAAAGAAGATTATTTCAAAAAAATAGAATGTT TTGATAGTGTTGAAATTTCAGGAGTTGAAGATAGATTTAATGCTTCATTA

SEQ ID NO	Description	Sequence
		GGTACCTACCATGATTTGCTAAAAATTATTAAAGATAAAGATTTTTTGG ATAATGAAGAAAATGAAGATATCTTAGAGGATATTGTTTTAACATTGAC CTTATTTGAAGATAGGGAGATGATTGAGGAAAGACTTAAAACATATGCT CACCTCTTTGATGATAAGGTGATGAAACAGCTTAAACGTCGCCGTTATA CTGGTTGGGGACGTTTGTCTCGAAAATTGATTAATGGTATTAGGGATAA GCAATCTGGCAAAACAATATTAGATTTTTTGAAATCAGATGGTTTTGCC AATCGCAATTTTATGCAGCTGATCCATGATGATAGTTTGACATTTAAAG AAGACATTCAAAAAGCACAAGTGTCTGGACAAGGCGATAGTTTACATG AACATATTGCAAATTTAGCTGGTAGCCCTGCTATTAAAAAAGGTATTTT ACAGACTGTAAAAGTTGTTGATGAATTGGTCAAAGTAATGGGGCGGCAT AAGCCAGAAAATATCGTTATTGAAATGGCACGTGAAAATCAGACAACTC AAAAGGGCCAGAAAAATTCGCGAGAGCGTATGAAACGAATCGAAGAAG GTATCAAAGAATTAGGAAGTCAGATTCTTAAAGAGCATCCTGTTGAAAA TACTCAATTGCAAAATGAAAAGCTCTATCTCTATTATCTCCAAAATGGA AGAGACATGTATGTGGACCAAGAATTAGATATTAATCGTTTAAGTGATT ATGATGTCGATCACATTGTTCCACAAAGTTTCCTTAAAGACGATTCAATA GACAATAAGGTCTTAACGCGTTCTGATAAAAATCGTGGTAAATCGGATA ACGTTCCAAGTGAAGAAGTAGTCAAAAAGATGAAAAACTATTGGAGAC AACTTCTAAACGCCAAGTTAATCACTCAACGTAAGTTTGATAATTTAAC GAAAGCTGAACGTGGAGGTTTGAGTGAACTTGATAAAGCTGGTTTTATC AAACGCCAATTGGTTGAAACTCGCCAAATCACTAAGCATGTGGCACAAA TTTTGGATAGTCGCATGAATACTAAATACGATGAAAATGATAAACTTAT TCGAGAGGTTAAAGTGATTACCTTAAAATCTAAATTAGTTTCTGACTTCC GAAAAGATTTCCAATTCTATAAAGTACGTGAGATTAACAATTACCATCA TGCCCATGATGCGTATCTAAATGCCGTCGTTGGAACTGCTTTGATTAAGA AATATCCAAAACTTGAATCGGAGTTTGTCTATGGTGATTATAAAGTTTAT GATGTTCGTAAAATGATTGCTAAGTCTGAGCAAGAAATAGGCAAAGCA ACCGCAAAATATTTCTTTTACTCTAATATCATGAACTTCTTCAAAACAGA AATTACACTTGCAAATGGAGAGATTCGCAAACGCCCTCTAATCGAAACT AATGGGGAAACTGGAGAAATTGTCTGGGATAAAGGGCGAGATTTTGCC ACAGTGCGCAAAGTATTGTCCATGCCCCAAGTCAATATTGTCAAGAAAA CAGAAGTACAGACAGGCGGATTCTCCAAGGAGTCAATTTTACCAAAAA GAAATTCGGACAAGCTTATTGCTCGTAAAAAAGACTGGGATCCAAAAA AATATGGTGGTTTTGATAGTCCAACGGTAGCTTATTCAGTCCTAGTGGTT GCTAAGGTGGAAAAAGGGAAATCGAAGAAGTTAAAATCCGTTAAAGAG TTACTAGGGATCACAATTATGGAAAGAAGTTCCTTTGAAAAAAATCCGA TTGACTTTTTAGAAGCTAAAGGATATAAGGAAGTTAAAAAAGACTTAAT CATTAAACTACCTAAATATAGTCTTTTTGAGTTAGAAAACGGTCGTAAA CGGATGCTGGCTAGTGCCGGAGAATTACAAAAAGGAAATGAGCTGGCT CTGCCAAGCAAATATGTGAATTTTTTATATTTAGCTAGTCATTATGAAAA GTTGAAGGGTAGTCCAGAAGATAACGAACAAAAACAATTGTTTGTGGA GCAGCATAAGCATTATTTAGATGAGATTATTGAGCAAATCAGTGAATTT TCTAAGCGTGTTATTTTAGCAGATGCCAATTTAGATAAAGTTCTTAGTGC ATATAACAAACATAGAGACAAACCAATACGTGAACAAGCAGAAAATAT TATTCATTTATTTACGTTGACGAATCTTGGAGCTCCCGCTGCTTTTAAAT ATTTTGATACAACAATTGATCGTAAACGATATACGTCTACAAAAGAAGT TTTAGATGCCACTCTTATCCATCAATCCATCACTGGTCTTTATGAAACAC GCATTGATTTGAGTCAGCTAGGAGGTGACTGA
2	S. pyogenes WT Cas9	MDKKYSIGLDIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD

SEQ ID NO	Description	Sequence
	AA Sequence	AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDHIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTST KEVLDATLIHQSITGLYETRIDLSQLGGD
3	dCas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTST KEVLDATLIHQSITGLYETRIDLSQLGGD

SEQ ID NO	Description	Sequence
4	inactive VRER SpCas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFVSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASARELQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKEYRST KEVLDATLIHQSITGLYETRIDLSQLGGD
5	inactive EQR SpCas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFESPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL

SEQ ID NO	Description	Sequence
		DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKQYRST KEVLDATLIHQSITGLYETRIDLSQLGGD
6	inactive VQR SpCas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFVSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKQYRST KEVLDATLIHQSITGLYETRIDLSQLGGD
7	inactive SPG SpCas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFLWPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA

SEQ ID NO	Description	Sequence
		KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAKQLQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKQYRST KEVLDATLIHQSITGLYETRIDLSQLGGD
8	inactive SpRY Cas9	MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGAL LFDSGETAERTRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIY LALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVD AKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAE DAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEI TKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYI DGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIH LGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTR KSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTL TLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIA NLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQK NSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVK KMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQIT KHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEI GKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFA TVRKVLSMPQVNIVKKTEVQTGGFSKESIRPKRNSDKLIARKKDWDPKKYG GFLWPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEA KGYKEVKKDLIIKLPKYSLFELENGRKRMLASAKQLQKGNELALPSKYVNF LYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANL DKVLSAYNKHRDKPIREQAENIIHLFTLTRLGAPRAFKYFDTTIDPKQYRST KEVLDATLIHQSITGLYETRIDLSQLGGD
9	SaCas9	MKRNYILGLDIGITSVGYGIIDYETRDVIDAGVRLFKEANVENNEGRRSKRG ARRLKRRRRHRIQRVKKLLFDYNLLTDHSELSGINPYEARVKGLSQKLSEEE FSAALLHLAKRRGVHNVNEVEEDTGNELSTKEQISRNSKALEEKYVAELQL ERLKKDGEVRGSINRFKTSDYVKEAKQLLKVQKAYHQLDQSFIDTYIDLLE TRRTYYEGPGEGSPFGWKDIKEWYEMLMGHCTYFPEELRSVKYAYNADLY NALNDLNNLVITRDENEKLEYYEKFQIIENVFKQKKKPTLKQIAKEILVNEE DIKGYRVTSTGKPEFTNLKVYHDIKDITARKEIIENAELLDQIAKILTIYQSSE DIQEELTNLNSELTQEEIEQISNLKGYTGTHNLSLKAINLILDELWHTNDNQI AIFNRLKLVPKKVDLSQQKEIPTTLVDDFILSPVVKRSFIQSIKVINAIIKKYG LPNDIIIELAREKNSKDAQKMINEMQKRNRQTNERIEEIIRTTGKENAKYLIE KIKLHDMQEGKCLYSLEAIPLEDLLNNPFNYEVDHIIPRSVSFDNSFNNKVL VKQEEASKKGNRTPFQYLSSSDSKISYETFKKHILNLAKGKGRISKTKKEYL LEERDINRFSVQKDFINRNLVDTRYATRGLMNLLRSYFRVNNLDVKVKSIN GGFTSFLRRKWKFKKERNKGYKHHAEDALIIANADFIFKEWKKLDKAKKV MENQMFEEKQAESMPEIETEQEYKEIFITPHQIKHIKDFKDYKYSHRVDKKP NRELINDTLYSTRKDDKGNTLIVNNLNGLYDKDNDKLKKLINKSPEKLLMY HHDPQTYQKLKLIMEQYGDEKNPLYKYYEETGNYLTKYSKKDNGPVIKKI KYYGNKLNAHLDITDDYPNSRNKVVKLSLKPYRFDVYLDNGVYKFVTVK NLDVIKKENYYEVNSKAYEEAKKLKKISNQAEFIASFYNNDLIKINGELYRV IGVNNDLLNRIEVNMIDITYREYLENMNDKRPPRIIKTIASKTQSIKKYSTDIL GNLYEVKSKKHPQIIKKG

SEQ ID NO	Description	Sequence
10	inactive KKH dSaCas9	MKRNYILGLAIGITSVGYGIIDYETRDVIDAGVRLFKEANVENNEGRRSKRG ARRLKRRRRHRIQRVKKLLFDYNLLTDHSELSGINPYEARVKGLSQKLSEEE FSAALLHLAKRRGVHNVNEVEEDTGNELSTKEQISRNSKALEEKYVAELQL ERLKKDGEVRGSINRFKTSDYVKEAKQLLKVQKAYHQLDQSFIDTYIDLLE TRRTYYEGPGEGSPFGWKDIKEWYEMLMGHCTYFPEELRSVKYAYNADLY NALNDLNNLVITRDENEKLEYYEKFQIIENVFKQKKKPTLKQIAKEILVNEE DIKGYRVTSTGKPEFTNLKVYHDIKDITARKEIIENAELLDQIAKILTIYQSSE DIQEELTNLNSELTQEEIEQISNLKGYTGTHNLSLKAINLILDELWHTNDNQI AIFNRLKLVPKKVDLSQQKEIPTTLVDDFILSPVVKRSFIQSIKVINAIIKKYG LPNDIIIELAREKNSKDAQKMINEMQKRNRQTNERIEEIIRTTGKENAKYLIE KIKLHDMQEGKCLYSLEAIPLEDLLNNPFNYEVDHIIPRSVSFDNSFNNKVL VKQEEASKKGNRTPFQYLSSSDSKISYETFKKHILNLAKGKGRISKTKKEYL LEERDINRFSVQKDFINRNLVDTRYATRGLMNLLRSYFRVNNLDVKVKSIN GGFTSFLRRKWKFKKERNKGYKHHAEDALIIANADFIFKEWKKLDKAKKV MENQMFEEKQAESMPEIETEQEYKEIFITPHQIKHIKDFKDYKYSHRVDKKP NRKLINDTLYSTRKDDKGNTLIVNNLNGLYDKDNDKLKKLINKSPEKLLMY HHDPQTYQKLKLIMEQYGDEKNPLYKYYEETGNYLTKYSKKDNGPVIKKI KYYGNKLNAHLDITDDYPNSRNKVVKLSLKPYRFDVYLDNGVYKFVTVK NLDVIKKENYYEVNSKCYEEAKKLKKISNQAEFIASFYKNDLIKINGELYRV IGVNNDLLNRIEVNMIDITYREYLENMNDKRPPHIIKTIASKTQSIKKYSTDIL GNLYEVKSKKHPQIIKKG
11	dNmeCas9	MAAFKPNSINYILGLAIGIASVGWAMVEIDEEENPIRLIDLGVRVFERAEVPK TGDSLAMARRLARSVRRLTRRRAHRLLRTRRLLKREGVLQAANFDENGLI KSLPNTPWQLRAAALDRKLTPLEWSAVLLHLIKHRGYLSQRKNEGETADK ELGALLKGVAGNAHALQTGDFRTPAELALNKFEKESGHIRNQRSDYSHTFS RKDLQAELILLFEKQKEFGNPHVSGGLKEGIETLLMTQRPALSGDAVQKML GHCTFEPAEPKAAKNTYTAERFIWLTKLNNLRILEQGSERPLTDTERATLMD EPYRKSKLTYAQARKLLGLEDTAFFKGLRYGKDNAEASTLMEMKAYHAIS RALEKEGLKDKKSPLNLSPELQDEIGTAFSLFKTDEDITGRLKDRIQPEILEA LLKHISFDKFVQISLKALRRIVPLMEQGKRYDEACAEIYGDHYGKKNTEEKI YLPPIPADEIRNPVVLRALSQARKVINGVVRRYGSPARIHIETAREVGKSFKD RKEIEKRQEENRKDREKAAAKFREYFPNFVGEPKSKDILKLRLYEQQHGKC LYSGKEINLGRLNEKGYVEIDAALPFSRTWDDSFNNKVLVLGSENQNKGNQ TPYEYFNGKDNSREWQEFKARVETSRFPRSKKQRILLQKFDEDGFKERNLN DTRYVNRFLCQFVADRMRLTGKGKKRVFASNGQITNLLRGFWGLRKVRAE NDRHHALDAVVVACSTVAMQQKITRFVRYKEMNAFDGKTIDKETGEVLH QKTHFPQPWEFFAQEVMIRVFGKPDGKPEFEEADTLEKLRTLLAEKLSSRPE AVHEYVTPLFVSRAPNRKMSGQGHMETVKSAKRLDEGVSVLRVPLTQLKL KDLEKMVNREREPKLYEALKARLEAHKDDPAKAFAEPFYKYDKAGNRTQ QVKAVRVEQVQKTGVWVRNHNGIADNATMVRVDVFEKGDKYYLVPIYS WQVAKGILPDRAVVQGKDEEDWQLIDDSFNFKFSLHPNDLVEVITKKARM FGYFASCHRGTGNINIRIHDLDHKIGKNGILEGIGVKTALSFQKYQIDELGKE IRPCRLKKRPPVR
12	dCjCas9	MARILAFAIGISSIGWAFSENDELKDCGVRIFTKVENPKTGESLALPRRLARS ARKRLARRKARLNHLKHLIANEFKLNYEDYQSFDESLAKAYKGSLISPYEL RFRALNELLSKQDFARVILHIAKRRGYDDIKNSDDKEKGAILKAIKQNEEKL ANYQSVGEYLYKEYFQKFKENSKEFTNVRNKKESYERCIAQSFLKDELKLIF KKQREFGFSFSKKFEEEVLSVAFYKRALKDFSHLVGNCSFFTDEKRAPKNSP LAFMFVALTRIINLLNNLKNTEGILYTKDDLNALLNEVLKNGTLTYKQTKK LLGLSDDYEFKGEKGTYFIEFKKYKEFIKALGEHNLSQDDLNEIAKDITLIKD EIKLKKALAKYDLNQNQIDSLSKLEFKDHLNISFKALKLVTPLMLEGKKYD EACNELNLKVAINEDKKDFLPAFNETYYKDEVTNPVVLRAIKEYRKVLNAL

SEQ ID NO	Description	Sequence
		LKKYGKVHKINIELAREVGKNHSQRAKIEKEQNENYKAKKDAELECEKLG LKINSKNILKLRLFKEQKEFCAYSGEKIKISDLQDEKMLEIDAIYPYSRSFDDS YMNKVLVFTKQNQEKLNQTPFEAFGNDSAKWQKIEVLAKNLPTKKQKRIL DKNYKDKEQKNFKDRNLNDTRYIARLVLNYTKDYLDFLPLSDDENTKLND TQKGSKVHVEAKSGMLTSALRHTWGFSAKDRNNHLHHAIDAVIIAYANNSI VKAFSDFKKEQESNSAELYAKKISELDYKNKRKFFEPFSGFRQKVLDKIDEI FVSKPERKKPSGALHEETFRKEEEFYQSYGGKEGVLKALELGKIRKVNGKI VKNGDMFRVDIFKHKKTNKFYAVPIYTMDFALKVLPNKAVARSKKGEIKD WILMDENYEFCFSLYKDSLILIQTKDMQEPEFVYYNAFTSSTVSLIVSKHDN KFETLSKNQKILFKNANEKEVIAKSIGIQNLKVFEKYIVSALGEVTKAEFRQR EDFKK
13	dSt1Cas9	MGSDLVLGLAIGIGSVGVGILNKVTGEIIHKNSRIFPAAQAENNLVRRTNRQ GRRLARRKKHRRVRLNRLFEESGLITDFTKISINLNPYQLRVKGLTDELSNE ELFIALKNMVKHRGISYLDDASDDGNSSVGDYAQIVKENSKQLETKTPGQI QLERYQTYGQLRGDFTVEKDGKKHRLINVFPTSAYRSEALRILQTQQEFNP QITDEFINRYLEILTGKRKYYHGPGNEKSRTDYGRYRTSGETLDNIFGILIGK CTFYPDEFRAAKASYTAQEFNLLNDLNNLTVPTETKKLSKEQKNQIINYVK NEKAMGPAKLFKYIAKLLSCDVADIKGYRIDKSGKAEIHTFEAYRKMKTLE TLDIEQMDRETLDKLAYVLTLNTEREGIQEALEHEFADGSFSQKQVDELVQ FRKANSSIFGKGWHNFSVKLMMELIPELYETSEEQMTILTRLGKQKTTSSSN KTKYIDEKLLTEEIYNPVVAKSVRQAIKIVNAAIKEYGDFDNIVIEMARETNE DDEKKAIQKIQKANKDEKDAAMLKAANQYNGKAELPHSVFHGHKQLATK IRLWHQQGERCLYTGKTISIHDLINNSNQFEVDAILPLSITFDDSLANKVLVY ATANQEKGQRTPYQALDSMDDAWSFRELKAFVRESKTLSNKKKEYLLTEE DISKFDVRKKFIERNLVDTRYASRVVLNALQEHFRAHKIDTKVSVVRGQFT SQLRRHWGIEKTRDTYHHHAVDALIIAASSQLNLWKKQKNTLVSYSEDQLL DIETGELISDDEYKESVFKAPYQHFVDTLKSKEFEDSILFSYQVDSKFNRKIS DATIYATRQAKVGKDKADETYVLGKIKDIYTQDGYDAFMKIYKKDKSKFL MYRHDPQTFEKVIEPILENYPNKQINEKGKEVPCNPFLKYKEEHGYIRKYSK KGNGPEIKSLKYYDSKLGNHIDITPKDSNNKVVLQSVSPWRADVYFNKTTG KYEILGLKYADLQFEKGTGTYKISQEKYNDIKKKEGVDSDSEFKFTLYKND LLLVKDTETKEQQLFRFLSRTMPKQKHYVELKPYDKQKFEGGEALIKVLGN VANSGQCKKGLGKSNISIYKVRTDVLGNQHIIKNEGDKPKLDF
14	dSt3Cas9	MTKPYSIGLAIGTNSVGWAVITDNYKVPSKKMKVLGNTSKKYIKKNLLGV LLFDSGITAEGRRLKRTARRRYTRRRNRILYLQEIFSTEMATLDDAFFQRLD DSFLVPDDKRDSKYPIFGNLVEEKVYHDEFPTIYHLRKYLADSTKKADLRL VYLALAHMIKYRGHFLIEGEFNSKNNDIQKNFQDFLDTYNAIFESDLSLENS KQLEEIVKDKISKLEKKDRILKLFPGEKNSGIFSEFLKLIVGNQADFRKCFNL DEKASLHFSKESYDEDLETLLGYIGDDYSDVFLKAKKLYDAILLSGFLTVTD NETEAPLSSAMIKRYNEHKEDLALLKEYIRNISLKTYNEVFKDDTKNGYAG YIDGKTNQEDFYVYLKNLLAEFEGADYFLEKIDREDFLRKQRTFDNGSIPYQ IHLQEMRAILDKQAKFYPFLAKNKERIEKILTFRIPYYVGPLARGNSDFAWSI RKRNEKITPWNFEDVIDKESSAEAFINRMTSFDLYLPEEKVLPKHSLLYETF NVYNELTKVRFIAESMRDYQFLDSKQKKDIVRLYFKDKRKVTDKDIIEYLH AIYGYDGIELKGIEKQFNSSLSTYHDLLNIINDKEFLDDSSNEAIIEEIIHTLTIF EDREMIKQRLSKFENIFDKSVLKKLSRRHYTGWGKLSAKLINGIRDEKSGNT ILDYLIDDGISNRNFMQLIHDDALSFKKKIQKAQIIGDEDKGNIKEVVKSLPG SPAIKKGILQSIKIVDELVKVMGGRKPESIVVEMARENQYTNQGKSNSQQRL KRLEKSLKELGSKILKENIPAKLSKIDNNALQNDRLYLYYLQNGKDMYTGD DLDIDRLSNYDIDHIIPQAFLKDNSIDNKVLVSSASARGKSDDFPSLEVVKKR KTFWYQLLKSKLISQRKFDNLTKAERGGLLPEDKAGFIQRQLVETRQITKH VARLLDEKFNNKKDENNRAVRTVKIITLKSTLVSQFRKDFELYKVREINDFH

SEQ ID NO	Description	Sequence
		HAHDAYLNAVIASALLKKYPKLEPEFVYGDYPKYNSFRERKSATEKVYFYS NIMNIFKKSISLADGRVIERPLIEVNEETGESVWNKESDLATVRRVLSYPQV NVVKKVEEQNHGLDRGKPKGLFNANLSSKPKPNSNENLVGAKEYLDPKKY GGYAGISNSFAVLVKGTIEKGAKKKITNVLEFQGISILDRINYRKDKLNFLLE KGYKDIELIIELPKYSLFELSDGSRRMLASILSTNNKRGEIHKGNQIFLSQKFV KLLYHAKRISNTINENHRKYVENHKKEFEELFYYILEFNENYVGAKKNGKL LNSAFQSWQNHSIDELCSSFIGPTGSERKGLFELTSRGSAADFEFLGVKIPRY RDYTPSSLLKDATLIHQSVTGLYETRIDLAKLGEG
15	F. novicida WT Cpf1	MSIYQEFVNKYSLSKTLRFELIPQGKTLENIKARGLILDDEKRAKDYKKAKQ IIDKYHQFFIEEILSSVCISEDLLQNYSDVYFKLKKSDDDNLQKDFKSAKDTI KKQISEYIKDSEKFKNLFNQNLIDAKKGQESDLILWLKQSKDNGIELFKANS DITDIDEALEIIKSFKGWTTYFKGFHENRKNVYSSNDIPTSIIYRIVDDNLPKF LENKAKYESLKDKAPEAINYEQIKKDLAEELTFDIDYKTSEVNQRVFSLDEV FEIANFNNYLNQSGITKFNTIIGGKFVNGENTKRKGINEYINLYSQQINDKTL KKYKMSVLFKQILSDTESKSFVIDKLEDDSDVVTTMQSFYEQIAAFKTVEEK SIKETLSLLFDDLKAQKLDLSKIYFKNDKSLTDLSQQVFDDYSVIGTAVLEYI TQQIAPKNLDNPSKKEQELIAKKTEKAKYLSLETIKLALEEFNKHRDIDKQC RFEEILANFAAIPMIFDEIAQNKDNLAQISIKYQNQGKKDLLQASAEDDVKAI KDLLDQTNNLLHKLKIFHISQSEDKANILDKDEHFYLVFEECYFELANIVPL YNKIRNYITQKPYSDEKFKLNFENSTLANGWDKNKEPDNTAILFIKDDKYY LGVMNKKNNKIFDDKAIKENKGEGYKKIVYKLLPGANKMLPKVFFSAKSIK FYNPSEDILRIRNHSTHTKNGSPQKGYEKFEFNIEDCRKFIDFYKQSISKHPE WKDFGFRFSDTQRYNSIDEFYREVENQGYKLTFENISESYIDSVVNQGKLYL FQIYNKDFSAYSKGRPNLHTLYWKALFDERNLQDVVYKLNGEAELFYRKQ SIPKKITHPAKEAIANKNKDNPKKESVFEYDLIKDKRFTEDKFFFHCPITINFK SSGANKFNDEINLLLKEKANDVHILSIDRGERHLAYYTLVDGKGNIIKQDTF NIIGNDRMKTNYHDKLAAIEKDRDSARKDWKKINNIKEMKEGYLSQVVHEI AKLVIEYNAIVVFEDLNFGFKRGRFKVEKQVYQKLEKMLIEKLNYLVFKDN EFDKTGGVLRAYQLTAPFETFKKMGKQTGIIYYVPAGFTSKICPVTGFVNQL YPKYESVSKSQEFFSKFDKICYNLDKGYFEFSFDYKNFGDKAAKGKWTIAS FGSRLINFRNSDKNHNWDTREVYPTKELEKLLKDYSIEYGHGECIKAAICGE SDKKFFAKLTSVLNTILQMRNSKTGTELDYLISPVADVNGNFFDSRQAPKN MPQDADANGAYHIGLKGLMLLGRIKNNQEGKKLNLVIKNEEYFEFVQNRN N
16	inactive FnCpf1	MSIYQEFVNKYSLSKTLRFELIPQGKTLENIKARGLILDDEKRAKDYKKAKQ IIDKYHQFFIEEILSSVCISEDLLQNYSDVYFKLKKSDDDNLQKDFKSAKDTI KKQISEYIKDSEKFKNLFNQNLIDAKKGQESDLILWLKQSKDNGIELFKANS DITDIDEALEIIKSFKGWTTYFKGFHENRKNVYSSNDIPTSIIYRIVDDNLPKF LENKAKYESLKDKAPEAINYEQIKKDLAEELTFDIDYKTSEVNQRVFSLDEV FEIANFNNYLNQSGITKFNTIIGGKFVNGENTKRKGINEYINLYSQQINDKTL KKYKMSVLFKQILSDTESKSFVIDKLEDDSDVVTTMQSFYEQIAAFKTVEEK SIKETLSLLFDDLKAQKLDLSKIYFKNDKSLTDLSQQVFDDYSVIGTAVLEYI TQQIAPKNLDNPSKKEQELIAKKTEKAKYLSLETIKLALEEFNKHRDIDKQC RFEEILANFAAIPMIFDEIAQNKDNLAQISIKYQNQGKKDLLQASAEDDVKAI KDLLDQTNNLLHKLKIFHISQSEDKANILDKDEHFYLVFEECYFELANIVPL YNKIRNYITQKPYSDEKFKLNFENSTLANGWDKNKEPDNTAILFIKDDKYY LGVMNKKNNKIFDDKAIKENKGEGYKKIVYKLLPGANKMLPKVFFSAKSIK FYNPSEDILRIRNHSTHTKNGSPQKGYEKFEFNIEDCRKFIDFYKQSISKHPE WKDFGFRFSDTQRYNSIDEFYREVENQGYKLTFENISESYIDSVVNQGKLYL FQIYNKDFSAYSKGRPNLHTLYWKALFDERNLQDVVYKLNGEAELFYRKQ SIPKKITHPAKEAIANKNKDNPKKESVFEYDLIKDKRFTEDKFFFHCPITINFK SSGANKFNDEINLLLKEKANDVHILSIARGERHLAYYTLVDGKGNIIKQDTF

SEQ ID NO	Description	Sequence
		NIIGNDRMKTNYHDKLAAIEKDRDSARKDWKKINNIKEMKEGYLSQVVHEI AKLVIEYNAIVVFEDLNFGFKRGRFKVEKQVYQKLEKMLIEKLNYLVFKDN EFDKTGGVLRAYQLTAPFETFKKMGKQTGIIYYVPAGFTSKICPVTGFVNQL YPKYESVSKSQEFFSKFDKICYNLDKGYFEFSFDYKNFGDKAAKGKWTIAS FGSRLINFRNSDKNHNWDTREVYPTKELEKLLKDYSIEYGHGECIKAAICGE SDKKFFAKLTSVLNTILQMRNSKTGTELDYLISPVADVNGNFFDSRQAPKN MPQDADANGAYHIGLKGLMLLGRIKNNQEGKKLNLVIKNEEYFEFVQNRN N
17	inactive dLbCpf1	MSKLEKFTNCYSLSKTLRFKAIPVGKTQENIDNKRLLVEDEKRAEDYKGVK KLLDRYYLSFINDVLHSIKLKNLNNYISLFRKKTRTEKENKELENLEINLRKE IAKAFKGNEGYKSLFKKDIIETILPEFLDDKDEIALVNSFNGFTTAFTGFFDN RENMFSEEAKSTSIAFRCINENLTRYISNMDIFEKVDAIFDKHEVQEIKEKILN SDYDVEDFFEGEFFNFVLTQEGIDVYNAIIGGFVTESGEKIKGLNEYINLYNQ KTKQKLPKFKPLYKQVLSDRESLSFYGEGYTSDEEVLEVFRNTLNKNSEIFS SIKKLEKLFKNFDEYSSAGIFVKNGPAISTISKDIFGEWNVIRDKWNAEYDDI HLKKKAVVTEKYEDDRRKSFKKIGSFSLEQLQEYADADLSVVEKLKEIIIQK VDEIYKVYGSSEKLFDADFVLEKSLKKNDAVVAIMKDLLDSVKSFENYIKA FFGEGKETNRDESFYGDFVLAYDILLKVDHIYDAIRNYVTQKPYSKDKFKL YFQNPQFMGGWDKDKETDYRATILRYGSKYYLAIMDKKYAKCLQKIDKD DVNGNYEKINYKLLPGPNKMLPKVFFSKKWMAYYNPSEDIQKIYKNGTFK KGDMFNLNDCHKLIDFFKDSISRYPKWSNAYDFNFSETEKYKDIAGFYREV EEQGYKVSFESASKKEVDKLVEEGKLYMFQIYNKDFSDKSHGTPNLHTMY FKLLFDENNHGQIRLSGGAELFMRRASLKKEELVVHPANSPIANKNPDNPK KTTTLSYDVYKDKRFSEDQYELHIPIAINKCPKNIFKINTEVRVLLKHDDNPY VIGIARGERNLLYIVVVDGKGNIVEQYSLNEIINNFNGIRIKTDYHSLLDKKE KERFEARQNWTSIENIKELKAGYISQVVHKICELVEKYDAVIALEDLNSGFK NSRVKVEKQVYQKFEKMLIDKLNYMVDKKSNPCATGGALKGYQITNKFES FKSMSTQNGFIFYIPAWLTSKIDPSTGFVNLLKTKYTSIADSKKFISSFDRIMY VPEEDLFEFALDYKNFSRTDADYIKKWKLYSYGNRIRIFRNPKKNNVFDWE EVCLTSAYKELFNKYGINYQQGDIRALLCEQSDKAFYSSFMALMSLMLQM RNSITGRTDVDFLISPVKNSDGIFYDSRNYEAQENAILPKNADANGAYNIAR KVLWAIGQFKKAEDEKLDKVKIAISNKEWLEYAQTSVKH
18	inactive AsCpf1	MTQFEGFTNLYQVSKTLRFELIPQGKTLKHIQEQGFIEEDKARNDHYKELKP IIDRIYKTYADQCLQLVQLDWENLSAAIDSYRKEKTEETRNALIEEQATYRN AIHDYFIGRTDNLTDAINKRHAEIYKGLFKAELFNGKVLKQLGTVTTTEHEN ALLRSFDKFTTYFSGFYENRKNVFSAEDISTAIPHRIVQDNFPKFKENCHIFT RLITAVPSLREHFENVKKAIGIFVSTSIEEVFSFPFYNQLLTQTQIDLYNQLLG GISREAGTEKIKGLNEVLNLAIQKNDETAHIIASLPHRFIPLFKQILSDRNTLSF ILEEFKSDEEVIQSFCKYKTLLRNENVLETAEALFNELNSIDLTHIFISHKKLE TISSALCDHWDTLRNALYERRISELTGKITKSAKEKVQRSLKHEDINLQEIIS AAGKELSEAFKQKTSEILSHAHAALDQPLPTTLKKQEEKEILKSQLDSLLGL YHLLDWFAVDESNEVDPEFSARLTGIKLEMEPSLSFYNKARNYATKKPYSV EKFKLNFQMPTLASGWDVNKEKNNGAILFVKNGLYYLGIMPKQKGRYKA LSFEPTEKTSEGFDKMYYDYFPDAAKMIPKCSTQLKAVTAHFQTHTTPILLS NNFIEPLEITKEIYDLNNPEKEPKKFQTAYAKKTGDQKGYREALCKWIDFTR DFLSKYTKTTSIDLSSLRPSSQYKDLGEYYAELNPLLYHISFQRIAEKEIMDA VETGKLYLFQIYNKDFAKGHHGKPNLHTLYWTGLFSPENLAKTSIKLNGQA ELFYRPKSRMKRMAHRLGEKMLNKKLKDQKTPIPDTLYQELYDYVNHRLS HDLSDEARALLPNVITKEVSHEIIKDRRFTSDKFFFHVPITLNYQAANSPSKF NQRVNAYLKEHPETPIIGIARGERNLIYITVIDSTGKILEQRSLNTIQQFDYQK KLDNREKERVAARQAWSVVGTIKDLKQGYLSQVIHEIVDLMIHYQAVVVL ENLNFGFKSKRTGIAEKAVYQQFEKMLIDKLNCLVLKDYPAEKVGGVLNP

SEQ ID NO	Description	Sequence
		YQLTDQFTSFAKMGTQSGFLFYVPAPYTSKIDPLTGFVDPFVWKTIKNHESR KHFLEGFDFLHYDVKTGDFILHFKMNRNLSFQRGLPGFMPAWDIVFEKNET QFDAKGTPFIAGKRIVPVIENHRFTGRYRDLYPANELIALLEEKGIVFRDGSN ILPKLLENDDSHAIDTMVALIRSVLQMRNSNAATGEDYINSPVRDLNGVCF DSRFQNPEWPMDADANGAYHIALKGQLLLNHLKESKDLKLQNGISNQDWL AYIQELRN
19	inactive enAsCpf1	MTQFEGFTNLYQVSKTLRFELIPQGKTLKHIQEQGFIEEDKARNDHYKELKP IIDRIYKTYADQCLQLVQLDWENLSAAIDSYRKEKTEETRNALIEEQATYRN AIHDYFIGRTDNLTDAINKRHAEIYKGLFKAELFNGKVLKQLGTVTTTEHEN ALLRSFDKFTTYFSGFYRNRKNVFSAEDISTAIPHRIVQDNFPKFKENCHIFT RLITAVPSLREHFENVKKAIGIFVSTSIEEVFSFPFYNQLLTQTQIDLYNQLLG GISREAGTEKIKGLNEVLNLAIQKNDETAHIIASLPHRFIPLFKQILSDRNTLSF ILEEFKSDEEVIQSFCKYKTLLRNENVLETAEALFNELNSIDLTHIFISHKKLE TISSALCDHWDTLRNALYERRISELTGKITKSAKEKVQRSLKHEDINLQEIIS AAGKELSEAFKQKTSEILSHAHAALDQPLPTTLKKQEEKEILKSQLDSLLGL YHLLDWFAVDESNEVDPEFSARLTGIKLEMEPSLSFYNKARNYATKKPYSV EKFKLNFQMPTLARGWDVNREKNNGAILFVKNGLYYLGIMPKQKGRYKA LSFEPTEKTSEGFDKMYYDYFPDAAKMIPKCSTQLKAVTAHFQTHTTPILLS NNFIEPLEITKEIYDLNNPEKEPKKFQTAYAKKTGDQKGYREALCKWIDFTR DFLSKYTKTTSIDLSSLRPSSQYKDLGEYYAELNPLLYHISFQRIAEKEIMDA VETGKLYLFQIYNKDFAKGHHGKPNLHTLYWTGLFSPENLAKTSIKLNGQA ELFYRPKSRMKRMAHRLGEKMLNKKLKDQKTPIPDTLYQELYDYVNHRLS HDLSDEARALLPNVITKEVSHEIIKDRRFTSDKFFFHVPITLNYQAANSPSKF NQRVNAYLKEHPETPIIGIARGERNLIYITVIDSTGKILEQRSLNTIQQFDYQK KLDNREKERVAARQAWSVVGTIKDLKQGYLSQVIHEIVDLMIHYQAVVVL ENLNFGFKSKRTGIAEKAVYQQFEKMLIDKLNCLVLKDYPAEKVGGVLNP YQLTDQFTSFAKMGTQSGFLFYVPAPYTSKIDPLTGFVDPFVWKTIKNHESR KHFLEGFDFLHYDVKTGDFILHFKMNRNLSFQRGLPGFMPAWDIVFEKNET QFDAKGTPFIAGKRIVPVIENHRFTGRYRDLYPANELIALLEEKGIVFRDGSN ILPKLLENDDSHAIDTMVALIRSVLQMRNSNAATGEDYINSPVRDLNGVCF DSRFQNPEWPMDADANGAYHIALKGQLLLNHLKESKDLKLQNGISNQDWL AYIQELRN
20	inactive HFAsCpf1	MTQFEGFTNLYQVSKTLRFELIPQGKTLKHIQEQGFIEEDKARNDHYKELKP IIDRIYKTYADQCLQLVQLDWENLSAAIDSYRKEKTEETRNALIEEQATYRN AIHDYFIGRTDNLTDAINKRHAEIYKGLFKAELFNGKVLKQLGTVTTTEHEN ALLRSFDKFTTYFSGFYRNRKNVFSAEDISTAIPHRIVQDNFPKFKENCHIFT RLITAVPSLREHFENVKKAIGIFVSTSIEEVFSFPFYNQLLTQTQIDLYNQLLG GISREAGTEKIKGLNEVLALAIQKNDETAHIIASLPHRFIPLFKQILSDRNTLSF ILEEFKSDEEVIQSFCKYKTLLRNENVLETAEALFNELNSIDLTHIFISHKKLE TISSALCDHWDTLRNALYERRISELTGKITKSAKEKVQRSLKHEDINLQEIIS AAGKELSEAFKQKTSEILSHAHAALDQPLPTTLKKQEEKEILKSQLDSLLGL YHLLDWFAVDESNEVDPEFSARLTGIKLEMEPSLSFYNKARNYATKKPYSV EKFKLNFQMPTLARGWDVNREKNNGAILFVKNGLYYLGIMPKQKGRYKA LSFEPTEKTSEGFDKMYYDYFPDAAKMIPKCSTQLKAVTAHFQTHTTPILLS NNFIEPLEITKEIYDLNNPEKEPKKFQTAYAKKTGDQKGYREALCKWIDFTR DFLSKYTKTTSIDLSSLRPSSQYKDLGEYYAELNPLLYHISFQRIAEKEIMDA VETGKLYLFQIYNKDFAKGHHGKPNLHTLYWTGLFSPENLAKTSIKLNGQA ELFYRPKSRMKRMAHRLGEKMLNKKLKDQKTPIPDTLYQELYDYVNHRLS HDLSDEARALLPNVITKEVSHEIIKDRRFTSDKFFFHVPITLNYQAANSPSKF NQRVNAYLKEHPETPIIGIARGERNLIYITVIDSTGKILEQRSLNTIQQFDYQK KLDNREKERVAARQAWSVVGTIKDLKQGYLSQVIHEIVDLMIHYQAVVVL ENLNFGFKSKRTGIAEKAVYQQFEKMLIDKLNCLVLKDYPAEKVGGVLNP

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		YQLTDQFTSFAKMGTQSGFLFYVPAPYTSKIDPLTGFVDPFVWKTIKNHESR KHFLEGFDFLHYDVKTGDFILHFKMNRNLSFQRGLPGFMPAWDIVFEKNET QFDAKGTPFIAGKRIVPVIENHRFTGRYRDLYPANELIALLEEKGIVFRDGSN ILPKLLENDDSHAIDTMVALIRSVLQMRNSNAATGEDYINSPVRDLNGVCF DSRFQNPEWPMDADANGAYHIALKGQLLLNHLKESKDLKLQNGISNQDWL AYIQELRN
21	inactive RVRAsCpf1	MTQFEGFTNLYQVSKTLRFELIPQGKTLKHIQEQGFIEEDKARNDHYKELKP IIDRIYKTYADQCLQLVQLDWENLSAAIDSYRKEKTEETRNALIEEQATYRN AIHDYFIGRTDNLTDAINKRHAEIYKGLFKAELFNGKVLKQLGTVTTTEHEN ALLRSFDKFTTYFSGFYENRKNVFSAEDISTAIPHRIVQDNFPKFKENCHIFT RLITAVPSLREHFENVKKAIGIFVSTSIEEVFSFPFYNQLLTQTQIDLYNQLLG GISREAGTEKIKGLNEVLNLAIQKNDETAHIIASLPHRFIPLFKQILSDRNTLSF ILEEFKSDEEVIQSFCKYKTLLRNENVLETAEALFNELNSIDLTHIFISHKKLE TISSALCDHWDTLRNALYERRISELTGKITKSAKEKVQRSLKHEDINLQEIIS AAGKELSEAFKQKTSEILSHAHAALDQPLPTTLKKQEEKEILKSQLDSLLGL YHLLDWFAVDESNEVDPEFSARLTGIKLEMEPSLSFYNKARNYATKKPYSV EKFKLNFQMPTLARGWDVNVEKNRGAILFVKNGLYYLGIMPKQKGRYKA LSFEPTEKTSEGFDKMYYDYFPDAAKMIPKCSTQLKAVTAHFQTHTTPILLS NNFIEPLEITKEIYDLNNPEKEPKKFQTAYAKKTGDQKGYREALCKWIDFTR DFLSKYTKTTSIDLSSLRPSSQYKDLGEYYAELNPLLYHISFQRIAEKEIMDA VETGKLYLFQIYNKDFAKGHHGKPNLHTLYWTGLFSPENLAKTSIKLNGQA ELFYRPKSRMKRMAHRLGEKMLNKKLKDQKTPIPDTLYQELYDYVNHRLS HDLSDEARALLPNVITKEVSHEIIKDRRFTSDKFFFHVPITLNYQAANSPSKF NQRVNAYLKEHPETPIIGIARGERNLIYITVIDSTGKILEQRSLNTIQQFDYQK KLDNREKERVAARQAWSVVGTIKDLKQGYLSQVIHEIVDLMIHYQAVVVL ENLNFGFKSKRTGIAEKAVYQQFEKMLIDKLNCLVLKDYPAEKVGGVLNP YQLTDQFTSFAKMGTQSGFLFYVPAPYTSKIDPLTGFVDPFVWKTIKNHESR KHFLEGFDFLHYDVKTGDFILHFKMNRNLSFQRGLPGFMPAWDIVFEKNET QFDAKGTPFIAGKRIVPVIENHRFTGRYRDLYPANELIALLEEKGIVFRDGSN ILPKLLENDDSHAIDTMVALIRSVLQMRNSNAATGEDYINSPVRDLNGVCF DSRFQNPEWPMDADANGAYHIALKGQLLLNHLKESKDLKLQNGISNQDWL AYIQELRN
22	RRAsCpf1	MTQFEGFTNLYQVSKTLRFELIPQGKTLKHIQEQGFIEEDKARNDHYKELKP IIDRIYKTYADQCLQLVQLDWENLSAAIDSYRKEKTEETRNALIEEQATYRN AIHDYFIGRTDNLTDAINKRHAEIYKGLFKAELFNGKVLKQLGTVTTTEHEN ALLRSFDKFTTYFSGFYENRKNVFSAEDISTAIPHRIVQDNFPKFKENCHIFT RLITAVPSLREHFENVKKAIGIFVSTSIEEVFSFPFYNQLLTQTQIDLYNQLLG GISREAGTEKIKGLNEVLNLAIQKNDETAHIIASLPHRFIPLFKQILSDRNTLSF ILEEFKSDEEVIQSFCKYKTLLRNENVLETAEALFNELNSIDLTHIFISHKKLE TISSALCDHWDTLRNALYERRISELTGKITKSAKEKVQRSLKHEDINLQEIIS AAGKELSEAFKQKTSEILSHAHAALDQPLPTTLKKQEEKEILKSQLDSLLGL YHLLDWFAVDESNEVDPEFSARLTGIKLEMEPSLSFYNKARNYATKKPYSV EKFKLNFQMPTLARGWDVNKEKNNGAILFVKNGLYYLGIMPKQKGRYKA LSFEPTEKTSEGFDKMYYDYFPDAAKMIPRCSTQLKAVTAHFQTHTTPILLS NNFIEPLEITKEIYDLNNPEKEPKKFQTAYAKKTGDQKGYREALCKWIDFTR DFLSKYTKTTSIDLSSLRPSSQYKDLGEYYAELNPLLYHISFQRIAEKEIMDA VETGKLYLFQIYNKDFAKGHHGKPNLHTLYWTGLFSPENLAKTSIKLNGQA ELFYRPKSRMKRMAHRLGEKMLNKKLKDQKTPIPDTLYQELYDYVNHRLS HDLSDEARALLPNVITKEVSHEIIKDRRFTSDKFFFHVPITLNYQAANSPSKF NQRVNAYLKEHPETPIIGIARGERNLIYITVIDSTGKILEQRSLNTIQQFDYQK KLDNREKERVAARQAWSVVGTIKDLKQGYLSQVIHEIVDLMIHYQAVVVL ENLNFGFKSKRTGIAEKAVYQQFEKMLIDKLNCLVLKDYPAEKVGGVLNP

SEQ ID NO	Description	Sequence
		YQLTDQFTSFAKMGTQSGFLFYVPAPYTSKIDPLTGFVDPFVWKTIKNHESR KHFLEGFDFLHYDVKTGDFILHFKMNRNLSFQRGLPGFMPAWDIVFEKNET QFDAKGTPFIAGKRIVPVIENHRFTGRYRDLYPANELIALLEEKGIVFRDGSN ILPKLLENDDSHAIDTMVALIRSVLQMRNSNAATGEDYINSPVRDLNGVCF DSRFQNPEWPMDADANGAYHIALKGQLLLNHLKESKDLKLQNGISNQDWL AYIQELRN
23	CasX	MEKRINKIRKKLSADNATKPVSRSGPMKTLLVRVMTDDLKKRLEKRRKKP EVMPQVISNNAANNLRMLLDDYTKMKEAILQVYWQEFKDDHVGLMCKFA QPASKKIDQNKLKPEMDEKGNLTTAGFACSQCGQPLFVYKLEQVSEKGKA YTNYFGRCNVAEHEKLILLAQLKPEKDSDEAVTYSLGKFGQRALDFYSIHV TKESTHPVKPLAQIAGNRYASGPVGKALSDACMGTIASFLSKYQDIIIEHQK VVKGNQKRLESLRELAGKENLEYPSVTLPPQPHTKEGVDAYNEVIARVRM WVNLNLWQKLKLSRDDAKPLLRLKGFPSFPVVERRENEVDWWNTINEVK KLIDAKRDMGRVFWSGVTAEKRNTILEGYNYLPNENDHKKREGSLENPKK PAKRQFGDLLLYLEKKYAGDWGKVFDEAWERIDKKIAGLTSHIEREEARN AEDAQSKAVLTDWLRAKASFVLERLKEMDEKEFYACEIQLQKWYGDLRG NPFAVEAENRVVDISGFSIGSDGHSIQYRNLLAWKYLENGKREFYLLMNYG KKGRIRFTDGTDIKKSGKWQGLLYGGGKAKVIDLTFDPDDEQLIILPLAFGT RQGREFIWNDLLSLETGLIKLANGRVIEKTIYNKKIGRDEPALFVALTFERRE VVDPSNIKPVNLIGVDRGENIPAVIALTDPEGCPLPEFKDSSGGPTDILRIGEG YKEKQRAIQAAKEVEQRRAGGYSRKFASKSRNLADDMVRNSARDLFYHA VTHDAVLVFENLSRGFGRQGKRTFMTERQYTKMEDWLTAKLAYEGLTSK TYLSKTLAQYTSKTCSNCGFTITTADYDGMLVRLKKTSDGWATTLNNKEL KAEGQITYYNRYKRQTVEKELSAELDRLSEESGNNDISKWTKGRRDEALFL LKKRFSHRPVQEQFVCLDCGHEVHADEQAALNIARSWLFLNSNSTEFKSYK SGKQPFVGAWQAFYKRRLKEVWKPNA
24	dCasX	MEKRINKIRKKLSADNATKPVSRSGPMKTLLVRVMTDDLKKRLEKRRKKP EVMPQVISNNAANNLRMLLDDYTKMKEAILQVYWQEFKDDHVGLMCKFA QPASKKIDQNKLKPEMDEKGNLTTAGFACSQCGQPLFVYKLEQVSEKGKA YTNYFGRCNVAEHEKLILLAQLKPEKDSDEAVTYSLGKFGQRALDFYSIHV TKESTHPVKPLAQIAGNRYASGPVGKALSDACMGTIASFLSKYQDIIIEHQK VVKGNQKRLESLRELAGKENLEYPSVTLPPQPHTKEGVDAYNEVIARVRM WVNLNLWQKLKLSRDDAKPLLRLKGFPSFPVVERRENEVDWWNTINEVK KLIDAKRDMGRVFWSGVTAEKRNTILEGYNYLPNENDHKKREGSLENPKK PAKRQFGDLLLYLEKKYAGDWGKVFDEAWERIDKKIAGLTSHIEREEARN AEDAQSKAVLTDWLRAKASFVLERLKEMDEKEFYACEIQLQKWYGDLRG NPFAVEAENRVVDISGFSIGSDGHSIQYRNLLAWKYLENGKREFYLLMNYG KKGRIRFTDGTDIKKSGKWQGLLYGGGKAKVIDLTFDPDDEQLIILPLAFGT RQGREFIWNDLLSLETGLIKLANGRVIEKTIYNKKIGRDEPALFVALTFERRE VVDPSNIKPVNLIGVARGENIPAVIALTDPEGCPLPEFKDSSGGPTDILRIGEG YKEKQRAIQAAKEVEQRRAGGYSRKFASKSRNLADDMVRNSARDLFYHA VTHDAVLVFANLSRGFGRQGKRTFMTERQYTKMEDWLTAKLAYEGLTSK TYLSKTLAQYTSKTCSNCGFTITTADYDGMLVRLKKTSDGWATTLNNKEL KAEGQITYYNRYKRQTVEKELSAELDRLSEESGNNDISKWTKGRRDEALFL LKKRFSHRPVQEQFVCLDCGHEVHAAEQAALNIARSWLFLNSNSTEFKSYK SGKQPFVGAWQAFYKRRLKEVWKPNA
25	CasY	MRKKLFKGYILHNKRLVYTGKAAIRSIKYPLVAPNKTALNNLSEKIIYDYEH LFGPLNVASYARNSNRYSLVDFWIDSLRAGVIWQSKSTSLIDLISKLEGSKSP SEKIFEQIDFELKNKLDKEQFKDIILLNTGIRSSSNVRSLRGRFLKCFKEEFRD TEEVIACVDKWSKDLIVEGKSILVSKQFLYWEEEFGIKIFPHFKDNHDLPKLT FFVEPSLEFSPHLPLANCLERLKKFDISRESLLGLDNNFSAFSNYFNELFNLLS RGEIKKIVTAVLAVSKSWENEPELEKRLHFLSEKAKLLGYPKLTSSWADYR

SEQ ID NO	Description	Sequence
		MIIGGKIKSWHSNYTEQLIKVREDLKKHQIALDKLQEDLKKVVDSSLREQIE AQREALLPLLDTMLKEKDFSDDLELYRFILSDFKSLLNGSYQRYIQTEEERK EDRDVTKKYKDLYSNLRNIPRFFGESKKEQFNKFINKSLPTIDVGLKILEDIR NALETVSVRKPPSITEEYVTKQLEKLSRKYKINAFNSNRFKQITEQVLRKYN NGELPKISEVFYRYPRESHVAIRILPVKISNPRKDISYLLDKYQISPDWKNSNP GEVVDLIEIYKLTLGWLLSCNKDFSMDFSSYDLKLFPEAASLIKNFGSCLSG YYLSKMIFNCITSEIKGMITLYTRDKFVVRYVTQMIGSNQKFPLLCLVGEKQ TKNFSRNWGVLIEEKGDLGEEKNQEKCLIFKDKTDFAKAKEVEIFKNNIWRI RTSKYQIQFLNRLFKKTKEWDLMNLVLSEPSLVLEEEWGVSWDKDKLLPL LKKEKSCEERLYYSLPLNLVPATDYKEQSAEIEQRNTYLGLDVGEFGVAYA VVRIVRDRIELLSWGFLKDPALRKIRERVQDMKKKQVMAVFSSSSTAVARV REMAIHSLRNQIHSIALAYKAKIIYEISISNFETGGNRMAKIYRSIKVSDVYRE SGADTLVSEMIWGKKNKQMGNHISSYATSYTCCNCARTPFELVIDNDKEYE KGGDEFIFNVGDEKKVRGFLQKSLLGKTIKGKEVLKSIKEYARPPIREVLLE GEDVEQLLKRRGNSYIYRCPFCGYKTDADIQAALNIACRGYISDNAKDAVK EGERKLDYILEVRKLWEKNGAVLRSAKFL
26	CasPhi	MADTPTLFTQFLRHHLPGQRFRKDILKQAGRILANKGEDATIAFLRGKSEES PPDFQPPVKCPIIACSRPLTEWPIYQASVAIQGYVYGQSLAEFEASDPGCSKD GLLGWFDKTGVCTDYFSVQGLNLIFQNARKRYIGVQTKVTNRNEKRHKKL KRINAKRIAEGLPELTSDEPESALDETGHLIDPPGLNTNIYCYQQVSPKPLAL SEVNQLPTAYAGYSTSGDDPIQPMVTKDRLSISKGQPGYIPEHQRALLSQKK HRRMRGYGLKARALLVIVRIQDDWAVIDLRSLLRNAYWRRIVQTKEPSTIT KLLKLVTGDPVLDATRMVATFTYKPGIVQVRSAKCLKNKQGSKLFSERYL NETVSVTSIDLGSNNLVAVATYRLVNGNTPELLQRFTLPSHLVKDFERYKQ AHDTLEDSIQKTAVASLPQGQQTEIRMWSMYGFREAQERVCQELGLADGSI PWNVMTATSTILTDLFLARGGDPKKCMFTSEPKKKKNSKQVLYKIRDRAW AKMYRTLLSKETREAWNKALWGLKRGSPDYARLSKRKEELARRCVNYTIS TAEKRAQCGRTIVALEDLNIGFFHGRGKQEPGWVGLFTRKKENRWLMQAL HKAFLELAHHRGYHVIEVNPAYTSQTCPVCRHCDPDNRDQHNREAFHCIGC GFRGNADLDVATHNIAMVAITGESLKRARGSVASKTPQPLAAE
27	dCasPhi	MPKPAVESEFSKVLKKHFPGERFRSSYMKRGGKILAAQGEEAVVAYLQGK SEEEPPNFQPPAKCHVVTKSRDFAEWPIMKASEAIQRYIYALSTTERAACKP GKSSESHAAWFAATGVSNHGYSHVQGLNLIFDHTLGRYDGVLKKVQLRNE KARARLESINASRADEGLPEIKAEEEEVATNETGHLLQPPGINPSFYVYQTIS PQAYRPRDEIVLPPEYAGYVRDPNAPIPLGVVRNRCDIQKGCPGYIPEWQRE AGTAISPKTGKAVTVPGLSPKKNKRMRRYWRSEKEKAQDALLVTVRIGTD WVVIDVRGLLRNARWRTIAPKDISLNALLDLFTGDPVIDVRRNIVTFTYTLD ACGTYARKWTLKGKQTKATLDKLTATQTVALVAIALGQTNPISAGISRVTQ ENGALQCEPLDRFTLPDDLLKDISAYRIAWDRNEEELRARSVEALPEAQQA EVRALDGVSKETARTQLCADFGLDPKRLPWDKMSSNTTFISEALLSNSVSR DQVFFTPAPKKGAKKKAPVEVMRKDRTWARAYKPRLSVEAQKLKNEALW ALKRTSPEYLKLSRRKEELCRRSINYVIEKTRRRTQCQIVIPVIEDLNVRFFH GSGKRLPGWDNFFTAKKENRWFIQGLHKAFSDLRTHRSFYVFEVRPERTSIT CPKCGHCEVGNRDGEAFQCLSCGKTCNADLDVATHNLTQVALTGKTMPK REEPRDAQGTAPARKTKKASKSKAPPAEREDQTPAQEPSQTS
28	Cas12f1 (Cas14a)	MIKVYRYEIVKPLDLDWKEFGTILRQLQQETRFALNKATQLAWEWMGFSS DYKDNHGEYPKSKDILGYTNVHGYAYHTIKTKAYRLNSGNLSQTIKRATD RFKAYQKEILRGDMSIPSYKRDIPLDLIKENISVNRMNHGDYIASLSLLSNPA KQEMNVKRKISVIIIVRGAGKTIMDRILSGEYQVSASQIIHDDRKNKWYLNIS YDFEPQTRVLDLNKIMGIDLGVAVAVYMAFQHTPARYKLEGGEIENFRRQ VESRRISMLRQGKYAGGARGGHGRDKRIKPIEQLRDKIANFRDTTNHRYSR YIVDMAIKEGCGTIQMEDLTNIRDIGSRFLQNWTYYDLQQKIIYKAEEAGIK

SEQ ID NO	Description	Sequence
		VIKIDPQYTSQRCSECGNIDSGNRIGQAIFKCRACGYEANADYNAARNIAIPN IDKIIAESIKSGGS
29	Cas12f2 (Cas14b)	NAMIAQKTIKIKLNPTKEQIIKLNSIIEEYIKVSNFTAKKIAEIQESFTDSGLTQ GTCSECGKEKTYRKYHLLKKDNKLFCITCYKRKYSQFTLQKVEFQNKTGLR NVAKLPKTYYTNAIRFASDTFSGFDEIIKKKQNRLNSIQNRLNFWKELLYNP SNRNEIKIKVVKYAPKTDTREHPHYYSEAEIKGRIKRLEKQLKKFKMPKYPE FTSETISLQRELYSWKNPDELKISSITDKNESMNYYGKEYLKRYIDLINSQTP QILLEKENNSFYLCFPITKNIEMPKIDDTFEPVGIDWGITRNIAVVSILDSKTK KPKFVKFYSAGYILGKRKHYKSLRKHFGQKKRQDKINKLGTKEDRFIDSNI HKLAFLIVKEIRNHSNKPIILMENITDNREEAEKSMRQNILLHSVKSRLQNYI AYKALWNNIPTNLVKPEHTSQICNRCGHQDRENRPKGSKLFKCVKCNYMS NADFNASINIARKFYIGEYEPFYKDNEKMKSGVNSISM
30	Cas12f3 (Cas14c)	MEVQKTVMKTLSLRILRPLYSQEIEKEIKEEEKERRKQAGGTGELDGGFYK KLEKKHSEMFSFDRLNLLLNQLQREIAKVYNHAISELYIATIAQGNKSNKHY ISSIVYNRAYGYFYNAYIALGICSKVEANFRSNELLTQQSALPTAKSDNFPIV LHKQKGAEGEDGGFRISTEGSDLIFEIPIPFYEYNGENRKEPYKWVKKGGQK PVLKLILSTFRRQRNKGWAKDEGTDAEIRKVTEGKYQVSQIEINRGKKLGE HQKWFANFSIEQPIYERKPNRSIVGGLDVGIRSPLVCAINNSFSRYSVDSNDV FKFSKQVFAFRRRLLSKNSLKRKHGHAAHKLEPITEMTEKNDKFRKKIIER WAKEVTNFFVKNQVGIVQIEDLSTMKDREDHFFNQYLRGFWPYYQMQTLI ENKLKEYGIEVKRVQAKYTSQLCSNPNCRYWNNYFNFEYRKVNKFPKFKC EKCNLEISADYNAARNLSTPDIEKFVAKATKGINLPEK
31	C2c8	MKVLEFKIHPTEEQVSKIDQSLAACKLLWNLSIALKEESKQRYYRKKHKFD EFSPEIWGLSYSGHYDEKEFKTLKDKEKKLLIGNPCCKIAYFKKTSNGKEYT PLNSIPIRRFMNAENIDKDAVNYLNRKKLAFYFRENTAKFIGEIETEFKKGFF KSVIKPAYDAAKKGIRGIPRFKGRRDKVETLVNGQPETIKIKSNGVIVSSKIG LLKIRGLDRLQGKAPRMAKITRKATGYYLQLTIETDDTIYKESDKCVGLDM GAVAIFTDDLGRQSEAKRYAKIQKKRLNRLQRQASRQKDNSNNQRKTYAK LARVHEKIARQRKGRNAQLAHKITSEYQSVILEDLNLKNMTAAAKPKERED GDGYKQNGKKRKSGLNKALLDNAIGQLRTFIENKANERGRKIIRVNPKHTS QTCPNCGNIDKANRVSQSKFKCVSCGYEAHADQNAAANILIRGLRDEFLRA IGSLYKFPVSMIGKYPGLAGEFTPDLDANQESIGDAPIENAEHSISKQMKQE GNRTPTQPENGSQSLIFLSAPPQPCGDSHGTNNPKALPNKASKRSSKKPRGA IPENPDQLTIWDLLD
32	human DNMT1	MPARTAPARVPTLAVPAISLPDDVRRRLKDLERDSLTEKECVKEKLNLLHE FLQTEIKNQLCDLETKLRKEELSEEGYLAKVKSLLNKDLSLENGAHAYNRE VNGRLENGNQARSEARRVGMADANSPPKPLSKPRTPRRSKSDGEAKPEPSP SPRITRKSTRQTTITSHFAKGPAKRKPQEESERAKSDESIKEEDKDQDEKRRR VTSRERVARPLPAEEPERAKSGTRTEKEEERDEKEEKRLRSQTKEPTPKQKL KEEPDREARAGVQADEDEDGDEKDEKKHRSQPKDLAAKRRPEEKEPEKVN PQISDEKDEDEKEEKRRKTTPKEPTEKKMARAKTVMNSKTHPPKCIQCGQY LDDPLKYGQHPPDAVDEPQMLTNEKLSIFDANESGFESYEALPQHKLTCFS VYCKHGHLCPIDTGLIEKNIELFFSGSAKPIYDDDPSLEGGVNGKNLGPINE WWITGFDGGEKALIGFSTSFAEYILMDPSPEYAPIFGLMQEKIYISKIVVEFL QSNSDSTYEDLINKIETTVPPSGLNLNRFTEDSLLRHAQFVVEQVESYDEAG DSDEQPIFLTPCMRDLIKLAGVTLGQRRAQARRQTIRHSTREKDRGPTKATT TKLVYQIFDTFFAEQIEKDDREDKENAFKRRRCGVCEVCQQPECGKCKACK DMVKFGGSGRSKQACQERRCPNMAMKEADDDEEVDDNIPEMPSPKKMHQ GKKKKQNKNRISWVGEAVKTDGKKSYYKKVCIDAETLEVGDCVSVIPDDS SKPLYLARVTALWEDSSNGQMFHAHWFCAGTDTVLGATSDPLELFLVDEC EDMQLSYIHSKVKVIYKAPSENWAMEGGMDPESLLEGDDGKTYFYQLWY DQDYARFESPPKTQPTEDNKFKFCVSCARLAEMRQKEIPRVLEQLEDLDSR

SEQ ID NO	Description	Sequence
		VLYYSATKNGILYRVGDGVYLPPEAFTFNIKLSSPVKRPRKEPVDEDLYPEH YRKYSDYIKGSNLDAPEPYRIGRIKEIFCPKKSNGRPNETDIKIRVNKFYRPE NTHKSTPASYHADINLLYWSDEEAVVDFKAVQGRCTVEYGEDLPECVQVY SMGGPNRFYFLEAYNAKSKSFEDPPNHARSPGNKGKGKGKGKGKPKSQAC EPSEPEIEIKLPKLRTLDVFSGCGGLSEGFHQAGISDTLWAIEMWDPAAQAF RLNNPGSTVFTEDCNILLKLVMAGETTNSRGQRLPQKGDVEMLCGGPPCQ GFSGMNRFNSRTYSKFKNSLVVSFLSYCDYYRPRFFLLENVRNFVSFKRSM VLKLTLRCLVRMGYQCTFGVLQAGQYGVAQTRRRAIILAAAPGEKLPLFPE PLHVFAPRACQLSVVVDDKKFVSNITRLSSGPFRTITVRDTMSDLPEVRNGA SALEISYNGEPQSWFQRQLRGAQYQPILRDHICKDMSALVAARMRHIPLAP GSDWRDLPNIEVRLSDGTMARKLRYTHHDRKNGRSSSGALRGVCSCVEAG KACDPAARQFNTLIPWCLPHTGNRHNHWAGLYGRLEWDGFFSTTVTNPEP MGKQGRVLHPEQHRVVSVRECARSQGFPDTYRLFGNILDKHRQVGNAVPP PLAKAIGLEIKLCMLAKARESASAKIKEEEAAKD
33	human DNMT3A	MPAMPSSGPGDTSSSAAEREEDRKDGEEQEEPRGKEERQEPSTTARKVGRP GRKRKHPPVESGDTPKDPAVISKSPSMAQDSGASELLPNGDLEKRSEPQPEE GSPAGGQKGGAPAEGEGAAETLPEASRAVENGCCTPKEGRGAPAEAGKEQ KETNIESMKMEGSRGRLRGGLGWESSLRQRPMPRLTFQAGDPYYISKRKRD EWLARWKREAEKKAKVIAGMNAVEENQGPGESQKVEEASPPAVQQPTDP ASPTVATTPEPVGSDAGDKNATKAGDDEPEYEDGRGFGIGELVWGKLRGF SWWPGRIVSWWMTGRSRAAEGTRWVMWFGDGKFSVVCVEKLMPLSSFC SAFHQATYNKQPMYRKAIYEVLQVASSRAGKLFPVCHDSDESDTAKAVEV QNKPMIEWALGGFQPSGPKGLEPPEEEKNPYKEVYTDMWVEPEAAAYAPP PPAKKPRKSTAEKPKVKEIIDERTRERLVYEVRQKCRNIEDICISCGSLNVTL EHPLFVGGMCQNCKNCFLECAYQYDDDGYQSYCTICCGGREVLMCGNNN CCRCFCVECVDLLVGPGAAQAAIKEDPWNCYMCGHKGTYGLLRRREDWP SRLQMFFANNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLG IQVDRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDLVI GGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPFFWLFE NVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWGNLPGMNRPLA STVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGKDQHFPVFMNEKEDI LWCTEMERVFGFPVHYTDVSNMSRLARQRLLGRSWSVPVIRHLFAPLKEYF ACV
34	human DNMT3A catalytic domain	NHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQVDRYIASE VCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDLVIGGSPCNDLSI VNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPFFWLFENVVAMGVSD KRDISRFLESNPVMIDAKEVSAAHRARYFWGNLPGMNRPLASTVNDKLEL QECLEHGRIAKFSKVRTITTRSNSIKQGKDQHFPVFMNEKEDILWCTEMERV FGFPVHYTDVSNMSRLARQRLLGRSWSVPVIRHLFAPLKEYFACV
35	human DNMT3B	MKGDTRHLNGEEDAGGREDSILVNGACSDQSSDSPPILEAIRTPEIRGRRSSS RLSKREVSSLLSYTQDLTGDGDGEDGDGSDTPVMPKLFRETRTRSESPAVR TRNNNSVSSRERHRPSPRSTRGRQGRNHVDESPVEFPATRSLRRRATASAGT PWPSPPSSYLTIDLTDDTEDTHGTPQSSSTPYARLAQDSQQGGMESPQVEAD SGDGDSSEYQDGKEFGIGDLVWGKIKGFSWWPAMVVSWKATSKRQAMSG MRWVQWFGDGKFSEVSADKLVALGLFSQHFNLATFNKLVSYRKAMYHAL EKARVRAGKTFPSSPGDSLEDQLKPMLEWAHGGFKPTGIEGLKPNNTQPVV NKSKVRRAGSRKLESRKYENKTRRRTADDSATSDYCPAPKRLKTNCYNNG KDRGDEDQSREQMASDVANNKSSLEDGCLSCGRKNPVSFHPLFEGGLCQT CRDRFLELFYMYDDDGYQSYCTVCCEGRELLLCSNTSCCRCFCVECLEVLV GTGTAAEAKLQEPWSCYMCLPQRCHGVLRRRKDWNVRLQAFFTSDTGLE YEAPKLYPAIPAARRRPIRVLSLFDGIATGYLVLKELGIKVGKYVASEVCEES IAVGTVKHEGNIKYVNDVRNITKKNIEEWGPFDLVIGGSPCNDLSNVNPAR

SEQ ID NO	Description	Sequence
		KGLYEGTGRLFFEFYHLLNYSRPKEGDDRPFFWMFENVVAMKVGDKRDIS RFLECNPVMIDAIKVSAAHRARYFWGNLPGMNRPVIASKNDKLELQDCLE YNRIAKLKKVQTITTKSNSIKQGKNQLFPVVMNGKEDVLWCTELERIFGFP VHYTDVSNMGRGARQKLLGRSWSVPVIRHLFAPLKDYFACE
36	mouse DNMT3C	MRGGSRHLSNEEDVSGCEDCIIISGTCSDQSSDPKTVPLTQVLEAVCTVENR GCRTSSQPSKRKASSLISYVQDLTGDGDEDRDGEVGGSSGSGTPVMPQLFC ETRIPSKTPAPLSWQANTSASTPWLSPASPYPIIDLTDEDVIPQSISTPSVDWS QDSHQEGMDTTQVDAESRDGGNIEYQVSADKLLLSQSCILAAFYKLVPYRE SIYRTLEKARVRAGKACPSSPGESLEDQLKPMLEWAHGGFKPTGIEGLKPN KKQPENKSRRRTTNDPAASESSPPKRLKTNSYGGKDRGEDEESREQMASDV TNNKGNLEDHCLSCGRKDPVSFHPLFEGGLCQSCRDRFLELFYMYDEDGY QSYCTVCCEGRELLLCSNTSCCRCFCVECLEVLVGAGTAEDVKLQEPWSCY MCLPQRCHGVLRRRKDWNMRLQDFFTTDPDLEEFEPPKLYPAIPAAKRRPI RVLSLFDGIATGYLVLKELGIKVEKYIASEVCAESIAVGTVKHEGQIKYVDD IRNITKEHIDEWGPFDLVIGGSPCNDLSCVNPVRKGLFEGTGRLFFEFYRLLN YSCPEEEDDRPFFWMFENVVAMEVGDKRDISRFLECNPVMIDAIKVSAAHR ARYFWGNLPGMNRPVMASKNDKLELQDCLEFSRTAKLKKVQTITTKSNSIR QGKNQLFPVVMNGKDDVLWCTELERIFGFPEHYTDVSNMGRGARQKLLG RSWSVPVIRHLFAPLKDHFACE
37	human DNMT3L	MAAIPALDPEAEPSMDVILVGSSELSSSVSPGTGRDLIAYEVKANQRNIEDIC ICCGSLQVHTQHPLFEGGICAPCKDKFLDALFLYDDDGYQSYCSICCSGETL LICGNPDCTRCYCFECVDSLVGPGTSGKVHAMSNWVCYLCLPSSRSGLLQR RRKWRSQLKAFYDRESENPLEMFETVPVWRRQPVRVLSLFEDIKKELTSLG FLESGSDPGQLKHVVDVTDTVRKDVEEWGPFDLVYGATPPLGHTCDRPPS WYLFQFHRLLQYARPKPGSPRPFFWMFVDNLVLNKEDLDVASRFLEMEPV TIPDVHGGSLQNAVRVWSNIPAIRSSRHWALVSEEELSLLAQNKQSSKLAA KWPTKLVKNCFLPLREYFKYFSTELTSSL
38	human DNMT3L catalytic domain	NPLEMFETVPVWRRQPVRVLSLFEDIKKELTSLGFLESGSDPGQLKHVVDV TDTVRKDVEEWGPFDLVYGATPPLGHTCDRPPSWYLFQFHRLLQYARPKP GSPRPFFWMFVDNLVLNKEDLDVASRFLEMEPVTIPDVHGGSLQNAVRVW SNIPAIRSRHWALVSEEELSLLAQNKQSSKLAAKWPTKLVKNCFLPLREYFK YFSTELTSSL
39	mouse DNMT3L	MGSRETPSSCSKTLETLDLETSDSSSPDADSPLEEQWLKSSPALKEDSVDVV LEDCKEPLSPSSPPTGREMIRYEVKVNRRSIEDICLCCGTLQVYTRHPLFEGG LCAPCKDKFLESLFLYDDDGHQSYCTICCSGGTLFICESPDCTRCYCFECVDI LVGPGTSERINAMACWVCFLCLPFSRSGLLQRRKRWRHQLKAFHDQEGAG PMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGGGTLKYVED VTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYMFQFHRILQYALPR QESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVTLQDVRGRDYQNAMRV WSNIPGLKSKHAPLTPKEEEYLQAQVRSRSKLDAPKVDLLVKNCLLPLREY FKYFSQNSLPL
40	mouse DNMT3L catalytic domain	GPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGGGTLKYVE DVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYMFQFHRILQYALP RQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVTLQDVRGRDYQNAMR VWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSKLDAPKVDLLVKNCLLPLRE YFKYFSQNSLPL
41	human TRDMT1 (DNMT2)	MEPLRVLELYSGVGGMHHALRESCIPAQVVAAIDVNTVANEVYKYNFPHT QLLAKTIEGITLEEFDRLSFDMILMSPPCQPFTRIGRQGDMTDSRTNSFLHILD ILPRLQKLPKYILLENVKGFEVSSTRDLLIQTIENCGFQYQEFLLSPTSLGIPNS RLRYFLIAKLQSEPLPFQAPGQVLMEFPKIESVHPQKYAMDVENKIQEKNVE PNISFDGSIQCSGKDAILFKLETAEEIHRKNQQDSDLSVKMLKDFLEDDTDV NQYLLPPKSLLRYALLLDIVQPTCRRSVCFTKGYGSYIEGTGSVLQTAEDVQ

SEQ ID NO	Description	Sequence
		VENIYKSLTNLSQEEQITKLLILKLRYFTPKEIANLLGFPPEFGFPEKITVKQR YRLLGNSLNVHVVAKLIKILYE
42	M. penetrans M MpeI	MNSNKDKIKVIKVFEAFAGIGSQFKALKNIARSKNWEIQHSGMVEWFVDAI VSYVAIHSKNFNPKIEQLDKDILSISNDSKMPISEYGIKKINNTIKASYLNYAK KHFNNLFDIKKVNKDNFPKNIDIFTYSFPCQDLSVQGLQKGIDKELNTRSGL LWEIERILEEIKNSFSKEEMPKYLLMENVKNLLSHKNKKNYNTWLKQLEKF GYKSKTYLLNSKNFDNCQNRERVFCLSIRDDYLEKTGFKFKELEKVKNPPK KIKDILVDSSNYKYLNLNKYETTTFRETKSNIISRSLKNYTTFNSENYVYNIN GIGPTLTASGANSRIKIETQQGVRYLTPLECFKYMQFDVNDFKKVQSTNLIS ENKMIYIAGNSIPVKILEAIFNTLEFVNNEE
43	S. monobiae M SssI	MSKVENKTKKLRVFEAFAGIGAQRKALEKVRKDEYEIVGLAEWYVPAIVM YQAIHNNFHTKLEYKSVSREEMIDYLENKTLSWNSKNPVSNGYWKRKKDD ELKIIYNAIKLSEKEGNIFDIRDLYKRTLKNIDLLTYSFPCQDLSQQGIQKGM KRGSGTRSGLLWEIERALDSTEKNDLPKYLLMENVGALLHKKNEEELNQW KQKLESLGYQNSIEVLNAADFGSSQARRRVFMISTLNEFVELPKGDKKPKSI KKVLNKIVSEKDILNNLLKYNLTEFKKTKSNINKASLIGYSKFNSEGYVYDP EFTGPTLTASGANSRIKIKDGSNIRKMNSDETFLYIGFDSQDGKRVNEIEFLT ENQKIFVCGNSISVEVLEAIIDKIGG
44	H. parainfluenzae M HpaII	MKDVLDDNLLEEPAAQYSLFEPESNPNLREKFTFIDLFAGIGGFRIAMQNLG GKCIFSSEWDEQAQKTYEANFGDLPYGDITLEETKAFIPEKFDILCAGFPCQA FSIAGKRGGFEDTRGTLFFDVAEIIRRHQPKAFFLENVKGLKNHDKGRTLKT ILNVLREDLGYFVPEPAIVNAKNFGVPQNRERIYIVGFHKSTGVNSFSYPEPL DKIVTFADIREEKTVPTKYYLSTQYIDTLRKHKERHESKGNGFGYEIIPDDGI ANAIVVGGMGRERNLVIDHRITDFTPTTNIKGEVNREGIRKMTPREWARLQ GFPDSYVIPVSDASAYKQFGNSVAVPAIQATGKKILEKLGNLYD
45	A. luteus M AluI	MSKANAKYSFVDLFAGIGGFHAALAATGGVCEYAVEIDREAAAVYERNW NKPALGDITDDANDEGVTLRGYDGPIDVLTGGFPCQPFSKSGAQHGMAETR GTLFWNIARIIEEREPTVLILENVRNLVGPRHRHEWLTIIETLRFFGYEVSGAP AIFSPHLLPAWMGGTPQVRERVFITATLVPERMRDERIPRTETGEIDAEAIGP KPVATMNDRFPIKKGGTELFHPGDRKSGWNLLTSGIIREGDPEPSNVDLRLT ETETLWIDAWDDLESTIRRATGRPLEGFPYWADSWTDFRELSRLVVIRGFQ APEREVVGDRKRYVARTDMPEGFVPASVTRPAIDETLPAWKQSHLRRNYD FFERHFAEVVAWAYRWGVYTDLFPASRRKLEWQAQDAPRLWDTVMHFRP SGIRAKRPTYLPALVAITQTSIVGPLERRLSPRETARLQGLPEWFDFGEQRAA ATYKQMGNGVNVGVVRHILREHVRRDRALLKLTPAGQRIINAVLADEPDA TVGALGAAE
46	H. aegyptius M HaeIII	MNLISLFSGAGGLDLGFQKAGFRIICANEYDKSIWKTYESNHSAKLIKGDIS KISSDEFPKCDGIIGGPPCQSWSEGGSLRGIDDPRGKLFYEYIRILKQKKPIFF LAENVKGMMAQRHNKAVQEFIQEFDNAGYDVHIILLNANDYGVAQDRKR VFYIGFRKELNINYLPPIPHLIKPTFKDVIWDLKDNPIPALDKNKTNGNKCIY PNHEYFIGSYSTIFMSRNRVRQWNEPAFTVQASGRQCQLHPQAPVMLKVSK NLNKFVEGKEHLYRRLTVRECARVQGFPDDFIFHYESLNDGYKMIGNAVPV NLAYEIAKTIKSALEICKGN
47	H. haemolyticus M HhaI	MIEIKDKQLTGLRFIDLFAGLGGFRLALESCGAECVYSNEWDKYAQEVYEM NFGEKPEGDITQVNEKTIPDHDILCAGFPCQAFSISGKQKGFEDSRGTLFFDI ARIVREKKPKVVFMENVKNFASHDNGNTLEVVKNTMNELDYSFHAKVLN ALDYGIPQKRERIYMICFRNDLNIQNFQFPKPFELNTFVKDLLLPDSEVEHLV IDRKDLVMTNQEIEQTTPKTVRLGIVGKGGQGERIYSTRGIAITLSAYGGGIF AKTGGYLVNGKTRKLHPRECARVMGYPDSYKVHPSTSQAYKQFGNSVVIN VLQYIAYNIGSSLNFKPY
48	Moraxella M MspI	MKPEILKLIRSKLDLTQKQASEIIEVSDKTWQQWESGKTEMHPAYYSFLQE KLKDKINFEELSAQKTLQKKIFDKYNQNQITKNAEELAEITHIEERKDAYSS

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		DFKFIDLFSGIGGIRQSFEVNGGKCVFSSEIDPFAKFTYYTNFGVVPFGDITKV EATTIPQHDILCAGFPCQPFSHIGKREGFEHPTQGTMFHEIVRIIETKKTPVLF LENVPGLINHDDGNTLKVIIETLEDMGYKVHHTVLDASHFGIPQKRKRFYL VAFLNQNIHFEFPKPPMISKDIGEVLESDVTGYSISEHLQKSYLFKKDDGKPS LIDKNTTGAVKTLVSTYHKIQRLTGTFVKDGETGIRLLTTNECKAIMGFPKD FVIPVSRTQMYRQMGNSVVVPVVTKIAEQISLALKTVNQQSPQENFELELV
49	Ascobolus Masc1	MSERRYEAGMTVALHEGSFLKIQRVYIRQYHADNRREHMLVGPLFRRTKY LKALSKKVNEVAIVHESIHVPVQDVIGVRELIITNRPFPECRKGDEHTGRLVC RWVYNLDERAKGREYKKQRYIRRITEAEADPEYRVEDRVLRRRWFQEGYI GDEISYKEHGNGDIVDIRSESPLQVLDGWGGDLVDLENGEETSIPGPCRSAS SYGRLMKPPLAQAADSNTSRKYTFGDTFCGGGGVSLGARQAGLEVKWAF DMNPNAGANYRRNFPNTDFFLAEAEQFIQLSVGISQHVDILHLSPPCQTFSR AHTIAGKNDENNEASFFAVVNLIKAVRPRLFTVEETDGIMDRQSRQFIDTAL MGITELGYSFRICVLNAIEYGVCQNRKRLIIIGAAPGEELPPFPLPTHQDFFSK DPRRDLLPAVTLDDALSTITPESTDHHLNHVWQPAEWKTPYDAHRPFKNAI RAGGGEYDIYPDGRRKFTVRELACIQGFPDEYEFVGTLTDKRRIIGNAVPPP LSAAIMSTLRQWMTEKDFERME
50	Arabidopsis MET1	MVENGAKAAKRKKRPLPEIQEVEDVPRTRRPRRAAACTSFKEKSIRVCEKS ATIEVKKQQIVEEEFLALRLTALETDVEDRPTRRLNDFVLFDSDGVPQPLEM LEIHDIFVSGAILPSDVCTDKEKEKGVRCTSFGRVEHWSISGYEDGSPVIWIS TELADYDCRKPAASYRKVYDYFYEKARASVAVYKKLSKSSGGDPDIGLEE LLAAVVRSMSSGSKYFSSGAAIIDFVISQGDFIYNQLAGLDETAKKHESSYV EIPVLVALREKSSKIDKPLQRERNPSNGVRIKEVSQVAESEALTSDQLVDGT DDDRRYAILLQDEENRKSMQQPRKNSSSGSASNMFYIKINEDEIANDYPLPS YYKTSEEETDELILYDASYEVQSEHLPHRMLHNWALYNSDLRFISLELLPM KQCDDIDVNIFGSGVVTDDNGSWISLNDPDSGSQSHDPDGMCIFLSQIKEW MIEFGSDDIISISIRTDVAWYRLGKPSKLYAPWWKPVLKTARVGISILTFLRV ESRVARLSFADVTKRLSGLQANDKAYISSDPLAVERYLVVHGQIILQLFAVY PDDNVKRCPFVVGLASKLEDRHHTKWIIKKKKISLKELNLNPRAGMAPVAS KRKAMQATTTRLVNRIWGEFYSNYSPEDPLQATAAENGEDEVEEEGGNGE EEVEEEGENGLTEDTVPEPVEVQKPHTPKKIRGSSGKREIKWDGESLGKTSA GEPLYQQALVGGEMVAVGGAVTLEVDDPDEMPAIYFVEYMFESTDHCKM LHGRFLQRGSMTVLGNAANERELFLTNECMTTQLKDIKGVASFEIRSRPWG HQYRKKNITADKLDWARALERKVKDLPTEYYCKSLYSPERGGFFSLPLSDI GRSSGFCTSCKIREDEEKRSTIKLNVSKTGFFINGIEYSVEDFVYVNPDSIGGL KEGSKTSFKSGRNIGLRAYVVCQLLEIVPKESRKADLGSFDVKVRRFYRPED VSAEKAYASDIQELYFSQDTVVLPPGALEGKCEVRKKSDMPLSREYPISDHI FFCDLFFDTSKGSLKQLPANMKPKFSTIKDDTLLRKKKGKGVESEIESEIVKP VEPPKEIRLATLDIFAGCGGLSHGLKKAGVSDAKWAIEYEEPAGQAFKQNH PESTVFVDNCNVILRAIMEKGGDQDDCVSTTEANELAAKLTEEQKSTLPLP GQVDFINGGPPCQGFSGMNRFNQSSWSKVQCEMILAFLSFADYFRPRYFLL ENVRTFVSFNKGQTFQLTLASLLEMGYQVRFGILEAGAYGVSQSRKRAFIW AAAPEEVLPEWPEPMHVFGVPKLKISLSQGLHYAAVRSTALGAPFRPITVRD TIGDLPSVENGDSRTNKEYKEVAVSWFQKEIRGNTIALTDHICKAMNELNLI RCKLIPTRPGADWHDLPKRKVTLSDGRVEEMIPFCLPNTAERHNGWKGLY GRLDWQGNFPTSVTDPQPMGKVGMCFHPEQHRILTVRECARSQGFPDSYEF AGNINHKHRQIGNAVPPPLAFALGRKLKEALHLKKSPQHQP
51	Ascobolus Masc2	MELTPELSGVSTDLGGGGSIFAHWRMKEESPAPTEILDDLNVLEWEKTTRD YSKEDLRIADQLFSIEDEHQSLPFETADAEDGTPTEEEEEKELPMRTLDNFVL YDASDLELAALDLIGTELNIHAVGTVGPIYTEGEEDEQEDEDEDVSPPVRTG TQATSASVTQMTVELYIRNIVQYEFCFNDDGTVETWIQTTNAHYKLLQPAK CYTSLYRPVNDCLNVITAIITLAPESTTMSLKDLLKVMDDKAQAVSYEEVE

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		RMSEFIVQHLDQWMETAPKKKSKLIEKSKVYIDLNNLAGIDMVSGVRPPPV RRVTGRSSAPKKRIVRNMNDAVLLHQNETTVTNWIHQLSAGMFGRALNVL GAETADVENLTCDPASAKFVVPQRRLHKRLKWETRGHIPVSEEEYKHIYQG KKYAKFFEAVRAVDESKLTIKLGDLVYVLDQDPKVTQTQFATAGREGRKK GAEKEKIQVRFGRVLSIRQPDSNSKDAQNVFIHVQWLVLGCDTILQEMASR RELFLTDSCDTVFADVIYGVAKLTPLGAKDIPTVEFHESMATMMGENEFFV RFKYNYQDGSFTDLKDVDAEQIGTLQPRVNTHRNPGYCSNCRIKYDNERTG DKWIYENDTEGEPRLFRSSKGWCIYAQEFVYLQPVEKQPGTTFRVGYISEIN KSSVIVELLARVDDDDKSGHISYSDPRHLYFTGTDIKVTFDKIIRKCFVFHDS GDQKAKAPLMYGTLQRDLYYYRYEKRKGKAELVPVREIRSIHEQTLNDWE SRTQIERHGAVSGKKLKGLDIFAGCGGLTLGLDLSGAVDTKWDIEFAPSAA NTLALNFPDAQVFNQCANVLLSRAIQSEDEGSLDIEYDLQGRVLPDLPKKG EVDFIYGGPPCQGFSGVNRYKKGNDIKNSLVATFLSYVDHYKPRFVLLENV KGLITTKLGNSKNAEGKWEGGISNGVVKFIYRTLISMNYQCRIGLVQSGEY GVPQSRPRVIFLAARMGERLPDLPEPMHAFEVLDSQYALPHIKRYHTTQNG VAPLPRITIGEAVSDLPKFQYANPGVWPRHDPYSSAKAQPSDKTIEKFSVSK ATSFVGYLLQPYHSRPQSEFQRRLRTKLVPSDEPAEKTSLLTTKLVTAHVTR LFNKETTQRIVCVPMWPGADHRSLPKEMRPWCLVDPNSQAEKHRFWPGLF GRLGMEDFFSTALTDVQPCGKQGKVLHPTQRRVYTVRELARAQGFPDWFA FTDGDADSGLGGVKKWHRNIGNAVPVPLGEQIGRCIGYSVWWKDDMIAQ LREDGADEDEEMIDGNDQWVEELNTQMAADMPGLPLLVTHLLNLCVYRR LYGPNAKEFLPARVYDKKLEGGRRRLVWAML
52	Neurospora Dim2	MDSPDRSHGGMFIDVPAETMGFQEDYLDMFASVLSQGLAKEGDYAHHQPL PAGKEECLEPIAVATTITPSPDDPQLQLQLELEQQFQTESGLNGVDPAPAPES EDEADLPDGFSDESPDDDFVVQRSKHITVDLPVSTLINPRSTFQRIDENDNLV PPPQSTPERVAVEDLLKAAKAAGKNKEDYIEFELHDFNFYVNYAYHPQEM RPIQLVATKVLHDKYYFDGVLKYGNTKHYVTGMQVLELPVGNYGASLHS VKGQIWVRSKHNAKKEIYYLLKKPAFEYQRYYQPFLWIADLGKHVVDYCT RMVERKREVTLGCFKSDFIQWASKAHGKSKAFQNWRAQHPSDDFRTSVAA NIGYIWKEINGVAGAKRAAGDQLFRELMIVKPGQYFRQEVPPGPVVTEGDR TVAATIVTPYIKECFGHMILGKVLRLAGEDAEKEKEVKLAKRLKIENKNAT KADTKDDMKNDTATESLPTPLRSLPVQVLEATPIESDIVSIVSSDLPPSENNP PPLTNGSVKPKAKANPKPKPSTQPLHAAHVKYLSQELVNKIKVGDVISTPR DDSSNTDTKWKPTDTDDHRWFGLVQRVHTAKTKSSGRGLNSKSFDVIWFY RPEDTPCCAMKYKWRNELFLSNHCTCQEGHHARVKGNEVLAVHPVDWFG TPESNKGEFFVRQLYESEQRRWITLQKDHLTCYHNQPPKPPTAPYKPGDTV LATLSPSDKFSDPYEVVEYFTQGEKETAFVRLRKLLRRRKVDRQDAPANEL VYTEDLVDVRAERIVGKCIMRCFRPDERVPSPYDRGGTGNMFFITHRQDHG RCVPLDTLPPTLRQGFNPLGNLGKPKLRGMDLYCGGGNFGRGLEEGGVVE MRWANDIWDKAIHTYMANTPDPNKTNPFLGSVDDLLRLALEGKFSDNVPR PGEVDFIAAGSPCPGFSLLTQDKKVLNQVKNQSLVASFASFVDFYRPKYGV LENVSGIVQTFVNRKQDVLSQLFCALVGMGYQAQLILGDAWAHGAPQSRE RVFLYFAAPGLPLPDPPLPSHSHYRVKNRNIGFLCNGESYVQRSFIPTAFKFV SAGEGTADLPKIGDGKPDACVRFPDHRLASGITPYIRAQYACIPTHPYGMNF IKAWNNGNGVMSKSDRDLFPSEGKTRTSDASVGWKRLNPKTLFPTVTTTS NPSDARMGPGLHWDEDRPYTVQEMRRAQGYLDEEVLVGRTTDQWKLVG NSVSRHMALAIGLKFREAWLGTLYDESAVVATATATATTAAAVGVTVPV MEEPGIGTTESSRPSRSPVHTAVDLDDSKSERSRSTTPATVLSTSSAAGDGSA NAAGLEDDDNDDMEMMEVTRKRSSPAVDEEGMRPSKVQKVEVTVASPAS RRSSRQASRNPTASPSSKASKATTHEAPAPEELESDAESYSETYDKEGFDGD YHSGHEDQYSEEDEEEEYAEPETMTVNGMTIVKL

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53	Drosophila dDnmt2	MVFRVLELFSGIGGMHYAFNYAQLDGQIVAALDVNTVANAVYAHNYGSN LVKTRNIQSLSVKEVTKLQANMLLMSPPCQPHTRQGLQRDTEDKRSDALTH LCGLIPECQELEYILMENVKGFESSQARNQFIESLERSGFHWREFILTPTQFN VPNTRYRYYCIARKGADFPFAGGKIWEEMPGAIAQNQGLSQIAEIVEENVSP DFLVPDDVLTKRVLVMDIIHPAQSRSMCFTKGYTHYTEGTGSAYTPLSEDE SHRIFELVKEIDTSNQDASKSEKILQQRLDLLHQVRLRYFTPREVARLMSFPE NFEFPPETTNRQKYRLLGNSINVKVVGELIKLLTIK
54	S. pombe Pmt1	MLSTKRLRVLELYSGIGGMHYALNLANIPADIVCAIDINPQANEIYNLNHGK LAKHMDISTLTAKDFDAFDCKLWTMSPSCQPFTRIGNRKDILDPRSQAFLNI LNVLPHVNNLPEYILIENVQGFEESKAAEECRKVLRNCGYNLIEGILSPNQFN IPNSRSRWYGLARLNFKGEWSIDDVFQFSEVAQKEGEVKRIRDYLEIERDW SSYMVLESVLNKWGHQFDIVKPDSSSCCCFTRGYTHLVQGAGSILQMSDHE NTHEQFERNRMALQLRYFTAREVARLMGFPESLEWSKSNVTEKCMYRLLG NSINVKVVSYLISLLLEPLNF
55	Arabidopsis DRM1	MVMSHIFLISQIQEVEHGDSDDVNWNTDDDELAIDNFQFSPSPVHISATSPNS IQNRISDETVASFVEMGFSTQMIARAIEETAGANMEPMMILETLFNYSASTE ASSSKSKVINHFIAMGFPEEHVIKAMQEHGDEDVGEITNALLTYAEVDKLRE SEDMNININDDDDDNLYSLSSDDEEDELNNSSNEDRILQALIKMGYLREDA AIAIERCGEDASMEEVVDFICAAQMARQFDEIYAEPDKKELMNNNKKRRTY TETPRKPNTDQLISLPKEMIGFGVPNHPGLMMHRPVPIPDIARGPPFFYYENV AMTPKGVWAKISSHLYDIVPEFVDSKHFCAAARKRGYIHNLPIQNRFQIQPP QHNTIQEAFPLTKRWWPSWDGRTKLNCLLTCIASSRLTEKIREALERYDGET PLDVQKWVMYECKKWNLVWVGKNKLAPLDADEMEKLLGFPRDHTRGGG ISTTDRYKSLGNSFQVDTVAYHLSVLKPLFPNGINVLSLFTGIGGGEVALHR LQIKMNVVVSVEISDANRNILRSFWEQTNQKGILREFKDVQKLDDNTIERL MDEYGGFDLVIGGSPCNNLAGGNRHHRVGLGGEHSSLFFDYCRILEAVRRK ARHMRR
56	Arabadopsis DRM2	MVIWNNDDDDFLEIDNFQSSPRSSPIHAMQCRVENLAGVAVTTSSLSSPTET TDLVQMGFSDEVFATLFDMGFPVEMISRAIKETGPNVETSVIIDTISKYSSDC EAGSSKSKAIDHFLAMGFDEEKVVKAIQEHGEDNMEAIANALLSCPEAKKL PAAVEEEDGIDWSSSDDDTNYTDMLNSDDEKDPNSNENGSKIRSLVKMGFS ELEASLAVERCGENVDIAELTDFLCAAQMAREFSEFYTEHEEQKPRHNIKK RRFESKGEPRSSVDDEPIRLPNPMIGFGVPNEPGLITHRSLPELARGPPFFYYE NVALTPKGVWETISRHLFEIPPEFVDSKYFCVAARKRGYIHNLPINNRFQIQP PPKYTIHDAFPLSKRWWPEWDKRTKLNCILTCTGSAQLTNRIRVALEPYNE EPEPPKHVQRYVIDQCKKWNLVWVGKNKAAPLEPDEMESILGFPKNHTRG GGMSRTERFKSLGNSFQVDTVAYHLSVLKPIFPHGINVLSLFTGIGGGEVAL HRLQIKMKLVVSVEISKVNRNILKDFWEQTNQTGELIEFSDIQHLTNDTIEGL MEKYGGFDLVIGGSPCNNLAGGNRVSRVGLEGDQSSLFFEYCRILEVVRAR MRGS
57	Arabadopsis CMT1	MAARNKQKKRAEPESDLCFAGKPMSVVESTIRWPHRYQSKKTKLQAPTKK PANKGGKKEDEEIIKQAKCHFDKALVDGVLINLNDDVYVTGLPGKLKFIAK VIELFEADDGVPYCRFRWYYRPEDTLIERFSHLVQPKRVFLSNDENDNPLTC IWSKVNIAKVPLPKITSRIEQRVIPPCDYYYDMKYEVPYLNFTSADDGSDAS SSLSSDSALNCFENLHKDEKFLLDLYSGCGAMSTGFCMGASISGVKLITKWS VDINKFACDSLKLNHPETEVRNEAAEDFLALLKEWKRLCEKFSLVSSTEPVE SISELEDEEVEENDDIDEASTGAELEPGEFEVEKFLGIMFGDPQGTGEKTLQL MVRWKGYNSSYDTWEPYSGLGNCKEKLKEYVIDGFKSHLLPLPGTVYTVC GGPPCQGISGYNRYRNNEAPLEDQKNQQLLVFLDIIDFLKPNYVLMENVVD LLRFSKGFLARHAVASFVAMNYQTRLGMMAAGSYGLPQLRNRVFLWAAQ PSEKLPPYPLPTHEVAKKFNTPKEFKDLQVGRIQMEFLKLDNALTLADAISD LPPVTNYVANDVMDYNDAAPKTEFENFISLKRSETLLPAFGGDPTRRLFDH

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		QPLVLGDDDLERVSYIPKQKGANYRDMPGVLVHNNKAEINPRFRAKLKSG KNVVPAYAISFIKGKSKKPFGRLWGDEIVNTVVTRAEPHNQCVIHPMQNRV LSVRENARLQGFPDCYKLCGTIKEKYIQVGNAVAVPVGVALGYAFGMASQ GLTDDEPVIKLPFKYPECMQAKDQI
58	Arabadopsis CMT2	MLSPAKCESEEAQAPLDLHSSSRSEPECLSLVLWCPNPEEAAPSSTRELIKLP DNGEMSLRRSTTLNCNSPEENGGEGRVSQRKSSRGKSQPLLMLTNGCQLRR SPRFRALHANFDNVCSVPVTKGGVSQRKFSRGKSQPLLTLTNGCQLRRSPR FRAVDGNFDSVCSVPVTGKFGSRKRKSNSALDKKESSDSEGLTFKDIAVIAK SLEMEIISECQYKNNVAEGRSRLQDPAKRKVDSDTLLYSSINSSKQSLGSNK RMRRSQRFMKGTENEGEENLGKSKGKGMSLASCSFRRSTRLSGTVETGNT ETLNRRKDCGPALCGAEQVRGTERLVQISKKDHCCEAMKKCEGDGLVSSK QELLVFPSGCIKKTVNGCRDRTLGKPRSSGLNTDDIHTSSLKISKNDTSNGLT MTTALVEQDAMESLLQGKTSACGAADKGKTREMHVNSTVIYLSDSDEPSSI EYLNGDNLTQVESGSALSSGGNEGIVSLDLNNPTKSTKRKGKRVTRTAVQE QNKRSICFFIGEPLSCEEAQERWRWRYELKERKSKSRGQQSEDDEDKIVAN VECHYSQAKVDGHTFSLGDFAYIKGEEEETHVGQIVEFFKTTDGESYFRVQ WFYRATDTIMERQATNHDKRRLFYSTVMNDNPVDCLISKVTVLQVSPRVG LKPNSIKSDYYFDMEYCVEYSTFQTLRNPKTSENKLECCADVVPTESTESIL KKKSFSGELPVLDLYSGCGGMSTGLSLGAKISGVDVVTKWAVDQNTAACK SLKLNHPNTQVRNDAAGDFLQLLKEWDKLCKRYVFNNDQRTDTLRSVNST KETSGSSSSSDDDSDSEEYEVEKLVDICFGDHDKTGKNGLKFKVHWKGYRS DEDTWELAEELSNCQDAIREFVTSGFKSKILPLPGRVGVICGGPPCQGISGYN RHRNVDSPLNDERNQQIIVFMDIVEYLKPSYVLMENVVDILRMDKGSLGRY ALSRLVNMRYQARLGIMTAGCYGLSQFRSRVFMWGAVPNKNLPPFPLPTH DVIVRYGLPLEFERNVVAYAEGQPRKLEKALVLKDAISDLPHVSNDEDREK LPYESLPKTDFQRYIRSTKRDLTGSAIDNCNKRTMLLHDHRPFHINEDDYAR VCQIPKRKGANFRDLPGLIVRNNTVCRDPSMEPVILPSGKPLVPGYVFTFQQ GKSKRPFARLWWDETVPTVLTVPTCHSQALLHPEQDRVLTIRESARLQGFP DYFQFCGTIKERYCQIGNAVAVSVSRALGYSLGMAFRGLARDEHLIKLPQN FSHSTYPQLQETIPH
59	Arabadopsis CMT3	MAPKRKRPATKDDTTKSIPKPKKRAPKRAKTVKEEPVTVVEEGEKHVARFL DEPIPESEAKSTWPDRYKPIEVQPPKASSRKKTKDDEKVEIIRARCHYRRAIV DERQIYELNDDAYVQSGEGKDPFICKIIEMFEGANGKLYFTARWFYRPSDT VMKEFEILIKKKRVFFSEIQDTNELGLLEKKLNILMIPLNENTKETIPATENCD FFCDMNYFLPYDTFEAIQQETMMAISESSTISSDTDIREGAAAISEIGECSQET EGHKKATLLDLYSGCGAMSTGLCMGAQLSGLNLVTKWAVDMNAHACKS LQHNHPETNVRNMTAEDFLFLLKEWEKLCIHFSLRNSPNSEEYANLHGLNN VEDNEDVSEESENEDDGEVFTVDKIVGISFGVPKKLLKRGLYLKVRWLNYD DSHDTWEPIEGLSNCRGKIEEFVKLGYKSGILPLPGGVDVVCGGPPCQGISG HNRFRNLLDPLEDQKNKQLLVYMNIVEYLKPKFVLMENVVDMLKMAKGY LARFAVGRLLQMNYQVRNGMMAAGAYGLAQFRLRFFLWGALPSEIIPQFP LPTHDLVHRGNIVKEFQGNIVAYDEGHTVKLADKLLLKDVISDLPAVANSE KRDEITYDKDPTTPFQKFIRLRKDEASGSQSKSKSKKHVLYDHHPLNLNIND YERVCQVPKRKGANFRDFPGVIVGPGNVVKLEEGKERVKLESGKTLVPDY ALTYVDGKSCKPFGRLWWDEIVPTVVTRAEPHNQVIIHPEQNRVLSIRENA RLQGFPDDYKLFGPPKQKYIQVGNAVAVPVAKALGYALGTAFQGLAVGK DPLLTLPEGFAFMKPTLPSELA
60	Neurospora Rid	MAEQNPFVIDDEDDVIQIHDEEEVEEEVAEVIDITEDDIEPSELDRAFGSRPK EETLPSLLLRDQGFIVRPGMTVELKAPIGRFAISFVRVNSIVKVRQAHVNNV TIRGHGFTRAKEMNGMLPKQLNECCLVASIDTRDPRP

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61	E. coli strain 12 hsdM	MNNNDLVAKLWKLCDNLRDGGVSYQNYVNELASLLFLKMCKETGQEAE YLPEGYRWDDLKSRIGQEQLQFYRKMLVHLGEDDKKLVQAVFHNVSTTIT EPKQITALVSNMDSLDWYNGAHGKSRDDFGDMYEGLLQKNANETKSGAG QYFTPRPLIKTIIHLLKPQPREVVQDPAAGTAGFLIEADRYVKSQTNDLDDL DGDTQDFQIHRAFIGLELVPGTRRLALMNCLLHDIEGNLDHGGAIRLGNTL GSDGENLPKAHIVATNPPFGSAAGTNITRTFVHPTSNKQLCFMQHIIETLHPG GRAAVVVPDNVLFEGGKGTDIRRDLMDKCHLHTILRLPTGIFYAQGVKTNV LFFTKGTVANPNQDKNCTDDVWVYDLRTNMPSFGKRTPFTDEHLQPFERV YGEDPHGLSPRTEGEWSFNAEETEVADSEENKNTDQHLATSRWRKFSREWI RTAKSDSLDISWLKDKDSIDADSLPEPDVLAAEAMGELVQALSELDALMRE LGASDEADLQRQLLEEAFGGVKE
62	E. coli strain 12 hsdS	MSAGKLPEGWVIAPVSTVTTLIRGVTYKKEQAINYLKDDYLPLIRANNIQN GKFDTTDLVFVPKNLVKESQKISPEDIVIAMSSGSKSVVGKSAHQHLPFECS FGAFCGVLRPEKLIFSGFIAHFTKSSLYRNKISSLSAGANINNIKPASFDLINIPI PPLAEQKIIAEKLDTLLAQVDSTKARFEQIPQILKRFRQAVLGGAVNGKLTE KWRNFEPQHSVFKKLNFESILTELRNGLSSKPNESGVGHPILRISSVRAGHV DQNDIRFLECSESELNRHKLQDGDLLFTRYNGSLEFVGVCGLLKKLQHQNL LYPDKLIRARLTKDALPEYIEIFFSSPSARNAMMNCVKTTSGQKGISGKDIKS QVVLLPPVKEQAEIVRRVEQLFAYADTIEKQVNNALARVNNLTQSILAKAF RGELTAQWRAENPDLISGENSAAALLEKIKAERAASGGKKASRKKS
63	T. aquaticus M TaqI	MGLPPLLSLPSNSAPRSLGRVETPPEVVDFMVSLAEAPRGGRVLEPACAHGP FLRAFREAHGTAYRFVGVEIDPKALDLPPWAEGILADFLLWEPGEAFDLILG NPPYGIVGEASKYPIHVFKAVKDLYKKAFSTWKGKYNLYGAFLEKAVRLL KPGGVLVFVVPATWLVLEDFALLREFLAREGKTSVYYLGEVFPQKKVSAV VIRFQKSGKGLSLWDTQESESGFTPILWAEYPHWEGEIIRFETEETRKLEISG MPLGDLFHIRFAARSPEFKKHPAVRKEPGPGLVPVLTGRNLKPGWVDYEKN HSGLWMPKERAKELRDFYATPHLVVAHTKGTRVVAAWDERAYPWREEFH LLPKEGVRLDPSSLVQWLNSEAMQKHVRTLYRDFVPHLTLRMLERLPVRR EYGFHTSPESARNF
64	E. coli M EcoDam	MKKNRAFLKWAGGKYPLLDDIKRHLPKGECLVEPFVGAGSVFLNTDFSRYI LADINSDLISLYNIVKMRTDEYVQAARELFVPETNCAEVYYQFREEFNKSQ DPFRRAVLFLYLNRYGYNGLCRYNLRGEFNVPFGRYKKPYFPEAELYHFAE KAQNAFFYCESYADSMARADDASVVYCDPPYAPLSATANFTAYHTNSFTL EQQAHLAEIAEGLVERHIPVLISNHDTMLTREWYQRAKLHVVKVRRSISSN GGTRKKVDELLALYKPGVVSPAKK
65	C. crescentus M CcrMI	MKFGPETIIHGDCIEQMNALPEKSVDLIFADPPYNLQLGGDLLRPDNSKVDA VDDHWDQFESFAAYDKFTREWLKAARRVLKDDGAIWVIGSYHNIFRVGV AVQDLGFWILNDIVWRKSNPMPNFKGTRFANAHETLIWASKSQNAKRYTF NYDALKMANDEVQMRSDWTIPLCTGEERIKGADGQKAHPTQKPEALLYRV ILSTTKPGDVILDPFFGVGTTGAAAKRLGRKFIGIEREAEYLEHAKARIAKVV PIAPEDLDVMGSKRAEPRVPFGTIVEAGLLSPGDTLYCSKGTHVAKVRPDGS ITVGDLSGSIHKIGALVQSAPACNGWTYWHFKTDAGLAPIDVLRAQVRAG MN
66	C. difficile CamA	MDDISQDNFLLSKEYENSLDVDTKKASGIYYTPKIIVDYIVKKTLKNHDIIKN PYPRILDISCGCGNFLLEVYDILYDLFEENIYELKKKYDENYWTVDNIHRHIL NYCIYGADIDEKAISILKDSLTNKKVVNDLDESDIKINLFCCDSLKKKWRYK FDYIVGNPPYIGHKKLEKKYKKFLLEKYSEVYKDKADLYFCFYKKIIDILKQ GGIGSVITPRYFLESLSGKDLREYIKSNVNVQEIVDFLGANIFKNIGVSSCILT FDKKKTKETYIDVFKIKNEDICINKFETLEELLKSSKFEHFNINQRLLSDEWIL VNKDDETFYNKIQEKCKYSLEDIAISFQGIITGCDKAFILSKDDVKLNLVDD KFLKCWIKSKNINKYIVDKSEYRLIYSNDIDNENTNKRILDEIIGLYKTKLEN RRECKSGIRKWYELQWGREKLFFERKKIMYPYKSNENRFAIDYDNNFSSAD

SEQ ID NO	Description	Sequence
		VYSFFIKEEYLDKFSYEYLVGILNSSVYDKYFKITAKKMSKNIYDYYPNKV MKIRIFRDNNYEEIENLSKQIISILLNKSIDKGKVEKLQIKMDNLIMDSLGI
67	ZIM3	MNNSQGRVTFEDVTVNFTQGEWQRLNPEQRNLYRDVMLENYSNLVSVGQ GETTKPDVILRLEQGKEPWLEEEEVLGSGRAEKNGDIGGQIWKPKDVKESL
68	ZNF436	MAATLLMAGSQAPVTFEDMAMYLTREEWRPLDAAQRDLYRDVMQENYG NVVSLDFEIRSENEVNPKQEISEDVQFGTTSERPAENAEENPESEEGFESGDR SERQW
69	ZNF257	MLENYRNLVFLGIAVSKPDLITCLEQGKEPCNMKRHEMVAKPPVMCSHIAE DLCPERDIKYFFQKVILRRYDKCEHENLQLRKGCKSVDECKVCK
70	ZNF675	MGLLTFRDVAIEFSLEEWQCLDTAQRNLYKNVILENYRNLVFLGIAVSKQD LITCLEQEKEPLTVKRHEMVNEPPVMCSHFAQEFWPEQNIKDSF
71	ZNF490	MLQMQNSEHHGQSIKTQTDSISLEDVAVNFTLEEWALLDPGQRNIYRDVM RATFKNLACIGEKWKDQDIEDEHKNQGRNLRSPMVEALCENKEDCPCGKS TSQIPDLNTNLETPTG
72	ZNF320	MALSQGLLTFRDVAIEFSQEEWKCLDPAQRTLYRDVMLENYRNLVSLDISS KCMMNTLSSTGQGNTEVIHTGTLQRQASYHIGAFCSQEIEKDIHDFVFQ
73	ZNF331	MAQGLVTFADVAIDFSQEEWACLNSAQRDLYWDVMLENYSNLVSLDLES AYENKSLPTKKNIHEIRASKRNSDRRSKSLGRNWICEGTLERPQRSRGR
74	ZNF816	MLREEATKKSKEKEPGMALPQGRLTFRDVAIEFSLEEWKCLNPAQRALYR AVMLENYRNLEFVDSSLKSMMEFSSTRHSITGEVIHTGTLQRHKSHHIGDFC FPEMKKDIHHFEFQWQ
75	ZNF680	MPGPPGSLEMGPLTFRDVAIEFSLEEWQCLDTAQRNLYRKVMFENYRNLVF LGIAVSKPHLITCLEQGKEPWNRKRQEMVAKPPVIYSHFTEDLWPEHSIKDS F
76	ZNF41	MSPPWSPALAAEGRGSSCEASVSFEDVTVDFSKEEWQHLDPAQRRLYWDV TLENYSHLLSVGYQIPKSEAAFKLEQGEGPWMLEGEAPHQSCSGEAIGKMQ QQGIPGGIFFHC
77	ZNF189	MASPSPPPESKEEWDYLDPAQRSLYKDVMMENYGNLVSLDVLNRDKDEEP TVKQEIEEIEEEVEPQGVIVTRIKSEIDQDPMGRETFELVGRLDKQRGIFLWEI PRESL
78	ZNF528	MALTQGPLKFMDVAIEFSQEEWKCLDPAQRTLYRDVMLENYRNLVSLGIC LPDLSVTSMLEQKRDPWTLQSEEKIANDPDGRECIKGVNTERSSKLGSN
79	ZNF543	MAASAQVSVTFEDVAVTFTQEEWGQLDAAQRTLYQEVMLETCGLLMSLG CPLFKPELIYQLDHRQELWMATKDLSQSSYPGDNTKPKTTEPTFSHLALPE
80	ZNF554	MFSQEERMAAGYLPRWSQELVTFEDVSMDFSQEEWELLEPAQKNLYREV MLENYRNVVSLEALKNQCTDVGIKEGPLSPAQTSQVTSLSSWTGYLLFQPV ASSHLEQREALWIEEKGTPQASCSDWMTVLRNQDSTYKKVALQE
81	ZNF140	MSQGSVTFRDVAIDFSQEEWKWLQPAQRDLYRCVMLENYGHLVSLGLSIS KPDVVSLLEQGKEPWLGKREVKRDLFSVSESSGEIKDFSPKNVIYDD
82	ZNF610	MEEAQKRKAKESGMALPQGRLTFMDVAIEFSQEEWKSLDPGQRALYRDV MLENYRNLVFLGRSCVLGSNAENKPIKNQLGLTLESHLSELQLFQAGRKIY RSNQVEKFTNHR
83	ZNF264	MAAAVLTDRAQVSVTFDDVAVTFTKEEWGQLDLAQRTLYQEVMLENCGL LVSLGCPVPKAELICHLEHGQEPWTRKEDLSQDTCPGDKGKPKTTEPTTCEP ALSE
84	ZNF350	MIQAQESITLEDVAVDFTWEEWQLLGAAQKDLYRDVMLENYSNLVAVGY QASKPDALFKLEQGEQLWTIEDGIHSGACSDIWKVDHVLERLQSESLVNR
85	ZNF8	MEGVAGVMSVGPPAARLQEPVTFRDVAVDFTQEEWGQLDPTQRILYRDV MLETFGHLLSIGPELPKPEVISQLEQGTELWVAERGTTQGCHPAWEPRSESQ ASRKEEGLPEE

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86	ZNF582	MSLGSELFRDVAIVFSQEEWQWLAPAQRDLYRDVMLETYSNLVSLGLAVS KPDVISFLEQGKEPWMVERVVSGGLCPVLESRYDTKELFPKQHVYEV
87	ZNF30	MAHKYVGLQYHGSVTFEDVAIAFSQQEWESLDSSQRGLYRDVMLENYRN LVSMAGHSRSKPHVIALLEQWKEPEVTVRKDGRRWCTDLQLEDDTIGCKE MPTSEN
88	ZNF324	MAFEDVAVYFSQEEWGLLDTAQRALYRRVMLDNFALVASLGLSTSRPRVV IQLERGEEPWVPSGTDTTLSRTTYRRRNPGSWSLTEDRDVSG
89	ZNF98	MLENYRNLVFVGIAASKPDLITCLEQGKEPWNVKRHEMVTEPPVVYSYFA QDLWPKQGKKNYFQKVILRTYKKCGRENLQLRKYCKSMDECKVHKECYN GLNQC
90	ZNF669	MHFRRPDPCREPLASPIQDSVAFEDVAVNFTQEEWALLDSSQKNLYREVMQ ETCRNLASVGSQWKDQNIEDHFEKPGKDIRNHIVQRLCESKEDGQYGEVVS QIPNLDLNENISTGLKPCECSICGK
91	ZNF677	MALSQGLFTFKDVAIEFSQEEWECLDPAQRALYRDVMLENYRNLLSLDED NIPPEDDISVGFTSKGLSPKENNKEELYHLVILERKESHGINNFDLKEVWEN MPKFDSLW
92	ZNF596	MTFEDIIVDFTQEEWALLDTSQRKLFQDVMLENISHLVSIGKQLCKSVVLSQ LEQVEKLSTQRISLLQGREVGIKHQEIPFIHHIYQKGTSTISTMRS
93	ZNF214	MAVTFEDVTIIFTWEEWKFLDSSQKRLYREVMWENYTNVMSVENWNESY KSQEEKFRYLEYENFSYWQGWWNAGAQMYENQNYGETVQGTDSKDLTQ QDRSQC
94	ZNF37A	MITSQGSVSFRDVTVGFTQEEWQHLDPAQRTLYRDVMLENYSHLVSVGYC IPKPEVILKLEKGEEPWILEEKFPSQSHLELINTSRNYSIMKFNEFNKG
95	ZNF34	MFEDVAVYLSREEWGRLGPAQRGLYRDVMLETYGNLVSLGVGPAGPKPG VISQLERGDEPWVLDVQGTSGKEHLRVNSPALGTRTEYKELTSQETFGEED PQGSEPVEACDHIS
96	ZNF250	METYGNVVSLGLPGSKPDIISQLERGEDPWVLDRKGAKKSQGLWSDYSDN LKYDHTTACTQQDSLSCPWECETKGESQNTDLSPKPLISEQTVILGKTPLGRI DQENNETKQ
97	ZNF547	MAEMNPAQGHVVFEDVAIYFSQEEWGHLDEAQRLLYRDVMLENLALLSSL GCCHGAEDEEAPLEPGVSVGVSQVMAPKPCLSTQNTQPCETCSSLLKDILRL
98	ZNF273	MLDNYRNLVFLGIAVSKPDLITCLEQGKEPCNMKRHAMVAKPPVVCSHFA QDLWPKQGLKDS
99	ZNF354A	MAAGQREARPQVSLTFEDVAVLFTRDEWRKLAPSQRNLYRDVMLENYRN LVSLGLPFTKPKVISLLQQGEDPWEVEKDGSGVSSLGSKSSHKTTKSTQTQD SSFQ
100	ZFP82	MALRSVMFSDVSIDFSPEEWEYLDLEQKDLYRDVMLENYSNLVSLGCFISK PDVISSLEQGKEPWKVVRKGRRQYPDLETKYETKKLSLENDIYEIN
101	ZNF224	MTTFKEAMTFKDVAVVFTEEELGLLDLAQRKLYRDVMLENFRNLLSVGHQ AFHRDTFHFLREEKIWMMKTAIQREGNSGDKIQTEMETVSEAGTHQEW
102	ZNF33A	MFQVEQKSQESVSFKDVTVGFTQEEWQHLDPSQRALYRDVMLENYSNLVS VGYCVHKPEVIFRLQQGEEPWKQEEEFPSQSFPEVWTADHLKERSQENQSK HL
103	ZNF45	MTKSKEAVTFKDVAVVFSEEELQLLDLAQRKLYRDVMLENFRNVVSVGH QSTPDGLPQLEREEKLWMMKMATQRDNSSGAKNLKEMETLQEVGLRYLP
104	ZNF175	MSQKPQVLGPEKQDGSCEASVSFEDVTVDFSREEWQQLDPAQRCLYRDVM LELYSHLFAVGYHIPNPEVIFRMLKEKEPRVEEAEVSHQRCQEREFGLEIPQ KEISKKASFQ
105	ZNF595	MELVTFRDVAIEFSPEEWKCLDPAQQNLYRDVMLENYRNLVSLGFVISNPD LVTCLEQIKEPCNLKIHETAAKPPAICSPFSQDLSPVQGIEDSF

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106	ZNF184	MSTLLQGGHNLLSSASFQESVTFKDVIVDFTQEEWKQLDPGQRDLFRDVTL ENYTHLVSIGLQVSKPDVISQLEQGTEPWIMEPSIPVGTCADWETRLENSVS APEPDISEE
107	ZNF419	MDPAQVPVAADLLTDHEEGYVTFEDVAVYFSQEEWRLLDDAQRLLYRNV MLENFTLLASLGLASSKTHEITQLESWEEPFMPAWEVVTSAIPRGCWHGAE AEEAPEQIASVG
108	ZFP28-1	MKKLEAVGTGIEPKAMSQGLVTFGDVAVDFSQEEWEWLNPIQRNLYRKV MLENYRNLASLGLCVSKPDVISSLEQGKEPWTVKRKMTRAWCPDLKAVW KIKELPLKKDFCEG
109	ZFP28-2	MSLLGEHWDYDALFETQPGLVTIKNLAVDFRQQLHPAQKNFCKNGIWENN SDLGSAGHCVAKPDLVSLLEQEKEPWMVKRELTGSLFSGQRSVHETQELFP KQDSYAE
110	ZNF18	MLALAASQPARLEERLIRDRDLGASLLPAAPQEQWRQLDSTQKEQYWDLIL ETYGKMVSGAGISHPKSDLTNSIEFGEELAGIYLHVNEKIPRPTCIGDRQEND KENLNLENH
111	ZNF213	MEGRPGETTDTCFVSGVHGPVALGDIPFYFSREEWGTLDPAQRDLFWDIKR ENSRNTTLGFGLKGQSEKSLLQEMVPVVPGQTGSDVTVSWSPEEAEAWESE NRPRAALGPVVGARRGRPPTRRRQFRDLA
112	ZNF394	MVAVVRALQRALDGTSSQGMVTFEDTAVSLTWEEWERLDPARRDFCRES AQKDSGSTVPPSLESRVENKELIPMQQILEEAEPQGQLQEAFQGKRPLFSKC GSTHEDRVEKQSGDP
113	ZFP1	MNKSQGSVSFTDVTVDFTQEEWEQLDPSQRILYMDVMLENYSNLLSVEVW KADDQMERDHRNPDEQARQFLILKNQTPIEERGDLFGKALNLNTDFVSLRQ VPYKYDLYEKTL
114	ZFP14	MAHGSVTFRDVAIDFSQEEWEFLDPAQRDLYRDVMWENYSNFISLGPSISK PDVITLLDEERKEPGMVVREGTRRYCPDLESRYRTNTLSPEKDIYEIYSFQW DIMER
115	ZNF416	MAAAVLRDSTSVPVTAEAKLMGFTQGCVTFEDVAIYFSQEEWGLLDEAQR LLYRDVMLENFALITALVCWHGMEDEETPEQSVSVEGVPQVRTPEASPSTQ KIQSCDMCVPFLTDILHLTDLPGQELYLTGACAVFHQDQK
116	ZNF557	MLPPTAASQREGHTEGGELVNELLKSWLKGLVTFEDVAVEFTQEEWALLD PAQRTLYRDVMLENCRNLASLGNQVDKPRLISQLEQEDKVMTEERGILSGT CPDVENPFKAKGLTPKLHVFRKEQSRNMKMER
117	ZNF566	MAQESVMFSDVSVDFSQEEWECLNDDQRDLYRDVMLENYSNLVSMGHSIS KPNVISYLEQGKEPWLADRELTRGQWPVLESRCETKKLFLKKEIYEIESTQW EIMEK
118	ZNF729	MPGAPGSLEMGPLTFRDVTIEFSLEEWQCLDTVQQNLYRDVMLENYRNLV FLGMAVFKPDLITCLKQGKEPWNMKRHEMVTKPPVMRSHFTQDLWPDQS TKDSFQEVILRTYAR
119	ZIM2	MAGSQFPDFKHLGTFLVFEELVTFEDVLVDFSPEELSSLSAAQRNLYREVM LENYRNLVSLGHQFSKPDIISRLEEEESYAMETDSRHTVICQGE
120	ZNF254	MPGPPRSLEMGLLTFRDVAIEFSLEEWQHLDIAQQNLYRNVMLENYRNLAF LGIAVSKPDLITCLEQGKEPWNMKRHE
121	ZNF764	MAPPLAPLPPRDPNGAGPEWREPGAVSFADVAVYFCREEWGCLRPAQRAL YRDVMRETYGHLSALGIGGNKPALISWVEEEAELWGPAAQDPE
122	ZNF785	MGPPLAPRPAHVPGEAGPRRTRESRPGAVSFADVAVYFSPEEWECLRPAQR ALYRDVMRETFGHLGALGFSVPKPAFISWVEGEVEAWSPEAQDPDGESS
123	ZNF10 (KOX1)	MDAKSLTAWSRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKN LVSLGYQLTKPDVILRLEKGEEPWLVEREIHQETHPDSETAFEIKSSVSSRSIF KDKQSCDIKMEGMARNDLWYLSLEEVWKCRDQLDKYQENPERHLRQVAF TQKKVLTQERVSESGKYGGNCLLPAQLVLREYFHKRDSHTKSLKHDLVLN

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		GHQDSCASNSNECGQTFCQNIHLIQFARTHTGDKSYKCPDNDNSLTHGSSL GISKGIHREKPYECKECGKFFSWRSNLTRHQLIHTGEKPYECKECGKSFSRSS HLIGHQKTHTGEEPYECKECGKSFSWFSHLVTHQRTHTGDKLYTCNQCGKS FVHSSRLIRHQRTHTGEKPYECPECGKSFRQSTHLILHQRTHVRVRPYECNE CGKSYSQRSHLVVHHRIHTGLKPFECKDCGKCFSRSSHLYSHQRTHTGEKP YECHDCGKSFSQSSALIVHQRIHTGEKPYECCQCGKAFIRKNDLIKHQRIHV GEETYKCNQCGIIFSQNSPFIVHQIAHTGEQFLTCNQCGTALVNTSNLIGYQT NHIRENAY
124	CBX5 (chromosha dow domain)	MGKKTKRTADSSSSEDEEEYVVEKVLDRRVVKGQVEYLLKWKGFSEEHNT WEPEKNLDCPELISEFMKKYKKMKEGENNKPREKSESNKRKSNFSNSADDI KSKKKREQSNDIARGFERGLEPEKIIGATDSCGDLMFLMKWKDTDEADLVL AKEANVKCPQIVIAFYEERLTWHAYPEDAENKEKETAKS
125	RYBP (YAF2_RY BP component of PRC1)	MTMGDKKSPTRPKRQAKPAADEGFWDCSVCTFRNSAEAFKCSICDVRKGT STRKPRINSQLVAQQVAQQYATPPPPKKEKKEKVEKQDKEKPEKDKEISPS VTKKNTNKKTKPKSDILKDPPSEANSIQSANATTKTSETNHTSRPRLKNVDR STAQQLAVTVGNVTVIITDFKEKTRSSSTSSSTVTSSAGSEQQNQSSSGSEST DKGSSRSSTPKGDMSAVNDESF
126	YAF2 (YAF2_RY BP component of PRC1)	MGDKKSPTRPKRQPKPSSDEGYWDCSVCTFRNSAEAFKCMMCDVRKGTST RKPRPVSQLVAQQVTQQFVPPTQSKKEKKDKVEKEKSEKETTSKKNSHKK TRPRLKNVDRSSAQHLEVTVGDLTVIITDFKEKTKSPPASSAASADQHSQSG SSSDNTERGMSRSSSPRGEASSLNGESH
127	MGA (componen t of PRC1.6)	MEEKQQIILANQDGGTVAGAAPTFFVILKQPGNGKTDQGILVTNQDACALA SSVSSPVKSKGKICLPADCTVGGITVTLDNNSMWNEFYHRSTEMILTKQGR RMFPYCRYWITGLDSNLKYILVMDISPVDNHRYKWNGRWWEPSGKAEPH VLGRVFIHPESPSTGHYWMHQPVSFYKLKLTNNTLDQEGHIILHSMHRYLP RLHLVPAEKAVEVIQLNGPGVHTFTFPQTEFFAVTAYQNIQITQLKIDYNPF AKGFRDDGLNNKPQRDGKQKNSSDQEGNNISSSSGHRVRLTEGQGSEIQPG DLDPLSRGHETSGKGLEKTSLNIKRDFLGFMDTDSALSEVPQLKQEISECLIA SSFEDDSRVASPLDQNGSFNVVIKEEPLDDYDYELGECPEGVTVKQEETDEE TDVYSNSDDDPILEKQLKRHNKVDNPEADHLSSKWLPSSPSGVAKAKMFK LDTGKMPVVYLEPCAVTRSTVKISELPDNMLSTSRKDKSSMLAELEYLPTYI ENSNETAFCLGKESENGLRKHSPDLRVVQKYPLLKEPQWKYPDISDSISTER ILDDSKDSVGDSLSGKEDLGRKRTTMLKIATAAKVVNANQNASPNVPGKR GRPRKLKLCKAGRPPKNTGKSLISTKNTPVSPGSTFPDVKPDLEDVDGVLFV SFESKEALDIHAVDGTTEESSSLQASTTNDSGYRARISQLEKELIEDLKTLRH KQVIHPGLQEVGLKLNSVDPTMSIDLKYLGVQLPLAPATSFPFWNLTGTNP ASPDAGFPFVSRTGKTNDFTKIKGWRGKFHSASASRNEGGNSESSLKNRSA FCSDKLDEYLENEGKLMETSMGFSSNAPTSPVVYQLPTKSTSYVRTLDSVL KKQSTISPSTSYSLKPHSVPPVSRKAKSQNRQATFSGRTKSSYKSILPYPVSP KQKYSHVILGDKVTKNSSGIISENQANNFVVPTLDENIFPKQISLRQAQQQQ QQQQGSRPPGLSKSQVKLMDLEDCALWEGKPRTYITEERADVSLTTLLTAQ ASLKTKPIHTIIRKRAPPCNNDFCRLGCVCSSLALEKRQPAHCRRPDCMFGC TCLKRKVVLVKGGSKTKHFQRKAAHRDPVFYDTLGEEAREEEEGIREEEEQ LKEKKKRKKLEYTICETEPEQPVRHYPLWVKVEGEVDPEPVYIPTPSVIEPM KPLLLPQPEVLSPTVKGKLLTGIKSPRSYTPKPNPVIREEDKDPVYLYFESM MTCARVRVYERKKEDQRQPSSSSSPSPSFQQQTSCHSSPENHNNAKEPDSEQ QPLKQLTCDLEDDSDKLQEKSWKSSCNEGESSSTSYMHQRSPGGPTKLIEIIS DCNWEEDRNKILSILSQHINSNMPQSLKVGSFIIELASQRKSRGEKNPPVYSS RVKISMPSCQDQDDMAEKSGSETPDGPLSPGKMEDISPVQTDALDSVRERL HGGKGLPFYAGLSPAGKLVAYKRKPSSSTSGLIQVASNAKVAASRKPRTLL PSTSNSKMASSSGTATNRPGKNLKAFVPAKRPIAARPSPGGVFTQFVMSKV

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		GALQQKIPGVSTPQTLAGTQKFSIRPSPVMVVTPVVSSEPVQVCSPVTAAVT TTTPQVFLENTTAVTPMTAISDVETKETTYSSGATTTGVVEVSETNTSTSVT STQSTATVNLTKTTGITTPVASVAFPKSLVASPSTITLPVASTASTSLVVVTA AASSSMVTTPTSSLGSVPIILSGINGSPPVSQRPENAAQIPVATPQVSPNTVKR AGPRLLLIPVQQGSPTLRPVSNTQLQGHRMVLQPVRSPSGMNLFRHPNGQI VQLLPLHQLRGSNTQPNLQPVMFRNPGSVMGIRLPAPSKPSETPPSSTSSSAF SVMNPVIQAVGSSSAVNVITQAPSLLSSGASFVSQAGTLTLRISPPEPQSFAS KTGSETKITYSSGGQPVGTASLIPLQSGSFALLQLPGQKPVPSSILQHVASLQ MKRESQNPDQKDETNSIKREQETKKVLQSEGEAVDPEANVIKQNSGAATSE ETLNDSLEDRGDHLDEECLPEEGCATVKPSEHSCITGSHTDQDYKDVNEEY GARNRKSSKEKVAVLEVRTISEKASNKTVQNLSKVQHQKLGDVKVEQQKG FDNPEENSSEFPVTFKEESKFELSGSKVMEQQSNLQPEAKEKECGDSLEKDR ERWRKHLKGPLTRKCVGASQECKKEADEQLIKETKTCQENSDVFQQEQGIS DLLGKSGITEDARVLKTECDSWSRISNPSAFSIVPRRAAKSSRGNGHFQGHL LLPGEQIQPKQEKKGGRSSADFTVLDLEEDDEDDNEKTDDSIDEIVDVVSDY QSEEVDDVEKNNCVEYIEDDEEHVDIETVEELSEEINVAHLKTTAAHTQSFK QPSCTHISADEKAAERSRKAPPIPLKLKPDYWSDKLQKEAEAFAYYRRTHT ANERRRRGEMRDLFEKLKITLGLLHSSKVSKSLILTRAFSEIQGLTDQADKLI GQKNLLTRKRNILIRKVSSLSGKTEEVVLKKLEYIYAKQQALEAQKRKKKM GSDEFDISPRISKQQEGSSASSVDLGQMFINNRRGKPLILSRKKDQATENTSP LNTPHTSANLVMTPQGQLLTLKGPLFSGPVVAVSPDLLESDLKPQVAGSAV ALPENDDLFMMPRIVNVTSLATEGGLVDMGGSKYPHEVPDSKPSDHLKDT VRNEDNSLEDKGRISSRGNRDGRVTLGPTQVFLANKDSGYPQIVDVSNMQ KAQEFLPKKISGDMRGIQYKWKESESRGERVKSKDSSFHKLKMKDLKDSSI EMELRKVTSAIEEAALDSSELLTNMEDEDDTDETLTSLLNEIAFLNQQLNDD SVGLAELPSSMDTEFPGDARRAFISKVPPGSRATFQVEHLGTGLKELPDVQG ESDSISPLLLHLEDDDFSENEKQLAEPASEPDVLKIVIDSEIKDSLLSNKKAID GGKNTSGLPAEPESVSSPPTLHMKTGLENSNSTDTLWRPMPKLAPLGLKVA NPSSDADGQSLKVMPCLAPIAAKVGSVGHKMNLTGNDQEGRESKVMPTLA PVVAKLGNSGASPSSAGK
128	CBX1 (chromosha dow)	MGKKQNKKKVEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKWKGFSDED NTWEPEENLDCPDLIAEFLQSQKTAHETDKSEGGKRKADSDSEDKGEESKP KKKKEESEKPRGFARGLEPERIIGATDSSGELMFLMKWKNSDEADLVPAKE ANVKCPQVVISFYEERLTWHSYPSEDDDKKDDKN
129	SCMH1 (SAM_1/S PM)	MLVCYSVLACEILWDLPCSIMGSPLGHFTWDKYLKETCSVPAPVHCFKQSY TPPSNEFKISMKLEAQDPRNTTSTCIATVVGLTGARLRLRLDGSDNKNDFW RLVDSAEIQPIGNCEKNGGMLQPPLGFRLNASSWPMFLLKTLNGAEMAPIRI FHKEPPSPSHNFFKMGMKLEAVDRKNPHFICPATIGEVRGSEVLVTFDGWR GAFDYWCRFDSRDIFPVGWCSLTGDNLQPPGTKVVIPKNPYPASDVNTEKP SIHSSTKTVLEHQPGQRGRKPGKKRGRTPKTLISHPISAPSKTAEPLKFPKKR GPKPGSKRKPRTLLNPPPASPTTSTPEPDTSTVPQDAATIPSSAMQAPTVCIY LNKNGSTGPHLDKKKVQQLPDHFGPARASVVLQQAVQACIDCAYHQKTVF SFLKQGHGGEVISAVFDREQHTLNLPAVNSITYVLRFLEKLCHNLRSDNLFG NQPFTQTHLSLTAIEYSHSHDRYLPGETFVLGNSLARSLEPHSDSMDSASNP TNLVSTSQRHRPLLSSCGLPPSTASAVRRLCSRGVLKGSNERRDMESFWKL NRSPGSDRYLESRDASRLSGRDPSSWTVEDVMQFVREADPQLGPHADLFRK HEIDGKALLLLRSDMMMKYMGLKLGPALKLSYHIDRLKQGKF
130	MPP8 (Chromodo main)	MEQVAEGARVTAVPVSAADSTEELAEVEEGVGVVGEDNDAAARGAEAFG DSEEDGEDVFEVEKILDMKTEGGKVLYKVRWKGYTSDDDTWEPEIHLEDC KEVLLEFRKKIAENKAKAVRKDIQRLSLNNDIFEANSDSDQQSETKEDTSPK KKKKKLRQREEKSPDDLKKKKAKAGKLKDKSKPDLESSLESLVFDLRTKK RISEAKEELKESKKPKKDEVKETKELKKVKKGEIRDLKTKTREDPKENRKT

SEQ ID NO	Description	Sequence
		KKEKFVESQVESESSVLNDSPFPEDDSEGLHSDSREEKQNTKSARERAGQD MGLEHGFEKPLDSAMSAEEDTDVRGRRKKKTPRKAEDTRENRKLENKNAF LEKKTVPKKQRNQDRSKSAAELEKLMPVSAQTPKGRRLSGEERGLWSTDS AEEDKETKRNESKEKYQKRHDSDKEEKGRKEPKGLKTLKEIRNAFDLFKLT PEEKNDVSENNRKREEIPLDFKTIDDHKTKENKQSLKERRNTRDETDTWAY IAAEGDQEVLDSVCQADENSDGRQQILSLGMDLQLEWMKLEDFQKHLDGK DENFAATDAIPSNVLRDAVKNGDYITVKVALNSNEEYNLDQEDSSGMTLV MLAAAGGQDDLLRLLITKGAKVNGRQKNGTTALIHAAEKNFLTTVAILLEA GAFVNVQQSNGETALMKACKRGNSDIVRLVIECGADCNILSKHQNSALHFA KQSNNVLVYDLLKNHLETLSRVAEETIKDYFEARLALLEPVFPIACHRLCEG PDFSTDFNYKPPQNIPEGSGILLFIFHANFLGKEVIARLCGPCSVQAVVLNDK FQLPVFLDSHFVYSFSPVAGPNKLFIRLTEAPSAKVKLLIGAYRVQLQ
131	SUMO3 (Rad60-SLD)	MSEEKPKEGVKTENDHINLKVAGQDGSVVQFKIKRHTPLSKLMKAYCERQ GLSMRQIRFRFDGQPINETDTPAQLEMEDEDTIDVFQQQTGGVPESSLAGHS F
132	HERC2 (Cyt-b5)	MPSESFCLAAQARLDSKWLKTDIQLAFTRDGLCGLWNEMVKDGEIVYTGT ESTQNGELPPRKDDSVEPSGTKKEDLNDKEKKDEEETPAPIYRAKSILDSWV WGKQPDVNELKECLSVLVKEQQALAVQSATTTLSALRLKQRLVILERYFIA LNRTVFQENVKVKWKSSGISLPPVDKKSSRPAGKGVEGLARVGSRAALSFA FAFLRRAWRSGEDADLCSELLQESLDALRALPEASLFDESTVSSVWLEVVE RATRFLRSVVTGDVHGTPATKGPGSIPLQDQHLALAILLELAVQRGTLSQM LSAILLLLQLWDSGAQETDNERSAQGTSAPLLPLLQRFQSIICRKDAPHSEGD MHLLSGPLSPNESFLRYLTLPQDNELAIDLRQTAVVVMAHLDRLATPCMPP LCSSPTSHKGSLQEVIGWGLIGWKYYANVIGPIQCEGLANLGVTQIACAEKR FLILSRNGRVYTQAYNSDTLAPQLVQGLASRNIVKIAAHSDGHHYLALAAT GEVYSWGCGDGGRLGHGDTVPLEEPKVISAFSGKQAGKHVVHIACGSTYS AAITAEGELYTWGRGNYGRLGHGSSEDEAIPMLVAGLKGLKVIDVACGSG DAQTLAVTENGQVWSWGDGDYGKLGRGGSDGCKTPKLIEKLQDLDVVKV RCGSQFSIALTKDGQVYSWGKGDNQRLGHGTEEHVRYPKLLEGLQGKKVI DVAAGSTHCLALTEDSEVHSWGSNDQCQHFDTLRVTKPEPAALPGLDTKHI VGIACGPAQSFAWSSCSEWSIGLRVPFVVDICSMTFEQLDLLLRQVSEGMD GSADWPPPQEKECVAVATLNLLRLQLHAAISHQVDPEFLGLGLGSILLNSLK QTVVTLASSAGVLSTVQSAAQAVLQSGWSVLLPTAEERARALSALLPCAVS GNEVNISPGRRFMIDLLVGSLMADGGLESALHAAITAEIQDIEAKKEAQKEK EIDEQEANASTFHRSRTPLDKDLINTGICESSGKQCLPLVQLIQQLLRNIASQT VARLKDVARRISSCLDFEQHSRERSASLDLLLRFQRLLISKLYPGESIGQTSDI SSPELMGVGSLLKKYTALLCTHIGDILPVAASIASTSWRHFAEVAYIVEGDF TGVLLPELVVSIVLLLSKNAGLMQEAGAVPLLGGLLEHLDRFNHLAPGKER DDHEELAWPGIMESFFTGQNCRNNEEVTLIRKADLENHNKDGGFWTVIDG KVYDIKDFQTQSLTGNSILAQFAGEDPVVALEAALQFEDTRESMHAFCVGQ YLEPDQEIVTIPDLGSLSSPLIDTERNLGLLLGLHASYLAMSTPLSPVEIECAK WLQSSIFSGGLQTSQIHYSYNEEKDEDHCSSPGGTPASKSRLCSHRRALGDH SQAFLQAIADNNIQDHNVKDFLCQIERYCRQCHLTTPIMFPPEHPVEEVGRL LLCCLLKHEDLGHVALSLVHAGALGIEQVKHRTLPKSVVDVCRVVYQAKC SLIKTHQEQGRSYKEVCAPVIERLRFLFNELRPAVCNDLSIMSKFKLLSSLPR WRRIAQKIIRERRKKRVPKKPESTDDEEKIGNEESDLEEACILPHSPINVDKR PIAIKSPKDKWQPLLSTVTGVHKYKWLKQNVQGLYPQSPLLSTIAEFALKEE PVDVEKMRKCLLKQLERAEVRLEGIDTILKLASKNFLLPSVQYAMFCGWQ RLIPEGIDIGEPLTDCLKDVDLIPPFNRMLLEVTFGKLYAWAVQNIRNVLMD ASAKFKELGIQPVPLQTITNENPSGPSLGTIPQARFLLVMLSMLTLQHGANN LDLLLNSGMLALTQTALRLIGPSCDNVEEDMNASAQGASATVLEETRKETA PVQLPVSGPELAAMMKIGTRVMRGVDWKWGDQDGPPPGLGRVIGELGED

SEQ ID NO	Description	Sequence
		GWIRVQWDTGSTNSYRMGKEGKYDLKLAELPAAAQPSAEDSDTEDDSEAE QTERNIHPTAMMFTSTINLLQTLCLSAGVHAEIMQSEATKTLCGLLRMLVES GTTDKTSSPNRLVYREQHRSWCTLGFVRSIALTPQVCGALSSPQWITLLMK VVEGHAPFTATSLQRQILAVHLLQAVLPSWDKTERARDMKCLVEKLFDFL GSLLTTCSSDVPLLRESTLRRRRVRPQASLTATHSSTLAEEVVALLRTLHSLT QWNGLINKYINSQLRSITHSFVGRPSEGAQLEDYFPDSENPEVGGLMAVLA VIGGIDGRLRLGGQVMHDEFGEGTVTRITPKGKITVQFSDMRTCRVCPLNQ LKPLPAVAFNVNNLPFTEPMLSVWAQLVNLAGSKLEKHKIKKSTKQAFAG QVDLDLLRCQQLKLYILKAGRALLSHQDKLRQILSQPAVQETGTVHTDDGA VVSPDLGDMSPEGPQPPMILLQQLLASATQPSPVKAIFDKQELEAAALAVC QCLAVESTHPSSPGFEDCSSSEATTPVAVQHIRPARVKRRKQSPVPALPIVVQ LMEMGFSRRNIEFALKSLTGASGNASSLPGVEALVGWLLDHSDIQVTELSD ADTVSDEYSDEEVVEDVDDAAYSMSTGAVVTESQTYKKRADFLSNDDYA VYVRENIQVGMMVRCCRAYEEVCEGDVGKVIKLDRDGLHDLNVQCDWQ QKGGTYWVRYIHVELIGYPPPSSSSHIKIGDKVRVKASVTTPKYKWGSVTH QSVGVVKAFSANGKDIIVDFPQQSHWTGLLSEMELVPSIHPGVTCDGCQMF PINGSRFKCRNCDDFDFCETCFKTKKHNTRHTFGRINEPGQSAVFCGRSGKQ LKRCHSSQPGMLLDSWSRMVKSLNVSSSVNQASRLIDGSEPCWQSSGSQGK HWIRLEIFPDVLVHRLKMIVDPADSSYMPSLVVVSGGNSLNNLIELKTININP SDTTVPLLNDCTEYHRYIEIAIKQCRSSGIDCKIHGLILLGRIRAEEEDLAAVP FLASDNEEEEDEKGNSGSLIRKKAAGLESAATIRTKVFVWGLNDKDQLGGL KGSKIKVPSFSETLSALNVVQVAGGSKSLFAVTVEGKVYACGEATNGRLGL GISSGTVPIPRQITALSSYVVKKVAVHSGGRHATALTVDGKVFSWGEGDDG KLGHFSRMNCDKPRLIEALKTKRIRDIACGSSHSAALTSSGELYTWGLGEYG RLGHGDNTTQLKPKMVKVLLGHRVIQVACGSRDAQTLALTDEGLVFSWG DGDFGKLGRGGSEGCNIPQNIERLNGQGVCQIECGAQFSLALTKSGVVWT WGKGDYFRLGHGSDVHVRKPQVVEGLRGKKIVHVAVGALHCLAVTDSGQ VYAWGDNDHGQQGNGTTTVNRKPTLVQGLEGQKITRVACGSSHSVAWTT VDVATPSVHEPVLFQTARDPLGASYLGVPSDADSSAASNKISGASNSKPNRP SLAKILLSLDGNLAKQQALSHILTALQIMYARDAVVGALMPAAMIAPVECP SFSSAAPSDASAMASPMNGEECMLAVDIEDRLSPNPWQEKREIVSSEDAVT PSAVTPSAPSASARPFIPVTDDLGAASIIAETMTKTKEDVESQNKAAGPEPQA LDEFTSLLIADDTRVVVDLLKLSVCSRAGDRGRDVLSAVLSGMGTAYPQV ADMLLELCVTELEDVATDSQSGRLSSQPVVVESSHPYTDDTSTSGTVKIPGA EGLRVEFDRQCSTERRHDPLTVMDGVNRIVSVRSGREWSDWSSELRIPGDE LKWKFISDGSVNGWGWRFTVYPIMPAAGPKELLSDRCVLSCPSMDLVTCL LDFRLNLASNRSIVPRLAASLAACAQLSALAASHRMWALQRLRKLLTTEFG QSININRLLGENDGETRALSFTGSALAALVKGLPEALQRQFEYEDPIVRGGK QLLHSPFFKVLVALACDLELDTLPCCAETHKWAWFRRYCMASRVAVALD KRTPLPRLFLDEVAKKIRELMADSENMDVLHESHDIFKREQDEQLVQWMN RRPDDWTLSAGGSGTIYGWGHNHRGQLGGIEGAKVKVPTPCEALATLRPV QLIGGEQTLFAVTADGKLYATGYGAGGRLGIGGTESVSTPTLLESIQHVFIK KVAVNSGGKHCLALSSEGEVYSWGEAEDGKLGHGNRSPCDRPRVIESLRGI EVVDVAAGGAHSACVTAAGDLYTWGKGRYGRLGHSDSEDQLKPKLVEAL QGHRVVDIACGSGDAQTLCLTDDDTVWSWGDGDYGKLGRGGSDGCKVP MKIDSLTGLGVVKVECGSQFSVALTKSGAVYTWGKGDYHRLGHGSDDHV RRPRQVQGLQGKKVIAIATGSLHCVCCTEDGEVYTWGDNDEGQLGDGTTN AIQRPRLVAALQGKKVNRVACGSAHTLAWSTSKPASAGKLPAQVPMEYNH LQEIPIIALRNRLLLLHHLSELFCPCIPMFDLEGSLDETGLGPSVGFDTLRGILI SQGKEAAFRKVVQATMVRDRQHGPVVELNRIQVKRSRSKGGLAGPDGTKS VFGQMCAKMSSFGPDSLLLPHRVWKVKFVGESVDDCGGGYSESIAEICEEL QNGLTPLLIVTPNGRDESGANRDCYLLSPAARAPVHSSMFRFLGVLLGIAIR

SEQ ID NO	Description	Sequence
		TGSPLSLNLAEPVWKQLAGMSLTIADLSEVDKDFIPGLMYIRDNEATSEEFE AMSLPFTVPSASGQDIQLSSKHTHITLDNRAEYVRLAINYRLHEFDEQVAAV REGMARVVPVPLLSLFTGYELETMVCGSPDIPLHLLKSVATYKGIEPSASLIQ WFWEVMESFSNTERSLFLRFVWGRTRLPRTIADFRGRDFVIQVLDKYNPPD HFLPESYTCFFLLKLPRYSCKQVLEEKLKYAIHFCKSIDTDDYARIALTGEPA ADDSSDDSDNEDVDSFASDSTQDYLTGH
133	BIN1 (SH3_9)	MAEMGSKGVTAGKIASNVQKKLTRAQEKVLQKLGKADETKDEQFEQCVQ NFNKQLTEGTRLQKDLRTYLASVKAMHEASKKLNECLQEVYEPDWPGRDE ANKIAENNDLLWMDYHQKLVDQALLTMDTYLGQFPDIKSRIAKRGRKLVD YDSARHHYESLQTAKKKDEAKIAKPVSLLEKAAPQWCQGKLQAHLVAQT NLLRNQAEEELIKAQKVFEEMNVDLQEELPSLWNSRVGFYVNTFQSIAGLE ENFHKEMSKLNQNLNDVLVGLEKQHGSNTFTVKAQPSDNAPAKGNKSPSP PDGSPAATPEIRVNHEPEPAGGATPGATLPKSPSQLRKGPPVPPPPKHTPSKE VKQEQILSLFEDTFVPEISVTTPSQFEAPGPFSEQASLLDLDFDPLPPVTSPVK APTPSGQSIPWDLWEPTESPAGSLPSGEPSAAEGTFAVSWPSQTAEPGPAQP AEASEVAGGTQPAAGAQEPGETAASEAASSSLPAVVVETFPATVNGTVEGG SGAGRLDLPPGFMFKVQAQHDYTATDTDELQLKAGDVVLVIPFQNPEEQD EGWLMGVKESDWNQHKELEKCRGVFPENFTERVP
134	PCGF2 (RING finger protein domain)	MHRTTRIKITELNPHLMCALCGGYFIDATTIVECLHSFCKTCIVRYLETNKY CPMCDVQVHKTRPLLSIRSDKTLQDIVYKLVPGLFKDEMKRRRDFYAAYPL TEVPNGSNEDRGEVLEQEKGALSDDEIVSLSIEFYEGARDRDEKKGPLENGD GDKEKTGVRFLRCPAAMTVMHLAKFLRNKMDVPSKYKVEVLYEDEPLKE YYTLMDIAYIYPWRRNGPLPLKYRVQPACKRLTLATVPTPSEGTNTSGASE CESVSDKAPSPATLPATSSSLPSPATPSHGSPSSHGPPATHPTSPTPPSTASGA TTAANGGSLNCLQTPSSTSRGRKMTVNGAPVPPLT
135	TOX (HMG box)	MDVRFYPPPAQPAAAPDAPCLGPSPCLDPYYCNKFDGENMYMSMTEPSQD YVPASQSYPGPSLESEDFNIPPITPPSLPDHSLVHLNEVESGYHSLCHPMNHN GLLPFHPQNMDLPEITVSNMLGQDGTLLSNSISVMPDIRNPEGTQYSSHPQM AAMRPRGQPADIRQQPGMMPHGQLTTINQSQLSAQLGLNMGGSNVPHNSP SPPGSKSATPSPSSSVHEDEGDDTSKINGGEKRPASDMGKKPKTPKKKKKK DPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDGLGEEQ KQVYKKKTEAAKKEYLKQLAAYRASLVSKSYSEPVDVKTSQPPQLINSKPS VFHGPSQAHSALYLSSHYHQQPGMNPHLTAMHPSLPRNIAPKPNNQMPVT VSIANMAVSPPPPLQISPPLHQHLNMQQHQPLTMQQPLGNQLPMQVQSALH SPTMQQGFTLQPDYQTIINPTSTAAQVVTQAMEYVRSGCRNPPPQPVDWNN DYCSSGGMQRDKALYLT
136	FOXA1 (HNF3A C-terminal domain)	MLGTVKMEGHETSDWNSYYADTQEAYSSVPVSNMNSGLGSMNSMNTYM TMNTMTTSGNMTPASFNMSYANPGLGAGLSPGAVAGMPGGSAGAMNSM TAAGVTAMGTALSPSGMGAMGAQQAASMNGLGPYAAAMNPCMSPMAY APSNLGRSRAGGGGDAKTFKRSYPHAKPPYSYISLITMAIQQAPSKMLTLSE IYQWIMDLFPYYRQNQQRWQNSIRHSLSFNDCFVKVARSPDKPGKGSYWT LHPDSGNMFENGCYLRRQKRFKCEKQPGAGGGGGSGSGGSGAKGGPESRK DPSGASNPSADSPLHRGVHGKTGQLEGAPAPGPAASPQTLDHSGATATGGA SELKTPASSTAPPISSGPGALASVPASHPAHGLAPHESQLHLKGDPHYSFNHP FSINNLMSSSEQQHKLDFKAYEQALQYSPYGSTLPASLPLGSASVTTRSPIEP SALEPAYYQGVYSRPVLNTS
137	FOXA2 (HNF3B C-terminal domain)	MLGAVKMEGHEPSDWSSYYAEPEGYSSVSNMNAGLGMNGMNTYMSMSA AAMGSGSGNMSAGSMNMSSYVGAGMSPSLAGMSPGAGAMAGMGGSAG AAGVAGMGPHLSPSLSPLGGQAAGAMGGLAPYANMNSMSPMYGQAGLSR ARDPKTYRRSYTHAKPPYSYISLITMAIQQSPNKMLTLSEIYQWIMDLFPFY RQNQQRWQNSIRHSLSFNDCFLKVPRSPDKPGKGSFWTLHPDSGNMFENG CYLRRQKRFKCEKQLALKEAAGAAGSGKKAAAGAQASQAQLGEAAGPAS

SEQ ID NO	Description	Sequence
		ETPAGTESPHSSASPCQEHKRGGLGELKGTPAAALSPPEPAPSPGQQQQAAA HLLGPPHHPGLPPEAHLKPEHHYAFNHPFSINNLMSSEQQHHHSHHHHQPH KMDLKAYEQVMHYPGYGSPMPGSLAMGPVTNKTGLDASPLAADTSYYQG VYSRPIMNSS
138	IRF2BP1 (IRF-2BP1_2 N-terminal domain)	MASVQASRRQWCYLCDLPKMPWAMVWDFSEAVCRGCVNFEGADRIELLI DAARQLKRSHVLPEGRSPGPPALKHPATKDLAAAAAQGPQLPPPQAQPQPS GTGGGVSGQDRYDRATSSGRLPLPSPALEYTLGSRLANGLGREEAVAEGAR RALLGSMPGLMPPGLLAAAVSGLGSRGLTLAPGLSPARPLFGSDFEKEKQQ RNADCLAELNEAMRGRAEEWHGRPKAVREQLLALSACAPFNVRFKKDHG LVGRVFAFDATARPPGYEFELKLFTEYPCGSGNVYAGVLAVARQMFHDAL REPGKALASSGFKYLEYERRHGSGEWRQLGELLTDGVRSFREPAPAEALPQ QYPEPAPAALCGPPPRAPSRNLAPTPRRRKASPEPEGEAAGKMTTEEQQQR HWVAPGGPYSAETPGVPSPIAALKNVAEALGHSPKDPGGGGGPVRAGGAS PAASSTAQPPTQHRLVARNGEAEVSPTAGAEAVSGGGSGTGATPGAPLCCT LCRERLEDTHFVQCPSVPGHKFCFPCSREFIKAQGPAGEVYCPSGDKCPLVG SSVPWAFMQGEIATILAGDIKVKKERDP
139	IRF2BP2 (IRF-2BP1_2 N-terminal domain)	MAAAVAVAAASRRQSCYLCDLPRMPWAMIWDFTEPVCRGCVNYEGADR VEFVIETARQLKRAHGCFPEGRSPPGAAASAAAKPPPLSAKDILLQQQQQLG HGGPEAAPRAPQALERYPLAAAAERPPRLGSDFGSSRPAASLAQPPTPQPPP VNGILVPNGFSKLEEPPELNRQSPNPRRGHAVPPTLVPLMNGSATPLPTALG LGGRAAASLAAVSGTAAASLGSAQPTDLGAHKRPASVSSSAAVEHEQREA AAKEKQPPPPAHRGPADSLSTAAGAAELSAEGAGKSRGSGEQDWVNRPKT VRDTLLALHQHGHSGPFESKFKKEPALTAGRLLGFEANGANGSKAVARTA RKRKPSPEPEGEVGPPKINGEAQPWLSTSTEGLKIPMTPTSSFVSPPPPTASPH SNRTTPPEAAQNGQSPMAALILVADNAGGSHASKDANQVHSTTRRNSNSPP SPSSMNQRRLGPREVGGQGAGNTGGLEPVHPASLPDSSLATSAPLCCTLCH ERLEDTHFVQCPSVPSHKFCFPCSRQSIKQQGASGEVYCPSGEKCPLVGSNV PWAFMQGEIATILAGDVKVKKERDS
140	IRF2BPL IRF-2BP1_2 N-terminal domain	MSAAQVSSSRRQSCYLCDLPRMPWAMIWDFSEPVCRGCVNYEGADRIEFVI ETARQLKRAHGCFQDGRSPGPPPPVGVKTVALSAKEAAAAAAAAAAAAAA AAQQQQQQQQQQQQQQQQQQQQQQQQQQQLNHVDGSSKPAVLAAPSGLER YGLSAAAAAAAAAAAAAVEQRSRFEYPPPPVSLGSSSHTARLPNGLGGPNGF PKPTPEEGPPELNRQSPNSSSAAASVASRRGTHGGLVTGLPNPGGGGGPQLT VPPNLLPQTLLNGPASAAVLPPPPPHALGSRGPPTPAPPGAPGGPACLGGTP GVSATSSSASSSTSSSVAEVGVGAGGKRPGSVSSTDQERELKEKQRNAEAL AELSESLRNRAEEWASKPKMVRDTLLTLAGCTPYEVRFKKDHSLLGRVFAF DAVSKPGMDYELKLFIEYPTGSGNVYSSASGVAKQMYQDCMKDFGRGLSS GFKYLEYEKKHGSGDWRLLGDLLPEAVRFFKEGVPGADMLPQPYLDASCP MLPTALVSLSRAPSAPPGTGALPPAAPSGRGAAASLRKRKASPEPPDSAEGA LKLGEEQQRQQWMANQSEALKLTMSAGGFAAPGHAAGGPPPPPPLGPHS NRTTPPESAPQNGPSPMAALMSVADTLGTAHSPKDGSSVHSTTASARRNSS SPVSPASVPGQRRLASRNGDLNLQVAPPPPSAHPGMDQVHPQNIPDSPMAN SGPLCCTICHERLEDTHFVQCPSVPSHKFCFPCSRESIKAQGATGEVYCPSGE KCPLVGSNVPWAFMQGEIATILAGDVKVKKERDP
141	HOXA13 (homeodomain)	MTASVLLHPRWIEPTVMFLYDNGGGLVADELNKNMEGAAAAAAAAAAA AAAGAGGGGFPHPAAAAAGGNFSVAAAAAAAAAAAANQCRNLMAHPAP LAPGAASAYSSAPGEAPPSAAAAAAAAAAAAAAAAAAASSSGGPGPAGPAG AEAAKQCSPCSAAAQSSSGPAALPYGYFGSGYYPCARMGPHPNAIKSCAQP ASAAAAAAFADKYMDTAGPAAEEFSSRAKEFAFYHQGYAAGPYHHHQPM PGYLDMPVVPGLGGPGESRHEPLGLPMESYQPWALPNGWNGQMYCPKEQ AQPPHLWKSTLPDVVSHPSDASSYRRGRKKRVPYTKVQLKELEREYATNK FITKDKRRRISATTNLSERQVTIWFQNRRVKEKKVINKLKTTS

SEQ ID NO	Description	Sequence
142	HOXB13 (homeodomain)	MEPGNYATLDGAKDIEGLLGAGGGRNLVAHSPLTSHPAAPTLMPAVNYAP LDLPGSAEPPKQCHPCPGVPQGTSPAPVPYGYFGGGYYSCRVSRSSLKPCA QAATLAAYPAETPTAGEEYPSRPTEFAFYPGYPGTYQPMASYLDVSVVQTL GAPGEPRHDSLLPVDSYQSWALAGGWNSQMCCQGEQNPPGPFWKAAFAD SSGQHPPDACAFRRGRKKRIPYSKGQLRELEREYAANKFITKDKRRKISAAT SLSERQITIWFQNRRVKEKKVLAKVKNSATP
143	HOXC13 (homeodomain)	MTTSLLLHPRWPESLMYVYEDSAAESGIGGGGGGGGGGTGGAGGGCSGAS PGKAPSMDGLGSSCPASHCRDLLPHPVLGRPPAPLGAPQGAVYTDIPAPEA ARQCAPPPAPPTSSSATLGYGYPFGGSYYGCRLSHNVNLQQKPCAYHPGDK YPEPSGALPGDDLSSRAKEFAFYPSFASSYQAMPGYLDVSVVPGISGHPEPR HDALIPVEGYQHWALSNGWDSQVYCSKEQSQSAHLWKSPFPDVVPLQPEV SSYRRGRKKRVPYTKVQLKELEKEYAASKFITKEKRRRISATTNLSERQVTI WFQNRRVKEKKVVSKSKAPHLHST
144	HOXA11 (homeodomain)	MDFDERGPCSSNMYLPSCTYYVSGPDFSSLPSFLPQTPSSRPMTYSYSSNLP QVQPVREVTFREYAIEPATKWHPRGNLAHCYSAEELVHRDCLQAPSAAGV PGDVLAKSSANVYHHPTPAVSSNFYSTVGRNGVLPQAFDQFFETAYGTPEN LASSDYPGDKSAEKGPPAATATSAAAAAAATGAPATSSSDSGGGGGCRET AAAAEEKERRRRPESSSSPESSSGHTEDKAGGSSGQRTRKKRCPYTKYQIRE LEREFFFSVYINKEKRLQLSRMLNLTDRQVKIWFQNRRMKEKKINRDRLQY YSANPLL
145	HOXC11 (homeodomain)	MFNSVNLGNFCSPSRKERGADFGERGSCASNLYLPSCTYYMPEFSTVSSFLP QAPSRQISYPYSAQVPPVREVSYGLEPSGKWHHRNSYSSCYAAADELMHRE CLPPSTVTEILMKNEGSYGGHHHPSAPHATPAGFYSSVNKNSVLPQAFDRFF DNAYCGGGDPPAEPPCSGKGEAKGEPEAPPASGLASRAEAGAEAEAEEENT NPSSSGSAHSVAKEPAKGAAPNAPRTRKKRCPYSKFQIRELEREFFFNVYIN KEKRLQLSRMLNLTDRQVKIWFQNRRMKEKKLSRDRLQYFSGNPLL
146	HOXC10 (homeodomain)	MTCPRNVTPNSYAEPLAAPGGGERYSRSAGMYMQSGSDFNCGVMRGCGL APSLSKRDEGSSPSLALNTYPSYLSQLDSWGDPKAAYRLEQPVGRPLSSCSY PPSVKEENVCCMYSAEKRAKSGPEAALYSHPLPESCLGEHEVPVPSYYRAS PSYSALDKTPHCSGANDFEAPFEQRASLNPRAEHLESPQLGGKVSFPETPKS DSQTPSPNEIKTEQSLAGPKGSPSESEKERAKAADSSPDTSDNEAKEEIKAEN TTGNWLTAKSGRKKRCPYTKHQTLELEKEFLFNMYLTRERRLEISKTINLTD RQVKIWFQNRRMKLKKMNRENRIRELTSNFNFT
147	HOXA10 (homeodomain)	MSARKGYLLPSPNYPTTMSCSESPAANSFLVDSLISSGRGEAGGGGGGAGG GGGGGYYAHGGVYLPPAADLPYGLQSCGLFPTLGGKRNEAASPGSGGGGG GLGPGAHGYGPSPIDLWLDAPRSCRMEPPDGPPPPQQQPPPPPQPPQPAPQ ATSCSFAQNIKEESSYCLYDSADKCPKVSATAAELAPFPRGPPPDGCALGTS SGVPVPGYFRLSQAYGTAKGYGSGGGGAQQLGAGPFPAQPPGRGFDLPPA LASGSADAARKERALDSPPPPTLACGSGGGSQGDEEAHASSSAAEELSPAPS ESSKASPEKDSLGNSKGENAANWLTAKSGRKKRCPYTKHQTLELEKEFLFN MYLTRERRLEISRSVHLTDRQVKIWFQNRRMKLKKMNRENRIRELTANFNF S
148	HOXB9 (homeodomain)	MSISGTLSSYYVDSIISHESEDAPPAKFPSGQYASSRQPGHAEHLEFPSCSFQP KAPVFGASWAPLSPHASGSLPSVYHPYIQPQGVPPAESRYLRTWLEPAPRGE AAPGQGQAAVKAEPLLGAPGELLKQGTPEYSLETSAGREAVLSNQRPGYG DNKICEGSEDKERPDQTNPSANWLHARSSRKKRCPYTKYQTLELEKEFLFN MYLTRDRRHEVARLLNLSERQVKIWFQNRRMKMKKMNKEQGKE
149	HOXA9 (homeodomain)	MATTGALGNYYVDSFLLGADAADELSVGRYAPGTLGQPPRQAATLAEHPD FSPCSFQSKATVFGASWNPVHAAGANAVPAAVYHHHHHHPYVHPQAPVA AAAPDGRYMRSWLEPTPGALSFAGLPSSRPYGIKPEPLSARRGDCPTLDTHT LSLTDYACGSPPVDREKQPSEGAFSENNAENESGGDKPPIDPNNPAANWLH

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		ARSTRKKRCPYTKHQTLELEKEFLFNMYLTRDRRYEVARLLNLTERQVKIW FQNRRMKMKKINKDRAKDE
150	ZFP28_HUMAN	NKKLEAVGTGIEPKAMSQGLVTFGDVAVDFSQEEWEWLNPIQRNLYRKVM LENYRNLASLGLCVSKPDVISSLEQGKEPW
151	ZN334_HUMAN	KMKKFQIPVSFQDLTVNFTQEEWQQLDPAQRLLYRDVMLENYSNLVSVGY HVSKPDVIFKLEQGEEPWIVEEFSNQNYPD
152	ZN568_HUMAN	CSQESALSEEEEDTTRPLETVTFKDVAVDLTQEEWEQMKPAQRNLYRDVM LENYSNLVTVGCQVTKPDVIFKLEQEEEPW
153	ZN37A_HUMAN	ITSQGSVSFRDVTVGFTQEEWQHLDPAQRTLYRDVMLENYSHLVSVGYCIP KPEVILKLEKGEEPWILEEKFPSQSHLEL
154	ZN181_HUMAN	PQVTFNDVAIDFTHEEWGWLSSAQRDLYKDVMVQNYENLVSVAGLSVTK PYVITLLEDGKEPWMMEKKLSKGMIPDWESR
155	ZN510_HUMAN	PLRFSTLFQEQQKMNISQASVSFKDVTIEFTQEEWQQMAPVQKNLYRDVML ENYSNLVSVGYCCFKPEVIFKLEQGEEPW
156	ZN862_HUMAN	QDPSAEGLSEEVPVVFEELPVVFEDVAVYFTREEWGMLDKRQKELYRDVM RMNYELLASLGPAAAKPDLISKLERRAAPW
157	ZN140_HUMAN	SQGSVTFRDVAIDFSQEEWKWLQPAQRDLYRCVMLENYGHLVSLGLSISKP DVVSLLEQGKEPWLGKREVKRDLFSVSES
158	ZN208_HUMAN	GSLTFRDVAIEFSLEEWQCLDTAQQNLYRNVMLENYRNLVFLGIAAFKPDL IIFLEEGKESWNMKRHEMVEESPVICSHF
159	ZN248_HUMAN	NKSQEQVSFKDVCVDFTQEEWYLLDPAQKILYRDVILENYSNLVSVGYCIT KPEVIFKIEQGEEPWILEKGFPSQCHPER
160	ZN571_HUMAN	PHLLVTFRDVAIDFSQEEWECLDPAQRDLYRDVMLENYSNLISLDLESSCVT KKLSPEKEIYEMESLQWENMGKRINHHL
161	ZN699_HUMAN	EEERKTAELQKNRIQDSVVFEDVAVDFTQEEWALLDLAQRNLYRDVMLEN FQNLASLGYPLHTPHLISQWEQEEDLQTVK
162	ZN726_HUMAN	GLLTFRDVAIEFSLEEWQCLDTAQKNLYRNVMLENYRNLAFLGIAVSKPDL IICLEKEKEPWNMKRDEMVDEPPGICPHF
163	ZIK1_HUMAN	RAPTQVTVSPETHMDLTKGCVTFEDIAIYFSQDEWGLLDEAQRLLYLEVML ENFALVASLGCGHGTEDEETPSDQNVSVG
164	ZNF2_HUMAN	AAVSPTTRCQESVTFEDVAVVFTDEEWSRLVPIQRDLYKEVMLENYNSIVS LGLPVPQPDVIFQLKRGDKPWMVDLHGSE
165	Z705F_HUMAN	HSLEKVTFEDVAIDFTQEEWDMMDTSKRKLYRDVMLENISHLVSLGYQISK SYIILQLEQGKELWREGRVFLQDQNPDRE
166	ZNF14_HUMAN	DSVSFEDVAVNFTLEEWALLDSSQKKLYEDVMQETFKNLVCLGKKWEDQ DIEDDHRNQGKNRRCHMVERLCESRRGSKCG
167	ZN471_HUMAN	NVEVVKVMPQDLVTFKDVAIDFSQEEWQWMNPAQKRLYRSMMLENYQS LVSLGLCISKPYVISLLEQGREPWEMTSEMTR
168	ZN624_HUMAN	TQPDEDLHLQAEETQLVKESVTFKDVAIDFTLEEWRLMDPTQRNLHKDVM LENYRNLVSLGLAVSKPDMISHLENGKGPW
169	ZNF84_HUMAN	TMLQESFSFDDLSVDFTQKEWQLLDPSQKNLYKDVMLENYSSLVSLGYEV MKPDVIFKLEQGEEPWVGDGEIPSSDSPEV
170	ZNF7_HUMAN	EVVTFGDVAVHFSREEWQCLDPGQRALYREVMLENHSSVAGLAGFLVFKP ELISRLEQGEEPWVLDLQGAEGTEAPRTSK
171	ZN891_HUMAN	RNAEEERMIAVFLTTWLQEPMTFKDVAVEFTQEEWMMLDSAQRSLYRDV MLENYRNLTSVEYQLYRLTVISPLDQEEIRN
172	ZN337_HUMAN	GPQGARRQAFLAFGDVTVDFTQKEWRLLSPAQRALYREVTLENYSHLVSL GILHSKPELIRRLEQGEVPWGEERRRRPGP
173	Z705G_HUMAN	HSLKKLTFEDVAIDFTQEEWAMMDTSKRKLYRDVMLENISHLVSLGYQISK SYIILQLEQGKELWREGRVFLQDQNPNRE

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174	ZN529_HUMAN	MPEVEFPDQFFTVLTMDHELVTLRDVVINFSQEEWEYLDSAQRNLYWDVMMENYSNLLSLDLESRNETKHLSVGKDIIQN
175	ZN729_HUMAN	PGAPGSLEMGPLTFRDVTIEFSLEEWQCLDTVQQNLYRDVMLENYRNLVFLGMAVFKPDLITCLKQGKEPWNMKRHEMVT
176	ZN419_HUMAN	RDPAQVPVAADLLTDHEEGYVTFEDVAVYFSQEEWRLLDDAQRLLYRNVMLENFTLLASLGLASSKTHEITQLESWEEPF
177	Z705A_HUMAN	HSLKKVTFEDVAIDFTQEEWAMMDTSKRKLYRDVMLENISHLVSLGYQISKSYIILQLEQGKELWREGREFLQDQNPDRE
178	ZNF45_HUMAN	TKSKEAVTFKDVAVVFSEEELQLLDLAQRKLYRDVMLENFRNVVSVGHQSTPDGLPQLEREEKLWMMKMATQRDNSSGAK
179	ZN302_HUMAN	SQVTFSDVAIDFSHEEWACLDSAQRDLYKDVMVQNYENLVSVGLSVTKPYVIMLLEDGKEPWMMEKKLSKAYPFPLSHSV
180	ZN486_HUMAN	PGPLRSLEMESLQFRDVAVEFSLEEWHCLDTAQQNLYRDVMLENYRHLVFLGIIVSKPDLITCLEQGIKPLTMKRHEMIA
181	ZN621_HUMAN	LQTTWPQESVTFEDVAVYFTQNQWASLDPAQRALYGEVMLENYANVASLVAFPFPKPALISHLERGEAPWGPDPWDTEIL
182	ZN688_HUMAN	APLLAPRPGETRPGCRKPGTVSFADVAVYFSPEEWGCLRPAQRALYRDVMQETYGHLGALGFPGPKPALISWMEQESEAW
183	ZN33A_HUMAN	NKVEQKSQESVSFKDVTVGFTQEEWQHLDPSQRALYRDVMLENYSNLVSVGYCVHKPEVIFRLQQGEEPWKQEEEFPSQS
184	ZN554_HUMAN	CFSQEERMAAGYLPRWSQELVTFEDVSMDFSQEEWELLEPAQKNLYREVMLENYRNVVSLEALKNQCTDVGIKEGPLSPA
185	ZN878_HUMAN	DSVAFEDVAVNFTQEEWALLDPSQKNLYREVMQETLRNLTSIGKKWNNQYIEDEHQNPRRNLRRLIGERLSESKESHQHG
186	ZN772_HUMAN	MGPAQVPMNSEVIVDPIQGQVNFEDVFVYFSQEEWVLLDEAQRLLYRDVMLENFALMASLGHTSFMSHIVASLVMGSEPW
187	ZN224_HUMAN	TTFKEAMTFKDVAVVFTEEELGLLDLAQRKLYRDVMLENFRNLLSVGHQAFHRDTFHFLREEKIWMMKTAIQREGNSGDK
188	ZN184_HUMAN	DSTLLQGGHNLLSSASFQEAVTFKDVIVDFTQEEWKQLDPGQRDLFRDVTLENYTHLVSIGLQVSKPDVISQLEQGTEPW
189	ZN544_HUMAN	EARSMLVPPQASVCFEDVAMAFTQEEWEQLDLAQRTLYREVTLETWEHIVSLGLFLSKSDVISQLEQEEDLCRAEQEAPR
190	ZNF57_HUMAN	DSVVFEDVAVDFTLEEWALLDSAQRDLYRDVMLETFRNLASVDDGTQFKANGSVSLQDMYGQEKSKEQTIPNFTGNNSCA
191	ZN283_HUMAN	EESHGALISSCNSRTMTDGLVTFRDVAIDFSQEEWECLDPAQRDLYVDVMLENYSNLVSLDLESKTYETKKIFSENDIFE
192	ZN549_HUMAN	VITPQIPMVTEEFVKPSQGHVTFEDIAVYFSQEEWGLLDEAQRCLYHDVMLENFSLMASVGCLHGIEAEEAPSEQTLSAQ
193	ZN211_HUMAN	VQLRPQTRMATALRDPASGSVTFEDVAVYFSWEEWDLLDEAQKHLYFDVMLENFALTSSLGCWCGVEHEETPSEQRISGE
194	ZN615_HUMAN	MQAQESLTLEDVAVDFTWEEWQFLSPAQKDLYRDVMLENYSNLVAVGYQASKPDALSKLERGEETCTTEDEIYSRICSEI
195	ZN253_HUMAN	GPLQFRDVAIEFSLEEWHCLDTAQRNLYRDVMLENYRNLVFLGIVVSKPDLVTCLEQGKKPLTMERHEMIAKPPVMSSHF
196	ZN226_HUMAN	NMFKEAVTFKDVAVAFTEEELGLLGPAQRKLYRDVMVENFRNLLSVGHPPFKQDVSPIERNEQLWIMTTATRRQGNLGEK
197	ZN730_HUMAN	GALTFRDVAIEFSLEEWQCLDTEQQNLYRNVMLDNYRNLVFLGIAVSKPDLITCLEQEKEPWNLKTHDMVAKPPVICSHI
198	Z585A_HUMAN	SPQKSSALAPEDHGSSYEGSVSFRDVAIDFSREEWRHLDPSQRNLYRDVMLETYSHLLSVGYQVPEAEVVMLEQGKEPWA

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199	ZN732_HUMAN	ELLTFRDVAIEFSPEEWKCLDPAQQNLYRDVMLENYRNLISLGVAISNPDLVIYLEQRKEPYKVKIHETVAKHPAVCSHF
200	ZN681_HUMAN	EPLKFRDVAIEFSLEEWQCLDTIQQNLYRNVMLENYRNLVFLGIVVSKPDLITCLEQEKEPWTRKRHRMVAEPPVICSHF
201	ZN667_HUMAN	PSARGKSKSKAPITFGDLAIYFSQEEWEWLSPIQKDLYEDVMLENYRNLVSLGLSFRRPNVITLLEKGKAPWMVEPVRRR
202	ZN649_HUMAN	TKAQESLTLEDVAVDFTWEEWQFLSPAQKDLYRDVMLENYSNLVSVGYQAGKPDALTKLEQGEPLWTLEDEIHSPAHPEI
203	ZN470_HUMAN	SQEEVEVAGIKLCKAMSLGSVTFTDVAIDFSQDEWEWLNLAQRSLYKKVMLENYRNLVSVGLCISKPDVISLLEQEKDPW
204	ZN484_HUMAN	TKSLESVSFKDVTVDFSRDEWQQLDLAQKSLYREVMLENYFNLISVGCQVPKPEVIFSLEQEEPCMLDGEIPSQSRPDGD
205	ZN431_HUMAN	SGCPGAERNLLVYSYFEKETLTFRDVAIEFSLEEWECLNPAQQNLYMNVMLENYKNLVFLGVAVSKQDPVTCLEQEKEPW
206	ZN382_HUMAN	PLQGSVSFKDVTVDFTQEEWQQLDPAQKALYRDVMLENYCHFVSVGFHMAKPDMIRKLEQGEELWTQRIFPSYSYLEEDG
207	ZN254_HUMAN	PGPPRSLEMGLLTFRDVAIEFSLEEWQHLDIAQQNLYRNVMLENYRNLAFLGIAVSKPDLITCLEQGKEPWNMKRHEMVD
208	ZN124_HUMAN	SGHPGSWEMNSVAFEDVAVNFTQEEWALLDPSQKNLYRDVMQETFRNLASIGNKGEDQSIEDQYKNSSRNLRHIISHSGN
209	ZN607_HUMAN	SYGSITFGDVAIDFSHQEWEYLSLVQKTLYQEVMMENYDNLVSLAGHSVSKPDLITLLEQGKEPWMIVREETRGECTDLD
210	ZN317_HUMAN	DLFVCSGLEPHTPSVGSQESVTFQDVAVDFTEKEWPLLDSSQRKLYKDVMLENYSNLTSLGYQVGKPSLISHLEQEEEPR
211	ZN620_HUMAN	FQTAWRQEPVTFEDVAVYFTQNEWASLDSVQRALYREVMLENYANVASLAFPFTTPVLVSQLEQGELPWGLDPWEPMGRE
212	ZN141_HUMAN	ELLTFRDVAIEFSPEEWKCLDPDQQNLYRDVMLENYRNLVSLGVAISNPDLVTCLEQRKEPYNVKIHKIVARPPAMCSHF
213	ZN584_HUMAN	AGEAEAQLDPSLQGLVMFEDVTVYFSREEWGLLNVTQKGLYRDVMLENFALVSSLGLAPSRSPVFTQLEDDEQSWVPSWV
214	ZN540_HUMAN	AHALVTFRDVAIDFSQKEWECLDTTQRKLYRDVMLENYNNLVSLGYSGSKPDVITLLEQGKEPCVVARDVTGRQCPGLLS
215	ZN75D_HUMAN	KRIKHWKMASKLILPESLSLLTFEDVAVYFSEEEWQLLNPLEKTLYNDVMQDIYETVISLGLKLKNDTGNDHPISVSTSE
216	ZN555_HUMAN	DSVVFEDVAVDFTLEEWALLDSAQRDLYRDVMLETFQNLASVDDETQFKASGSVSQQDIYGEKIPKESKIATFTRNVSWA
217	ZN658_HUMAN	NMSQASVSFQDVTVEFTREEWQHLGPVERTLYRDVMLENYSHLISVGYCITKPKVISKLEKGEEPWSLEDEFLNQRYPGY
218	ZN684_HUMAN	ISFQESVTFQDVAVDFTAEEWQLLDCAERTLYWDVMLENYRNLISVGCPITKTKVILKVEQGQEPWMVEGANPHESSPES
219	RBAK_HUMAN	NTLQGPVSFKDVAVDFTQEEWQQLDPDEKITYRDVMLENYSHLVSVGYDTTKPNVIIKLEQGEEPWIMGGEFPCQHSPEA
220	ZN829_HUMAN	HPEEEERMHDELLQAVSKGPVMFRDVSIDFSQEEWECLDADQMNLYKEVMLENFSNLVSVGLSNSKPAVISLLEQGKEPW
221	ZN582_HUMAN	SLGSELFRDVAIVFSQEEWQWLAPAQRDLYRDVMLETYSNLVSLGLAVSKPDVISFLEQGKEPWMVERVVSGGLCPVLES
222	ZN112_HUMAN	TKFQEMVTFKDVAVVFTEEELGLLDSVQRKLYRDVMLENFRNLLLVAHQPFKPDLISQLEREEKLLMVETETPRDGCSGR
223	ZN716_HUMAN	AKRPGPPGSREMGLLTFRDIAIEFSLAEWQCLDHAQQNLYRDVMLENYRNLVSLGIAVSKPDLITCLEQNKEPQNIKRNE

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224	HKR1_HUMAN	TCMVHRQTMSCSGAGGITAFVAFRDVAVYFTQEEWRLLSPAQRTLHREVMLETYNHLVSLEIPSSKPKLIAQLERGEAPW
225	ZN350_HUMAN	IQAQESITLEDVAVDFTWEEWQLLGAAQKDLYRDVMLENYSNLVAVGYQASKPDALFKLEQGEQLWTIEDGIHSGACSDI
226	ZN480_HUMAN	AQKRRKRKAKESGMALPQGHLTFRDVAIEFSQAEWKCLDPAQRALYKDVMLENYRNLVSLGISLPDLNINSMLEQRREPW
227	ZN416_HUMAN	DSTSVPVTAEAKLMGFTQGCVTFEDVAIYFSQEEWGLLDEAQRLLYRDVMLENFALITALVCWHGMEDEETPEQSVSVEG
228	ZNF92_HUMAN	GPLTFRDVKIEFSLEEWQCLDTAQRNLYRDVMLENYRNLVFLGIAVSKPDLITWLEQGKEPWNLKRHEMVDKTPVMCSHF
229	ZN100_HUMAN	SGCPGAERSLLVQSYFEKGPLTFRDVAIEFSLEEWQCLDSAQQGLYRKVMLENYRNLVFLAGIALTKPDLITCLEQGKEP
230	ZN736_HUMAN	GVLTFRDVAVEFSPEEWECLDSAQQRLYRDVMLENYGNLVSLGLAIFKPDLMTCLEQRKEPWKVKRQEAVAKHPAGSFHF
231	ZNF74_HUMAN	KENLEDISGWGLPEARSKESVSFKDVAVDFTQEEWGQLDSPQRALYRDVMLENYQNLLALGPPLHKPDVISHLERGEEPW
232	CBX1_HUMAN	EESEKPRGFARGLEPERIIGATDSSGELMFLMKWKNSDEADLVPAKEANVKCPQVVISFYEERLTWHSYPSEDDDKKDDK
233	ZN443_HUMAN	ASVALEDVAVNFTREEWALLGPCQKNLYKDVMQETIRNLDCVVMKWKDQNIEDQYRYPRKNLRCRMLERFVESKDGTQCG
234	ZN195_HUMAN	TLLTFRDVAIEFSLEEWKCLDLAQQNLYRDVMLENYRNLFSVGLTVCKPGLITCLEQRKEPWNVKRQEAADGHPEMGFHH
235	ZN530_HUMAN	AAALRAPTQQVFVAFEDVAIYFSQEEWELLDEMQRLLYRDVMLENFAVMASLGCWCGAVDEGTPSAESVSVEELSQGRTP
236	ZN782_HUMAN	NTFQASVSFQDVTVEFSQEEWQHMGPVERTLYRDVMLENYSHLVSVGYCFTKPELIFTLEQGEDPWLLEKEKGFLSRNSP
237	ZN791_HUMAN	DSVAFEDVSVSFSQEEWALLAPSQKKLYRDVMQETFKNLASIGEKWEDPNVEDQHKNQGRNLRSHTGERLCEGKEGSQCA
238	ZN331_HUMAN	AQGLVTFADVAIDFSQEEWACLNSAQRDLYWDVMLENYSNLVSLDLESAYENKSLPTEKNIHEIRASKRNSDRRSKSLGR
239	Z354C_HUMAN	AVDLLSAQEPVTFRDVAVFFSQDEWLHLDSAQRALYREVMLENYSSLVSLGIPFSMPKLIHQLQQGEDPCMVEREVPSDT
240	ZN157_HUMAN	SPQRFPALIPGEPGRSFEGSVSFEDVAVDFTRQEWHRLDPAQRTMHKDVMLETYSNLASVGLCVAKPEMIFKLERGEELW
241	ZN727_HUMAN	RVLTFRDVAVEFSPEEWECLDSAQQRLYRDVMLENYGNLFSLGLAIFKPDLITYLEQRKEPWNARRQKTVAKHPAGSLHF
242	ZN550_HUMAN	AETKDAAQMLVTFKDVAVTFTREEWRQLDLAQRTLYREVMLETCGLLVSLGHRVPKPELVHLLEHGQELWIVKRGLSHAT
243	ZN793_HUMAN	IEYQIPVSFKDVVVGFTQEEWHRLSPAQRALYRDVMLETYSNLVSVGYEGTKPDVILRLEQEEAPWIGEAACPGCHCWED
244	ZN235_HUMAN	TKFQEAVTFKDVAVAFTEEELGLLDSAQRKLYRDVMLENFRNLVSVGHQSFKPDMISQLEREEKLWMKELQTQRGKHSGD
245	ZNF8_HUMAN	DEGVAGVMSVGPPAARLQEPVTFRDVAVDFTQEEWGQLDPTQRILYRDVMLETFGHLLSIGPELPKPEVISQLEQGTELW
246	ZN724_HUMAN	GPLTFMDVAIEFSVEEWQCLDTAQQNLYRNVMLENYRNLVFLGIAVSKPDLITCLEQGKEPWNMERHEMVAKPPGMCCYF
247	ZN573_HUMAN	HQVGLIRSYNSKTMTCFQELVTFRDVAIDFSRQEWEYLDPNQRDLYRDVMLENYRNLVSLGGHSISKPVVVDLLERGKEP
248	ZN577_HUMAN	NATIVMSVRREQGSSSGEGSLSFEDVAVGFTREEWQFLDQSQKVLYKEVMLENYINLVSIGYRGTKPDSLFKLEQGEPPG

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249	ZN789_HUMAN	FPPARGKELLSFEDVAMYFTREEWGHLNWGQKDLYRDVMLENYRNMVLLGFQFPKPEMICQLENWDEQWILDLPRTGNRK
250	ZN718_HUMAN	ELLTFKDVAIEFSPEEWKCLDTSQQNLYRDVMLENYRNLVSLGVSISNPDLVTSLEQRKEPYNLKIHETAARPPAVCSHF
251	ZN300_HUMAN	MKSQGLVSFKDVAVDFTQEEWQQLDPSQRTLYRDVMLENYSHLVSMGYPVSKPDVISKLEQGEEPWIIKGDISNWIYPDE
252	ZN383_HUMAN	AEGSVMFSDVSIDFSQEEWDCLDPVQRDLYRDVMLENYGNLVSMGLYTPKPQVISLLEQGKEPWMVGRELTRGLCSDLES
253	ZN429_HUMAN	GPLTFTDVAIEFSLEEWQCLDTAQQNLYRNVMLENYRNLVFLGIAVSKPDLITCLEKEKEPCKMKRHEMVDEPPVVCSHF
254	ZN677_HUMAN	ALSQGLFTFKDVAIEFSQEEWECLDPAQRALYRDVMLENYRNLLSLDEDNIPPEDDISVGFTSKGLSPKENNKEELYHLV
255	ZN850_HUMAN	NMEGLVMFQDLSIDFSQEEWECLDAAQKDLYRDVMMENYSSLVSLGLSIPKPDVISLLEQGKEPWMVSRDVLGGWCRDSE
256	ZN454_HUMAN	AVSHLPTMVQESVTFKDVAILFTQEEWGQLSPAQRALYRDVMLENYSNLVSLGLLGPKPDTFSQLEKREVWMPEDTPGGF
257	ZN257_HUMAN	GPLTIRDVTVEFSLEEWHCLDTAQQNLYRDVMLENYRNLVFLGIAVSKPDLITCLEQGKEPCNMKRHEMVAKPPVMCSHI
258	ZN264_HUMAN	AAAVLTDRAQVSVTFDDVAVTFTKEEWGQLDLAQRTLYQEVMLENCGLLVSLGCPVPKAELICHLEHGQEPWTRKEDLSQ
259	ZFP82_HUMAN	ALRSVMFSDVSIDFSPEEWEYLDLEQKDLYRDVMLENYSNLVSLGCFISKPDVISSLEQGKEPWKVVRKGRRQYPDLETK
260	ZFP14_HUMAN	AHGSVTFRDVAIDFSQEEWEFLDPAQRDLYRDVMWENYSNFISLGPSISKPDVITLLDEERKEPGMVVREGTRRYCPDLE
261	ZN485_HUMAN	APRAQIQGPLTFGDVAVAFTRIEWRHLDAAQRALYRDVMLENYGNLVSVGLLSSKPKLITQLEQGAEPWTEVREAPSGTH
262	ZN737_HUMAN	GPLQFRDVAIEFSLEEWHCLDTAQRNLYRNVMLENYRNLVFLGIVVSKPDLITCLEQGKKPLTMKKHEMVANPSVTCSHF
263	ZNF44_HUMAN	TLPRGQPEVLEWGLPKDQDSVAFEDVAVNFTHEEWALLGPSQKNLYRDVMRETIRNLNCIGMKWENQNIDDQHQNLRRNP
264	ZN596_HUMAN	PSPDSMTFEDIIVDFTQEEWALLDTSQRKLFQDVMLENISHLVSIGKQLCKSVVLSQLEQVEKLSTQRISLLQGREVGIK
265	ZN565_HUMAN	EESREIRAGQIVLKAMAQGLVTFRDVAIEFSLEEWKCLEPAQRDLYREVTLENFGHLASLGLSISKPDVVSLLEQGKEPW
266	ZN543_HUMAN	AASAQVSVTFEDVAVTFTQEEWGQLDAAQRTLYQEVMLETCGLLMSLGCPLFKPELIYQLDHRQELWMATKDLSQSSYPG
267	ZFP69_HUMAN	RESLEDEVTPGLPTAESQELLTFKDISIDFTQEEWGQLAPAHQNLYREVMLENYSNLVSVGYQLSKPSVISQLEKGEEPW
268	SUMO1_HUMAN	EGEYIKLKVIGQDSSEIHFKVKMTTHLKKLKESYCQRQGVPMNSLRFLFEGQRIADNHTPKELGMEEEDVIEVYQEQTGG
269	ZNF12_HUMAN	NKSLGPVSFKDVAVDFTQEEWQQLDPEQKITYRDVMLENYSNLVSVGYHIIKPDVISKLEQGEEPWIVEGEFLLQSYPDE
270	ZN169_HUMAN	SPGLLTTRKEALMAFRDVAVAFTQKEWKLLSSAQRTLYREVMLENYSHLVSLGIAFSKPKLIEQLEQGDEPWREENEHLL
271	ZN433_HUMAN	MFQDSVAFEDVAVTFTQEEWALLDPSQKNLCRDVMQETFRNLASIGKKWKPQNIYVEYENLRRNLRIVGERLFESKEGHQ
272	SUMO3_HUMAN	ENDHINLKVAGQDGSVVQFKIKRHTPLSKLMKAYCERQGLSMRQIRFRFDGQPINETDTPAQLEMEDEDTIDVFQQQTGG
273	ZNF98_HUMAN	PGPLGSLEMGVLTFRDVALEFSLEEWQCLDTAQQNLYRNVMLENYRNLVFVGIAASKPDLITCLEQGKEPWNVKRHEMVT

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274	ZN175_HUMAN	LSQKPQVLGPEKQDGSCEASVSFEDVTVDFSREEWQQLDPAQRCLYRDVMLELYSHLFAVGYHIPNPEVIFRMLKEKEPR
275	ZN347_HUMAN	ALTQGQVTFRDVAIEFSQEEWTCLDPAQRTLYRDVMLENYRNLASLGISCFDLSIISMLEQGKEPFTLESQVQIAGNPDG
276	ZNF25_HUMAN	NKFQGPVTLKDVIVEFTKEEWKLLTPAQRTLYKDVMLENYSHLVSVGYHVNKPNAVFKLKQGKEPWILEVEFPHRGFPED
277	ZN519_HUMAN	ELLTFRDVAIEFSPEEWKCLDPAQQNLYRDVMLENYRNLVSLAVYSYYNQGILPEQGIQDSFKKATLGRYGSCGLENICL
278	Z585B_HUMAN	SPQKSSALAPEDHGSSYEGSVSFRDVAIDFSREEWRHLDLSQRNLYRDVMLETYSHLLSVGYQVPKPEVVMLEQGKEPWA
279	ZIM3_HUMAN	NNSQGRVTFEDVTVNFTQGEWQRLNPEQRNLYRDVMLENYSNLVSVGQGETTKPDVILRLEQGKEPWLEEEEVLGSGRAE
280	ZN517_HUMAN	AMALPMPGPQEAVVFEDVAVYFTRIEWSCLAPDQQALYRDVMLENYGNLASLGFLVAKPALISLLEQGEEPGALILQVAE
281	ZN846_HUMAN	DSSQHLVTFEDVAVDFTQEEWTLLDQAQRDLYRDVMLENYKNLIILAGSELFKRSLMSGLEQMEELRTGVTGVLQELDLQ
282	ZN230_HUMAN	TTFKEAVTFKDVAVFFTEEELGLLDPAQRKLYQDVMLENFTNLLSVGHQPFHPFHFLREEKFWMMETATQREGNSGGKTI
283	ZNF66_HUMAN	GPLQFRDVAIEFSLEEWHCLDMAQRNLYRDVMLENYRNLVFLGIVVSKPDLITHLEQGKKPSTMQRHEMVANPSVLCSHF
284	ZFP1_HUMAN	NKSQGSVSFTDVTVDFTQEEWEQLDPSQRILYMDVMLENYSNLLSVEVWKADDQMERDHRNPDEQARQFLILKNQTPIEE
285	ZN713_HUMAN	EEEEMNDGSQMVRSQESLTFQDVAVDFTREEWDQLYPAQKNLYRDVMLENYRNLVALGYQLCKPEVIAQLELEEEWVIER
286	ZN816_HUMAN	EEATKKSKEKEPGMALPQGRLTFRDVAIEFSLEEWKCLNPAQRALYRAVMLENYRNLEFVDSSLKSMMEFSSTRHSITGE
287	ZN426_HUMAN	EKTPAGRIVADCLTDCYQDSVTFDDVAVDFTQEEWTLLDSTQRSLYSDVMLENYKNLATVGGQIIKPSLISWLEQEESRT
288	ZN674_HUMAN	AMSQESLTFKDVFVDFTLEEWQQLDSAQKNLYRDVMLENYSHLVSVGHLVGKPDVIFRLGPGDESWMADGGTPVRTCAGE
289	ZN627_HUMAN	DSVAFEDVAVNFTLEEWALLDPSQKNLYRDVMRETFRNLASVGKQWEDQNIEDPFKIPRRNISHIPERLCESKEGGQGEE
290	ZNF20_HUMAN	MFQDSVAFEDVAVSFTQEEWALLDPSQKNLYRDVMQETFKNLTSVGKTWKVQNIEDEYKNPRRNLSLMREKLCESKESHH
291	Z587B_HUMAN	AVVATLRLSAQGTVTFEDVAVKFTQEEWNLLSEAQRCLYRDVTLENLALMSSLGCWCGVEDEAAPSKQSIYIQRETQVRT
292	ZN316_HUMAN	EEEEEDEDEDDLLTAGCQELVTFEDVAVYFSLEEWERLEADQRGLYQEVMQENYGILVSLGYPIPKPDLIFRLEQGEEPW
293	ZN233_HUMAN	TKFQEMVTFKDVAVVFTREELGLLDLAQRKLYQDVMLENFRNLLSVGYQPFKLDVILQLGKEDKLRMMETEIQGDGCSGH
294	ZN611_HUMAN	EEAAQKRKGKEPGMALPQGRLTFRDVAIEFSLAEWKCLNPSQRALYREVMLENYRNLEAVDISSKCMMKEVLSTGQGNTE
295	ZN556_HUMAN	DTVVFEDVVVDFTLEEWALLNPAQRKLYRDVMLETFKHLASVDNEAQLKASGSISQQDTSGEKLSLKQKIEKFTRKNIWA
296	ZN234_HUMAN	TTFKEGLTFKDVAVVFTEEELGLLDPVQRNLYQDVMLENFRNLLSVGHHPFKHDVFLLEKEKKLDIMKTATQRKGKSADK
297	ZN560_HUMAN	SALQQEFWKIQTSNGIQMDLVTFDSVAVEFTQEEWTLLDPAQRNLYSDVMLENYKNLSSVGYQLFKPSLISWLEEEEELS
298	ZNF77_HUMAN	DCVIFEEVAVNFTPEEWALLDHAQRSLYRDVMLETCRNLASLDCYIYVRTSGSSSQRDVFGNGISNDEEIVKFTGSDSWS

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299	ZN682_HUMAN	ELLTFRDVTIEFSLEEWEFLNPAQQSLYRKVMLENYRNLVSLGLTVSKPELISRLEQRQEPWNVKRHETIAKPPAMSSHY
300	ZN614_HUMAN	IKTQESLTLEDVAVEFSWEEWQLLDTAQKNLYRDVMVENYNHLVSLGYQTSKPDVLSKLAHGQEPWTTDAKIQNKNCPGI
301	ZN785_HUMAN	PAHVPGEAGPRRTRESRPGAVSFADVAVYFSPEEWECLRPAQRALYRDVMRETFGHLGALGFSVPKPAFISWVEGEVEAW
302	ZN445_HUMAN	GCPGDQVTPTRSLTAQLQETMTFKDVEVTFSQDEWGWLDSAQRNLYRDVMLENYRNMASLVGPFTKPALISWLEAREPWG
303	ZFP30_HUMAN	ARDLVMFRDVAVDFSQEEWECLNSYQRNLYRDVILENYSNLVSLAGCSISKPDVITLLEQGKEPWMVVRDEKRRWTLDLE
304	ZN225_HUMAN	TTLKEAVTFKDVAVVFTEEELRLLDLAQRKLYREVMLENFRNLLSVGHQSLHRDTFHFLKEEKFWMMETATQREGNLGGK
305	ZN551_HUMAN	SPPSPRSSMAAVALRDSAQGMTFEDVAIYFSQEEWELLDESQRFLYCDVMLENFAHVTSLGYCHGMENEAIASEQSVSIQ
306	ZN610_HUMAN	DEEAQKRKAKESGMALPQGRLTFMDVAIEFSQEEWKSLDPGQRALYRDVMLENYRNLVFLGICLPDLSIISMLKQRREPL
307	ZN528_HUMAN	ALTQGPLKFMDVAIEFSQEEWKCLDPAQRTLYRDVMLENYRNLVSLGICLPDLSVTSMLEQKRDPWTLQSEEKIANDPDG
308	ZN284_HUMAN	TMFKEAVTFKDVAVVFTEEELGLLDVSQRKLYRDVMLENFRNLLSVGHQLSHRDTFHFQREEKFWIMETATQREGNSGGK
309	ZN418_HUMAN	QGTVAFEDVAVNFSQEEWSLLSEVQRCLYHDVMLENWVLISSLGCWCGSEDEEAPSKKSISIQRVSQVSTPGAGVSPKKA
310	MPP8_HUMAN	AEAFGDSEEDGEDVFEVEKILDMKTEGGKVLYKVRWKGYTSDDDTWEPEIHLEDCKEVLLEFRKKIAENKAKAVRKDIQR
311	ZN490_HUMAN	VLQMQNSEHHGQSIKTQTDSISLEDVAVNFTLEEWALLDPGQRNIYRDVMRATFKNLACIGEKWKDQDIEDEHKNQGRNL
312	ZN805_HUMAN	AMALTDPAQVSVTFDDVAVTFTQEEWGQLDLAQRTLYQEVMLENCGLLVSLGCPVPRPELIYHLEHGQEPWTRKEDLSQG
313	Z780B_HUMAN	VHGSVTFRDVAIDFSQEEWECLQPDQRTLYRDVMLENYSHLISLGSSISKPDVITLLEQEKEPWIVVSKETSRWYPDLES
314	ZN763_HUMAN	DPVACEDVAVNFTQEEWALLDISQRKLYREVMLETFRNLTSIGKKWKDQNIEYEYQNPRRNFRSLIEGNVNEIKEDSHCG
315	ZN285_HUMAN	IKFQERVTFKDVAVVFTKEELALLDKAQINLYQDVMLENFRNLMLVRDGIKNNILNLQAKGLSYLSQEVLHCWQIWKQRI
316	ZNF85_HUMAN	GPLTFRDVAIEFSLKEWQCLDTAQRNLYRNVMLENYRNLVFLGITVSKPDLITCLEQGKEAWSMKRHEIMVAKPTVMCSH
317	ZN223_HUMAN	TMSKEAVTFKDVAVVFTEEELGLLDLAQRKLYRDVMLENFRNLLSVGHQPFHRDTFHFLREEKFWMMDIATQREGNSGGK
318	ZNF90_HUMAN	GPLEFRDVAIEFSLEEWHCLDTAQQNLYRDVMLENYRHLVFLGIVVTKPDLITCLEQGKKPFTVKRHEMIAKSPVMCFHF
319	ZN557_HUMAN	GHTEGGELVNELLKSWLKGLVTFEDVAVEFTQEEWALLDPAQRTLYRDVMLENCRNLASLGNQVDKPRLISQLEQEDKVM
320	ZN425_HUMAN	AEPASVTVTFDDVALYFSEQEWEILEKWQKQMYKQEMKTNYETLDSLGYAFSKPDLITWMEQGRMLLISEQGCLDKTRRT
321	ZN229_HUMAN	HSQASAISQDREEKIMSQEPLSFKDVAVVFTEEELELLDSTQRQLYQDVMQENFRNLLSVGERNPLGDKNGKDTEYIQDE
322	ZN606_HUMAN	GSLEEGRRATGLPAAQVQEPVTFKDVAVDFTQEEWGQLDLVQRTLYRDVMLETYGHLLSVGNQIAKPEVISLLEQGEEPW
323	ZN155_HUMAN	TTFKEAVTFKDVAVVFTEEELGLLDPAQRKLYRDVMLENFRNLLSVGHQPFHQDTCHFLREEKFWMMGTATQREGNSGGK

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324	ZN222_HUMAN	AKLYEAVTFKDVAVIFTEEELGLLDPAQRKLYRDVMLENFRNLLSVGGKIQTEMETVPEAGTHEEFSCKQIWEQIASDLT
325	ZN442_HUMAN	RSDLFLPDSQTNEERKQYDSVAFEDVAVNFTQEEWALLGPSQKSLYRDVMWETIRNLDCIGMKWEDTNIEDQHRNPRRSL
326	ZNF91_HUMAN	PGTPGSLEMGLLTFRDVAIEFSPEEWQCLDTAQQNLYRNVMLENYRNLAFLGIALSKPDLITYLEQGKEPWNMKQHEMVD
327	ZN135_HUMAN	TPGVRVSTDPEQVTFEDVVVGFSQEEWGQLKPAQRTLYRDVMLDTFRLLVSVGHWLPKPNVISLLEQEAELWAVESRLPQ
328	ZN778_HUMAN	EQTQAAGMVAGWLINCYQDAVTFDDVAVDFTQEEWTLLDPSQRDLYRDVMLENYENLASVEWRLKTKGPALRQDRSWFRA
329	RYBP_HUMAN	PSEANSIQSANATTKTSETNHTSRPRLKNVDRSTAQQLAVTVGNVTVIITDFKEKTRSSSTSSSTVTSSAGSEQQNQSSS
330	ZN534_HUMAN	ALTQGQLSFSDVAIEFSQEEWKCLDPGQKALYRDVMLENYRNLVSLGEDNVRPEACICSGICLPDLSVTSMLEQKRDPWT
331	ZN586_HUMAN	AAAAALRAPAQSSVTFEDVAVNFSLEEWSLLNEAQRCLYRDVMLETLTLISSLGCWHGGEDEAAPSKQSTCIHIYKDQGG
332	ZN567_HUMAN	AQGSVSFNDVTVDFTQEEWQHLDHAQKTLYMDVMLENYCHLISVGCHMTKPDVILKLERGEEPWTSFAGHTCLEENWKAE
333	ZN440_HUMAN	DPVAFKDVAVNFTQEEWALLDISQRKLYREVMLETFRNLTSLGKRWKDQNIEYEHQNPRRNFRSLIEEKVNEIKDDSHCG
334	ZN583_HUMAN	SKDLVTFGDVAVNFSQEEWEWLNPAQRNLYRKVMLENYRSLVSLGVSVSKPDVISLLEQGKEPWMVKKEGTRGPCPDWEY
335	ZN441_HUMAN	DSVAFEDVAINFTCEEWALLGPSQKSLYRDVMQETIRNLDCIGMIWQNHDIEEDQYKDLRRNLRCHMVERACEIKDNSQC
336	ZNF43_HUMAN	GPLTFMDVAIEFCLEEWQCLDIAQQNLYRNVMLENYRNLVFLGIAVSKPDLITCLEQEKEPWEPMRRHEMVAKPPVMCSH
337	CBX5_HUMAN	QSNDIARGFERGLEPEKIIGATDSCGDLMFLMKWKDTDEADLVLAKEANVKCPQIVIAFYEERLTWHAYPEDAENKEKET
338	ZN589_HUMAN	ALPAKDSAWPWEEKPRYLGPVTFEDVAVLFTEAEWKRLSLEQRNLYKEVMLENLRNLVSLAESKPEVHTCPSCPLAFGSQ
339	ZNF10_HUMAN	DAKSLTAWSRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLGYQLTKPDVILRLEKGEEPWLVEREIHQ
340	ZN563_HUMAN	DAVAFEDVAVNFTQEEWALLGPSQKNLYRYVMQETIRNLDCIRMIWEEQNTEDQYKNPRRNLRCHMVERFSESKDSSQCG
341	ZN561_HUMAN	EKTKVERMVEDYLASGYQDSVTFDDVAVDFTPEEWALLDTTEKYLYRDVMLENYMNLASVEWEIQPRTKRSSLQQGFLKN
342	ZN136_HUMAN	DSVAFEDVDVNFTQEEWALLDPSQKNLYRDVMWETMRNLASIGKKWKDQNIKDHYKHRGRNLRSHMLERLYQTKDGSQRG
343	ZN630_HUMAN	IESQEPVTFEDVAVDFTQEEWQQLNPAQKTLHRDVMLETYNHLVSVGCSGIKPDVIFKLEHGKDPWIIESELSRWIYPDR
344	ZN527_HUMAN	AVGLCKAMSQGLVTFRDVALDFSQEEWEWLKPSQKDLYRDVMLENYRNLVWLGLSISKPNMISLLEQGKEPWMVERKMSQ
345	ZN333_HUMAN	DKVEEEAMAPGLPTACSQEPVTFADVAVVFTPEEWVFLDSTQRSLYRDVMLENYRNLASVADQLCKPNALSYLEERGEQW
346	Z324B_HUMAN	TFEDVAVYFSQEEWGLLDTAQRALYRHVMLENFTLVTSLGLSTSRPRVVIQLERGEEPWVPSGKDMTLARNTYGRLNSGS
347	ZN786_HUMAN	AEPPRLPLTFEDVAIYFSEQEWQDLEAWQKELYKHVMRSNYETLVSLDDGLPKPELISWIEHGGEPFRKWRESQKSGNII
348	ZN709_HUMAN	DSVVFEDVAVNFTQEEWALLGPSQKKLYRDVMQETFVNLASIGENWEEKNIEDHKNQGRKLRSHMVERLCERKEGSQFGE

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349	ZN792_HUMAN	AAAALRDPAQGCVTFEDVTIYFSQEEWVLLDEAQRLLYCDVMLENFALIAS LGLISFRSHIVSQLEMGKEPWVPDSVDMT
350	ZN599_HUMAN	AAPALALVSFEDVVVTFTGEEWGHLDLAQRTLYQEVMLETCRLLVSLGHP VPKPELIYLLEHGQELWTVKRGLSQSTCAG
351	ZN613_HUMAN	IKSQESLTLEDVAVEFTWEEWQLLGPAQKDLYRDVMLENYSNLVSVGYQA SKPDALFKLEQGEPWTVENEIHSQICPEIK
352	ZF69B_HUMAN	GESLESRVTLGSLTAESQELLTFKDVSVDFTQEEWGQLAPAHRNLYREVML ENYGNLVSVGCQLSKPGVISQLEKGEEPW
353	ZN799_HUMAN	ASVALEDVAVNFTREEWALLGPCQKNLYKDVMQETIRNLDCVGMKWKD QNIEDQYRYPRKNLRCRMLERFVESKDGTQCG
354	ZN569_HUMAN	TESQGTVTFKDVAIDFTQEEWKRLDPAQRKLYRNVMLENYNNLITVGYPFT KPDVIFKLEQEEEPWVMEEEVLRRHWQGE
355	ZN564_HUMAN	DSVASEDVAVNFTLEEWALLDPSQKKLYRDVMRETFRNLACVGKKWEDQ SIEDWYKNQGRILRNHMEEGLSESKEYDQCG
356	ZN546_HUMAN	EETQGELTSSCGSKTMANVSLAFRDVSIDLSQEEWECLDAVQRDLYKDVM LENYSNLVSLGYTIPKPDVITLLEQEKEPW
357	ZFP92_HUMAN	AAILLTTRPKVPVSFEDVSVYFTKTEWKLLDLRQKVLYKRVMLENYSHLVS LGFSFSKPHLISQLERGEGPWVADIPRTW
358	YAF2_HUMAN	KDKVEKEKSEKETTSKKNSHKKTRPRLKNVDRSSAQHLEVTVGDLTVIITD FKEKTKSPPASSAASADQHSQSGSSSDNT
359	ZN723_HUMAN	GPLTFTDVAIKFSLEEWQFLDTAQQNLYRDVMLENYRNLVFLGVGVSKPD LITCLEQGKEPWNMKRHKMVAKPPVVCSHF
360	ZNF34_HUMAN	RKPNPQAMAALFLSAPPQAEVTFEDVAVYLSREEWGRLGPAQRGLYRDVM LETYGNLVSLGVGPAGPKPGVISQLERGDE
361	ZN439_HUMAN	LSLSPILLYTCEMFQDPVAFKDVAVNFTQEEWALLDISQKNLYREVMLETF WNLTSIGKKWKDQNIEYEYQNPRRNFRSV
362	ZFP57_HUMAN	AAGEPRSLLFFQKPVTFEDVAVNFTQEEWDCLDASQRVLYQDVMSETFKN LTSVARIFLHKPELITKLEQEEEQWRETRV
363	ZNF19_HUMAN	AAMPLKAQYQEMVTFEDVAVHFTKTEWTGLSPAQRALYRSVMLENFGNL TALGYPVPKPALISLLERGDMAWGLEAQDDP
364	ZN404_HUMAN	ARVPLTFSDVAIDFSQEEWEYLNSDQRDLYRDVMLENYTNLVSLDFNFTTE SNKLSSEKRNYEVNAYHQETWKRNKTFNL
365	ZN274_HUMAN	ASRLPTAWSCEPVTFEDVTLGFTPEEWGLLDLKQKSLYREVMLENYRNLVS VEHQLSKPDVVSQLEEAEDFWPVERGIPQ
366	CBX3_HUMAN	SKKKRDAADKPRGFARGLDPERIIGATDSSGELMFLMKWKDSDEADLVLA KEANMKCPQIVIAFYEERLTWHSCPEDEAQ
367	ZNF30_HUMAN	AHKYVGLQYHGSVTFEDVAIAFSQQEWESLDSSQRGLYRDVMLENYRNLV SMGHSRSKPHVIALLEQWKEPEVTVRKDGR
368	ZN250_HUMAN	AAARLLPVPAGPQPLSFQAKLTFEDVAVLLSQDEWDRLCPAQRGLYRNVM METYGNVVSLGLPGSKPDIISQLERGEDPW
369	ZN570_HUMAN	AVGLLKAMYQELVTFRDVAVDFSQEEWDCLDSSQRHLYSNVMLENYRILV SLGLCFSKPSVILLLEQGKAPWMVKRELTK
370	ZN675_HUMAN	GLLTFRDVAIEFSLEEWQCLDTAQRNLYKNVILENYRNLVFLGIAVSKQDLI TCLEQEKEPLTVKRHEMVNEPPVMCSHF
371	ZN695_HUMAN	GLLAFRDVALEFSPEEWECLDPAQRSLYRDVMLENYRNLISLGEDSFNMQF LFHSLAMSKPELIICLEARKEPWNVNTEK
372	ZN548_HUMAN	NLTEGRVVFEDVAIYFSQEEWGHLDEAQRLLYRDVMLENLALLSSLGSWH GAEDEEAPSQQGFSVGVSEVTASKPCLSSQ
373	ZN132_HUMAN	GPAQHTSWPCGSAVPTLKSMVTFEDVAVYFSQEEWELLDAAQRHLYHSV MLENLELVTSLGSWHGVEGEGAHPKQNVSVE

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374	ZN738_HUMAN	SGYPGAERNLLEYSYFEKGPLTFRDVVIEFSQEEWQCLDTAQQDLYRKVML ENFRNLVFLGIDVSKPDLITCLEQGKDPW
375	ZN420_HUMAN	ARKLVMFRDVAIDFSQEEWECLDSAQRDLYRDVMLENYSNLVSLDLPSRC ASKDLSPEKNTYETELSQWEMSDRLENCDL
376	ZN626_HUMAN	GPLQFRDVAIEFSLEEWHCLDTAQRNLYRNVMLENYSNLVFLGITVSKPDLI TCLEQGRKPLTMKRNEMIAKPSVMCSHF
377	ZN559_HUMAN	VAGWLTNYSQDSVTFEDVAVDFTQEEWTLLDQTQRNLYRDVMLENYKNL VAVDWESHINTKWSAPQQNFLQGKTSSVVEM
378	ZN460_HUMAN	AAAWMAPAQESVTFEDVAVTFTQEEWGQLDVTQRALYVEVMLETCGLLV ALGDSTKPETVEPIPSHLALPEEVSLQEQLA
379	ZN268_HUMAN	VLEWLFISQEQPKITKSWGPLSFMDVFVDFTWEEWQLLDPAQKCLYRSVM LENYSNLVSLGYQHTKPDIIFKLEQGEELC
380	ZN304_HUMAN	AAAVLMDRVQSCVTFEDVFVYFSREEWELLEEAQRFLYRDVMLENFALVA TLGFWCEAEHEAPSEQSVSVEGVSQVRTAE
381	ZIM2_HUMAN	AGSQFPDFKHLGTFLVFEELVTFEDVLVDFSPEELSSLSAAQRNLYREVMLE NYRNLVSLGHQFSKPDIISRLEEEESYA
382	ZN605_HUMAN	IQSQISFEDVAVDFTLEEWQLLNPTQKNLYRDVMLENYSNLVFLEVWLDNP KMWLRDNQDNLKSMERGHKYDVFGKIFNS
383	ZN844_HUMAN	DLVAFEDVAVNFTQEEWSLLDPSQKNLYREVMQETLRNLASIGEKWKDQN IEDQYKNPRNNLRSLLGERVDENTEENHCG
384	SUMO5_HUMAN	KDEDIKLRVIGQDSSEIHFKVKMTTPLKKLKKSYCQRQGVPVNSLRFLFEGQ RIADNHTPEELGMEEEDVIEVYQEQIGG
385	ZN101_HUMAN	DSVAFEDVAVNFTQEEWALLSPSQKNLYRDVTLETFRNLASVGIQWKDQDI ENLYQNLGIKLRSLVERLCGRKEGNEHRE
386	ZN783_HUMAN	RNFWILRLPPGSKGEAPKVPVTFDDVAVYFSELEWGKLEDWQKELYKHVM RGNYETLVSLDYAISKPDILTRIERGEEPC
387	ZN417_HUMAN	AAAAPRRPTQQGTVTFEDVAVNFSQEEWCLLSEAQRCLYRDVMLENLALIS SLGCWCGSKDEEAPCKQRISVQRESQSRT
388	ZN182_HUMAN	SGEDSGSFYSWQKAKREQGLVTFEDVAVDFTQEEWQYLNPPQRTLYRDV MLETYSNLVFVGQQVTKPNLILKLEVEECPA
389	ZN823_HUMAN	DSVAFEDVAVNFTQEEWALLGPSQKSLYRNVMQETIRNLDCIEMKWEDQN IGDQCQNAKRNLRSHTCEIKDDSQCGETFG
390	ZN177_HUMAN	AAGWLTTWSQNSVTFQEVAVDFSQEEWALLDPAQKNLYKDVMLENFRNL ASVGYQLCRHSLISKVDQEQLKTDERGILQG
391	ZN197_HUMAN	ENPRNQLMALMLLTAQPQELVMFEEVSVCFTSEEWACLGPIQRALYWDVM LENYGNVTSLEWETMTENEEVTSKPSSSQR
392	ZN717_HUMAN	LETYNSLVSLQELVSFEEVAVHFTWEEWQDLDDAQRTLYRDVMLETYSSL VSLGHCITKPEMIFKLEQGAEPWIVEETPN
393	ZN669_HUMAN	RHFRRPEPCREPLASPIQDSVAFEDVAVNFTQEEWALLDSSQKNLYREVMQ ETCRNLASVGSQWKDQNIEDHFEKPGKDI
394	ZN256_HUMAN	AAAELTAPAQGIVTFEDVAVYFSWKEWGLLDEAQKCLYHDVMLENLTLTT SLGGSGAGDEEAPYQQSTSPQRVSQVRIPK
395	ZN251_HUMAN	AATFQLPGHQEMPLTFQDVAVYFSQAEGRQLGPQQRALYRDVMLENYGN VASLGFPVPKPELISQLEQGKELWVLNLLGA
396	CBX4_HUMAN	RSEAGEPPSSLQVKPETPASAAVAVAAAAAPTTTAEKPPAEAQDEPAESLSE FKPFFGNIIITDVTANCLTVTFKEYVTV
397	PCGF2_HUMAN	HRTTRIKITELNPHLMCALCGGYFIDATTIVECLHSFCKTCIVRYLETNKYCP MCDVQVHKTRPLLSIRSDKTLQDIVYK
398	CDY2_HUMAN	ASQEFEVEAIVDKRQDKNGNTQYLVRWKGYDKQDDTWEPEQHLMNCEKC VHDFNRRQTEKQKKLTWTTTSRIFSNNARRR

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399	CDYL2_HUMAN	ASGDLYEVERIVDKRKNKKGKWEYLIRWKGYGSTEDTWEPEHHLLHCEEFIDEFNGLHMSKDKRIKSGKQSSTSKLLRDS
400	HERC2_HUMAN	TLIRKADLENHNKDGGFWTVIDGKVYDIKDFQTQSLTGNSILAQFAGEDPVVALEAALQFEDTRESMHAFCVGQYLEPDQ
401	ZN562_HUMAN	EKTKIGTMVEDHRSNSYQDSVTFDDVAVEFTPEEWALLDTTQKYLYRDVMLENYMNLASVDFFFCLTSEWEIQPRTKRSS
402	ZN461_HUMAN	AHELVMFRDVAIDVSQEEWECLNPAQRNLYKEVMLENYSNLVSLGLSVSKPAVISSLEQGKEPWMVVREETGRWCPGTWK
403	Z324A_HUMAN	AFEDVAVYFSQEEWGLLDTAQRALYRRVMLDNFALVASLGLSTSRPRVVIQLERGEEPWVPSGTDTTLSRTTYRRRNPGS
404	ZN766_HUMAN	AQLRRGHLTFRDVAIEFSQEEWKCLDPVQKALYRDVMLENYRNLVSLGICLPDLSIISMMKQRTEPWTVENEMKVAKNPD
405	ID2_HUMAN	SDHSLGISRSKTPVDDPMSLLYNMNDCYSKLKELVPSIPQNKKVSKMEILQHVIDYILDLQIALDSHPTIVSLHHQRPGQ
406	TOX_HUMAN	KDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDGLGEEQKQVYKKKTEAAKKEYLKQLAAYRASLVSK
407	ZN274_HUMAN	QEEKQEDAAICPVTVLPEEPVTFQDVAVDFSREEWGLLGPTQRTEYRDVMLETFGHLVSVGWETTLENKELAPNSDIPEE
408	SCMH1_HUMAN	DASRLSGRDPSSWTVEDVMQFVREADPQLGPHADLFRKHEIDGKALLLLRSDMMMKYMGLKLGPALKLSYHIDRLKQGKF
409	ZN214_HUMAN	AVTFEDVTIIFTWEEWKFLDSSQKRLYREVMWENYTNVMSVENWNESYKSQEEKFRYLEYENFSYWQGWWNAGAQMYENQ
410	CBX7_HUMAN	ELSAIGEQVFAVESIRKKRVRKGKVEYLVKWKGWPPKYSTWEPEEHILDPRLVMAYEEKEERDRASGYRKRGPKPKRLLL
411	ID1_HUMAN	GGAGARLPALLDEQQVNVLLYDMNGCYSRLKELVPTLPQNRKVSKVEILQHVIDYIRDLQLELNSESEVGTPGGRGLPVR
412	CREM_HUMAN	VVMAASPGSLHSPQQLAEEATRKRELRLMKNREAAKECRRRKKEYVKCLESRVAVLEVQNKKLIEELETLKDICSPKTDY
413	SCX_HUMAN	GGGPGGRPGREPRQRHTANARERDRTNSVNTAFTALRTLIPTEPADRKLSKIETLRLASSYISHLGNVLLAGEACGDGQP
414	ASCL1_HUMAN	SGFGYSLPQQQPAAVARRNERERNRVKLVNLGFATLREHVPNGAANKKMSKVETLRSAVEYIRALQQLLDEHDAVSAAFQ
415	ZN764_HUMAN	APLPPRDPNGAGPEWREPGAVSFADVAVYFCREEWGCLRPAQRALYRDVMRETYGHLSALGIGGNKPALISWVEEEAELW
416	SCML2_HUMAN	KQGFSKDPSTWSVDEVIQFMKHTDPQISGPLADLFRQHEIDGKALFLLKSDVMMKYMGLKLGPALKLCYYIEKLKEGKYS
417	TWST1_HUMAN	SGGGSPQSYEELQTQRVMANVRERQRTQSLNEAFAALRKIIPTLPSDKLSKIQTLKLAARYIDFLYQVLQSDELDSKMAS
418	CREB1_HUMAN	IAPGVVMASSPALPTQPAEEAARKREVRLMKNREAARECRRKKKEYVKCLENRVAVLENQNKTLIEELKALKDLYCHKSD
419	TERF1_HUMAN	SRIPVSKSQPVTPEKHRARKRQAWLWEEDKNLRSGVRKYGEGNWSKILLHYKFNNRTSVMLKDRWRTMKKLKLISSDSED
420	ID3_HUMAN	SLAIARGRGKGPAAEEPLSLLDDMNHCYSRLRELVPGVPRGTQLSQVEILQRVIDYILDLQVVLAEPAPGPPDGPHLPIQ
421	CBX8_HUMAN	GSGPPSSGGGLYRDMGAQGGRPSLIARIPVARILGDPEEESWSPSLTNLEKVVVTDVTSNFLTVTIKESNTDQGFFKEKR
422	CBX4_HUMAN	ELPAVGEHVFAVESIEKKRIRKGRVEYLVKWRGWSPKYNTWEPEENILDPRLLIAFQNRERQEQLMGYRKRGPKPKPLVV
423	GSX1_HUMAN	VDSSSNQLPSSKRMRTAFTSTQLLELEREFASNMYLSRLRRIEIATYLNLSEKQVKIWFQNRRVKHKKEGKGSNHRGGGG

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424	NKX22_HUMAN	TPGGGGDAGKKRKRRVLFSKAQTYELERRFRQQRYLSAPEREHLASLIRLTPTQVKIWFQNHRYKMKRARAEKGMEVTPL
425	ATF1_HUMAN	QTVVMTSPVTLTSQTTKTDDPQLKREIRLMKNREAARECRRKKKEYVKCLENRVAVLENQNKTLIEELKTLKDLYSNKSV
426	TWST2_HUMAN	KGSPSAQSFEELQSQRILANVRERQRTQSLNEAFAALRKIIPTLPSDKLSKIQTLKLAARYIDFLYQVLQSDEMDNKMTS
427	ZNF17_HUMAN	NLTEDYMVFEDVAIHFSQEEWGILNDVQRHLHSDVMLENFALLSSVGCWHGAKDEEAPSKQCVSVGVSQVTTLKPALSTQ
428	TOX3_HUMAN	KDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDSLGEEQKQVYKRKTEAAKKEYLKALAAYRASLVSK
429	TOX4_HUMAN	KDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDSLGEEQKQVYKRKTEAAKKEYLKALAAYKDNQECQ
430	ZMYM3_HUMAN	LDGSTWDFCSEDCKSKYLLWYCKAARCHACKRQGKLLETIHWRGQIRHFCNQQCLLRFYSQQNQPNLDTQSGPESLLNSQ
431	I2BP1_HUMAN	ASVQASRRQWCYLCDLPKMPWAMVWDFSEAVCRGCVNFEGADRIELLIDAARQLKRSHVLPEGRSPGPPALKHPATKDLA
432	RHXF1_HUMAN	MEGPQPENMQPRTRRTKFTLLQVEELESVFRHTQYPDVPTRRELAENLGVTEDKVRVWFKNKRARCRRHQRELMLANELR
433	SSX2_HUMAN	PKIMPKKPAEEGNDSEEVPEASGPQNDGKELCPPGKPTTSEKIHERSGPKRGEHAWTHRLRERKQLVIYEEISDPEEDDE
434	I2BPL_HUMAN	SAAQVSSSRRQSCYLCDLPRMPWAMIWDFSEPVCRGCVNYEGADRIEFVIETARQLKRAHGCFQDGRSPGPPPPVGVKTV
435	ZN680_HUMAN	PGPPGSLEMGPLTFRDVAIEFSLEEWQCLDTAQRNLYRKVMFENYRNLVFLGIAVSKPHLITCLEQGKEPWNRKRQEMVA
436	CBX1_HUMAN	NKKKVEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKWKGFSDEDNTWEPEENLDCPDLIAEFLQSQKTAHETDKSEGGKR
437	TRI68_HUMAN	LANVVEKVRLLRLHPGMGLKGDLCERHGEKLKMFCKEDVLIMCEACSQSPEHEAHSVVPMEDVAWEYKWELHEALEHLKK
438	HXA13_HUMAN	VVSHPSDASSYRRGRKKRVPYTKVQLKELEREYATNKFITKDKRRRISATTNLSERQVTIWFQNRRVKEKKVINKLKTTS
439	PHC3_HUMAN	ENSDLLPVAQTEPSIWTVDDVWAFIHSLPGCQDIADEFRAQEIDGQALLLLKEDHLMSAMNIKLGPALKICARINSLKES
440	TCF24_HUMAN	AGPGGGSRSGSGRPAAANAARERSRVQTLRHAFLELQRTLPSVPPDTKLSKLDVLLLATTYIAHLTRSLQDDAEAPADAG
441	CBX3_HUMAN	QNGKSKKVEEAEPEEFVVEKVLDRRVVNGKVEYFLKWKGFTDADNTWEPEENLDCPELIEAFLNSQKAGKEKDGTKRKSL
442	HXB13_HUMAN	QHPPDACAFRRGRKKRIPYSKGQLRELEREYAANKFITKDKRRKISAATSLSERQITIWFQNRRVKEKKVLAKVKNSATP
443	HEY1_HUMAN	SMSPTTSSQILARKRRRGIIEKRRRDRINNSLSELRRLVPSAFEKQGSAKLEKAEILQMTVDHLKMLHTAGGKGYFDAHA
444	PHC2_HUMAN	LVGMGHHFLPSEPTKWNVEDVYEFIRSLPGCQEIAEEFRAQEIDGQALLLLKEDHLMSAMNIKLGPALKIYARISMLKDS
445	ZNF81_HUMAN	PANEDAPQPGEHGSACEVSVSFEDVTVDFSREEWQQLDSTQRRLYQDVMLENYSHLLSVGFEVPKPEVIFKLEQGEGPWT
446	FIGLA_HUMAN	GYSSTENLQLVLERRRVANAKERERIKNLNRGFARLKALVPFLPQSRKPSKVDILKGATEYIQVLSDLLEGAKDSKKQDP
447	SAM11_HUMAN	EEAPAPEDVTKWTVDDVCSFVGGLSGCGEYTRVFREQGIDGETLPLLTEEHLLTNMGLKLGPALKIRAQVARRLGRVFYV
448	KMT2B_HUMAN	GGTLAHTPRRSLPSHHGKKMRMARCGHCRGCLRVQDCGSCVNCLDKPKFGGPNTKKQCCVYRKCDKIEARKMERLAKKGR

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449	HEY2_HUMAN	LNSPTTTSQIMARKKRRGIIEKRRRDRINNSLSELRRLVPTAFEKQGSAKLEKAEILQMTVDHLKMLQATGGKGYFDAHA
450	JDP2_HUMAN	QPVKSELDEEEERRKRRREKNKVAAARCRNKKKERTEFLQRESERLELMNAELKTQIEELKQERQQLILMLNRHRPTCIV
451	HXC13_HUMAN	LQPEVSSYRRGRKKRVPYTKVQLKELEKEYAASKFITKEKRRRISATTNLSERQVTIWFQNRRVKEKKVVSKSKAPHLHS
452	ASCL4_HUMAN	LPVPLDSAFEPAFLRKRNERERQRVRCVNEGYARLRDHLPRELADKRLSKVETLRAAIDYIKHLQELLERQAWGLEGAAG
453	HHEX_HUMAN	SPFLQRPLHKRKGGQVRFSNDQTIELEKKFETQKYLSPPERKRLAKMLQLSERQVKTWFQNRRAKWRRLKQENPQSNKKE
454	HERC2_HUMAN	IAIATGSLHCVCCTEDGEVYTWGDNDEGQLGDGTTNAIQRPRLVAALQGKKVNRVACGSAHTLAWSTSKPASAGKLPAQV
455	GSX2_HUMAN	GGSDASQVPNGKRMRTAFTSTQLLELEREFSSNMYLSRLRRIEIATYLNLSEKQVKIWFQNRRVKHKKEGKGTQRNSHAG
456	BIN1_HUMAN	RLDLPPGFMFKVQAQHDYTATDTDELQLKAGDVVLVIPFQNPEEQDEGWLMGVKESDWNQHKELEKCRGVFPENFTERVP
457	ETV7_HUMAN	GICKLPGRLRIQPALWSREDVLHWLRWAEQEYSLPCTAEHGFEMNGRALCILTKDDFRHRAPSSGDVLYELLQYIKTQRR
458	ASCL3_HUMAN	PNYRGCEYSYGPAFTRKRNERERQRVKCVNEGYAQLRHHLPEEYLEKRLSKVETLRAAIKYINYLQSLLYPDKAETKNNP
459	PHC1_HUMAN	LHGINPVFLSSNPSRWSVEEVYEFIASLQGCQEIAEEFRSQEIDGQALLLLKEEHLMSAMNIKLGPALKICAKINVLKET
460	OTP_HUMAN	QAGQQQGQQKQKRHRTRFTPAQLNELERSFAKTHYPDIFMREELALRIGLTESRVQVWFQNRRAKWKKRKKTTNVFRAPG
461	I2BP2_HUMAN	AAAVAVAAASRRQSCYLCDLPRMPWAMIWDFTEPVCRGCVNYEGADRVEFVIETARQLKRAHGCFPEGRSPPGAAASAAA
462	VGLL2_HUMAN	FSSQTPASIKEEEGSPEKERPPEAEYINSRCVLFTYFQGDISSVVDEHFSRALSQPSSYSPSCTSSKAPRSSGPWRDCSF
463	HXA11_HUMAN	DKAGGSSGQRTRKKRCPYTKYQIRELEREFFFSVYINKEKRLQLSRMLNLTDRQVKIWFQNRRMKEKKINRDRLQYYSAN
464	PDLI4_HUMAN	GAPLSGLQGLPECTRCGHGIVGTIVKARDKLYHPECFMCSDCGLNLKQRGYFFLDERLYCESHAKARVKPPEGYDVVAVY
465	ASCL2_HUMAN	RRPATAETGGGAAAVARRNERERNRVKLVNLGFQALRQHVPHGGASKKLSKVETLRSAVEYIRALQRLLAEHDAVRNALA
466	CDX4_HUMAN	TVQVTGKTRTKEKYRVVYTDHQRLELEKEFHCNRYITIQRKSELAVNLGLSERQVKIWFQNRRAKERKMIKKKISQFENS
467	ZN860_HUMAN	EEAAQKRKEKEPGMALPQGHLTFRDVAIEFSLEEWKCLDPTQRALYRAMMLENYRNLHSVDISSKCMMKKFSSTAQGNTE
468	LMBL4_HUMAN	DIRASQVARWTVDEVAEFVQSLLGCEEHAKCFKKEQIDGKAFLLLTQTDIVKVMKIKLGPALKIYNSILMFRHSQELPEE
469	PDIP3_HUMAN	LSPLEGTKMTVNNLHPRVTEEDIVELFCVCGALKRARLVHPGVAEVVFVKKDDAITAYKKYNNRCLDGQPMKCNLHMNGN
470	NKX25_HUMAN	DNAERPRARRRRKPRVLFSQAQVYELERRFKQQRYLSAPERDQLASVLKLTSTQVKIWFQNRRYKCKRQRQDQTLELVGL
471	CEBPB_HUMAN	SQVKSKAKKTVDKHSDEYKIRRERNNIAVRKSRDKAKMRNLETQHKVLELTAENERLQKKVEQLSRELSTLRNLFKQLPE
472	ISL1_HUMAN	KRDYIRLYGIKCAKCSIGFSKNDFVMRARSKVYHIECFRCVACSRQLIPGDEFALREDGLFCRADHDVVERASLGAGDPL
473	CDX2_HUMAN	SLGSQVKTRTKDKYRVVYTDHQRLELEKEFHYSRYITIRRKAELAATLGLSERQVKIWFQNRRAKERKINKKKLQQQQQQ

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474	PROP1_HUMAN	QGGQRGRPHSRRRHRTTFSPVQLEQLESAFGRNQYPDIWARESLARDTGLSEARIQVWFQNRRAKQRKQERSLLQPLAHL
475	SIN3B_HUMAN	DALTYLDQVKIRFGSDPATYNGFLEIMKEFKSQSIDTPGVIRRVSQLFHEHPDLIVGFNAFLPLGYRIDIPKNGKLNIQS
476	SMBT1_HUMAN	RLHLDSNPLKWSVADVVRFIRSTDCAPLARIFLDQEIDGQALLLLTLPTVQECMDLKLGPAIKLCHHIERIKFAFYEQFA
477	HXC11_HUMAN	AKGAAPNAPRTRKKRCPYSKFQIRELEREFFFNVYINKEKRLQLSRMLNLTDRQVKIWFQNRRMKEKKLSRDRLQYFSGN
478	HXC10_HUMAN	TTGNWLTAKSGRKKRCPYTKHQTLELEKEFLFNMYLTRERRLEISKTINLTDRQVKIWFQNRRMKLKKMNRENRIRELTS
479	PRS6A_HUMAN	YLVSNVIELLDVDPNDQEEDGANIDLDSQRKGKCAVIKTSTRQTYFLPVIGLVDAEKLKPGDLVGVNKDSYLILETLPTE
480	VSX1_HUMAN	KASPTLGKRKKRRHRTVFTAHQLEELEKAFSEAHYPDVYAREMLAVKTELPEDRIQVWFQNRRAKWRKREKRWGGSSVMA
481	NKX23_HUMAN	EESERPKPRSRRKPRVLFSQAQVFELERRFKQQRYLSAPEREHLASSLKLTSTQVKIWFQNRRYKCKRQRQDKSLELGAH
482	MTG16_HUMAN	VVPGSRQEEVIDHKLTEREWAEEWKHLNNLLNCIMDMVEKTRRSLTVLRRCQEADREELNHWARRYSDAEDTKKGPAPAA
483	HMX3_HUMAN	ESPEKKPACRKKKTRTVFSRSQVFQLESTFDMKRYLSSSERAGLAASLHLTETQVKIWFQNRRNKWKRQLAAELEAANLS
484	HMX1_HUMAN	RGGVGVGGGRKKKTRTVFSRSQVFQLESTFDLKRYLSSAERAGLAASLQLTETQVKIWFQNRRNKWKRQLAAELEAASLS
485	KIF22_HUMAN	ELLAHGRQKILDLLNEGSARDLRSLQRIGPKKAQLIVGWRELHGPFSQVEDLERVEGITGKQMESFLKANILGLAAGQRC
486	CSTF2_HUMAN	ESPYGETISPEDAPESISKAVASLPPEQMFELMKQMKLCVQNSPQEARNMLLQNPQLAYALLQAQVVMRIVDPEIALKIL
487	CEBPE_HUMAN	AGPLHKGKKAVNKDSLEYRLRRERNNIAVRKSRDKAKRRILETQQKVLEYMAENERLRSRVEQLTQELDTLRNLFRQIPE
488	DLX2_HUMAN	IRIVNGKPKKVRKPRTIYSSFQLAALQRRFQKTQYLALPERAELAASLGLTQTQVKIWFQNRRSKFKKMWKSGEIPSEQH
489	ZMYM3_HUMAN	TVYQFCSPSCWTKFQRTSPEGGIHLSCHYCHSLFSGKPEVLDWQDQVFQFCCRDCCEDFKRLRGVVSQCEHCRQEKLLHE
490	PPARG_HUMAN	TMVDTEMPFWPTNFGISSVDLSVMEDHSHSFDIKPFTTVDFSSISTPHYEDIPFTRTDPVVADYKYDLKLQEYQSAIKVE
491	PRIC1_HUMAN	GRHHAELLKPRCSACDEIIFADECTEAEGRHWHMKHFCCLECETVLGGQRYIMKDGRPFCCGCFESLYAEYCETCGEHIG
492	UNC4_HUMAN	DPDKESPGCKRRRTRTNFTGWQLEELEKAFNESHYPDVFMREALALRLDLVESRVQVWFQNRRAKWRKKENTKKGPGRPA
493	BARX2_HUMAN	TEQPTPRQKKPRRSRTIFTELQLMGLEKKFQKQKYLSTPDRLDLAQSLGLTQLQVKTWYQNRRMKWKKMVLKGGQEAPTK
494	ALX3_HUMAN	SMELAKNKSKKRRNRTTFSTFQLEELEKVFQKTHYPDVYAREQLALRTDLTEARVQVWFQNRRAKWRKRERYGKIQEGRN
495	TCF15_HUMAN	GGGGGAGPVVVVRQRQAANARERDRTQSVNTAFTALRTLIPTEPVDRKLSKIETVRLASSYIAHLANVLLLGDSADDGQP
496	TERA_HUMAN	IDDTVEGITGNLFEVYLKPYFLEAYRPIRKGDIFLVRGGMRAVEFKVVETDPSPYCIVAPDTVIHCEGEPIKREDEEESL
497	VSX2_HUMAN	SALNQTKKRKKRRHRTIFTSYQLEELEKAFNEAHYPDVYAREMLAMKTELPEDRIQVWFQNRRAKWRKREKCWGRSSVMA
498	HXD12_HUMAN	DGLPWGAAPGRARKKRKPYTKQQIAELENEFLVNEFINRQKRKELSNRLNLSDQQVKIWFQNRRMKKKRVVLREQALALY

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499	CDX1_HUMAN	GGGGSGKTRTKDKYRVVYTDHQRLELEKEFHYSRYITIRRKSELAANLGLTERQVKIWFQNRRAKERKVNKKKQQQQQPP
500	TCF23_HUMAN	TRAGGLALGRSEASPENAARERSRVRTLRQAFLALQAALPAVPPDTKLSKLDVLVLAASYIAHLTRTLGHELPGPAWPPF
501	ALX1_HUMAN	KCDSNVSSSKKRRHRTTFTSLQLEELEKVFQKTHYPDVYVREQLALRTELTEARVQVWFQNRRAKWRKRERYGQIQQAKS
502	HXA10_HUMAN	NAANWLTAKSGRKKRCPYTKHQTLELEKEFLFNMYLTRERRLEISRSVHLTDRQVKIWFQNRRMKLKKMNRENRIRELTA
503	RX_HUMAN	LSEEEQPKKKHRRNRTTFTTYQLHELERAFEKSHYPDVYSREELAGKVNLPEVRVQVWFQNRRAKWRRQEKLEVSSMKLQ
504	CXXC5_HUMAN	HMAGLAEYPMQGELASAISSGKKKRKRCGMCAPCRRRINCEQCSSCRNRKTGHQICKFRKCEELKKKPSAALEKVMLPTG
505	SCML1_HUMAN	SITKHPSTWSVEAVVLFLKQTDPLALCPLVDLFRSHEIDGKALLLLTSDVLLKHLGVKLGTAVKLCYYIDRLKQGKCFEN
506	NFIL3_HUMAN	ACRRKREFIPDEKKDAMYWEKRRKNNEAAKRSREKRRLNDLVLENKLIALGEENATLKAELLSLKLKFGLISSTAYAQEI
507	DLX6_HUMAN	EIRFNGKGKKIRKPRTIYSSLQLQALNHRFQQTQYLALPERAELAASLGLTQTQVKIWFQNKRSKFKKLLKQGSNPHESD
508	MTG8_HUMAN	GLHGTRQEEMIDHRLTDREWAEEWKHLDHLLNCIMDMVEKTRRSLTVLRRCQEADREELNYWIRRYSDAEDLKKGGGSSS
509	CBX8_HUMAN	ELSAVGERVFAAEALLKRRIRKGRMEYLVKWKGWSQKYSTWEPEENILDARLLAAFEEREREMELYGPKKRGPKPKTFLL
510	CEBPD_HUMAN	AREKSAGKRGPDRGSPEYRQRRERNNIAVRKSRDKAKRRNQEMQQKLVELSAENEKLHQRVEQLTRDLAGLRQFFKQLPS
511	SEC13_HUMAN	SGGCDNLIKLWKEEEDGQWKEEQKLEAHSDWVRDVAWAPSIGLPTSTIASCSQDGRVFIWTCDDASSNTWSPKLLHKFND
512	FIP1_HUMAN	VKGVDLDAPGSINGVPLLEVDLDSFEDKPWRKPGADLSDYFNYGFNEDTWKAYCEKQKRIRMGLEVIPVTSTTNKITAED
513	ALX4_HUMAN	KADSESNKGKKRRNRTTFTSYQLEELEKVFQKTHYPDVYAREQLAMRTDLTEARVQVWFQNRRAKWRKRERFGQMQQVRT
514	LHX3_HUMAN	TAKQREAEATAKRPRTTITAKQLETLKSAYNTSPKPARHVREQLSSETGLDMRVVQVWFQNRRAKEKRLKKDAGRQRWGQ
515	PRIC2_HUMAN	GRHHAECLKPRCAACDEIIFADECTEAEGRHWHMKHFCCFECETVLGGQRYIMKEGRPYCCHCFESLYAEYCDTCAQHIG
516	MAGI3_HUMAN	IIGGDRPDEFLQVKNVLKDGPAAQDGKIAPGDVIVDINGNCVLGHTHADVVQMFQLVPVNQYVNLTLCRGYPLPDDSEDP
517	NELL1_HUMAN	CCPECDTRVTSQCLDQNGHKLYRSGDNWTHSCQQCRCLEGEVDCWPLTCPNLSCEYTAILEGECCPRCVSDPCLADNITY
518	PRRX1_HUMAN	LNSEEKKKRKQRRNRTTFNSSQLQALERVFERTHYPDAFVREDLARRVNLTEARVQVWFQNRRAKFRRNERAMLANKNAS
519	MTG8R_HUMAN	GLNGGYQDELVDHRLTEREWADEWKHLDHALNCIMEMVEKTRRSMAVLRRCQESDREELNYWKRRYNENTELRKTGTELV
520	RAX2_HUMAN	GPGEEAPKKKHRRNRTTFTTYQLHQLERAFEASHYPDVYSREELAAKVHLPEVRVQVWFQNRRAKWRRQERLESGSGAVA
521	DLX3_HUMAN	VRMVNGKPKKVRKPRTIYSSYQLAALQRRFQKAQYLALPERAELAAQLGLTQTQVKIWFQNRRSKFKKLYKNGEVPLEHS
522	DLX1_HUMAN	EVRFNGKGKKIRKPRTIYSSLQLQALNRRFQQTQYLALPERAELAASLGLTQTQVKIWFQNKRSKFKKLMKQGGAALEGS
523	NKX26_HUMAN	GRSEQPKARQRRKPRVLFSQAQVLALERRFKQQRYLSAPEREHLASALQLTSTQVKIWFQNRRYKCKRQRQDKSLELAGH

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524	NAB1_HUMAN	LPRTLGELQLYRILQKANLLSYFDAFIQQGGDDVQQLCEAGEEEFLEIMALV GMASKPLHVRRLQKALRDWVTNPGLFNQ
525	SAMD7_HUMAN	NLSLDEDIQKWTVDDVHSFIRSLPGCSDYAQVFKDHAIDGETLPLLTEEHLR GTMGLKLGPALKIQSQVSQHVGSMFYKK
526	PITX3_HUMAN	SPEDGSLKKKQRRQRTHFTSQQLQELEATFQRNRYPDMSTREEIAVWTNLT EARVRVWFKNRRAKWRKRERSQQAELCKG
527	WDR5_HUMAN	SNLLVSASDDKTLKIWDVSSGKCLKTLKGHSNYVFCCNFNPQSNLIVSGSFD ESVRIWDVKTGKCLKTLPAHSDPVSAVH
528	MEOX2_HUMAN	GNYKSEVNSKPRKERTAFTKEQIRELEAEFAHHNYLTRLRRYEIAVNLDLTE RQVKVWFQNRRMKWKRVKGGQQGAAARE
529	NAB2_HUMAN	LPRTLGELQLYRVLQRANLLSYYETFIQQGGDDVQQLCEAGEEEFLEIMAL VGMATKPLHVRRLQKALREWATNPGLFSQ
530	DHX8_HUMAN	PEEPTIGDIYNGKVTSIMQFGCFVQLEGLRKRWEGLVHISELRREGRVANVA DVVSKGQRVKVKVLSFTGTKTSLSMKDV
531	FOXA2_HUMAN	YAFNHPFSINNLMSSEQQHHHSHHHHQPHKMDLKAYEQVMHYPGYGSPM PGSLAMGPVTNKTGLDASPLAADTSYYQGVY
532	CBX6_HUMAN	TAAAGPAPPTAPEPAGASSEPEAGDWRPEMSPCSNVVVTDVTSNLLTVTIK EFCNPEDFEKVAAGVAGAAGGGGSIGASK
533	EMX2_HUMAN	FLLHNALARKPKRIRTAFSPSQLLRLEHAFEKNHYVVGAERKQLAHSLSLTE TQVKVWFQNRRTKFKRQKLEEEGSDSQQ
534	CPSF6_HUMAN	KRIALYIGNLTWWTTDEDLTEAVHSLGVNDILEIKFFENRANGQSKGFALV GVGSEASSKKLMDLLPKRELHGQNPVVTP
535	HXC12_HUMAN	SGAPWYPINSRSRKKRKPYSKLQLAELEGEFLVNEFITRQRRRELSDRLNLS DQQVKIWFQNRRMKKKRLLLREQALSFF
536	KDM4B_HUMAN	SDNLYPESITSRDCVQLGPPSEGELVELRWTDGNLYKAKFISSVTSHIYQVEF EDGSQLTVKRGDIFTLEEELPKRVRSR
537	LMBL3_HUMAN	GIPASKVSKWSTDEVSEFIQSLPGCEEHGKVFKDEQIDGEAFLLMTQTDIVKI MSIKLGPALKIFNSILMFKAAEKNSHN
538	PHX2A_HUMAN	EPSGLHEKRKQRRIRTTFTSAQLKELERVFAETHYPDIYTREELALKIDLTEA RVQVWFQNRRAKFRKQERAASAKGAAG
539	EMX1_HUMAN	LLLHGPFARKPKRIRTAFSPSQLLRLERAFEKNHYVVGAERKQLAGSLSLSE TQVKVWFQNRRTKYKRQKLEEEGPESEQ
540	NC2B_HUMAN	SSGNDDDLTIPRAAINKMIKETLPNVRVANDARELVVNCCTEFIHLISSEANE ICNKSEKKTISPEHVIQALESLGFGSY
541	DLX4_HUMAN	ERRPQAPAKKLRKPRTIYSSLQLQHLNQRFQHTQYLALPERAQLAAQLGLT QTQVKIWFQNKRSKYKKLLKQNSGGQEGD
542	SRY_HUMAN	NVQDRVKRPMNAFIVWSRDQRRKMALENPRMRNSEISKQLGYQWKMLTE AEKWPFFQEAQKLQAMHREKYPNYKYRPRRK
543	ZN777_HUMAN	EITRLAVWAAVQAVERKLEAQAMRLLTLEGRTGTNEKKIADCEKTAVEFA NHLESKWVVLGTLLQEYGLLQRRLENMENL
544	NELL1_HUMAN	CEKDIDECSEGIIECHNHSRCVNLPGWYHCECRSGFHDDGTYSLSGESCIDID ECALRTHTCWNDSACINLAGGFDCLCP
545	ZN398_HUMAN	AAISLWTVVAAVQAIERKVEIHSRRLLHLEGRTGTAEKKLASCEKTVTELG NQLEGKWAVLGTLLQEYGLLQRRLENLEN
546	GATA3_HUMAN	GQNRPLIKPKRRLSAARRAGTSCANCQTTTTTLWRRNANGDPVCNACGLY YKLHNINRPLTMKKEGIQTRNRKMSSKSKK
547	BSH_HUMAN	HAELPGKHCRRRKARTVFSDSQLSGLEKRFEIQRYLSTPERVELATALSLSE TQVKTWFQNRRMKHKKQLRKSQDEPKAP
548	SF3B4_HUMAN	QDATVYVGGLDEKVSEPLLWELFLQAGPVVNTHMPKDRVTGQHQGYGFV EFLSEEDADYAIKIMNMIKLYGKPIRVNKAS

SEQ ID NO	Description	Sequence
549	TEAD1_HUMAN	PIDNDAEGVWSPDIEQSFQEALAIYPPCGRRKIILSDEGKMYGRNELIARYIKLRTGKTRTRKQVSSHIQVLARRKSRDF
550	TEAD3_HUMAN	GLDNDAEGVWSPDIEQSFQEALAIYPPCGRRKIILSDEGKMYGRNELIARYIKLRTGKTRTRKQVSSHIQVLARKKVREY
551	RGAP1_HUMAN	DSVGTPQSNGGMRLHDFVSKTVIKPESCVPCGKRIKFGKLSLKCRDCRVVSHPECRDRCPLPCIPTLIGTPVKIGEGMLA
552	PHF1_HUMAN	SAPHSMTASSSSVSSPSPGLPRRSAPPSPLCRSLSPGTGGGVRGGVGYLSRGDPVRVLARRVRPDGSVQYLVEWGGGGIF
553	FOXA1_HUMAN	GDPHYSFNHPFSINNLMSSSEQQHKLDFKAYEQALQYSPYGSTLPASLPLGSASVTTRSPIEPSALEPAYYQGVYSRPVL
554	GATA2_HUMAN	GQNRPLIKPKRRLSAARRAGTCCANCQTTTTTLWRRNANGDPVCNACGLYYKLHNVNRPLTMKKEGIQTRNRKMSNKSKK
555	FOXO3_HUMAN	DSLSGSSLYSTSANLPVMGHEKFPSDLDLDMFNGSLECDMESIIRSELMDADGLDFNFDSLISTQNVVGLNVGNFTGAKQ
556	ZN212_HUMAN	TEISLWTVVAAIQAVEKKMESQAARLQSLEGRTGTAEKKLADCEKMAVEFGNQLEGKWAVLGTLLQEYGLLQRRLENVEN
557	IRX4_HUMAN	MDSGTRRKNATRETTSTLKAWLQEHRKNPYPTKGEKIMLAIITKMTLTQVSTWFANARRRLKKENKMTWPPRNKCADEKR
558	ZBED6_HUMAN	NIEKQIYLPSTRAKTSIVWHFFHVDPQYTWRAICNLCEKSVSRGKPGSHLGTSTLQRHLQARHSPHWTRANKFGVASGEE
559	LHX4_HUMAN	AKQNDDSEAGAKRPRTTITAKQLETLKNAYKNSPKPARHVREQLSSETGLDMRVVQVWFQNRRAKEKRLKKDAGRHRWGQ
560	SIN3A_HUMAN	DALSYLDQVKLQFGSQPQVYNDFLDIMKEFKSQSIDTPGVISRVSQLFKGHPDLIMGFNTFLPPGYKIEVQTNDMVNVTT
561	RBBP7_HUMAN	DDHTVCLWDINAGPKEGKIVDAKAIFTGHSAVVEDVAWHLLHESLFGSVADDQKLMIWDTRSNTTSKPSHLVDAHTAEVN
562	NKX61_HUMAN	GSILLDKDGKRKHTRPTFSGQQIFALEKTFEQTKYLAGPERARLAYSLGMTESQVKVWFQNRRTKWRKKHAAEMATAKKK
563	TRI68_HUMAN	DPTALVEAIVEEVACPICMTFLREPMSIDCGHSFCHSCLSGLWEIPGESQNWGYTCPLCRAPVQPRNLRPNWQLANVVEK
564	R51A1_HUMAN	QSLPKKVSLSSDTTRKPLEIRSPSAESKKPKWVPPAASGGSRSSSSPLVVVSVKSPNQSLRLGLSRLARVKPLHPNATST
565	MB3L1_HUMAN	AKSSQRKQRDCVNQCKSKPGLSTSIPLRMSSYTFKRPVTRITPHPGNEVRYHQWEESLEKPQQVCWQRRLQGLQAYSSAG
566	DLX5_HUMAN	VRMVNGKPKKVRKPRTIYSSFQLAALQRRFQKTQYLALPERAELAASLGLTQTQVKIWFQNKRSKIKKIMKNGEMPPEHS
567	NOTC1_HUMAN	LQCNNHACGWDGGDCSLNFNDPWKNCTQSLQCWKYFSDGHCDSQCNSAGCLFDGFDCQRAEGQCNPLYDQYCKDHFSDGH
568	TERF2_HUMAN	ETWVEEDELFQVQAAPDEDSTTNITKKQKWTVEESEWVKAGVQKYGEGNWAAISKNYPFVNRTAVMIKDRWRTMKRLGMN
569	ZN282_HUMAN	AEISLWTVVAAIQAVERKVDAQASQLLNLEGRTGTAEKKLADCEKTAVEFGNHMESKWAVLGTLLQEYGLLQRRLENLEN
570	RGS12_HUMAN	LEKRTLFRLDLVPINRSVGLKAKPTKPVTEVLRPVVARYGLDLSGLLVRLSGEKEPLDLGAPISSLDGQRVVLEEKDPSR
571	ZN840_HUMAN	PNCLSSSMQLPHGGGRHQELVRFRDVAVVFSPEEWDHLTPEQRNLYKDVMLDNCKYLASLGNWTYKAHVMSSLKQGKEPW
572	SPI2B_HUMAN	DDYKEGDLRIMPESSESPPTEREPGGVVDGLIGKHVEYTKEDGSKRIGMVIHQVEAKPSVYFIKFDDDFHIYVYDLVKKS
573	PAX7_HUMAN	SEPDLPLKRKQRRSRTTFTAEQLEELEKAFERTHYPDIYTREELAQRTKLTEARVQVWFSNRRARWRKQAGANQLAAFNH

SEQ ID NO	Description	Sequence
574	NKX62_HUMAN	AGGVLDKDGKKKHSRPTFSGQQIFALEKTFEQTKYLAGPERARLAYSLGMTESQVKVWFQNRRTKWRKRHAVEMASAKKK
575	ASXL2_HUMAN	DVMSFSVTVTTIPASQAMNPSSHGQTIPVQAFSEENSIEGTPSKCYCRLKAMIMCKGCGAFCHDDCIGPSKLCVSCLVVR
576	FOXO1_HUMAN	GGYSSVSSCNGYGRMGLLHQEKLPSDLDGMFIERLDCDMESIIRNDLMDGDTLDFNFDNVLPNQSFPHSVKTTTHSWVSG
577	GATA3_HUMAN	GGSPTGFGCKSRPKARSSTGRECVNCGATSTPLWRRDGTGHYLCNACGLYHKMNGQNRPLIKPKRRLSAARRAGTSCANC
578	GATA1_HUMAN	GQNRPLIRPKKRLIVSKRAGTQCTNCQTTTTTLWRRNASGDPVCNACGLYYKLHQVNRPLTMRKDGIQTRNRKASGKGKK
579	ZMYM5_HUMAN	PVALLRKQNFQPTAQQQLTKPAKITCANCKKPLQKGQTAYQRKGSAHLFCSTTCLSSFSHKRTQNTRSIICKKDASTKKA
580	ZN783_HUMAN	TEITLWTVVAAIQALEKKVDSCLTRLLTLEGRTGTAEKKLADCEKTAVEFGNQLEGKWAVLGTLLQEYGLLQRRLENVEN
581	SPI2B_HUMAN	KKQRGRPSSQPRRNIVGCRISHGWKEGDEPITQWKGTVLDQVPINPSLYLVKYDGIDCVYGLELHRDERVLSLKILSDRV
582	LRP1_HUMAN	WTCDLDDDCGDRSDESASCAYPTCFPLTQFTCNNGRCININWRCDNDNDCGDNSDEAGCSHSCSSTQFKCNSGRCIPEHW
583	MIXL1_HUMAN	PKGAAAPSASQRRKRTSFSAEQLQLLELVFRRTRYPDIHLRERLAALTLLPESRIQVWFQNRRAKSRRQSGKSFQPLARP
584	SGT1_HUMAN	KIKYDWYQTESQVVITLMIKNVQKNDVNVEFSEKELSALVKLPSGEDYNLKLELLHPIIPEQSTFKVLSTKIEIKLKKPE
585	LMCD1_HUMAN	DPSKEVEYVCELCKGAAPPDSPVVYSDRAGYNKQWHPTCFVCAKCSEPLVDLIYFWKDGAPWCGRHYCESLRPRCSGCDE
586	CEBPA_HUMAN	GSGAGKAKKSVDKNSNEYRVRRERNNIAVRKSRDKAKQRNVETQQKVLELTSDNDRLRKRVEQLSRELDTLRGIFRQLPE
587	GATA2_HUMAN	GPASSFTPKQRSKARSCSEGRECVNCGATATPLWRRDGTGHYLCNACGLYHKMNGQNRPLIKPKRRLSAARRAGTCCANC
588	SOX14_HUMAN	KPSDHIKRPMNAFMVWSRGQRRKMAQENPKMHNSEISKRLGAEWKLLSEAEKRPYIDEAKRLRAQHMKEHPDYKYRPRRK
589	WTIP_HUMAN	LYSGFQQTADKCSVCGHLIMEMILQALGKSYHPGCFRCSVCNECLDGVPFTVDVENNIYCVRDYHTVFAPKCASCARPIL
590	PRP19_HUMAN	HPSQDLVFSASPDATIRIWSVPNASCVQVVRAHESAVTGLSLHATGDYLLSSSDDQYWAFSDIQTGRVLTKVTDETSGCS
591	CBX6_HUMAN	ELSAVGERVFAAESIIKRRIRKGRIEYLVKWKGWAIKYSTWEPEENILDSRLIAAFEQKERERELYGPKKRGPKPKTFLL
592	NKX11_HUMAN	RTGSDSKSGKPRRARTAFTYEQLVALENKFKATRYLSVCERLNLALSLSLTETQVKIWFQNRRTKWKKQNPGADTSAPTG
593	RBBP4_HUMAN	VWDLSKIGEEQSPEDAEDGPPELLFIHGGHTAKISDFSWNPNEPWVICSVSEDNIMQVWQMAENIYNDEDPEGSVDPEGQ
594	DMRT2_HUMAN	ERCTPAGGGAEPRKLSRTPKCARCRNHGVVSCLKGHKRFCRWRDCQCANCLLVVERQRVMAAQVALRRQQATEDKKGLSG
595	SMCA2_HUMAN	SQPGALIPGDPQAMSQPNRGPSPFSPVQLHQLRAQILAYKMLARGQPLPETLQLAVQGKRTLPGLQQQQQQQQQQQQQQ
596	ZNF10	MDAKSLTAWSRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLGYQLTKPDVILRLEKGEEPWLVEREIHQETHPDSETAFEIKSSVSSRSIFKDKQSCDIKMEGMARNDLWYLSLEEVWKCRDQLDKYQENPERHLRQVAFTQKKVLTQERVSESGKYGGNCLLPAQLVLREYFHKRDSHTKSLKHDLVLNGHQDSCASNSNECGQTFCQNIHLIQFARTHTGDKSYKCPDNDNSLTHGSSLGISKGIHREKPYECKECGKFFSWRSNLTRHQLIHTGEKPYECKECGKSFSRSSHLIGHQKTHTGEEPYECKECGKSFSWFSHLVTHQRTHTGDKLYTCNQCGKS

SEQ ID NO	Description	Sequence
		FVHSSRLIRHQRTHTGEKPYECPECGKSFRQSTHLILHQRTHVRVRPYECNE CGKSYSQRSHLVVHHRIHTGLKPFECKDCGKCFSRSSHLYSHQRTHTGEKP YECHDCGKSFSQSSALIVHQRIHTGEKPYECCQCGKAFIRKNDLIKHQRIHV GEETYKCNQCGIIFSQNSPFIVHQIAHTGEQFLTCNQCGTALVNTSNLIGYQT NHIRENAY
597	KAP1	MAASAAAASAAAASAASGSPGPGEGSAGGEKRSTAPSAAASASASAAASSP AGGGAEALELLEHCGVCRERLRPEREPRLLPCLHSACSACLGPAAPAAANS SGDGGAAGDGTVVDCPVCKQQCFSKDIVENYFMRDSGSKAATDAQDANQ CCTSCEDNAPATSYCVECSEPLCETCVEAHQRVKYTKDHTVRSTGPAKSRD GERTVYCNVHKHEPLVLFCESCDTLTCRDCQLNAHKDHQYQFLEDAVRNQ RKLLASLVKRLGDKHATLQKSTKEVRSSIRQVSDVQKRVQVDVKMAILQI MKELNKRGRVLVNDAQKVTEGQQERLERQHWTMTKIQKHQEHILRFASW ALESDNNTALLLSKKLIYFQLHRALKMIVDPVEPHGEMKFQWDLNAWTKS AEAFGKIVAERPGTNSTGPAPMAPPRAPGPLSKQGSGSSQPMEVQEGYGFG SGDDPYSSAEPHVSGVKRSRSGEGEVSGLMRKVPRVSLERLDLDLTADSQP PVFKVFPGSTTEDYNLIVIERGAAAAATGQPGTAPAGTPGAPPLAGMAIVKE EETEAAIGAPPTATEGPETKPVLMALAEGPGAEGPRLASPSGSTSSGLEVVA PEGTSAPGGGPGTLDDSATICRVCQKPGDLVMCNQCEFCFHLDCHLPALQD VPGEEWSCSLCHVLPDLKEEDGSLSLDGADSTGVVAKLSPANQRKCERVLL ALFCHEPCRPLHQLATDSTFSLDQPGGTLDLTLIRARLQEKLSPPYSSPQEFA QDVGRMFKQFNKLTEDKADVQSIIGLQRFFETRMNEAFGDTKFSAVLVEPP PMSLPGAGLSSQELSGGPGDGP
598	MECP2	MVAGMLGLREEKSEDQDLQGLKDKPLKFKKVKKDKKEEKEGKHEPVQPS AHHSAEPAEAGKAETSEGSGSAPAVPEASASPKQRRSIIRDRGPMYDDPTLP EGWTRKLKQRKSGRSAGKYDVYLINPQGKAFRSKVELIAYFEKVGDTSLDP NDFDFTVTGRGSPSRREQKPPKKPKSPKAPGTGRGRGRPKGSGTTRPKAAT SEGVQVKRVLEKSPGKLLVKMPFQTSPGGKAEGGGATTSTQVMVIKRPGR KRKAEADPQAIPKKRGRKPGSVVAAAAAEAKKKAVKESSIRSVQETVLPIK KRKTRETVSIEVKEVVKPLLVSTLGEKSGKGLKTCKSPGRKSKESSPKGRSS SASSPPKKEHHHHHHHSESPKAPVPLLPPLPPPPPEPESSEDPTSPPEPQDLSS SVCKEEKMPRGGSLESDGCPKEPAKTQPAVATAATAAEKYKHRGEGERKD IVSSSMPRPNREEPVDSRTPVTERVS
599	human TET1	MSRSRHARPSRLVRKEDVNKKKKNSQLRKTTKGANKNVASVKTLSPGKLK QLIQERDVKKKTEPKPPVPVRSLLTRAGAARMNLDRTEVLFQNPESLTCNG FTMALRSTSLSRRLSQPPLVVAKSKKVPLSKGLEKQHDCDYKILPALGVKH SENDSVPMQDTQVLPDIETLIGVQNPSLLKGKSQETTQFWSQRVEDSKINIPT HSGPAAEILPGPLEGTRCGEGLFSEETLNDTSGSPKMFAQDTVCAPFPQRAT PKVTSQGNPSIQLEELGSRVESLKLSDSYLDPIKSEHDCYPTSSLNKVIPDLN LRNCLALGGSTSPTSVIKFLLAGSKQATLGAKPDHQEAFEATANQQEVSDT TSFLGQAFGAIPHQWELPGADPVHGEALGETPDLPEIPGAIPVQGEVFGTILD QQETLGMSGSVVPDLPVFLPVPPNPIATFNAPSKWPEPQSTVSYGLAVQGAI QILPLGSGHTPQSSSNSEKNSLPPVMAISNVENEKQVHISFLPANTQGFPLAP ERGLFHASLGIAQLSQAGPSKSDRGSSQVSVTSTVHVVNTTVVTMPVPMVS TSSSSYTTLLPTLEKKKRKRCGVCEPCQQKTNCGECTYCKNRKNSHQICKK RKCEELKKKPSVVVPLEVIKENKRPQREKKPKVLKADFDNKPVNGPKSESM DYSRCGHGEEQKLELNPHTVENVTKNEDSMTGIEVEKWTQNKKSQLTDHV KGDFSANVPEAEKSKNSEVDKKRTKSPKLFVQTVRNGIKHVHCLPAETNVS FKKFNIEEFGKTLENNSYKFLKDTANHKNAMSSVATDMSCDHLKGRSNVL VFQQPGFNCSSIPHSSHSIINHHASIHNEGDQPKTPENIPSKEPKDGSPVQPSL LSLMKDRRLTLEQVVAIEALTQLSEAPSENSSPSKSEKDEESEQRTASLLNSC KAILYTVRKDLQDPNLQGEPPKLNHCPSLEKQSSCNTVVFNGQTTTLSNSHI NSATNQASTKSHEYSKVTNSLSLFIPKSNSSKIDTNKSIAQGIITLDNCSNDLH

SEQ ID NO	Description	Sequence
		QLPPRNNEVEYCNQLLDSSKKLDSDDLSCQDATHTQIEEDVATQLTQLASII KINYIKPEDKKVESTPTSLVTCNVQQKYNQEKGTIQQKPPSSVHNNHGSSLT KQKNPTQKKTKSTPSRDRRKKKPTVVSYQENDRQKWEKLSYMYGTICDIW IASKFQNFGQFCPHDFPTVFGKISSSTKIWKPLAQTRSIMQPKTVFPPLTQIKL QRYPESAEEKVKVEPLDSLSLFHLKTESNGKAFTDKAYNSQVQLTVNANQ KAHPLTQPSSPPNQCANVMAGDDQIRFQQVVKEQLMHQRLPTLPGISHETP LPESALTLRNVNVVCSGGITVVSTKSEEEVCSSSFGTSEFSTVDSAQKNFND YAMNFFTNPTKNLVSITKDSELPTCSCLDRVIQKDKGPYYTHLGAGPSVAA VREIMENRYGQKGNAIRIEIVVYTGKEGKSSHGCPIAKWVLRRSSDEEKVLC LVRQRTGHHCPTAVMVVLIMVWDGIPLPMADRLYTELTENLKSYNGHPTD RRCTLNENRTCTCQGIDPETCGASFSFGCSWSMYFNGCKFGRSPSPRRFRID PSSPLHEKNLEDNLQSLATRLAPIYKQYAPVAYQNQVEYENVARECRLGSK EGRPFSGVTACLDFCAHPHRDIHNMNNGSTVVCTLTREDNRSLGVIPQDEQ LHVLPLYKLSDTDEFGSKEGMEAKIKSGAIEVLAPRRKKRTCFTQPVPRSGK KRAAMMTEVLAHKIRAVEKKPIPRIKRKNNSTTTNNSKPSSLPTLGSNTETV QPEVKSETEPHFILKSSDNTKTYSLMPSAPHPVKEASPGFSWSPKTASATPAP LKNDATASCGFSERSSTPHCTMPSGRLSGANAAAADGPGISQLGEVAPLPTL SAPVMEPLINSEPSTGVTEPLTPHQPNHQPSFLTSPQDLASSPMEEDEQHSEA DEPPSDEPLSDDPLSPAEEKLPHIDEYWSDSEHIFLDANIGGVAIAPAHGSVLI ECARRELHATTPVEHPNRNHPTRLSLVFYQHKNLNKPQHGFELNKIKFEAK EAKNKKMKASEQKDQAANEGPEQSSEVNELNQIPSHKALTLTHDNVVTVS PYALTHVAGPYNHWV
600	human TET2	MEQDRTNHVEGNRLSPFLIPSPPICQTEPLATKLQNGSPLPERAHPEVNGDT KWHSFKSYYGIPCMKGSQNSRVSPDFTQESRGYSKCLQNGGIKRTVSEPSLS GLLQIKKLKQDQKANGERRNFGVSQERNPGESSQPNVSDLSDKKESVSSVA QENAVKDFTSFSTHNCSGPENPELQILNEQEGKSANYHDKNIVLLKNKAVL MPNGATVSASSVEHTHGELLEKTLSQYYPDCVSIAVQKTTSHINAINSQATN ELSCEITHPSHTSGQINSAQTSNSELPPKPAAVVSEACDADDADNASKLAAM LNTCSFQKPEQLQQQKSVFEICPSPAENNIQGTTKLASGEEFCSGSSSNLQAP GGSSERYLKQNEMNGAYFKQSSVFTKDSFSATTTPPPPSQLLLSPPPPLPQVP QLPSEGKSTLNGGVLEEHHHYPNQSNTTLLREVKIEGKPEAPPSQSPNPSTH VCSPSPMLSERPQNNCVNRNDIQTAGTMTVPLCSEKTRPMSEHLKHNPPIFG SSGELQDNCQQLMRNKEQEILKGRDKEQTRDLVPPTQHYLKPGWIELKAPR FHQAESHLKRNEASLPSILQYQPNLSNQMTSKQYTGNSNMPGGLPRQAYTQ KTTQLEHKSQMYQVEMNQGQSQGTVDQHLQFQKPSHQVHFSKTDHLPKA HVQSLCGTRFHFQQRADSQTEKLMSPVLKQHLNQQASETEPFSNSHLLQHK PHKQAAQTQPSQSSHLPQNQQQQQKLQIKNKEEILQTFPHPQSNNDQQREG SFFGQTKVEECFHGENQYSKSSEFETHNVQMGLEEVQNINRRNSPYSQTMK SSACKIQVSCSNNTHLVSENKEQTTHPELFAGNKTQNLHHMQYFPNNVIPK QDLLHRCFQEQEQKSQQASVLQGYKNRNQDMSGQQAAQLAQQRYLIHNH ANVFPVPDQGGSHTQTPPQKDTQKHAALRWHLLQKQEQQQTQQPQTESCH SQMHRPIKVEPGCKPHACMHTAPPENKTWKKVTKQENPPASCDNVQQKSII ETMEQHLKQFHAKSLFDHKALTLKSQKQVKVEMSGPVTVLTRQTTAAELD SHTPALEQQTTSSEKTPTKRTAASVLNNFIESPSKLLDTPIKNLLDTPVKTQY DFPSCRCVEQIIEKDEGPFYTHLGAGPNVAAIREIMEERFGQKGKAIRIERVI YTGKEGKSSQGCPIAKWVVRRSSSEEKLLCLVRERAGHTCEAAVIVILILVW EGIPLSLADKLYSELTETLRKYGTLTNRRCALNEERTCACQGLDPETCGASF SFGCSWSMYYNGCKFARSKIPRKFKLLGDDPKEEEKLESHLQNLSTLMAPT YKKLAPDAYNNQIEYEHRAPECRLGLKEGRPFSGVTACLDFCAHAHRDLH NMQNGSTLVCTLTREDNREFGGKPEDEQLHVLPLYKVSDVDEFGSVEAQE EKKRSGAIQVLSSFRRKVRMLAEPVKTCRQRKLEAKKAAAEKLSSLENSSN KNEKEKSAPSRTKQTENASQAKQLAELLRLSGPVMQQSQQPQPLQKQPPQP

SEQ ID NO	Description	Sequence
		QQQQRPQQQQPHHPQTESVNSYSASGSTNPYMRRPNPVSPYPNSSHTSDIY GSTSPMNFYSTSSQAAGSYLNSSNPMNPYPGLLNQNTQYPSYQCNGNLSVD NCSPYLGSYSPQSQPMDLYRYPSQDPLSKLSLPPIHTLYQPRFGNSQSFTSKY LGYGNQNMQGDGFSSCTIRPNVHHVGKLPPYPTHEMDGHFMGATSRLPPN LSNPNMDYKNGEHHSPSHIIHNYSAAPGMFNSSLHALHLQNKENDMLSHT ANGLSKMLPALNHDRTACVQGGLHKLSDANGQEKQPLALVQGVASGAED NDEVWSDSEQSFLDPDIGGVAVAPTHGSILIECAKRELHATTPLKNPNRNHP TRISLVFYQHKSMNEPKHGLALWEAKMAEKAREKEEECEKYGPDYVPQKS HGKKVKREPAEPHETSEPTYLRFIKSLAERTMSVTTDSTVTTSPYAFTRVTG PYNRYI
601	human TET3	MSQFQVPLAVQPDLPGLYDFPQRQVMVGSFPGSGLSMAGSESQLRGGGDG RKKRKRCGTCEPCRRLENCGACTSCTNRRTHQICKLRKCEVLKKKVGLLKE VEIKAGEGAGPWGQGAAVKTGSELSPVDGPVPGQMDSGPVYHGDSRQLSA SGVPVNGAREPAGPSLLGTGGPWRVDQKPDWEAAPGPAHTARLEDAHDL VAFSAVAEAVSSYGALSTRLYETFNREMSREAGNNSRGPRPGPEGCSAGSE DLDTLQTALALARHGMKPPNCNCDGPECPDYLEWLEGKIKSVVMEGGEER PRLPGPLPPGEAGLPAPSTRPLLSSEVPQISPQEGLPLSQSALSIAKEKNISLQT AIAIEALTQLSSALPQPSHSTPQASCPLPEALSPPAPFRSPQSYLRAPSWPVVP PEEHSSFAPDSSAFPPATPRTEFPEAWGTDTPPATPRSSWPMPRPSPDPMAEL EQLLGSASDYIQSVFKRPEALPTKPKVKVEAPSSSPAPAPSPVLQREAPTPSS EPDTHQKAQTALQQHLHHKRSLFLEQVHDTSFPAPSEPSAPGWWPPPSSPV PRLPDRPPKEKKKKLPTPAGGPVGTEKAAPGIKPSVRKPIQIKKSRPREAQPL FPPVRQIVLEGLRSPASQEVQAHPPAPLPASQGSAVPLPPEPSLALFAPSPSRD SLLPPTQEMRSPSPMTALQPGSTGPLPPADDKLEELIRQFEAEFGDSFGLPGP PSVPIQDPENQQTCLPAPESPFATRSPKQIKIESSGAVTVLSTTCFHSEEGGQE ATPTKAENPLTPTLSGFLESPLKYLDTPTKSLLDTPAKRAQAEFPTCDCVEQI VEKDEGPYYTHLGSGPTVASIRELMEERYGEKGKAIRIEKVIYTGKEGKSSR GCPIAKWVIRRHTLEEKLLCLVRHRAGHHCQNAVIVILILAWEGIPRSLGDT LYQELTDTLRKYGNPTSRRCGLNDDRTCACQGKDPNTCGASFSFGCSWSM YFNGCKYARSKTPRKFRLAGDNPKEEEVLRKSFQDLATEVAPLYKRLAPQA YQNQVTNEEIAIDCRLGLKEGRPFAGVTACMDFCAHAHKDQHNLYNGCTV VCTLTKEDNRCVGKIPEDEQLHVLPLYKMANTDEFGSEENQNAKVGSGAIQ VLTAFPREVRRLPEPAKSCRQRQLEARKAAAEKKKIQKEKLSTPEKIKQEAL ELAGITSDPGLSLKGGLSQQGLKPSLKVEPQNHFSSFKYSGNAVVESYSVLG NCRPSDPYSMNSVYSYHSYYAQPSLTSVNGFHSKYALPSFSYYGFPSSNPVF PSQFLGPGAWGHSGSSGSFEKKPDLHALHNSLSPAYGGAEFAELPSQAVPT DAHHPTPHHQQPAYPGPKEYLLPKAPLLHSVSRDPSPFAQSSNCYNRSIKQE PVDPLTQAEPVPRDAGKMGKTPLSEVSQNGGPSHLWGQYSGGPSMSPKRT NGVGGSWGVFSSGESPAIVPDKLSSFGASCLAPSHFTDGQWGLFPGEGQQA ASHSGGRLRGKPWSPCKFGNSTSALAGPSLTEKPWALGAGDFNSALKGSPG FQDKLWNPMKGEEGRIPAAGASQLDRAWQSFGLPLGSSEKLFGALKSEEKL WDPFSLEEGPAEEPPSKGAVKEEKGGGGAEEEEEELWSDSEHNFLDENIGG VAVAPAHGSILIECARRELHATTPLKKPNRCHPTRISLVFYQHKNLNQPNHG LALWEAKMKQLAERARARQEEAARLGLGQQEAKLYGKKRKWGGTVVAE PQQKEKKGVVPTRQALAVPTDSAVTVSSYAYTKVTGPYSRWI
602	human TDG	MEAENAGSYSLQQAQAFYTFPFQQLMAEAPNMAVVNEQQMPEEVPAPAP AQEPVQEAPKGRKRKPRTTEPKQPVEPKKPVESKKSGKSAKSKEKQEKITD TFKVKRKVDRFNGVSEAELLTKTLPDILTFNLDIVIIGINPGLMAAYKGHHY PGPGNHFWKCLFMSGLSEVQLNHMDDHTLPGKYGIGFTNMVERTTPGSKD LSSKEFREGGRILVQKLQKYQPRIAVFNGKCIYEIFSKEVFGVKVKNLEFGL QPHKIPDTETLCYVMPSSSARCAQFPRAQDKVHYYIKLKDLRDQLKGIERN MDVQEVQYTFDLQLAQEDAKKMAVKEEKYDPGYEAAYGGAYGENPCSSE

SEQ ID NO	Description	Sequence
		PCGFSSNGLIESVELRGESAFSGIPNGQWMTQSFTDQIPSFSNHCGTQEQEEE SHA
603	arabidopsis ROS1	MEKQRREESSFQQPPWIPQTPMKPFSPICPYTVEDQYHSSQLEERRFVGNKD MSGLDHLSFGDLLALANTASLIFSGQTPIPTRNTEVMQKGTEEVESLSSVSN NVAEQILKTPEKPKRKKHRPKVRREAKPKREPKPRAPRKSVVTDGQESKTP KRKYVRKKVEVSKDQDATPVESSAAVETSTRPKRLCRRVLDFEAENGENQ TNGDIREAGEMESALQEKQLDSGNQELKDCLLSAPSTPKRKRSQGKRKGV QPKKNGSNLEEVDISMAQAAKRRQGPTCCDMNLSGIQYDEQCDYQKMHW LYSPNLQQGGMRYDAICSKVFSGQQHNYVSAFHATCYSSTSQLSANRVLTV EERREGIFQGRQESELNVLSDKIDTPIKKKTTGHARFRNLSSMNKLVEVPEH LTSGYCSKPQQNNKILVDTRVTVSKKKPTKSEKSQTKQKNLLPNLCRFPPSF TGLSPDELWKRRNSIETISELLRLLDINREHSETALVPYTMNSQIVLFGGGAG AIVPVTPVKKPRPRPKVDLDDETDRVWKLLLENINSEGVDGSDEQKAKWW EEERNVFRGRADSFIARMHLVQGDRRFTPWKGSVVDSVVGVFLTQNVSDH LSSSAFMSLASQFPVPFVPSSNFDAGTSSMPSIQITYLDSEETMSSPPDHNHSS VTLKNTQPDEEKDYVPSNETSRSSSEIAISAHESVDKTTDSKEYVDSDRKGS SVEVDKTDEKCRVLNLFPSEDSALTCQHSMVSDAPQNTERAGSSSEIDLEGE YRTSFMKLLQGVQVSLEDSNQVSPNMSPGDCSSEIKGFQSMKEPTKSSVDS SEPGCCSQQDGDVLSCQKPTLKEKGKKVLKEEKKAFDWDCLRREAQARA GIREKTRSTMDTVDWKAIRAADVKEVAETIKSRGMNHKLAERIQGFLDRLV NDHGSIDLEWLRDVPPDKAKEYLLSFNGLGLKSVECVRLLTLHHLAFPVDT NVGRIAVRLGWVPLQPLPESLQLHLLEMYPMLESIQKYLWPRLCKLDQKTL YELHYQMITFGKVFCTKSKPNCNACPMKGECRHFASAFASARLALPSTEKG MGTPDKNPLPLHLPEPFQREQGSEVVQHSEPAKKVTCCEPIIEEPASPEPETA EVSIADIEEAFFEDPEEIPTIRLNMDAFTSNLKKIMEHNKELQDGNMSSALVA LTAETASLPMPKLKNISQLRTEHRVYELPDEHPLLAQLEKREPDDPCSYLLA IWTPGETADSIQPSVSTCIFQANGMLCDEETCFSCNSIKETRSQIVRGTILIPCR TAMRGSFPLNGTYFQVNEVFADHASSLNPINVPRELIWELPRRTVYFGTSVP TIFKGLSTEKIQACFWKGYVCVRGFDRKTRGPKPLIARLHFPASKLKGQQA NLA
604	arabidopsis DME	MNSRADPGDRYFRVPLENQTQQEFMGSWIPFTPKKPRSSLMVDERVINQDL NGFPGGEFVDRGFCNTGVDHNGVFDHGAHQGVTNLSMMINSLAGSHAQA WSNSERDLLGRSEVTSPLAPVIRNTTGNVEPVNGNFTSDVGMVNGPFTQSG TSQAGYNEFELDDLLNPDQMPFSFTSLLSGGDSLFKVRQYGPPACNKPLYN LNSPIRREAVGSVCESSFQYVPSTPSLFRTGEKTGFLEQIVTTTGHEIPEPKSD KSMQSIMDSSAVNATEATEQNDGSRQDVLEFDLNKTPQQKPSKRKRKFMP KVVVEGKPKRKPRKPAELPKVVVEGKPKRKPRKAATQEKVKSKETGSAKK KNLKESATKKPANVGDMSNKSPEVTLKSCRKALNFDLENPGDARQGDSES EIVQNSSGANSFSEIRDAIGGTNGSFLDSVSQIDKTNGLGAMNQPLEVSMGN QPDKLSTGAKLARDQQPDLLTRNQQCQFPVATQNTQFPMENQQAWLQMK NQLIGFPFGNQQPRMTIRNQQPCLAMGNQQPMYLIGTPRPALVSGNQQLGG PQGNKRPIFLNHQTCLPAGNQLYGSPTDMHQLVMSTGGQQHGLLIKNQQP GSLIRGQQPCVPLIDQQPATPKGFTHLNQMVATSMSSPGLRPHSQSQVPTTY LHVESVSRILNGTTGTCQRSRAPAYDSLQQDIHQGNKYILSHEISNGNGCKK ALPQNSSLPTPIMAKLEEARGSKRQYHRAMGQTEKHDLNLAQQIAQSQDV ERHNSSTCVEYLDAAKKTKIQKVVQENLHGMPPEVIEIEDDPTDGARKGKN TASISKGASKGNSSPVKKTAEKEKCIVPKTPAKKGRAGRKKSVPPPAHASEI QLWQPTPPKTPLSRSKPKGKGRKSIQDSGKARGPSGELLCQDSIAEIIYRMQ NLYLGDKEREQEQNAMVLYKGDGALVPYESKKRKPRPKVDIDDETTRIWN LLMGKGDEKEGDEEKDKKKEKWWEEERRVFRGRADSFIARMHLVQGDRR FSPWKGSVVDSVIGVFLTQNVSDHLSSSAFMSLAARFPPKLSSSREDERNVR SVVVEDPEGCILNLNEIPSWQEKVQHPSDMEVSGVDSGSKEQLRDCSNSGIE

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		RFNFLEKSIQNLEEEVLSSQDSFDPAIFQSCGRVGSCSCSKSDAEFPTTRCET KTVSGTSQSVQTGSPNLSDEICLQGNERPHLYEGSGDVQKQETTNVAQKKP DLEKTMNWKDSVCFGQPRNDTNWQTTPSSSYEQCATRQPHVLDIEDFGMQ GEGLGYSWMSISPRVDRVKNKNVPRRFFRQGGSVPREFTGQIIPSTPHELPG MGLSGSSSAVQEHQDDTQHNQQDEMNKASHLQKTFLDLLNSSEECLTRQS STKQNITDGCLPRDRTAEDVVDPLSNNSSLQNILVESNSSNKEQTAVEYKET NATILREMKGTLADGKKPTSQWDSLRKDVEGNEGRQERNKNNMDSIDYEA IRRASISEISEAIKERGMNNMLAVRIKDFLERIVKDHGGIDLEWLRESPPDKA KDYLLSIRGLGLKSVECVRLLTLHNLAFPVDTNVGRIAVRMGWVPLQPLPE SLQLHLLELYPVLESIQKFLWPRLCKLDQRTLYELHYQLITFGKVFCTKSRP NCNACPMRGECRHFASAYASARLALPAPEERSLTSATIPVPPESYPPVAIPMI ELPLPLEKSLASGAPSNRENCEPIIEEPASPGQECTEITESDIEDAYYNEDPDEI PTIKLNIEQFGMTLREHMERNMELQEGDMSKALVALHPTTTSIPTPKLKNIS RLRTEHQVYELPDSHRLLDGMDKREPDDPSPYLLAIWTPGETANSAQPPEQ KCGGKASGKMCFDETCSECNSLREANSQTVRGTLLIPCRTAMRGSFPLNGT YFQVNELFADHESSLKPIDVPRDWIWDLPRRTVYFGTSVTSIFRGLSTEQIQF CFWKGFVCVRGFEQKTRAPRPLMARLHFPASKLKNNKT
605	arabidopsis DML2	MEVEGEVREKEARVKGRQPETEVLHGLPQEQSIFNNMQHNHQPDSDRRRL SLENLPGLYNMSCTQLLALANATVATGSSIGASSSSLSSQHPTDSWINSWK MDSNPWTLSKMQKQQYDVSTPQKFLCDLNLTPEELVSTSTQRTEPESPQITL KTPGKSLSETDHEPHDRIKKSVLGTGSPAAVKKRKIARNDEKSQLETPTLKR KKIRPKVVREGKTKKASSKAGIKKSSIAATATKTSEESNYVRPKRLTRRSIRF DFDLQEEDEEFCGIDFTSAGHVEGSSGEENLTDTTLGMFGHVPKGRRGQRR SNGFKKTDNDCLSSMLSLVNTGPGSFMESEEDRPSDSQISLGRQRSIMATRP RNFRSLKKLLQRIIPSKRDRKGCKLPRGLPKLTVASKLQLKVFRKKRSQRNR VASQFNARILDLQWRRQNPTGTSLADIWERSLTIDAITKLFEELDINKEGLCL PHNRETALILYKKSYEEQKAIVKYSKKQKPKVQLDPETSRVWKLLMSSIDC DGVDGSDEEKRKWWEEERNMFHGRANSFIARMRVVQGNRTFSPWKGSVV DSVVGVFLTQNVADHSSSSAYMDLAAEFPVEWNFNKGSCHEEWGSSVTQE TILNLDPRTGVSTPRIRNPTRVIIEEIDDDENDIDAVCSQESSKTSDSSITSADQ SKTMLLDPFNTVLMNEQVDSQMVKGKGHIPYTDDLNDLSQGISMVSSAST HCELNLNEVPPEVELCSHQQDPESTIQTQDQQESTRTEDVKKNRKKPTTSKP KKKSKESAKSTQKKSVDWDSLRKEAESGGRKRERTERTMDTVDWDALRC TDVHKIANIIIKRGMNNMLAERIKAFLNRLVKKHGSIDLEWLRDVPPDKAK EYLLSINGLGLKSVECVRLLSLHQIAFPVDTNVGRIAVRLGWVPLQPLPDEL QMHLLELYPVLESVQKYLWPRLCKLDQKTLYELHYHMITFGKVFCTKVKP NCNACPMKAECRHYSSARASARLALPEPEESDRTSVMIHERRSKRKPVVVN FRPSLFLYQEKEQEAQRSQNCEPIIEEPASPEPEYIEHDIEDYPRDKNNVGTSE DPWENKDVIPTIILNKEAGTSHDLVVNKEAGTSHDLVVLSTYAAAIPRRKLK IKEKLRTEHHVFELPDHHSILEGFERREAEDIVPYLLAIWTPGETVNSIQPPK QRCALFESNNTLCNENKCFQCNKTREEESQTVRGTILIPCRTAMRGGFPLNG TYFQTNEVFADHDSSINPIDVPTELIWDLKRRVAYLGSSVSSICKGLSVEAIK YNFQEGYVCVRGFDRENRKPKSLVKRLHCSHVAIRTKEKTEE
606	arabidopsis DML3	MLTDGSQHTYQNGETKNSKEHERKCDESAHLQDNSQTTHKKKEKKNSKE KHGIKHSESEHLQDDISQRVTGKGRRRNSKGTPKKLRFNRPRILEDGKKPRN PATTRLRTISNKRRKKDIDSEDEVIPELATPTKESFPKRRKNEKIKRSVARTL NFKQEIVLSCLEFDKICGPIFPRGKKRTTTRRRYDFLCFLLPMPVWKKQSRR SKRRKNMVRWARIASSSKLLEETLPLIVSHPTINGQADASLHIDDTLVRHVV SKQTKKSANNVIEHLNRQITYQKDHGLSSLADVPLHIEDTLIKSASSVLSERP IKKTKDIAKLIKDMGRLKINKKVTTMIKADKKLVTAKVNLDPETIKEWDVL MVNDSPSRSYDDKETEAKWKKEREIFQTRIDLFINRMHRLQGNRKFKQWK GSVVDSVVGVFLTQNTTDYLSSNAFMSVAAKFPVDAREGLSYYIEEPQDAK

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		SSECIILSDESISKVEDHENTAKRKNEKTGIIEDEIVDWNNLRRMYTKEGSRP EMHMDSVNWSDVRLSGQNVLETTIKKRGQFRILSERILKFLNDEVNQNGNI DLEWLRNAPSHLVKRYLLEIEGIGLKSAECVRLLGLKHHAFPVDTNVGRIA VRLGLVPLEPLPNGVQMHQLFEYPSMDSIQKYLWPRLCKLPQETLYELHYQ MITFGKVFCTKTIPNCNACPMKSECKYFASAYVSSKVLLESPEEKMHEPNTF MNAHSQDVAVDMTSNINLVEECVSSGCSDQAICYKPLVEFPSSPRAEIPEST DIEDVPFMNLYQSYASVPKIDFDLDALKKSVEDALVISGRMSSSDEEISKAL VIPTPENACIPIKPPRKMKYYNRLRTEHVVYVLPDNHELLHDFERRKLDDPS PYLLAIWQPGETSSSFVPPKKKCSSDGSKLCKIKNCSYCWTIREQNSNIFRGT ILIPCRTAMRGAFPLNGTYFQTNEVFADHETSLNPIVFRRELCKGLEKRALY CGSTVTSIFKLLDTRRIELCFWTGFLCLRAFDRKQRDPKELVRRLHTPPDER GPKFMSDDDI
607	Herpes strain 17 VP16	MDLLVDELFADMNADGASPPPPRPAGGPKNTPAAPPLYATGRLSQAQLMP SPPMPVPPAALFNRLLDDLGFSAGPALCTMLDTWNEDLFSALPTNADLYRE CKFLSTLPSDVVEWGDAYVPERTQIDIRAHGDVAFPTLPATRDGLGLYYEA LSRFFHAELRAREESYRTVLANFCSALYRYLRASVRQLHRQAHMRGRDRD LGEMLRATIADRYYRETARLARVLFLHLYLFLTREILWAAYAEQMMRPDL FDCLCCDLESWRQLAGLFQPFMFVNGALTVRGVPIEARRLRELNHIREHLN LPLVRSAATEEPGAPLTTPPTLHGNQARASGYFMVLIRAKLDSYSSFTTSPSE AVMREHAYSRARTKNNYGSTIEGLLDLPDDDAPEEAGLAAPRLSFLPAGHT RRLSTAPPTDVSLGDELHLDGEDVAMAHADALDDFDLDMLGDGDSPGPGF TPHDSAPYGALDMADFEFEQMFTDALGIDEYGG
608	Herpes strain 17 VP64	DALDDFDLDMLGSDALDDFDLDMLGSDALDDFDLDMLGSDALDDFDLDM
609	Herpes strain 17 VP160	DALDDFDLDMLGSDALDDFDLDMLGSDALDDFDLDMLGSDALDDFDLDM LGSDALDDFDLDMLGSSDALDDFDLDMLGSDALDDFDLDMLGSDALDDF DLDMLGSDALDDFDLDMLGSDALDDFDLDML
610	human HIF1alpha	MEGAGGANDKKKISSERRKEKSRDAARSRRSKESEVFYELAHQLPLPHNVS SHLDKASVMRLTISYLRVRKLLDAGDLDIEDDMKAQMNCFYLKALDGFV MVLTDDGDMIYISDNVNKYMGLTQFELTGHSVFDFTHPCDHEEMREMLTH RNGLVKKGKEQNTQRSFFLRMKCTLTSRGRTMNIKSATWKVLHCTGHIHV YDTNSNQPQCGYKKPPMTCLVLICEPIPHPSNIEIPLDSKTFLSRHSLDMKFS YCDERITELMGYEPEELLGRSIYEYYHALDSDHLTKTHHDMFTKGQVTTGQ YRMLAKRGGYVWVETQATVIYNTKNSQPQCIVCVNYVVSGIIQHDLIFSLQ QTECVLKPVESSDMKMTQLFTKVESEDTSSLFDKLKKEPDALTLLAPAAGD TIISLDFGSNDTETDDQQLEEVPLYNDVMLPSPNEKLQNINLAMSPLPTAETP KPLRSSADPALNQEVALKLEPNPESLELSFTMPQIQDQTPSPSDGSTRQSSPE PNSPSEYCFYVDSDMVNEFKLELVEKLFAEDTEAKNPFSTQDTDLDLEMLA PYIPMDDDFQLRSFDQLSPLESSSASPESASPQSTVTVFQQTQIQEPTANATT TTATTDELKTVTKDRMEDIKILIASPSPTHIHKETTSATSSPYRDTQSRTASPN RAGKGVIEQTEKSHPRSPNVLSVALSQRTTVPEEELNPKILALQNAQRKRK MEHDGSLFQAVGIGTLLQQPDDHAATTSLSWKRVKGCKSSEQNGMEQKTII LIPSDLACRLLGQSMDESGLPQLTSYDCEVNAPIQGSRNLLQGEELLRALDQ VN
611	human CITED2	MADHMMAMNHGRFPDGTNGLHHHPAHRMGMGQFPSPHHHQQQQPQHA FNALMGEHIHYGAGNMNATSGIRHAMGPGTVNGGHPPSALAPAARFNNSQ FMGPPVASQGGSLPASMQLQKLNNQYFNHHPYPHNHYMPDLHPAAGHQM NGTNQHFRDCNPKHSGGSSTPGGSGGSSTPGGSGSSSGGGAGSSNSGGGSG SGNMPASVAHVPAAMLPPNVIDTDFIDEEVLMSLVIEMGLDRIKELPELWL GQNEFDFMTDFVCKQQPSRVSC

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612	human Stat3	MAQWNQLQQLDTRYLEQLHQLYSDSFPMELRQFLAPWIESQDWAYAASK ESHATLVFHNLLGEIDQQYSRFLQESNVLYQHNLRRIKQFLQSRYLEKPMEI ARIVARCLWEESRLLQTAATAAQQGGQANHPTAAVVTEKQQMLEQHLQD VRKRVQDLEQKMKVVENLQDDFDFNYKTLKSQGDMQDLNGNNQSVTRQ KMQQLEQMLTALDQMRRSIVSELAGLLSAMEYVQKTLTDEELADWKRRQ QIACIGGPPNICLDRLENWITSLAESQLQTRQQIKKLEELQQKVSYKGDPIVQ HRPMLEERIVELFRNLMKSAFVVERQPCMPMHPDRPLVIKTGVQFTTKVRL LVKFPELNYQLKIKVCIDKDSGDVAALRGSRKFNILGTNTKVMNMEESNNG SLSAEFKHLTLREQRCGNGGRANCDASLIVTEELHLITFETEVYHQGLKIDL ETHSLPVVVISNICQMPNAWASILWYNMLTNNPKNVNFFTKPPIGTWDQVA EVLSWQFSSTTKRGLSIEQLTTLAEKLLGPGVNYSGCQITWAKFCKENMAG KGFSFWVWLDNIIDLVKKYILALWNEGYIMGFISKERERAILSTKPPGTFLLR FSESSKEGGVTFTWVEKDISGKTQIQSVEPYTKQQLNNMSFAEIIMGYKIMD ATNILVSPLVYLYPDIPKEEAFGKYCRPESQEHPEADPGSAAPYLKTKFICVT PTTCSNTIDLPMSPRTLDSLMQFGNNGEGAEPSAGGQFESLTFDMELTSECA TSPM
613	human p65	MDELFPLIFPAEPAQASGPYVEIIEQPKQRGMRFRYKCEGRSAGSIPGERSTD TTKTHPTIKINGYTGPGTVRISLVTKDPPHRPHPHELVGKDCRDGFYEAELC PDRCIHSFQNLGIQCVKKRDLEQAISQRIQTNNNPFQVPIEEQRGDYDLNAV RLCFQVTVRDPSGRPLRLPPVLSHPIFDNRAPNTAELKICRVNRNSGSCLGG DEIFLLCDKVQKEDIEVYFTGPGWEARGSFSQADVHRQVAIVFRTPPYADPS LQAPVRVSMQLRRPSDRELSEPMEFQYLPDTDDRHRIEEKRKRTYETFKSIM KKSPFSGPTDPRPPPRRIAVPSRSSASVPKPAPQPYPFTSSLSTINYDEFPTMV FPSGQISQASALAPAPPQVLPQAPAPAPAPAMVSALAQAPAPVPVLAPGPPQ AVAPPAPKPTQAGEGTLSEALLQLQFDDEDLGALLGNSTDPAVFTDLASVD NSEFQQLLNQGIPVAPHTTEPMLMEYPEAITRLVTGAQRPPDPAPAPLGAPG LPNGLLSGDEDFSSIADMDFSALLSQISS
614	human p53	MEEPQSDPSVEPPLSQETFSDLWKLLPENNVLSPLPSQAMDDLMLSPDDIEQ WFTEDPGPDEAPRMPEAAPPVAPAPAAPTPAAPAPAPSWPLSSSVPSQKTY QGSYGFRLGFLHSGTAKSVTCTYSPALNKMFCQLAKTCPVQLWVDSTPPPG TRVRAMAIYKQSQHMTEVVRRCPHHERCSDSDGLAPPQHLIRVEGNLRVE YLDDRNTFRHSVVVPYEPPEVGSDCTTIHYNYMCNSSCMGGMNRRPILTIIT LEDSSGNLLGRNSFEVRVCACPGRDRRTEEENLRKKGEPHHELPPGSTKRA LPNNTSSSPQPKKKPLDGEYFTLQIRGRERFEMFRELNEALELKDAQAGKEP GGSRAHSSHLKSKKGQSTSRHKKLMFKTEGPDSD
615	human ZNF473	MAEEFVTLKDVGMDFTLGDWEQLGLEQGDTFWDTALDNCQDLFLLDPPR PNLTSHPDGSEDLEPLAGGSPEATSPDVTETKNSPLMEDFFEEGFSQEIIEML SKDGFWNSNFGEACIEDTWLDSLLGDPESLLRSDIATNGESPTECKSHELKR GLSPVSTVSTGEDSMVHNVSEKTLTPAKSKEYRGEFFSYSDHSQQDSVQEG EKPYQCSECGKSFSGSYRLTQHWITHTREKPTVHQECEQGFDRNASLSVYP KTHTGYKFYVCNEYGTTFSQSTYLWHQKTHTGEKPCKSQDSDHPPSHDTQ PGEHQKTHTDSKSYNCNECGKAFTRIFHLTRHQKIHTRKRYECSKCQATFN LRKHLIQHQKTHAAKTTSECQECGKIFRHSSLLIEHQALHAGEEPYKCNERG KSFRHNSTLKIHQRVHSGEKPYKCSECGKAFHRHTHLNEHRRIHTGYRPHK CQECVRSFSRPSHLMRHQAIHTAEKPYSCAECKETFSDNNRLVQHQKMHT VKTPYECQECGERFICGSTLKCHESVHAREKQGFFVSGKILDQNPEQKEKCF KCNKCEKTFSCSKYLTQHERIHTRGVKPFECDQCGKAFGQSTRLIHHQRIHS RVRLYKWGEQGKAISSASLIKLQSFHTKEHPFKCNECGKTFSHSAHLSKHQ LIHAGENPFKCSKCDRVFTQRNYLVQHERTHARKKPLVCNECGKTFRQSSC LSKHQRIHSGEKPYVCDYCGKAFGLSAELVRHQRIHTGEKPYVCQECGKAF TQSSCLSIHRRVHTGEKPYRCGECGKAFAQKANLTQHQRIHTGEKPYSCNV

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		CGKAFVLSAHLNQHLRVHTQETLYQCQRCQKAFRCHSSLSRHQRVHNKQQ YCL
616	human FOXO1	MAEAPQVVEIDPDFEPLPRPRSCTWPLPRPEFSQSNSATSSPAPSGSAAANPD AAAGLPSASAAAVSADFMSNLSLLEESEDFPQAPGSVAAAVAAAAAAAAT GGLCGDFQGPEAGCLHPAPPQPPPPGPLSQHPPVPPAAAGPLAGQPRKSSSS RRNAWGNLSYADLITKAIESSAEKRLTLSQIYEWMVKSVPYFKDKGDSNSS AGWKNSIRHNLSLHSKFIRVQNEGTGKSSWWMLNPEGGKSGKSPRRRAAS MDNNSKFAKSRSRAAKKKASLQSGQEGAGDSPGSQFSKWPASPGSHSNDD FDNWSTFRPRTSSNASTISGRLSPIMTEQDDLGEGDVHSMVYPPSAAKMAS TLPSLSEISNPENMENLLDNLNLLSSPTSLTVSTQSSPGTMMQQTPCYSFAPP NTSLNSPSPNYQKYTYGQSSMSPLPQMPIQTLQDNKSSYGGMSQYNCAPGL LKELLTSDSPPHNDIMTPVDPGVAQPNSRVLGQNVMMGPNSVMSTYGSQA SHNKMMNPSSHTHPGHAQQTSAVNGRPLPHTVSTMPHTSGMNRLTQVKTP VQVPLPHPMQMSALGGYSSVSSCNGYGRMGLLHQEKLPSDLDGMFIERLD CDMESIIRNDLMDGDTLDFNFDNVLPNQSFPHSVKTTTHSWVSG
617	human Myb	MARRPRHSIYSSDEDDEDFEMCDHDYDGLLPKSGKRHLGKTRWTREEDEK LKKLVEQNGTDDWKVIANYLPNRTDVQCQHRWQKVLNPELIKGPWTKEE DQRVIELVQKYGPKRWSVIAKHLKGRIGKQCRERWHNHLNPEVKKTSWTE EEDRIIYQAHKRLGNRWAEIAKLLPGRTDNAIKNHWNSTMRRKVEQEGYL QESSKASQPAVATSFQKNSHLMGFAQAPPTAQLPATGQPTVNNDYSYYHIS EAQNVSSHVPYPVALHVNIVNVPQPAAAAIQRHYNDEDPEKEKRIKELELL LMSTENELKGQQVLPTQNHTCSYPGWHSTTIADHTRPHGDSAPVSCLGEHH STPSLPADPGSLPEESASPARCMIVHQGTILDNVKNLLEFAETLQFIDSFLNTS SNHENSDLEMPSLTSTPLIGHKLTVTTPFHRDQTVKTQKENTVFRTPAIKRSI LESSPRTPTPFKHALAAQEIKYGPLKMLPQTPSHLVEDLQDVIKQESDESGIV AEFQENGPPLLKKIKQEVESPTDKSGNFFCSHHWEGDSLNTQLFTQTSPVAD APNILTSSVLMAPASEDEDNVLKAFTVPKNRSLASPLQATKAQRLFQF
618	human CRTC1	MATSNNPRKFSEKIALHNQKQAEETAAFEEVMKDLSLTRAARLQLQKSQY LQLGPSRGQYYGGSLPNVNQIGSGTMDLPFQTPFQSSGLDTSRTTRHHGLV DRVYRERGRLGSPHRRPLSVDKHGRQADSCPYGTMYLSPPADTSWRRTNS DSALHQSTMTPTQPESFSSGSQDVHQKRVLLLTVPGMEETTSEADKNLSKQ AWDTKKTGSRPKSCEVPGINIFPSADQENTTALIPATHNTGGSLPDLTNIHFP SPLPTPLDPEEPTFPALSSSSSTGNLAANLTHLGIGGAGQGMSTPGSSPQHRP AGVSPLSLSTEARRQQASPTLSPLSPITQAVAMDALSLEQQLPYAFFTQAGS QQPPPQPQPPPPPPPASQQPPPPPPPQAPVRLPPGGPLLPSASLTRGPQPPPLA VTVPSSLPQSPPENPGQPSMGIDIASAPALQQYRTSAGSPANQSPTSPVSNQG FSPGSSPQHTSTLGSVFGDAYYEQQMAARQANALSHQLEQFNMMENAISSS SLYSPGSTLNYSQAAMMGLTGSHGSLPDSQQLGYASHSGIPNIILTVTGESPP SLSKELTSSLAGVGDVSFDSDSQFPLDELKIDPLTLDGLHMLNDPDMVLADP ATEDTFRMDRL
619	human Med9	MASAGVAAGRQAEDVLPPTSDQPLPDTKPLPPPQPPPVPAPQPQQSPAPRPQ SPARAREEENYSFLPLVHNIIKCMDKDSPEVHQDLNALKSKFQEMRKLISTM PGIHLSPEQQQQQLQSLREQVRTKNELLQKYKSLCMFEIPKE
620	human EGR3	MTGKLAEKLPVTMSSLLNQLPDNLYPEEIPSALNLFSGSSDSVVHYNQMAT ENVMDIGLTNEKPNPELSYSGSFQPAPGNKTVTYLGKFAFDSPSNWCQDNII SLMSAGILGVPPASGALSTQTSTASMVQPPQGDVEAMYPALPPYSNCGDLY SEPVSFHDPQGNPGLAYSPQDYQSAKPALDSNLFPMIPDYNLYHHPNDMGS IPEHKPFQGMDPIRVNPPPITPLETIKAFKDKQIHPGFGSLPQPPLTLKPIRPRK YPNRPSKTPLHERPHACPAEGCDRRFSRSDELTRHLRIHTGHKPFQCRICMR SFSRSDHLTTHIRTHTGEKPFACEFCGRKFARSDERKRHAKIHLKQKEKKAE KGGAPSASSAPPVSLAPVVTTCA

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621	human SMARCA2	MSTPTDPGAMPHPGPSPGPGPSPGPILGPSPGPGPSPGSVHSMMGPSPGPPSV SHPMPTMGSTDFPQEGMHQMHKPIDGIHDKGIVEDIHCGSMKGTGMRPPHP GMGPPQSPMDQHSQGYMSPHPSPLGAPEHVSSPMSGGGPTPPQMPPSQPGA LIPGDPQAMSQPNRGPSPFSPVQLHQLRAQILAYKMLARGQPLPETLQLAV QGKRTLPGLQQQQQQQQQQQQQQQQQQQQQQPQQQPPQPQTQQQQQP ALVNYNRPSGPGPELSGPSTPQKLPVPAPGGRPSPAPPAAAQPPAAAVPGPS VPQPAPGQPSPVLQLQQKQSRISPIQKPQGLDPVEILQEREYRLQARIAHRIQ ELENLPGSLPPDLRTKATVELKALRLLNFQRQLRQEVVACMRRDTTLETAL NSKAYKRSKRQTLREARMTEKLEKQQKIEQERKRRQKHQEYLNSILQHAK DFKEYHRSVAGKIQKLSKAVATWHANTEREQKKETERIEKERMRRLMAED EEGYRKLIDQKKDRRLAYLLQQTDEYVANLTNLVWEHKQAQAAKEKKKR RRRKKKAEENAEGGESALGPDGEPIDESSQMSDLPVKVTHTETGKVLFGPE APKASQLDAWLEMNPGYEVAPRSDSEESDSDYEEEDEEEESSRQETEEKILL DPNSEEVSEKDAKQIIETAKQDVDDEYSMQYSARGSQSYYTVAHAISERVE KQSALLINGTLKHYQLQGLEWMVSLYNNNLNGILADEMGLGKTIQTIALIT YLMEHKRLNGPYLIIVPLSTLSNWTYEFDKWAPSVVKISYKGTPAMRRSLV PQLRSGKFNVLLTTYEYIIKDKHILAKIRWKYMIVDEGHRMKNHHCKLTQV LNTHYVAPRRILLTGTPLQNKLPELWALLNFLLPTIFKSCSTFEQWFNAPFA MTGERVDLNEEETILIIRRLHKVLRPFLLRRLKKEVESQLPEKVEYVIKCDM SALQKILYRHMQAKGILLTDGSEKDKKGKGGAKTLMNTIMQLRKICNHPY MFQHIEESFAEHLGYSNGVINGAELYRASGKFELLDRILPKLRATNHRVLLF CQMTSLMTIMEDYFAFRNFLYLRLDGTTKSEDRAALLKKFNEPGSQYFIFLL STRAGGLGLNLQAADTVVIFDSDWNPHQDLQAQDRAHRIGQQNEVRVLRL CTVNSVEEKILAAAKYKLNVDQKVIQAGMFDQKSSSHERRAFLQAILEHEE ENEEEDEVPDDETLNQMIARREEEFDLFMRMDMDRRREDARNPKRKPRLM EEDELPSWIIKDDAEVERLTCEEEEEKIFGRGSRQRRDVDYSDALTEKQWLR AIEDGNLEEMEEEVRLKKRKRRRNVDKDPAKEDVEKAKKRRGRPPAEKLS PNPPKLTKQMNAIIDTVINYKDRCNVEKVPSNSQLEIEGNSSGRQLSEVFIQL PSRKELPEYYELIRKPVDFKKIKERIRNHKYRSLGDLEKDVMLLCHNAQTFN LEGSQIYEDSIVLQSVFKSARQKIAKEEESEDESNEEEEEDEEESESEAKSV KVKIKLNKKDDKGRDKGKGKKRPNRGKAKPVVSDFDSDEEQDEREQSEGS GTDDE
622	human Dpy30	MEPEQMLEGQTQVAENPHSEYGLTDNVERIVENEKINAEKSSKQKVDLQSL PTRAYLDQTVVPILLQGLAVLAKERPPNPIEFLASYLLKNKAQFEDRN
623	human NCOA3	MSGLGENLDPLASDSRKRKLPCDTPGQGLTCSGEKRRREQESKYIEELAELI SANLSDIDNFNVKPDKCAILKETVRQIRQIKEQGKTISNDDDVQKADVSSTG QGVIDKDSLGPLLLQALDGFLFVVNRDGNIVFVSENVTQYLQYKQEDLVNT SVYNILHEEDRKDFLKNLPKSTVNGVSWTNETQRQKSHTFNCRMLMKTPH DILEDINASPEMRQRYETMQCFALSQPRAMMEEGEDLQSCMICVARRITTG ERTFPSNPESFITRHDLSGKVVNIDTNSLRSSMRPGFEDIIRRCIQRFFSLNDG QSWSQKRHYQEAYLNGHAETPVYRFSLADGTIVTAQTKSKLFRNPVTNDR HGFVSTHFLQREQNGYRPNPNPVGQGIRPPMAGCNSSVGGMSMSPNQGLQ MPSSRAYGLADPSTTGQMSGARYGGSSNIASLTPGPGMQSPSSYQNNNYGL NMSSPPHGSPGLAPNQQNIMISPRNRGSPKIASHQFSPVAGVHSPMASSGNT GNHSFSSSSLSALQAISEGVGTSLLSTLSSPGPKLDNSPNMNITQPSKVSNQD SKSPLGFYCDQNPVESSMCQSNSRDHLSDKESKESSVEGAENQRGPLESKG HKKLLQLLTCSSDDRGHSSLTNSPLDSSCKESSVSVTSPSGVSSSTSGGVSST SNMHGSLLQEKHRILHKLLQNGNSPAEVAKITAEATGKDTSSITSCGDGNV VKQEQLSPKKKENNALLRYLLDRDDPSDALSKELQPQVEGVDNKMSQCTS STIPSSSQEKDPKIKTETSEEGSGDLDNLDAILGDLTSSDFYNNSISSNGSHLG TKQQVFQGTNSLGLKSSQSVQSIRPPYNRAVSLDSPVSVGSSPPVKNISAFP MLPKQPMLGGNPRMMDSQENYGSSMGGPNRNVTVTQTPSSGDWGLPNSK

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		AGRMEPMNSNSMGRPGGDYNTSLPRPALGGSIPTLPLRSNSIPGARPVLQQQ QQMLQMRPGEIPMGMGANPYGQAAASNQLGSWPDGMLSMEQVSHGTQN RPLLRNSLDDLVGPPSNLEGQSDERALLDQLHTLLSNTDATGLEEIDRALGI PELVNQGQALEPKQDAFQGQEAAVMMDQKAGLYGQTYPAQGPPMQGGF HLQGQSPSFNSMMNQMNQQGNFPLQGMHPRANIMRPRTNTPKQLRMQLQ QRLQGQQFLNQSRQALELKMENPTAGGAAVMRPMMQPQVSSQQGFLNAQ MVAQRSRELLSHHFRQQRVAMMMQQQQQQQQQQQQQQQQQQQQQQQQ QQQQQTQAFSPPPNVTASPSMDGLLAGPTMPQAPPQQFPYQPNYGMGQQP DPAFGRVSSPPNAMMSSRMGPSQNPMMQHPQAASIYQSSEMKGWPSGNLA RNSSFSQQQFAHQGNPAVYSMVHMNGSSGHMGQMNMNPMPMSGMPMGP DQKYC
624	human ZFP28	MRGAASASVREPTPLPGRGAPRTKPRAGRGPTVGTPATLALPARGRPRSRN GLASKGQRGAAPTGPGHRALPSRDTALPQERNKKLEAVGTGIEPKAMSQG LVTFGDVAVDFSQEEWEWLNPIQRNLYRKVMLENYRNLASLGLCVSKPDV ISSLEQGKEPWTVKRKMTRAWCPDLKAVWKIKELPLKKDFCEGKLSQAVIT ERLTSYNLEYSLLGEHWDYDALFETQPGLVTIKNLAVDFRQQLHPAQKNFC KNGIWENNSDLGSAGHCVAKPDLVSLLEQEKEPWMVKRELTGSLFSGQRS VHETQELFPKQDSYAEGVTDRTSNTKLDCSSFRENWDSDYVFGRKLAVGQ ETQFRQEPITHNKTLSKERERTYNKSGRWFYLDDSEEKVHNRDSIKNFQKSS VVIKQTGIYAGKKLFKCNECKKTFTQSSSLTVHQRIHTGEKPYKCNECGKA FSDGSSFARHQRCHTGKKPYECIECGKAFIQNTSLIRHWRYYHTGEKPFDCI DCGKAFSDHIGLNQHRRIHTGEKPYKCDVCHKSFRYGSSLTVHQRIHTGEK PYECDVCRKAFSHHASLTQHQRVHSGEKPFKCKECGKAFRQNIHLASHLRI HTGEKPFECAECGKSFSISSQLATHQRIHTGEKPYECKVCSKAFTQKAHLAQ HQKTHTGEKPYECKECGKAFSQTTHLIQHQRVHTGEKPYKCMECGKAFGD NSSCTQHQRLHTGQRPYECIECGKAFKTKSSLICHRRSHTGEKPYECSVCGK AFSHRQSLSVHQRIHSGKKPYECKECRKTFIQIGHLNQHKRVHTGERSYNY KKSRKVFRQTAHLAHHQRIHTGESSTCPSLPSTSNPVDLFPKFLWNPSSLPSP
625	human ZNF496	MPTALCPRVLAPKESEEPRKMRSPPGENPSPQGELPSPESSRRLFRRFRYQEA AGPREALQRLWDLCGGWLRPERHTKEQILELLVLEQFLAILPREIQSWVRA QEPESGEQAVAAVEALEREPGRPWQWLKHCEDPVVIDDGDSPLDQEQEQL PVEPHSDLAKNQDAQPITLAQCLGLPSRPPSQLSGDPVLQDAFLLQEENVRD TQQVTTLQLPPSRVSPFKDMILCFSEEDWSLLDPAQTGFYGEFIIGEDYGVS MPPNDLAAQPDLSQGEENEPRVPELQDLQGKEVPQVSYLDSPSLQPFQVEE RRKREELQVPEFQACPQTVVPQNTYPAGGNPRSLENSLDEEVTIEIVLSSSG DEDSQHGPYCTEELGSPTEKQRSLPASHRSSTEAGGEVQTSKKSYVCPNCG KIFRWRVNFIRHLRSRREQEKPHECSVCGELFSDSEDLDGHLESHEAQKPYR CGACGKSFRLNSHLLSHRRIHLQPDRLQPVEKREQAASEDADKGPKEPLEN GKAKLSFQCCECGKAFQRHDHLARHRSHFHLKDKARPFQCRYCVKSFTQN YDLLRHERLHMKRRSKQALNSY
626	human ZNF597	MASMPPTPEAQGPILFEDLAVYFSQEECVTLHPAQRSLSKDGTKESLEDAAL MGEEGKPEINQQLSLESMELDELALEKYPIAAPLVPYPEKSSEDGVGNPEAK ILSGTPTYKRRVISLLVTIENHTPLVELSEYLGTNTLSEILDSPWEGAKNVYK CPECDQNFSDHSYLVLHQKIHSGEKKHKCGDCGKIFNHRANLRTHRRIHTG EKPYKCAKCSASFRQHSHLSRHMNSHVKEKPYTCSICGRGFMWLPGLAQH QKSHSAENTYESTNCDKHFNEKPNLALPEETFVSGPQYQHTKCMKSFRQSL YPALSEKSHDEDSERCSDDGDNFFSFSKFKPLQCPDCDMTFPCFSELISHQNI HTEERPHKCKTCEESFALDSELACHQKSHMLAEPFKCTVCGKTFKSNLHLIT HKRTHIKNTT
627	human HSF1	MDLPVGPGAAGPSNVPAFLTKLWTLVSDPDTDALICWSPSGNSFHVFDQGQ FAKEVLPKYFKHNNMASFVRQLNMYGFRKVVHIEQGGLVKPERDDTEFQH PCFLRGQEQLLENIKRKVTSVSTLKSEDIKIRQDSVTKLLTDVQLMKGKQEC

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		MDSKLLAMKHENEALWREVASLRQKHAQQQKVVNKLIQFLISLVQSNRIL GVKRKIPLMLNDSGSAHSMPKYSRQFSLEHVHGSGPYSAPSPAYSSSSLYAP DAVASSGPIISDITELAPASPMASPGGSIDERPLSSSPLVRVKEEPPSPPQSPRV EEASPGRPSSVDTLLSPTALIDSILRESEPAPASVTALTDARGHTDTEGRPPSP PPTSTPEKCLSVACLDNLARTPQMSRVARLFPCPSSSPHGQVQPGNELSDHL DAMDSNLDNLQTMLSSHGFSVDTSALLDIQELLSPQEPPRPPEAENSSPDSA GALHSAAAVPAGPRLRGHREQRPAGAV
628	Epstein-barr virus strain B95-8 RTA	MRPKKDGLEDFLRLTPEIKKQLGSLVSDYCNVLNKEFTAGSVEITLRSYKIC KAFINEAKAHGREWGGLMATLNICNFWAILRNNRVRRRAENAGNDACSIA CPIVMRYVLDHLIVVTDRFFIQAPSNRVMIPATIGTAMYKLLKHSRVRAYTY SKVLGVDRAAIMASGKQVVEHLNRMEKEGLLSSKFKAFCKWVFTYPVLEE MFQTMVSSKTGHLTDDVKDVRALIKTLPRASYSSHAGQRSYVSGVLPACLL STKSKAVETPILVSGADRMDEELMGNDGGASHTEARYSESGQFHAFTDELE SLPSPTMPLKPGAQSADCGDSSSSSSDSGNSDTEQSEREEARAEAPRLRAPK SRRTSRPNRGQTPCPSNAAEPEQPWIAAVHQESDERPIFPHPSKPTFLPPVKR KKGLRDSREGMFLPKPEAGSAISDVFEGREVCQPKRIRPFHPPGSPWANRPL PASLAPTPTGPVHEPVGSLTPAPVPQPLDPAPAVTPEASHLLEDPDEETSQAV KALREMADTVIPQKEEAAICGQMDLSHPPPRGHLDELTTTLESMTEDLNLD SPLTPELNEILDTFLNDECLLHAMHISTGLSIFDTSLF
629	ABL1_HUMAN	KENLLAGPSENDPNLFVALYDFVASGDNTLSITKGEKLRVLGYNHNGEWC EAQTKNGQGWVPSNYITPVNSLEKHSWYHG
630	AF9_HUMAN	KSDKQIKNGECDKAYLDELVELHRRLMTLRERHILQQIVNLIEETGHFHITN TTFDFDLCSLDKTTVRKLQSYLETSGTS
631	ANM2_HUMAN	ECSEAGLLQEGVQPEEFVAIADYAATDETQLSFLRGEKILILRQTTADWWW GERAGCCGYIPANHVGKHVDEYDPEDTWQ
632	APBB1_HUMAN	GSPSYGSPEDTDSFWNPNAFETDSDLPAGWMRVQDTSGTYYWHIPTGTTQ WEPPGRASPSQGSSPQEESQLTWTGFAHGE
633	APC16_HUMAN	DLAPPRKALFTYPKGAGEMLEDGSERFLCESVFSYQVASTLKQVKHDQQV ARMEKLAGLVEELEADEWRFKPIEQLLGFT
634	BTK_HUMAN	PEPAAAPVSTSELKKVVALYDYMPMNANDLQLRKGDEYFILEESNLPWWR ARDKNGQEGYIPSNYVTEAEDSIEMYEWYS
635	CACO1_HUMAN	SGGEEANLLLPELGSAFYDMASGFTVGTLSETSTGGPATPTWKECPICKERF PAESDKDALEDHMDGHFFFSTQDPFTFE
636	CRTC2_HUMAN	GPNIILTGDSSPGFSKEIAAALAGVPGFEVSAAGLELGLGLEDELRMEPLGLE GLNMLSDPCALLPDPAVEESFRSDRLQ
637	CRTC3_HUMAN	NCGSLPNTILPEDSSTSLFKDLNSALAGLPEVSLNVDTPFPLEEELQIEPLSLD GLNMLSDSSMGLLDPSVEETFRADRL
638	CXXC1_HUMAN	AGEDSKSENGENAPIYCICRKPDINCFMIGCDNCNEWFHGDCIRITEKMAKA IREWYCRECREKDPKLEIRYRHKKSRER
639	DPF1_HUMAN	PLSLGEDFYREAIEHCRSYNARLCAERSLRLPFLDSQTGVAQNNCYIWMEK THRGPGLAPGQIYTYPARCWRKKRRLNIL
640	DPY30_HUMAN	EYGLTDNVERIVENEKINAEKSSKQKVDLQSLPTRAYLDQTVVPILLQGLAV LAKERPPNPIEFLASYLLKNKAQFEDRN
641	EGR3_HUMAN	TVTYLGKFAFDSPSNWCQDNIISLMSAGILGVPPASGALSTQTSTASMVQPP QGDVEAMYPALPPYSNCGDLYSEPVSFH
642	ENL_HUMAN	SKPEKILKKGTYDKAYTDELVELHRRLMALRERNVLQQIVNLIEETGHFNV TNTTFDFDLFSLDETTVRKLQSCLEAVAT
643	FIGN_HUMAN	LLVQRTEGFSGLDVAHLCQEAVVGPLHAMPATDLSAIMPSQLRPVTYQDFE NAFCKIQPSISQKELDMYVEWNKMFGCSQ
644	FOXO1_HUMAN	GGYSSVSSCNGYGRMGLLHQEKLPSDLDGMFIERLDCDMESIIRNDLMDGD TLDFNFDNVLPNQSFPHSVKTTTHSWVSG

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645	FOXO3_HUMAN	DSLSGSSLYSTSANLPVMGHEKFPSDLDLDMFNGSLECDMESIIRSELMDADGLDFNFDSLISTQNVVGLNVGNFTGAKQ
646	IKKA_HUMAN	LVGSSLEGAVTPQTSAWLPPTSAEHDHSLSCVVTPQDGETSAQMIEENLNCLGHLSTIIHEANEEQGNSMMNLDWSWLTE
647	IMA5_HUMAN	RLGEQEAKRNGTGINPYCALIEEAYGLDKIEFLQSHENQEIYQKAFDLIEHYFGTEDEDSSIAPQVDLNQQQYIFQQCEA
648	ITCH_HUMAN	SGLIIPLTISGGSGPRPLNPVTQAPLPPGWEQRVDQHGRVYYVDHVEKRTTWDRPEPLPPGWERRVDNMGRIYYVDHFTR
649	KIBRA_HUMAN	PRPELPLPEGWEEARDFDGKVYYIDHTNRTTSWIDPRDRYTKPLTFADCISDELPLGWEEAYDPQVGDYFIDHNTKTTQI
650	KPCI_HUMAN	QGHPFFRNVDWDMMEQKQVVPPFKPNISGEFGLDNFDSQFTNEPVQLTPDDDDIVRKIDQSEFEGFEYINPLLMSAEECV
651	KS6B2_HUMAN	HMNWDDLLAWRVDPPFRPCLQSEEDVSQFDTRFTRQTPVDSPDDTALSESANQAFLGFTYVAPSVLDSIKEGFSFQPKLR
652	MTA3_HUMAN	GAVNGAVGTTFQPQNPLLGRACESCYATQSHQWYSWGPPNMQCRLCAICWLYWKKYGGLKMPTQSEEEKLSPSPTTEDPR
653	MYB_HUMAN	EAQNVSSHVPYPVALHVNIVNVPQPAAAAIQRHYNDEDPEKEKRIKELELLLMSTENELKGQQVLPTQNHTCSYPGWHST
654	MYBA_HUMAN	FYIPVQIPGYQYVSPEGNCIEHVQPTSAFIQQPFIDEDPDKEKKIKELEMLLMSAENEVRRKRIPSQPGSFSSWSGSFLM
655	NCOA2_HUMAN	PFGSSPDDLLCPHPAAESPSDEGALLDQLYLALRNFDGLEEIDRALGIPELVSQSQAVDPEQFSSQDSNIMLEQKAPVFP
656	NCOA3_HUMAN	LRNSLDDLVGPPSNLEGQSDERALLDQLHTLLSNTDATGLEEIDRALGIPELVNQGQALEPKQDAFQGQEAAVMMDQKAG
657	NOTC1_HUMAN	LCHILDYSFGGGAGRDIPPPLIEEACELPECQEDAGNKVCSLQCNNHACGWDGGDCSLNFNDPWKNCTQSLQCWKYFSDG
658	NOTC1_HUMAN	LQCNNHACGWDGGDCSLNFNDPWKNCTQSLQCWKYFSDGHCDSQCNSAGCLFDGFDCQRAEGQCNPLYDQYCKDHFSDGH
659	NOTC2_HUMAN	EACNSHACQWDGGDCSLTMENPWANCSSPLPCWDYINNQCDELCNTVECLFDNFECQGNSKTCKYDKYCADHFKDNHCDQ
660	PRP19_HUMAN	TNKILTGGADKNVVVFDKSSEQILATLKGHTKKVTSVVFHPSQDLVFSASPDATIRIWSVPNASCVQVVRAHESAVTGLS
661	PYGO1_HUMAN	RHGHSSSDPVYPCGICTNEVNDDQDAILCEASCQKWFHRICTGMTETAYGLLTAEASAVWGCDTCMADKDVQLMRTRETF
662	PYGO2_HUMAN	SGPQPPPGLVYPCGACRSEVNDDQDAILCEASCQKWFHRECTGMTESAYGLLTTEASAVWACDLCLKTKEIQSVYIREGM
663	SAV1_HUMAN	HASGIGRVAATSLGNLTNHGSEDLPLPPGWSVDWTMRGRKYYIDHNTNTTHWSHPLEREGLPPGWERVESSEFGTYYVDH
664	SMCA2_HUMAN	SQPGALIPGDPQAMSQPNRGPSPFSPVQLHQLRAQILAYKMLARGQPLPETLQLAVQGKRTLPGLQQQQQQQQQQQQQQ
665	SMRC2_HUMAN	MYTKKNVPSKSKAAASATREWTEQETLLLLEALEMYKDDWNKVSEHVGSRTQDECILHFLRLPIEDPYLEDSEASLGPLA
666	STAT2_HUMAN	SQTVPEPDQGPVSQPVPEPDLPCDLRHLNTEPMEIFRNCVKIEEIMPNGDPLLAGQNTVDEVYVSRPSHFYTDGPLMPSD
667	T2EB_HUMAN	SSGYKFGVLAKIVNYMKTRHQRGDTHPLTLDEILDETQHLDIGLKQKQWLMTEALVNNPKIEVIDGKYAFKPKYNVRDKK
668	U2AF4_HUMAN	VEVQEHYDSFFEEVFTELQEKYGEIEEMNVCDNLGDHLVGNVYVKFRREEDGERAVAELSNRWFNGQAVHGELSPVTDFR
669	WBP4_HUMAN	YYDLISGASQWEKPEGFQGDLKKTAVKTVWVEGLSEDGFTYYYNTETGESRWEKPDDFIPHTSDLPSSKVNENSLGTLDE

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670	WWP1_HUMAN	AMQQFNQRYLYSASMLAAENDPYGPLPPGWEKRVDSTDRVYFVNHNTKTTQWEDPRTQGLQNEEPLPEGWEIRYTREGVR
671	WWP2_HUMAN	AMQHFSQRFLYQSSSASTDHDPLGPLPPGWEKRQDNGRVYYVNHNTRTTQWEDPRTQGMIQEPALPPGWEMKYTSEGVRY
672	WWTR1_HUMAN	GAAGSPAQQHAHLRQQSYDVTDELPLPPGWEMTFTATGQRYFLNHIEKITTWQDPRKAMNQPLNHMNLHPAVSSTPVPQR
673	ZFP28_HUMAN	LEYSLLGEHWDYDALFETQPGLVTIKNLAVDFRQQLHPAQKNFCKNGIWENNSDLGSAGHCVAKPDLVSLLEQEKEPWMV
674	ZN473_HUMAN	AEEFVTLKDVGMDFTLGDWEQLGLEQGDTFWDTALDNCQDLFLLDPPRPNLTSHPDGSEDLEPLAGGSPEATSPDVTETK
675	ZN496_HUMAN	QEENVRDTQQVTTLQLPPSRVSPFKDMILCFSEEDWSLLDPAQTGFYGEFIIGEDYGVSMPPNDLAAQPDLSQGEENEPR
676	ZN597_HUMAN	ASMPPTPEAQGPILFEDLAVYFSQEECVTLHPAQRSLSKDGTKESLEDAALMGEEGKPEINQQLSLESMELDELALEKYP
677	p300	MAENVVEPGPPSAKRPKLSSPALSASASDGTDFGSLFDLEHDLPDELINSTELGLTNGGDINQLQTSLGMVQDAASKHKQLSELLRSGSSPNLNMGVGGPGQVMASQAQQSSPGLGLINSMVKSPMTQAGLTSPNMGMGTSGPNQGPTQSTGMMNSPVNQPAMGMNTGMNAGMNPGMLAAGNGQGIMPNQVMNGSIGAGRGRQNMQYPNPGMGSAGNLLTEPLQQGSPQMGGQTGLRGPQPLKMGMMNNPNPYGSPYTQNPGQQIGASGLGLQIQTKTVLSNNLSPFAMDKKAVPGGGMPNMGQQPAPQVQQPGLVTPVAQGMGSGAHTADPEKRKLIQQQLVLLLHAHKCQRREQANGEVRQCNLPHCRTMKNVLNHMTHCQSGKSCQVAHCASSRQIISHWKNCTRHDCPVCLPLKNAGDKRNQQPILTGAPVGLGNPSSLGVGQQSAPNLSTVSQIDPSSIERAYAALGLPYQVNQMPTQPQVQAKNQQNQQPGQSPQGMRPMSNMSASPMGVNGGVGVQTPSLLSDSMLHSAINSQNPMMSENASVPSLGPMPTAAQPSTTGIRKQWHEDITQDLRNHLVHKLVQAIFPTPDPAALKDRRMENLVAYARKVEGDMYESANNRAEYYHLLAEKIYKIQKELEEKRRTRLQKQNMLPNAAGMVPVSMNPGPNMGQPQPGMTSNGPLPDPSMIRGSVPNQMMPRITPQSGLNQFGQMSMAQPPIVPRQTPPLQHHGQLAQPGALNPPMGYGPRMQQPSNQGQFLPQTQFPSQGMNVTNIPLAPSSGQAPVSQAQMSSSSCPVNSPIMPPGSQGSHIHCPQLPQPALHQNSPSPVPSRTPTPHHTPPSIGAQQPPATTIPAPVPTPPAMPPGPQSQALHPPPRQTPTPPTTQLPQQVQPSLPAAPSADQPQQQPRSQQSTAASVPTPTAPLLPPQPATPLSQPAVSIEGQVSNPPSTSSTEVNSQAIAEKQPSQEVKMEAKMEVDQPEPADTQPEDISESKVEDCKMESTETEERSTELKTEIKEEEDQPSTSATQSSPAPGQSKKKIFKPEELRQALMPTLEALYRQDPESLPFRQPVDPQLLGIPDYFDIVKSPMDLSTIKRKLDTGQYQEPWQYVDDIWLMFNNAWLYNRKTSRVYKYCSKLSEVFEQEIDPVMQSLGYCCGRKLEFSPQTLCCYGKQLCTIPRDATYYSYQNRYHFCEKCFNEIQGESVSLGDDPSQPQTTINKEQFSKRKNDTLDPELFVECTECGRKMHQICVLHHEIIWPAGFVCDGCLKKSARTRKENKFSAKRLPSTRLGTFLENRVNDFLRRQNHPESGEVTVRVVHASDKTVEVKPGMKARFVDSGEMAESFPYRTKALFAFEEIDGVDLCFFGMHVQEYGSDCPPPNQRRVYISYLDSVHFFRPKCLRTAVYHEILIGYLEYVKKLGYTTGHIWACPPSEGDDYIFHCHPPDQKIPKPKRLQEWYKKMLDKAVSERIVHDYKDIFKQATEDRLTSAKELPYFEGDFWPNVLEESIKELEQEEEERKREENTSNESTDVTKGDSKNAKKKNNKKTSKNKSSLSRGNKKKPGMPNVSNDLSQKLYATMEKHKEVFFVIRLIAGPAANSLPPIVDPDPLIPCDLMDGRDAFLTLARDKHLEFSSLRRAQWSTMCMLVELHTQSQDRFVYTCNECKHHVETRWHCTVCEDYDLCITCYNTKNHDHKMEKLGLGLDDESNNQQAAATQSPGDSRRLSIQRCIQSLVHACQCRNANCSLPSCQKMKRVVQHTKGCKRKTNGGCPICKQLIALCCYHAKHCQENKCPVPFCLNIKQKLRQQQLQHRLQQAQMLRRRMASMQRTGVVGQQQGLPSPTPATPTTPTGQQPTTPQTPQPTSQPQPTPPNSMPPYLPRTQAAGPVSQGKAAGQVTPPTPPQTAQPPLPGPPPAAVEMAM

SEQ ID NO	Description	Sequence
		QIQRAAETQRQMAHVQIFQRPIQHQMPPMTPMAPMGMNPPPMTRGPSGHL EPGMGPTGMQQQPPWSQGGLPQPQQLQSGMPRPAMMSVAQHGQPLNMA PQPGLGQVGISPLKPGTVSQQALQNLLRTLRSPSSPLQQQQVLSILHANPQLL AAFIKQRAAKYANSNPQPIPGQPGMPQGQPGLQPPTMPGQQGVHSNPAMQ NMNPMQAGVQRAGLPQQQPQQQLQPPMGGMSPQAQQMNMNHNTMPSQF RDILRRQQMMQQQQQQGAGPGIGPGMANHNQFQQPQGVGYPPQQQQRM QHHMQQMQQGNMGQIGQLPQALGAEAGASLQAYQQRLLQQQMGSPVQP NPMSPQQHMLPNQAQSPHLQGQQIPNSLSNQVRSPQPVPSPRPQSQPPHSSP SPRMQPQPSPHHVSPQTSSPHPGLVAAQANPMEQGHFASPDQNSMLSQLAS NPGMANLHGASATDLGLSTDNSDLNSNLSQSTLDIH
678	CREBBP	MAENLLDGPPNPKRAKLSSPGFSANDSTDFGSLFDLENDLPDELIPNGGELG LLNSGNLVPDAASKHKQLSELLRGGSGSSINPGIGNVSASSPVQQGLGGQA QGQPNSANMASLSAMGKSPLSQGDSSAPSLPKQAASTSGPTPAASQALNPQ AQKQVGLATSSPATSQTGPGICMNANFNQTHPGLLNSNSGHSLINQASQGQ AQVMNGSLGAAGRGRGAGMPYPTPAMQGASSSVLAETLTQVSPQMTGHA GLNTAQAGGMAKMGITGNTSPFGQPFSQAGGQPMGATGVNPQLASKQSM VNSLPTFPTDIKNTSVTNVPNMSQMQTSVGIVPTQAIATGPTADPEKRKLIQ QQLVLLLHAHKCQRREQANGEVRACSLPHCRTMKNVLNHMTHCQAGKAC QVAHCASSRQIISHWKNCTRHDCPVCLPLKNASDKRNQQTILGSPASGIQNT IGSVGTGQQNATSLSNPNPIDPSSMQRAYAALGLPYMNQPQTQLQPQVPGQ QPAQPQTHQQMRTLNPLGNNPMNIPAGGITTDQQPPNLISESALPTSLGATN PLMNDGSNSGNIGTLSTIPTAAPPSSTGVRKGWHEHVTQDLRSHLVHKLVQ AIFPTPDPAALKDRRMENLVAYAKKVEGDMYESANSRDEYYHLLAEKIYKI QKELEEKRRSRLHKQGILGNQPALPAPGAQPPVIPQAQPVRPPNGPLSLPVN RMQVSQGMNSFNPMSLGNVQLPQAPMGPRAASPMNHSVQMNSMGSVPG MAISPSRMPQPPNMMGAHTNNMMAQAPAQSQFLPQNQFPSSSGAMSVGM GQPPAQTGVSQGQVPGAALPNPLNMLGPQASQLPCPPVTQSPLHPTPPPAST AAGMPSLQHTTPPGMTPPQPAAPTQPSTPVSSSGQTPTPTPGSVPSATQTQST PTVQAAAQAQVTPQPQTPVQPPSVATPQSSQQQPTPVHAQPPGTPLSQAAA SIDNRVPTPSSVASAETNSQQPGPDVPVLEMKTETQAEDTEPDPGESKGEPR SEMMEEDLQGASQVKEETDIAEQKSEPMEVDEKKPEVKVEVKEEEESSSNG TASQSTSPSQPRKKIFKPEELRQALMPTLEALYRQDPESLPFRQPVDPQLLGI PDYFDIVKNPMDLSTIKRKLDTGQYQEPWQYVDDVWLMFNNAWLYNRKT SRVYKFCSKLAEVFEQEIDPVMQSLGYCCGRKYEFSPQTLCCYGKQLCTIPR DAAYYSYQNRYHFCEKCFTEIQGENVTLGDDPSQPQTTISKDQFEKKKNDT LDPEPFVDCKECGRKMHQICVLHYDIIWPSGFVCDNCLKKTGRPRKENKFS AKRLQTTRLGNHLEDRVNKFLRRQNHPEAGEVFVRVVASSDKTVEVKPGM KSRFVDSGEMSESFPYRTKALFAFEEIDGVDVCFFGMHVQEYGSDCPPPNT RRVYISYLDSIHFFRPRCLRTAVYHEILIGYLEYVKKLGYVTGHIWACPPSEG DDYIFHCHPPDQKIPKPKRLQEWYKKMLDKAFAERIIHDYKDIFKQATEDR LTSAKELPYFEGDFWPNVLEESIKELEQEEEERKKEESTAASETTEGSQGDS KNAKKKNNKKTNKNKSSISRANKKKPSMPNVSNDLSQKLYATMEKHKEV FFVIHLHAGPVINTLPPIVDPDPLLSCDLMDGRDAFLTLARDKHWEFSSLRR SKWSTLCMLVELHTQGQDRFVYTCNECKHHVETRWHCTVCEDYDLCINC YNTKSHAHKMVKWGLGLDDEGSSQGEPQSKSPQESRRLSIQRCIQSLVHAC QCRNANCSLPSCQKMKRVVQHTKGCKRKTNGGCPVCKQLIALCCYHAKH CQENKCPVPFCLNIKHKLRQQQIQHRLQQAQLMRRRMATMNTRNVPQQSL PSPTSAPPGTPTQQPSTPQTPQPPAQPQPSPVSMSPAGFPSVARTQPPTTVSTG KPTSQVPAPPPPAQPPPAAVEAARQIEREAQQQQHLYRVNINNSMPPGRTG MGTPGSQMAPVSLNVPRPNQVSGPVMPSMPPGQWQQAPLPQQQPMPGLPR PVISMQAQAAVAGPRMPSVQPPRSISPSALQDLLRTLKSPSSPQQQQQVLNI LKSNPQLMAAFIKQRTAKYVANQPGMQPQPGLQSQPGMQPQPGMHQQPSL

SEQ ID NO	Description	Sequence
		QNLNAMQAGVPRPGVPPQQQAMGGLNPQGQALNIMNPGHNPNMASMNP QYREMLRRQLLQQQQQQQQQQQQQQQQQQGSAGMAGGMAGHGQFQQP QGPGGYPPAMQQQQRMQQHLPLQGSSMGQMAAQMGQLGQMGQPGLGA DSTPNIQQALQQRILQQQQMKQQIGSPGQPNPMSPQQHMLSGQPQASHLPG QQIATSLSNQVRSPAPVQSPRPQSQPPHSSPSPRIQPQPSPHHVSPQTGSPHPG LAVTMASSIDQGHLGNPEQSAMLPQLNTPSRSALSSELSLVGDTTGDTLEKF VEGL
679	linker	SGSETPGTSESATPES
680	linker	SGGS
681	linker	SGGSSGSETPGTSESATPESSGGS
682	linker	SGGSSGGSSGSETPGTSESATPESSGGSSGGS
683	linker	GGSGGSPGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTE EGTSTE PSEGSAPGTSTEPSEGSAPGTSESATPESGPGSEPATSGGSGGS
684	XTEN linker	SGSETPGTSESATPES
685	XTEN linker	SGGSSGGSSGSETPGTSESATPES
686	XTEN linker	SGGSSGGSSGSETPGTSESATPES S GGSSGGSSGGSSGGS
687	XTEN linker	SGGSSGGSSGSETPGTSESATPESSGGSSGGSSGGSSGGSSGSETPGTSESATP ESSGGSSGGS
688	XTEN linker	PGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEP SEGSAP GTSTEPSEGSAPGTSESATPESGPGSEPATS
689	NLS	PKKKRKV
690	NLS	AVKRPAATKKAGQAKKKKLD
691	NLS	MSRRRKANPTKLSENAKKLAKEVEN
692	NLS	PAAKRVKLD
693	NLS	KLKIKRPVK
694	NLS	MDSLLMNRRKFLYQFKNVRWAKGRRETYLC
695	overlapping binding sites	GTACGTCGTCTT**TAGGTTGGGGGGAGGGGTT**TTATGCGATGGAGTTTC CCCACACTGA**GTGGGTGGAGACTGAAGTT**AGGCCAGCTTGGCACTTGA TGTAATTCTCCTTGGAATTTGCCCTTTTTGAGTTTGGATCTTGGTTCATTC TCAAGCCTCAGACAGTGGTTCAAAGTTTTTTTCTTCCATTTCAGGTGTCG TGACGCTAGCGCTACCGGTCGCCACCATGGTGAGCAAGGGCGCCGAGCT G**TTCACCGGCATCGTGCCCA**TCCTGATCGAGCTGAATGGCGATGTGAA TGGCCACAAGTTCAGCGTGAGC**GGC*GAGGGCGAGGGCGAT***GCC*ACCTA* CGGCAAGCTGACCCTGAAG**TTCATC*TGCACCACCGGC***AAGCTGC*CTGTG CCCTGGCCC
696	GFP-1 target binding site	TAGGTTgGGGGGAGGGGTT
697	GFP-2 target binding site	GTGGGTGGAGACtGAAGTT
698	GFP-3 target binding site	TGGGCAcGATGCCGGTGAA
699	GFP-4 target binding site	GGCGAGGGCGAGGGCGAT

SEQ ID NO	Description	Sequence
700	GFP-5 target binding site	GAGGGCGAGGGCGATGCC
701	GFP-6 target binding site	GCCGGTGGTGCAGATGAA
702	GFP-7 target binding site	GCAGCTtGCCGGTGGTGCA
703	Exemplary Zinc Finger Sequence	SRPGERPFQCRICMRNFS[F1]HTRTHTGEKPFQCRICMRNFS[F2]HLRTH[linker1]FQCRICMRNFS[F3]HTRTHTGEKPFQCRICMRNFS[F4]HLRTH[linker2]FQCRICMRNFS[F5]HTRTHTGEKPFQCRICMRNFS[F6]HLRTHLRGS
704	linker	TGSQKP
705	linker	TGGGGSQKP

Description	SEQ ID NO	F1 Sequence	SEQ ID NO	F2 Sequence	SEQ ID NO	F3 Sequence
GFP1-ZF1	716	HKSSLTR	757	RTEHLAR	798	QSAHLKR
GFP1-ZF2	717	HKSSLTR	758	RTEHLAR	799	TSAHLAR
GFP1-ZF3	718	IKAILTR	759	RREHLVR	800	QSAHLKR
GFP1-ZF4	719	IKAILTR	760	RREHLVR	801	TSAHLAR
GFP2-ZF1	720	TSTLLNR	761	QQTNLTR	802	DEANLRR
GFP2-ZF2	721	TSTLLNR	762	QQTNLTR	803	DEANLRR
GFP2-ZF3	722	TSTLLNR	763	QQTNLTR	804	DRGNLTR
GFP2-ZF4	723	TSTLLNR	764	QQTNLTR	805	DRGNLTR
GFP2-ZF5	724	HKSSLTR	765	QTNNLGR	806	DEANLRR
GFP2-ZF6	725	HKSSLTR	766	QTNNLGR	807	DEANLRR
GFP2-ZF7	726	HKSSLTR	767	QTNNLGR	808	DRGNLTR
GFP2-ZF8	727	HKSSLTR	768	QTNNLGR	809	DRGNLTR
GFP3-ZF1	728	QQTNLTR	769	IRHHLKR	810	DSSVLRR
GFP3-ZF2	729	QQTNLTR	770	IRHHLKR	811	DGSTLNR
GFP3-ZF3	730	RKPNLLR	771	EAHHLSR	812	DSSVLRR
GFP3-ZF4	731	RKPNLLR	772	EAHHLSR	813	DGSTLNR
GFP4-ZF1	732	VRHNLTR	773	ESGHLKR	814	RQDNLGR
GFP5-ZF1	733	DSSVLRR	774	LSTNLTR	815	LKEHLTR
GFP5-ZF2	734	DSSVLRR	775	LSTNLTR	816	LKEHLTR
GFP5-ZF3	735	DSSVLRR	776	LSTNLTR	817	SPSKLVR
GFP5-ZF4	736	DSSVLRR	777	LSTNLTR	818	SPSKLVR
GFP5-ZF5	737	DGSTLNR	778	VRHNLTR	819	LKEHLTR
GFP5-ZF6	738	DGSTLNR	779	VRHNLTR	820	LKEHLTR
GFP5-ZF7	739	DGSTLNR	780	VRHNLTR	821	SPSKLVR
GFP5-ZF8	740	DGSTLNR	781	VRHNLTR	822	SPSKLVR
GFP6-ZF1	741	RKPNLLR	782	VRHNLTR	823	DKAQLGR
GFP6-ZF2	742	RKPNLLR	783	VRHNLTR	824	DKAQLGR
GFP6-ZF3	743	RKPNLLR	784	VRHNLTR	825	QSTTLKR
GFP6-ZF4	744	RKPNLLR	785	VRHNLTR	826	QSTTLKR
GFP6-ZF5	745	QQTNLTR	786	VGSNLTR	827	DKAQLGR
GFP6-ZF6	746	QQTNLTR	787	VGSNLTR	828	DKAQLGR
GFP6-ZF7	747	QQTNLTR	788	VGSNLTR	829	QSTTLKR

GFP6-ZF8	748	QQTNLTR	789	VGSNLTR	830	QSTTLKR
GFP7-ZF1	749	QSTTLKR	790	VDHHLRR	831	EAHHLSR
GFP7-ZF2	750	QSTTLKR	791	VDHHLRR	832	EAHHLSR
GFP7-ZF3	751	QSTTLKR	792	VDHHLRR	833	RQSRLQR
GFP7-ZF4	752	QSTTLKR	793	VDHHLRR	834	RQSRLQR
GFP7-ZF5	753	DKAQLGR	794	EAHHLSR	835	EAHHLSR
GFP7-ZF6	754	DKAQLGR	795	EAHHLSR	836	EAHHLSR
GFP7-ZF7	755	DKAQLGR	796	EAHHLSR	837	RQSRLQR
GFP7-ZF8	756	DKAQLGR	797	EAHHLSR	838	RQSRLQR

Description	SEQ ID NO	F1 Sequence	SEQ ID NO	F2 Sequence	SEQ ID NO	F3 Sequence
GFP1-ZF1	839	RTEHLAR	880	HKSSLTR	921	RPESLAP
GFP1-ZF2	840	RREHLVR	881	HKSSLTR	922	RPESLAP
GFP1-ZF3	841	RTEHLAR	882	HKSSLTR	923	RPESLAP
GFP1-ZF4	842	RREHLVR	883	HKSSLTR	924	RPESLAP
GFP2-ZF1	843	QSAHLKR	884	IPNKLAR	925	RREVLEN
GFP2-ZF2	844	QSAHLKR	885	EAHHLSR	926	RKDALHV
GFP2-ZF3	845	QGGHLKR	886	IPNKLAR	927	RREVLEN
GFP2-ZF4	846	QGGHLKR	887	EAHHLSR	928	RKDALHV
GFP2-ZF5	847	QSAHLKR	888	IPNKLAR	929	RREVLEN
GFP2-ZF6	848	QSAHLKR	889	EAHHLSR	930	RKDALHV
GFP2-ZF7	849	QGGHLKR	890	IPNKLAR	931	RREVLEN
GFP2-ZF8	850	QGGHLKR	891	EAHHLSR	932	RKDALHV
GFP3-ZF1	851	LSTNLTR	892	QSTTLKR	933	RSDHLSL
GFP3-ZF2	852	VRHNLTR	893	QSTTLKR	934	RSDHLSL
GFP3-ZF3	853	LSTNLTR	894	QSTTLKR	935	RSDHLSL
GFP3-ZF4	854	VRHNLTR	895	QSTTLKR	936	RSDHLSL
GFP4-ZF1	855	KNHSLNN	896	RQDNLGR	937	KNHSLNN
GFP5-ZF1	856	RVDNLPR	897	LKEHLTR	938	RVDNLPR
GFP5-ZF2	857	RVDNLPR	898	SPSKLVR	939	RQDNLGR
GFP5-ZF3	858	RQDNLGR	899	LKEHLTR	940	RVDNLPR
GFP5-ZF4	859	RQDNLGR	900	SPSKLVR	941	RQDNLGR
GFP5-ZF5	860	RVDNLPR	901	LKEHLTR	942	RVDNLPR
GFP5-ZF6	861	RVDNLPR	902	SPSKLVR	943	RQDNLGR
GFP5-ZF7	862	RQDNLGR	903	LKEHLTR	944	RVDNLPR
GFP5-ZF8	863	RQDNLGR	904	SPSKLVR	945	RQDNLGR
GFP6-ZF1	864	EAHHLSR	905	RQSRLQR	946	KGDHLRR
GFP6-ZF2	865	EAHHLSR	906	EAHHLSR	947	DPSNLRR
GFP6-ZF3	866	VDHHLRR	907	RQSRLQR	948	KGDHLRR
GFP6-ZF4	867	VDHHLRR	908	EAHHLSR	949	DPSNLRR
GFP6-ZF5	868	EAHHLSR	909	RQSRLQR	950	KGDHLRR
GFP6-ZF6	869	EAHHLSR	910	EAHHLSR	951	DPSNLRR
GFP6-ZF7	870	VDHHLRR	911	RQSRLQR	952	KGDHLRR
GFP6-ZF8	871	VDHHLRR	912	EAHHLSR	953	DPSNLRR
GFP7-ZF1	872	DPSNLRR	913	QRSDLTR	954	QGGTLRR
GFP7-ZF2	873	DPSNLRR	914	TKQILGR	955	QSTTLKR
GFP7-ZF3	874	DSSVLRR	915	QRSDLTR	956	QGGTLRR
GFP7-ZF4	875	DSSVLRR	916	TKQILGR	957	QSTTLKR
GFP7-ZF5	876	DPSNLRR	917	QRSDLTR	958	QGGTLRR
GFP7-ZF6	877	DPSNLRR	918	TKQILGR	959	QSTTLKR

GFP7-ZF7	878	DSSVLRR	919	QRSDLTR	960	QGGTLRR
GFP7-ZF8	879	DSSVLRR	920	TKQILGR	961	QSTTLKR

SEQ ID NO	Description	Sequence
962	SPACER	GCCTACCGCAGGATGTTCGG
963	SPACER	GGCCCGGGGACGAGGCGTAG
964	SPACER	GCGCACGGCAGAGGAGCGCG
965	SPACER	GCCCTCGTTCGCCTCTTCTC
966	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
967	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
968	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
969	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
970	SPACER	GTGTCCAGGGACAATGAGCA
971	SPACER	GCGGCCCGGAGCCTACGAGG
972	SPACER	GCGGCGGCGGCAGCAGCTGCG
973	SPACER	GCCGGACTCGGACGCGTGGT
974	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
975	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
976	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
977	TRACR	GTTTAAGAGCTAAGCTGGAAACAGCATAGCAAGTTTAAATAAGG CTAGTCCGTTATCAACTTGAAAAAGTGGCACCGAGTCGGTGCTTT TTTT
998	DNMT3A-3L-ZF-KRAB (ZF is GFP1-ZF1)	MAPKKKRK*MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVL KDLGIQVDRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWG PFDLVIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDR PFFWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWGNL PGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGKDQHF PVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLGRSWSVPV IRHLFAPLKEYFACV***SSGNSNANSRGPSFSSGLVPLSLRGSHMGPME IYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGGGTLK YVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYMFQ FHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAV TLQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVR SRSKLDAPKVDLLVKNCLLPLREYFKYFSQNSLPL*SGGGGSGGGG SVGIHGVPSRPGERPFQCRICMRNFSHKSSLTRHTRTHTGEKPFQCRI CMRNFSRTEHLARHLRTHTGSQKPFQCRICMRNFSQSAHLKRHTRT HTGEKPFQCRICMRNFSRTEHLARHLRTHTGGGGSQKPFQCRICMRN FSHKSSLTRHTRTHTGEKPFQCRICMRNFSRPESLAPHLRTHLRGSGG GSMDAKSLTAWSRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVM

		LENYKNLVSLGYQLTKPDVILRLEKGEEPWLVEREIHQETHPDSETA FEIKSSV
999	DNMT3A/ L-dSpCas9-XTEN16-ZIM3	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMNNSQGRVTFEDVTVNFTQGEWQRLNPEQRNLYRDVMLENYSNL VSVGQGETTKPDVILRLEQGKEPWLEEEEVLGSGRAEKNGDIGGQIW KPKDVKESL
1000	DNMT3A/ L-dSpCas9-XTEN16-HP1b	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS

		HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMGKKQNKKKVEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKWK GFSDEDNTWEPEENLDCPDLIAEFLQSQKTAHETDKSEGGKRKADSD SEDKGEESKPKKKKEESEKPRGFARGLEPERIIGATDSSGELMFLMK WKNSDEADLVPAKEANVKCPQVVISFYEERLTWHSYPSEDDDKKD DKN
1001	DNMT3A/L-dSpCas9-XTEN16-RYBP	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK

		VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GPSEANSIQSANATTKTSETNHTSRPRLKNVDRSTAQQLAVTVGNVT VIITDFKEKTRSSSTSSSTVTSSAGSEQQNQSSS
1002	DNMT3A/ L- dSpCas9- XTEN16- ZFP28	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK

		NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GNKKLEAVGTGIEPKAMSQGLVTFGDVAVDFSQEEWEWLNPIQRNL YRKVMLENYRNLASLGLCVSKPDVISSLEQGKEPW
1003	DNMT3A/ L- dSpCas9- XTEN16- ZN627	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF

		RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GDSVAFEDVAVNFTLEEWALLDPSQKNLYRDVMRETFRNLASVGK QWEDQNIEDPFKIPRRNISHIPERLCESKEGGQGEE
1004	DNMT3A/L-dSpCas9-XTEN16-CDYL2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST

		GASGDLYEVERIVDKRKNKKGKWEYLIRWKGYGSTEDTWEPEHHL LHCEEFIDEFNGLHMSKDKRIKSGKQSSTSKLLRDS
1005	DNMT3A/ L- dSpCas9- XTEN16- TOX	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWD GLGEEQKQVYKKKTEAAKKEYLKQLAAYRASLVSK
1006	DNMT3A/ L- dSpCas9- XTEN16- SCMH1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG

		GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GDASRLSGRDPSSWTVEDVMQFVREADPQLGPHADLFRKHEIDGKA LLLLRSDMMMKYMGLKLGPALKLSYHIDRLKQGKF
1007	DNMT3A/L-dSpCas9-XTEN16-SCML2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL

		SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GKQGFSKDPSTWSVDEVIQFMKHTDPQISGPLADLFRQHEIDGKALF LLKSDVMMKYMGLKLGPALKLCYYIEKLKEGKYS
1008	DNMT3A/ L- dSpCas9- XTEN16- CBX8	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG

		FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GGSGPPSSGGGLYRDMGAQGGRPSLIARIPVARILGDPEEESWSPSLT NLEKVVVTDVTSNFLTVTIKESNTDQGFFKEKR
1009	DNMT3A/L-dSpCas9-XTEN16-TOX3	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG

		KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWD SLGEEQKQVYKRKTEAAKKEYLKALAAYRASLVSK
1010	DNMT3A/ L- dSpCas9- XTEN16- TOX4	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMWD SLGEEQKQVYKRKTEAAKKEYLKALAAYKDNQECQ
1011	DNMT3A/ L- dSpCas9-	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF

	XTEN16-I2BP1	FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GASVQASRRQWCYLCDLPKMPWAMVWDFSEAVCRGCVNFEGADR IELLIDAARQLKRSHVLPEGRSPGPPALKHPATKDLA
1012	DNMT3A/L-dSpCas9-XTEN16-MBD2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS

		EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMRAHPGGGRCCPEQEEGESAAGGSGAGGDSAIEQGGQGSALAPSP VSGVRREGARGGGRGRGRWKQAGRGGGVCGRGRGRGRGRGRGRG RGRGRGRPPSGGSGLGGDGGGCGGGGSGGGGAPRREPVPFPSGSAG PGPRGPRATESGKRMDCPALPPGWKKEEVIRKSGLSAGKSDVYYFSP SGKKFRSKPQLARYLGNTVDLSSFDFRTGKMMPSKLQKNKQRLRND PLNQNKGKPDLNTTLPIRQTASIFKQPVTKVTNHPSNKVKSDPQRMN EQPRQLFWEKRLQGLSASDVTEQIIKTMELPKGLQGVGPGSNDETLL SAVASALHTSSAPITGQVSAAVEKNPAVWLNTSQPLCKAFIVTDEDI RKQEERVQQVRKKLEEALMADILSRAADTEEMDIEMDSGDEA
1013	DNMT3A/ L- dSpCas9- XTEN16- SetDB1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN

		IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMSSLPGCIGLDAATATVESEEIAELQQAVVEELGISMEELRHFIDEE LEKMDCVQQRKKQLAELETWVIQKESEVAHVDQLFDDASRAVTNC ESLVKDFYSKLGLQYRDSSSEDESSRPTEIIEIPDEDDDVLSIDSGDAG SRTPKDQKLREAMAALRKSAQDVQKFMDAVNKKSSSQDLHKGTLS QMSGELSKDGDLIVSMRILGKKRTKTWHKGTLIAIQTVGPGKKYKV KFDNKGKSLLSGNHIAYDYHPPADKLYVGSRVVAKYKDGNQVWLY AGIVAETPNVKNKLRFLIFFDDGYASYVTQSELYPICRPLKKTWEDIE DISCRDFIEEYVTAYPNRPMVLLKSGQLIKTEWEGTWWKSRVEEVD GSLVRILFLDDKRCEWIYRGSTRLEPMFSMKTSSASALEKKQGQLRT RPNMGAVRSKGPVVQYTQDLTGTGTQFKPVEPPQPTAPPAPPFPPAP PLSPQAGDSDLESQLAQSRKQVAKKSTSFRPGSVGSGHSSPTSPALSE NVSGGKPGINQTYRSPLGSTASAPAPSALPAPPAPPVFHGMLERAPAE PSYRAPMEKLFYLPHVCSYTCLSRVRPMRNEQYRGKNPLLVPLLYD FRRMTARRRVNRKMGFHVIYKTPCGLCLRTMQEIERYLFETGCDFLF LEMFCLDPYVLVDRKFQPYKPFYYILDITYGKEDVPLSCVNEIDTTPP PQVAYSKERIPGKGVFINTGPEFLVGCDCKDGCRDKSKCACHQLTIQ ATACTPGGQINPNSGYQYKRLEECLPTGVYECNKRCKCDPNMCTNR LVQHGLQVRLQLFKTQNKGWGIRCLDDIAKGSFVCIYAGKILTDDFA DKEGLEMGDEYFANLDHIESVENFKEGYESDAPCSSDSSGVDLKDQ EDGNSGTEDPEESNDDSSDDNFCKDEDFSTSSVWRSYATRRQTRGQ KENGLSETTSKDSHPPDLGPPHIPVPPSIPVGGCNPPSSEETPKNKVAS WLSCNSVSEGGFADSDSHSSFKTNEGGEGRAGGSRMEAEKASTSGL GIKDEGDIKQAKKEDTDDRNKMSVVTESSRNYGYNPSPVKPEGLRR PPSKTSMHQSRRLMASAQSNPDDVLTLSSSTESEGESGTSRKPTAGQ TSATAVDSDDIQTISSGSEGDDFEDKKNMTGPMKRQVAVKSTRGFA LKSTHGIAIKSTNMASVDKGESAPVRKNTRQFYDGEESCYIIDAKLE GNLGRYLNHSCSPNLFVQNVFVDTHDLRFPWVAFFASKRIRAGTELT WDYNYEVGSVEGKELLCCCGAIECRGRLL

1014	DNMT3A/L-dSpCas9-XTEN16-MeCP2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMVAGMLGLREEKSEDQDLQGLKDKPLKFKKVKKDKKEEKEGKH EPVQPSAHHSAEPAEAGKAETSEGSGSAPAVPEASASPKQRRSIIRDR GPMYDDPTLPEGWTRKLKQRKSGRSAGKYDVYLINPQGKAFRSKVE LIAYFEKVGDTSLDPNDFDFTVTGRGSPSRREQKPPKKPKSPKAPGTG RGRGRPKGSGTTRPKAATSEGVQVKRVLEKSPGKLLVKMPFQTSPG GKAEGGGATTSTQVMVIKRPGRKRKAEADPQAIPKKRGRKPGSVVA AAAAEAKKKAVKESSIRSVQETVLPIKKRKTRETVSIEVKEVVKPLL VSTLGEKSGKGLKTCKSPGRKSKESSPKGRSSSASSPPKKEHHHHHH HSESPKAPVPLLPPLPPPPPEPESSEDPTSPPEPQDLSSSVCKEEKMPRG GSLESDGCPKEPAKTQPAVATAATAAEKYKHRGEGERKDIVSSSMP RPNREEPVDSRTPVTERVS

1015	DNMT3A/L-dSpCas9-XTEN16-Kap1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMAASAAAASAAAASAASGSPGPGEGSAGGEKRSTAPSAAASASAS AAASSPAGGGAEALELLEHCGVCRERLRPEREPRLLPCLHSACSACL GPAAPAAANSSGDGGAAGDGTVVDCPVCKQQCFSKDIVENYFMRD SGSKAATDAQDANQCCTSCEDNAPATSYCVECSEPLCETCVEAHQR VKYTKDHTVRSTGPAKSRDGERTVYCNVHKHEPLVLFCESCDTLTC RDCQLNAHKDHQYQFLEDAVRNQRKLLASLVKRLGDKHATLQKST KEVRSSIRQVSDVQKRVQVDVKMAILQIMKELNKRGRVLVNDAQK VTEGQQERLERQHWTMTKIQKHQEHILRFASWALESDNNTALLLSK KLIYFQLHRALKMIVDPVEPHGEMKFQWDLNAWTKSAEAFGKIVAE RPGTNSTGPAPMAPPRAPGPLSKQGSGSSQPMEVQEGYGFGSGDDP YSSAEPHVSGVKRSRSGEGEVSGLMRKVPRVSLERLDLDLTADSQPP VFKVFPGSTTEDYNLIVIERGAAAAATGQPGTAPAGTPGAPPLAGMA

		IVKEEETEAAIGAPPTATEGPETKPVLMALAEGPGAEGPRLASPSGST SSGLEVVAPEGTSAPGGGPGTLDDSATICRVCQKPGDLVMCNQCEFC FHLDCHLPALQDVPGEEWSCSLCHVLPDLKEEDGSLSLDGADSTGV VAKLSPANQRKCERVLLALFCHEPCRPLHQLATDSTFSLDQPGGTLD LTLIRARLQEKLSPPYSSPQEFAQDVGRMFKQFNKLTEDKADVQSIIG LQRFFETRMNEAFGDTKFSAVLVEPPPMSLPGAGLSSQELSGGPGDG P
1016	DNMT3A/ L- dSpCas9- XTEN16- HP1a	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMGKKTKRTADSSSSEDEEEYVVEKVLDRRVVKGQVEYLLKWKGF SEEHNTWEPEKNLDCPELISEFMKKYKKMKEGENNKPREKSESNKR KSNFSNSADDIKSKKKREQSNDIARGFERGLEPEKIIGATDSCGDLMF LMKWKGTDEADLVLAKEANVKCPQIVIAFYEERLTWHAYPEDAEN KEKETAKS

1017	DNMT3A/L-dSpCas9-XTEN16-EED	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMSEREVSTAPAGTDMPAAKKQKLSSDENSNPDLSGDENDDAVSIE SGTNTERPDTPTNTPNAPGRKSWGKGKWKSKKCKYSFKCVNSLKED HNQPLFGVQFNWHSKEGDPLVFATVGSNRVTLYECHSQGEIRLLQS YVDADADENFYTCAWTYDSNTSHPLLAVAGSRGIIRIINPITMQCIKH YVGHGNAINELKFHPRDPNLLLSVSKDHALRLWNIQTDTLVAIFGGV EGHRDEVLSADYDLLGEKIMSCGMDHSLKLWRINSKRMMNAIKESY DYNPNKTNRPFISQKIHFPDFSTRDIHRNYVDCVRWLGDLILSKSCEN AIVCWKPGKMEDDIDKIKPSESNVTILGRFDYSQCDIWYMRFSMDF WQKMLALGNQVGKLYVWDLEVEDPHKAKCTTLTHHKCGAAIRQT SFSRDSSILIAVCDDASIWRWDRLR
1018	DNMT3A/L-	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL

	dSpCas9-XTEN16-RBBP4	VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMADKEAAFDDAVEERVINEEYKIWKKNTPFLYDLVMTHALEWPS LTAQWLPDVTRPEGKDFSIHRLVLGTHTSDEQNHLVIASVQLPNDDA QFDASHYDSEKGEFGGFGSVSGKIEIEIKINHEGEVNRARYMPQNPCII ATKTPSSDVLVFDYTKHPSKPDPSGECNPDLRLRGHQKEGYGLSWN PNLSGHLLSASDDHTICLWDISAVPKEGKVVDAKTIFTGHTAVVEDV SWHLLHESLFGSVADDQKLMIWDTRSNNTSKPSHSVDAHTAEVNCL SFNPYSEFILATGSADKTVALWDLRNLKLKLHSFESHKDEIFQVQWS PHNETILASSGTDRRLNVWDLSKIGEEQSPEDAEDGPPELLFIHGGHT AKISDFSWNPNEPWVICSVSEDNIMQVWQMAENIYNDEDPEGSVDP EGQGS
1019	DNMT3A/L-dSpCas9-	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG

	XTEN16-RCOR1	NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMPAMVEKGPEVSGKRRGRNNAAASASAAAASAAASAACASPAAT AASGAAASSASAAAASAAAAPNNGQNKSLAAAAPNGNSSSNSWEE GSSGSSSDEEHGGGGMRVGPQYQAVVPDFDPAKLARRSQERDNLG MLVWSPNQNLSEAKLDEYIAIAKEKHGYNMEQALGMLFWHKHNIE KSLADLPNFTPFPDEWTVEDKVLFEQAFSFHGKTFHRIQQMLPDKSI ASLVKFYYSWKKTRTKTSVMDRHARKQKREREESEDELEEANGNN PIDIEVDQNKESKKEVPPTETVPQVKKEKHSTQAKNRAKRKPPKGMF LSQEDVEAVSANATAATTVLRQLDMELVSVKRQIQNIKQTNSALKE KLDGGIEPYRLPEVIQKCNARWTTEEQLLAVQAIRKYGRDFQAISDVI GNKSVVQVKNFFVNYRRRFNIDEVLQEWEAEHGKEETNGPSNQKPV KSPDNSIKMPEEEDEAPVLDVRYASAS
1020	DNMT3A/L-dSpCas9-XTEN16-EZH2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK

		DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GMGQTGKKSEKGPVCWRKRVKSEYMRLRQLKRFRRADEVKSMFSS NRQKILERTEILNQEWKQRRIQPVHILTSVSSLRGTRECSVTSDLDFPT QVIPLKTLNAVASVPIMYSWSPLQQNFMVEDETVLHNIPYMGDEVL DQDGTFIEELIKNYDGKVHGDRECGFINDEIFVELVNALGQYNDDDD DDDGDDPEEREEKQKDLEDHRDDKESRPPRKFPSDKIFEAISSMFPD KGTAEELKEKYKELTEQQLPGALPPECTPNIDGPNAKSVQREQSLHS FHTLFCRRCFKYDCFLHPFHATPNTYKRKNTETALDNKPCGPQCYQ HLEGAKEFAAALTAERIKTPPKRPGGRRRGRLPNNSSRPSTPTINVLE SKDTDSDREAGTETGGENNDKEEEEKKDETSSSSEANSRCQTPIKMK PNIEPPENVEWSGAEASMFRVLIGTYYDNFCAIARLIGTKTCRQVYEF RVKESSIIAPAPAEDVDTPPRKKKRKHRLWAAHCRKIQLKKDGSSNH VYNYQPCDHPRQPCDSSCPCVIAQNFCEKFCQCSSECQNRFPGCRCK AQCNTKQCPCYLAVRECDPDLCLTCGAADHWDSKNVSCKNCSIQR GSKKHLLLAPSDVAGWGIFIKDPVQKNEFISEYCGEIISQDEADRRGK VYDKYMCSFLFNLNNDFVVDATRKGNKIRFANHSVNPNCYAKVMM VNGDHRIGIFAKRAIQTGEELFFDYRYSQADALKYVGIEREMEIP

1021	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-ZIM3	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMNNSQGRVTF EDVTVNFTQGEWQRLNPEQRNLYRDVMLENYSNLVSVGQGETTKP DVILRLEQGKEPWLEEEEVLGSGRAEKNGDIGGQIWKPKDVKESL
1022	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-ZFP28	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG

		GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGNKKLEAVGTGI EPKAMSQGLVTFGDVAVDFSQEEWEWLNPIQRNLYRKVMLENYRN LASLGLCVSKPDVISSLEQGKEPW
1023	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-ZN627	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN

		IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGDSVAFEDVAV NFTLEEWALLDPSQKNLYRDVMRETFRNLASVGKQWEDQNIEDPFK IPRRNISHIPERLCESKEGGQGEE
1024	:DNMT3A/ L- dSpCas9- XTEN16- KOX1KR AB-RYBP	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK

		NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGPSEANSIQSAN ATTKTSETNHTSRPRLKNVDRSTAQQLAVTVGNVTVIITDFKEKTRS SSTSSSTVTSSAGSEQQNQSSS
1025	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-CDYL2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE

		EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGASGDLYEVERI VDKRKNKKGKWEYLIRWKGYGSTEDTWEPEHHLLHCEEFIDEFNGL HMSKDKRIKSGKQSSTSKLLRDS
1026	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-TOX	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG

		SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGKDPNEPQKPVS AYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDGLGEEQKQVYK KKTEAAKKEYLKQLAAYRASLVSK
1027	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-SCMH1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGDASRLSGRDPS SWTVEDVMQFVREADPQLGPHADLFRKHEIDGKALLLLRSDMMMK YMGLKLGPALKLSYHIDRLKQGKF

1028	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-SCML2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGKQGFSKDPST WSVDEVIQFMKHTDPQISGPLADLFRQHEIDGKALFLLKSDVMMKY MGLKLGPALKLCYYIEKLKEGKYS
1029	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-CBX8	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG

		GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGGSGPPSSGGGL YRDMGAQGGRPSLIARIPVARILGDPEEESWSPSLTNLEKVVVTDVT SNFLTVTIKESNTDQGFFKEKR
1030	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-TOX3	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN

		IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGKDPNEPQKPVS AYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDSLGEEQKQVYKR KTEAAKKEYLKALAAYRASLVSK
1031	:DNMT3A/ L- dSpCas9- XTEN16- KOX1KR AB-TOX4	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK

		NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGKDPNEPQKPVS AYALFFRDTQAAIKGQNPNATFGEVSKIVASMWDSLGEEQKQVYKR KTEAAKKEYLKALAAYKDNQECQ
1032	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-I2BP1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE

		EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGASVQASRRQW CYLCDLPKMPWAMVWDFSEAVCRGCVNFEGADRIELLIDAARQLK RSHVLPEGRSPGPPALKHPATKDLA
1033	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-MBD2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG

		SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMRAHPGGGRC CPEQEEGESAAGGSGAGGDSAIEQGGQGSALAPSPVSGVRREGARG GGRGRGRWKQAGRGGGVCGRGRGRGRGRGRGRGRGRGRGRPPSG GSGLGGDGGGCGGGGSGGGGAPRREPVPFPSGSAGPGPRGPRATES GKRMDCPALPPGWKKEEVIRKSGLSAGKSDVYYFSPSGKKFRSKPQ LARYLGNTVDLSSFDFRTGKMMPSKLQKNKQRLRNDPLNQNKGKP DLNTTLPIRQTASIFKQPVTKVTNHPSNKVKSDPQRMNEQPRQLFWE KRLQGLSASDVTEQIIKTMELPKGLQGVGPGSNDETLLSAVASALHT SSAPITGQVSAAVEKNPAVWLNTSQPLCKAFIVTDEDIRKQEERVQQ VRKKLEEALMADILSRAADTEEMDIEMDSGDEA
1034	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-MeCP2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN

		KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMVAGMLGLRE EKSEDQDLQGLKDKPLKFKKVKKDKKEEKEGKHEPVQPSAHHSAEP AEAGKAETSEGSGSAPAVPEASASPKQRRSIIRDRGPMYDDPTLPEG WTRKLKQRKSGRSAGKYDVYLINPQGKAFRSKVELIAYFEKVGDTS LDPNDFDFTVTGRGSPSRREQKPPKKPKSPKAPGTGRGRGRPKGSGT TRPKAATSEGVQVKRVLEKSPGKLLVKMPFQTSPGGKAEGGGATTS TQVMVIKRPGRKRKAEADPQAIPKKRGRKPGSVVAAAAAEAKKKA VKESSIRSVQETVLPIKKRKTRETVSIEVKEVVKPLLVSTLGEKSGKG LKTCKSPGRKSKESSPKGRSSSASSPPKKEHHHHHHHSESPKAPVPLL PPLPPPPPEPESSEDPTSPPEPQDLSSSVCKEEKMPRGGSLESDGCPKE PAKTQPAVATAATAAEKYKHRGEGERKDIVSSSMPRPNREEPVDSR TPVTERVS
1035	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-Kap1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG

		SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMAASAAAASA AAASAASGSPGPGEGSAGGEKRSTAPSAAASASASAAASSPAGGGA EALELLEHCGVCRERLRPEREPRLLPCLHSACSACLGPAAPAAANSS GDGGAAGDGTVVDCPVCKQQCFSKDIVENYFMRDSGSKAATDAQD ANQCCTSCEDNAPATSYCVECSEPLCETCVEAHQRVKYTKDHTVRS TGPAKSRDGERTVYCNVHKHEPLVLFCESCDTLTCRDCQLNAHKDH QYQFLEDAVRNQRKLLASLVKRLGDKHATLQKSTKEVRSSIRQVSD VQKRVQVDVKMAILQIMKELNKRGRVLVNDAQKVTEGQQERLERQ HWTMTKIQKHQEHILRFASWALESDNNTALLLSKKLIYFQLHRALK MIVDPVEPHGEMKFQWDLNAWTKSAEAFGKIVAERPGTNSTGPAP MAPPRAPGPLSKQGSGSSQPMEVQEGYGFGSGDDPYSSAEPHVSGV KRSRSGEGEVSGLMRKVPRVSLERLDLDLTADSQPPVFKVFPGSTTE DYNLIVIERGAAAAATGQPGTAPAGTPGAPPLAGMAIVKEEETEAAI GAPPTATEGPETKPVLMALAEGPGAEGPRLASPSGSTSSGLEVVAPE GTSAPGGGPGTLDDSATICRVCQKPGDLVMCNQCEFCFHLDCHLPA LQDVPGEEWSCSLCHVLPDLKEEDGSLSLDGADSTGVVAKLSPANQ RKCERVLLALFCHEPCRPLHQLATDSTFSLDQPGGTLDLTLIRARLQE KLSPPYSSPQEFAQDVGRMFKQFNKLTEDKADVQSIIGLQRFFETRM NEAFGDTKFSAVLVEPPPMSLPGAGLSSQELSGGPGDGP
1036	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-HP1a	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE

		EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMGKKTKRTAD SSSSEDEEEYVVEKVLDRRVVKGQVEYLLKWKGFSEEHNTWEPEKN LDCPELISEFMKKYKKMKEGENNKPREKSESNKRKSNFSNSADDIKS KKKREQSNDIARGFERGLEPEKIIGATDSCGDLMFLMKWKGTDEAD LVLAKEANVKCPQIVIAFYEERLTWHAYPEDAENKEKETAKS
1037	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-HP1b	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG

		KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMGKKQNKKK VEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKWKGFSDEDNTWEP EENLDCPDLIAEFLQSQKTAHETDKSEGGKRKADSDSEDKGEESKPK KKKEESEKPRGFARGLEPERIIGATDSSGELMFLMKWKNSDEADLVP AKEANVKCPQVVISFYEERLTWHSYPSEDDDKKDDKN
1038	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-EED	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG

		YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMSEREVSTAPA GTDMPAAKKQKLSSDENSNPDLSGDENDDAVSIESGTNTERPDTPTN TPNAPGRKSWGKGKWKSKKCKYSFKCVNSLKEDHNQPLFGVQFNW HSKEGDPLVFATVGSNRVTLYECHSQGEIRLLQSYVDADADENFYT CAWTYDSNTSHPLLAVAGSRGIIRIINPITMQCIKHYVGHGNAINELK FHPRDPNLLLSVSKDHALRLWNIQTDTLVAIFGGVEGHRDEVLSADY DLLGEKIMSCGMDHSLKLWRINSKRMMNAIKESYDYNPNKTNRPFI SQKIHFPDFSTRDIHRNYVDCVRWLGDLILSKSCENAIVCWKPGKME DDIDKIKPSESNVTILGRFDYSQCDIWYMRFSMDFWQKMLALGNQV GKLYVWDLEVEDPHKAKCTTLTHHKCGAAIRQTSFSRDSSILIAVCD DASIWRWDRLR
1039	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-RBBP4	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG

		YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMADKEAAFDD AVEERVINEEYKIWKKNTPFLYDLVMTHALEWPSLTAQWLPDVTRP EGKDFSIHRLVLGTHTSDEQNHLVIASVQLPNDDAQFDASHYDSEKG EFGGFGSVSGKIEIEIKINHEGEVNRARYMPQNPCIIATKTPSSDVLVF DYTKHPSKPDPSGECNPDLRLRGHQKEGYGLSWNPNLSGHLLSASD DHTICLWDISAVPKEGKVVDAKTIFTGHTAVVEDVSWHLLHESLFGS VADDQKLMIWDTRSNNTSKPSHSVDAHTAEVNCLSFNPYSEFILATG SADKTVALWDLRNLKLKLHSFESHKDEIFQVQWSPHNETILASSGTD RRLNVWDLSKIGEEQSPEDAEDGPPELLFIHGGHTAKISDFSWNPNEP WVICSVSEDNIMQVWQMAENIYNDEDPEGSVDPEGQGS
1040	:DNMT3A/ L- dSpCas9- XTEN16- KOX1KR AB- RCOR1	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMPAMVEKGPE

		VSGKRRGRNNAAASASAAAASAAASAACASPAATAASGAAASSAS AAAASAAAAPNNGQNKSLAAAAPNGNSSSNSWEEGSSGSSSDEEHG GGGMRVGPQYQAVVPDFDPAKLARRSQERDNLGMLVWSPNQNLSE AKLDEYIAIAKEKHGYNMEQALGMLFWHKHNIEKSLADLPNFTPFP DEWTVEDKVLFEQAFSFHGKTFHRIQQMLPDKSIASLVKFYYSWKK TRTKTSVMDRHARKQKREREESEDELEEANGNNPIDIEVDQNKESK KEVPPTETVPQVKKEKHSTQAKNRAKRKPPKGMFLSQEDVEAVSAN ATAATTVLRQLDMELVSVKRQIQNIKQTNSALKEKLDGGIEPYRLPE VIQKCNARWTTEEQLLAVQAIRKYGRDFQAISDVIGNKSVVQVKNFF VNYRRRFNIDEVLQEWEAEHGKEETNGPSNQKPVKSPDNSIKMPEEE DEAPVLDVRYASAS
1041	:DNMT3A/L-dSpCas9-XTEN16-KOX1KRAB-EZH2	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HMGPMEIYKTVSAWKRQPVRVLSLFRNIDKVLKSLGFLESGSGSGG GTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQPLGSSCDRCPGWYM FQFHRILQYALPRQESQRPFFWIFMDNLLLTEDDQETTTRFLQTEAVT LQDVRGRDYQNAMRVWSNIPGLKSKHAPLTPKEEEYLQAQVRSRSK LDAPKVDLLVKNCLLPLREYFKYFSQNSLPLGGPSSGAPPPSGGSPAG SPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPS EGSAPGTSTEPSEPKKKRKVYMDKKYSIGLAIGTNSVGWAVITDEYK VPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYT RRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGN IVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHF LIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARL SKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKL QLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTE ITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNG YAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTF DNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGP LARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDK NLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKK AIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGT YHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAH LFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDG FANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIK KGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRER MKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQ ELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSE EVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKR QLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDF RKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGD YKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRK RPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFS KESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKG KSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKY SLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKG SPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYN KHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVL DATLIHQSITGLYETRIDLSQLGGDPKKKRKVSGSETPGTSESATPEST GRTLVTFKDVFVDFTREEWKLLDTAQQIVYRNVMLENYKNLVSLG

		YQLTKPDVILRLEKGEEPSTEPSEGSAPGTSTEPSETGMGQTGKKSEK GPVCWRKRVKSEYMRLRQLKRFRRADEVKSMFSSNRQKILERTEIL NQEWKQRRIQPVHILTSVSSLRGTRECSVTSDLDFPTQVIPLKTLNAV ASVPIMYSWSPLQQNFMVEDETVLHNIPYMGDEVLDQDGTFIEELIK NYDGKVHGDRECGFINDEIFVELVNALGQYNDDDDDDDGDDPEERE EKQKDLEDHRDDKESRPPRKFPSDKIFEAISSMFPDKGTAEELKEKY KELTEQQLPGALPPECTPNIDGPNAKSVQREQSLHSFHTLFCRRCFKY DCFLHPFHATPNTYKRKNTETALDNKPCGPQCYQHLEGAKEFAAAL TAERIKTPPKRPGGRRRGRLPNNSSRPSTPTINVLESKDTDSDREAGT ETGGENNDKEEEEKKDETSSSSEANSRCQTPIKMKPNIEPPENVEWS GAEASMFRVLIGTYYDNFCAIARLIGTKTCRQVYEFRVKESSIIAPAP AEDVDTPPRKKKRKHRLWAAHCRKIQLKKDGSSNHVYNYQPCDHP RQPCDSSCPCVIAQNFCEKFCQCSSECQNRFPGCRCKAQCNTKQCPC YLAVRECDPDLCLTCGAADHWDSKNVSCKNCSIQRGSKKHLLLAPS DVAGWGIFIKDPVQKNEFISEYCGEIISQDEADRRGKVYDKYMCSFL FNLNNDFVVDATRKGNKIRFANHSVNPNCYAKVMMVNGDHRIGIF AKRAIQTGEELFFDYRYSQADALKYVGIEREMEIP
1042	Cas-ZIM3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMNNSQGRVTFEDVTVNFTQGEWQRLNPEQRNLYRDVMLENYSN LVSVGQGETTKPDVILRLEQGKEPWLEEEEVLGSGRAEKNGDIGGQI WKPKDVKESL
1043	Cas-ZNF554	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS

		DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMFSQEERMAAGYLPRWSQELVTFEDVSMDFSQEEWELLEPAQK NLYREVMLENYRNVVSLEALKNQCTDVGIKEGPLSPAQTSQVTSLSS WTGYLLFQPVASSHLEQREALWIEEKGTPQASCSDWMTVLRNQDST YKKVALQE
1044	Cas-ZNF264	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI

		ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAAAVLTDRAQVSVTFDDVAVTFTKEEWGQLDLAQRTLYQEV MLENCGLLVSLGCPVPKAELICHLEHGQEPWTRKEDLSQDTCPGDK GKPKTTEPTTCEPALSE
1045	Cas-ZNF354A	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAAGQREARPQVSLTFEDVAVLFTRDEWRKLAPSQRNLYRDVM LENYRNLVSLGLPFTKPKVISLLQQGEDPWEVEKDGSGVSSLGSKSS HKTTKSTQTQDSSFQ
1046	Cas-ZNF324	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH

		SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAFEDVAVYFSQEEWGLLDTAQRALYRRVMLDNFALVASLGLS TSRPRVVIQLERGEEPWVPSGTDTTLSRTTYRRRNPGSWSLTEDRDV SG
1047	Cas-ZFP28	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGNKKLEAVGTGIEPKAMSQGLVTFGDVAVDFSQEEWEWLNPIQRN LYRKVMLENYRNLASLGLCVSKPDVISSLEQGKEPW
1048	Cas-ZN627	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGDSVAFEDVAVNFTLEEWALLDPSQKNLYRDVMRETFRNLASVG KQWEDQNIEDPFKIPRRNISHIPERLCESKEGGQGEE
1049	Cas-ZN793	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGIEYQIPVSFKDVVVGFTQEEWHRLSPAQRALYRDVMLETYSNLVS VGYEGTKPDVILRLEQEEAPWIGEAACPGCHCWED
1050	Cas-ZN736	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGGVLTFRDVAVEFSPEEWECLDSAQQRLYRDVMLENYGNLVSLGL AIFKPDLMTCLEQRKEPWKVKRQEAVAKHPAGSFHF
1051	Cas-ZN577	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGNATIVMSVRREQGSSSGEGSLSFEDVAVGFTREEWQFLDQSQKVL YKEVMLENYINLVSIGYRGTKPDSLFKLEQGEPPG
1052	Cas-SUMO1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGEGEYIKLKVIGQDSSEIHFKVKMTTHLKKLKESYCQRQGVPMNSL RFLFEGQRIADNHTPKELGMEEEDVIEVYQEQTGG
1053	Cas-SUMO3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGENDHINLKVAGQDGSVVQFKIKRHTPLSKLMKAYCERQGLSMRQ IRFRFDGQPINETDTPAQLEMEDEDTIDVFQQQTGG
1054	Cas-MPP8	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGAEAFGDSEEDGEDVFEVEKILDMKTEGGKVLYKVRWKGYTSDD DTWEPEIHLEDCKEVLLEFRKKIAENKAKAVRKDIQR
1055	Cas-RYBP	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGPSEANSIQSANATTKTSETNHTSRPRLKNVDRSTAQQLAVTVGNV TVIITDFKEKTRSSSTSSSTVTSSAGSEQQNQSSS
1056	Cas-YAF2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGKDKVEKEKSEKETTSKKNSHKKTRPRLKNVDRSSAQHLEVTVGD LTVIITDFKEKTKSPPASSAASADQHSQSGSSSDNT
1057	Cas-SUMO5	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGKDEDIKLRVIGQDSSEIHFKVKMTTPLKKLKKSYCQRQGVPVNSL RFLFEGQRIADNHTPEELGMEEEDVIEVYQEQIGG
1058	Cas-CBX4	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGRSEAGEPPSSLQVKPETPASAAVAVAAAAAPTTTAEKPPAEAQDE PAESLSEFKPFFGNIIITDVTANCLTVTFKEYVTV
1059	Cas-PCGF2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGHRTTRIKITELNPHLMCALCGGYFIDATTIVECLHSFCKTCIVRYLE TNKYCPMCDVQVHKTRPLLSIRSDKTLQDIVYK
1060	Cas-CDY2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGASQEFEVEAIVDKRQDKNGNTQYLVRWKGYDKQDDTWEPEQHL MNCEKCVHDFNRRQTEKQKKLTWTTTSRIFSNNARRR
1061	Cas-CDYL2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGASGDLYEVERIVDKRKNKKGKWEYLIRWKGYGSTEDTWEPEHH LLHCEEFIDEFNGLHMSKDKRIKSGKQSSTSKLLRDS
1062	Cas-HERC2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGTLIRKADLENHNKDGGFWTVIDGKVYDIKDFQTQSLTGNSILAQF AGEDPVVALEAALQFEDTRESMHAFCVGQYLEPDQ
1063	Cas-ID2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSDHSLGISRSKTPVDDPMSLLYNMNDCYSKLKELVPSIPQNKKVS KMEILQHVIDYILDLQIALDSHPTIVSLHHQRPGQ
1064	Cas-TOX	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMW DGLGEEQKQVYKKKTEAAKKEYLKQLAAYRASLVSK
1065	Cas-SCMH1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGDASRLSGRDPSSWTVEDVMQFVREADPQLGPHADLFRKHEIDGK ALLLLRSDMMMKYMGLKLGPALKLSYHIDRLKQGKF
1066	Cas-CBX7	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGELSAIGEQVFAVESIRKKRVRKGKVEYLVKWKGWPPKYSTWEPE EHILDPRLVMAYEEKEERDRASGYRKRGPKPKRLLL
1067	Cas-ID1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGGGAGARLPALLDEQQVNVLLYDMNGCYSRLKELVPTLPQNRKV SKVEILQHVIDYIRDLQLELNSESEVGTPGGRGLPVR
1068	Cas-CREM	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGVVMAASPGSLHSPQQLAEEATRKRELRLMKNREAAKECRRRKK EYVKCLESRVAVLEVQNKKLIEELETLKDICSPKTDY
1069	Cas-SCX	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGGGGPGGRPGREPRQRHTANARERDRTNSVNTAFTALRTLIPTEPA DRKLSKIETLRLASSYISHLGNVLLAGEACGDGQP
1070	Cas-ASCL1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSGFGYSLPQQQPAAVARRNERERNRVKLVNLGFATLREHVPNGA ANKKMSKVETLRSAVEYIRALQQLLDEHDAVSAAFQ
1071	Cas-SCML2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGKQGFSKDPSTWSVDEVIQFMKHTDPQISGPLADLFRQHEIDGKAL FLLKSDVMMKYMGLKLGPALKLCYYIEKLKEGKYS
1072	Cas-TWST1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGSGGGSPQSYEELQTQRVMANVRERQRTQSLNEAFAALRKIIPTLP SDKLSKIQTLKLAARYIDFLYQVLQSDELDSKMAS
1073	Cas-CREB1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGIAPGVVMASSPALPTQPAEEAARKREVRLMKNREAARECRRKKK EYVKCLENRVAVLENQNKTLIEELKALKDLYCHKSD
1074	Cas-TERF1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSRIPVSKSQPVTPEKHRARKRQAWLWEEDKNLRSGVRKYGEGN WSKILLHYKFNNRTSVMLKDRWRTMKKLKLISSDSED
1075	Cas-ID3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSLAIARGRGKGPAAEEPLSLLDDMNHCYSRLRELVPGVPRGTQLS QVEILQRVIDYILDLQVVLAEPAPGPPDGPHLPIQ
1076	Cas-CBX8	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGGSGPPSSGGGLYRDMGAQGGRPSLIARIPVARILGDPEEESWSPSL TNLEKVVVTDVTSNFLTVTIKESNTDQGFFKEKR
1077	Cas-CBX4	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGELPAVGEHVFAVESIEKKRIRKGRVEYLVKWRGWSPKYNTWEPE ENILDPRLLIAFQNRERQEQLMGYRKRGPKPKPLVV
1078	Cas-GSX1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGVDSSSNQLPSSKRMRTAFTSTQLLELEREFASNMYLSRLRRIEIAT YLNLSEKQVKIWFQNRRVKHKKEGKGSNHRGGGG
1079	Cas- NKX22	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGTPGGGGDAGKKRKRRVLFSKAQTYELERRFRQQRYLSAPEREHL ASLIRLTPTQVKIWFQNHRYKMKRARAEKGMEVTPL
1080	Cas-ATF1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGQTVVMTSPVTLTSQTTKTDDPQLKREIRLMKNREAARECRRKKK EYVKCLENRVAVLENQNKTLIEELKTLKDLYSNKSV
1081	Cas-TWST2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGKGSPSAQSFEELQSQRILANVRERQRTQSLNEAFAALRKIIPTLPSD KLSKIQTLKLAARYIDFLYQVLQSDEMDNKMTS
1082	Cas-TOX3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMW DSLGEEQKQVYKRKTEAAKKEYLKALAAYRASLVSK
1083	Cas-TOX4	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGKDPNEPQKPVSAYALFFRDTQAAIKGQNPNATFGEVSKIVASMW DSLGEEQKQVYKRKTEAAKKEYLKALAAYKDNQECQ
1084	Cas-ZMYM3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGLDGSTWDFCSEDCKSKYLLWYCKAARCHACKRQGKLLETIHWR GQIRHFCNQQCLLRFYSQQNQPNLDTQSGPESLLNSQ
1085	Cas-I2BP1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGASVQASRRQWCYLCDLPKMPWAMVWDFSEAVCRGCVNFEGAD RIELLIDAARQLKRSHVLPEGRSPGPPALKHPATKDLA
1086	Cas-RHXF1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMEGPQPENMQPRTRRTKFTLLQVEELESVFRHTQYPDVPTRRELA ENLGVTEDKVRVWFKNKRARCRRHQRELMLANELR
1087	Cas-SSX2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGPKIMPKKPAEEGNDSEEVPEASGPQNDGKELCPPGKPTTSEKIHER SGPKRGEHAWTHRLRERKQLVIYEEISDPEEDDE
1088	Cas-I2BPL	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSAAQVSSSRRQSCYLCDLPRMPWAMIWDFSEPVCRGCVNYEGA DRIEFVIETARQLKRAHGCFQDGRSPGPPPPVGVKTV
1089	Cas-CBX1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGNKKKVEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKWKGFSDE DNTWEPEENLDCPDLIAEFLQSQKTAHETDKSEGGKR
1090	Cas-TRI68	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGLANVVEKVRLLRLHPGMGLKGDLCERHGEKLKMFCKEDVLIMC EACSQSPEHEAHSVVPMEDVAWEYKWELHEALEHLKK
1091	Cas-HXA13	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGVVSHPSDASSYRRGRKKRVPYTKVQLKELEREYATNKFITKDKR RRISATTNLSERQVTIWFQNRRVKEKKVINKLKTTS
1092	Cas-PHC3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGENSDLLPVAQTEPSIWTVDDVWAFIHSLPGCQDIADEFRAQEIDG QALLLLKEDHLMSAMNIKLGPALKICARINSLKES
1093	Cas-TCF24	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGAGPGGGSRSGSGRPAAANAARERSRVQTLRHAFLELQRTLPSVPP DTKLSKLDVLLLATTYIAHLTRSLQDDAEAPADAG
1094	Cas-HXB13	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGQHPPDACAFRRGRKKRIPYSKGQLRELEREYAANKFITKDKRRKI SAATSLSERQITIWFQNRRVKEKKVLAKVKNSATP
1095	Cas-HEY1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGSMSPTTSSQILARKRRRGIIEKRRRDRINNSLSELRRLVPSAFEKQG SAKLEKAEILQMTVDHLKMLHTAGGKGYFDAHA
1096	Cas-PHC2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL

		GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGLVGMGHHFLPSEPTKWNVEDVYEFIRSLPGCQEIAEEFRAQEIDG QALLLLKEDHLMSAMNIKLGPALKIYARISMLKDS
1097	Cas-FIGLA	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE

		TGGYSSTENLQLVLERRRVANAKERERIKNLNRGFARLKALVPFLPQ SRKPSKVDILKGATEYIQVLSDLLEGAKDSKKQDP
1098	Cas-SetDB1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMSSLPGCIGLDAATATVESEEIAELQQAVVEELGISMEELRHFIDE ELEKMDCVQQRKKQLAELETWVIQKESEVAHVDQLFDDASRAVTN CESLVKDFYSKLGLQYRDSSSEDESSRPTEIIEIPDEDDDVLSIDSGDA GSRTPKDQKLREAMAALRKSAQDVQKFMDAVNKKSSSQDLHKGTL SQMSGELSKDGDLIVSMRILGKKRTKTWHKGTLIAIQTVGPGKKYK VKFDNKGKSLLSGNHIAYDYHPPADKLYVGSRVVAKYKDGNQVWL YAGIVAETPNVKNKLRFLIFFDDGYASYVTQSELYPICRPLKKTWEDI EDISCRDFIEEYVTAYPNRPMVLLKSGQLIKTEWEGTWWKSRVEEV DGSLVRILFLDDKRCEWIYRGSTRLEPMFSMKTSSASALEKKQGQLR TRPNMGAVRSKGPVVQYTQDLTGTGTQFKPVEPPQPTAPPAPPFPPA PPLSPQAGDSDLESQLAQSRKQVAKKSTSFRPGSVGSGHSSPTSPALS ENVSGGKPGINQTYRSPLGSTASAPAPSALPAPPAPPVFHGMLERAPA EPSYRAPMEKLFYLPHVCSYTCLSRVRPMRNEQYRGKNPLLVPLLY DFRRMTARRRVNRKMGFHVIYKTPCGLCLRTMQEIERYLFETGCDF LFLEMFCLDPYVLVDRKFQPYKPFYYILDITYGKEDVPLSCVNEIDTT PPPQVAYSKERIPGKGVFINTGPEFLVGCDCKDGCRDKSKCACHQLT IQATACTPGGQINPNSGYQYKRLEECLPTGVYECNKRCKCDPNMCT NRLVQHGLQVRLQLFKTQNKGWGIRCLDDIAKGSFVCIYAGKILTD DFADKEGLEMGDEYFANLDHIESVENFKEGYESDAPCSSDSSGVDLK DQEDGNSGTEDPEESNDDSSDDNFCKDEDFSTSSVWRSYATRRQTR GQKENGLSETTSKDSHPPDLGPPHIPVPPSIPVGGCNPPSSEETPKNKV ASWLSCNSVSEGGFADSDSHSSFKTNEGGEGRAGGSRMEAEKASTS

		GLGIKDEGDIKQAKKEDTDDRNKMSVVTESSRNYGYNPSPVKPEGL RRPPSKTSMHQSRRLMASAQSNPDDVLTLSSSTESEGESGTSRKPTA GQTSATAVDSDDIQTISSGSEGDDFEDKKNMTGPMKRQVAVKSTRG FALKSTHGIAIKSTNMASVDKGESAPVRKNTRQFYDGEESCYIIDAKL EGNLGRYLNHSCSPNLFVQNVFVDTHDLRFPWVAFFASKRIRAGTEL TWDYNYEVGSVEGKELLCCCGAIECRGRLL
1099	Cas-MBD1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAEDWLDCPALGPGWKRREVFRKSGATCGRSDTYYQSPTGDRI RSKVELTRYLGPACDLTLFDFKQGILCYPAPKAHPVAVASKKRKKPS RPAKTRKRQVGPQSGEVRKEAPRDETKADTDTAPASFPAPGCCENC GISFSGDGTQRQRLKTLCKDCRAQRIAFNREQRMFKRVGCGECAAC QVTEDCGACSTCLLQLPHDVASGLFCKCERRRCLRIVERSRGCGVCR GCQTQEDCGHCPICLRPPRPGLRRQWKCVQRRCLRGKHARRKGGC DSKMAARRRPGAQPLPPPPPSQSPEPTEPHPRALAPSPPAEFIYYCVD EDELQPYTNRRQNRKCGACAACLRRMDCGRCDFCCDKPKFGGSNQ KRQKCRWRQCLQFAMKRLLPSVWSESEDGAGSPPPYRRRKRPSSAR RHHLGPTLKPTLATRTAQPDHTQAPTKQEAGGGFVLPPPGTDLVFLR EGASSPVQVPGPVAASTEALLQEAQCSGLSWVVALPQVKQEKADTQ DEWTPGTAVLTSPVLVPGCPSKAVDPGLPSVKQEPPDPEEDKEENKD DSASKLAPEEEAGGAGTPVITEIFSLGGTRFRDTAVWLPRSKDLKKP GARKQ
1100	Cas-MBD2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS

		DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMRAHPGGGRCCPEQEEGESAAGGSGAGGDSAIEQGGQGSALAPS PVSGVRREGARGGGRGRGRWKQAGRGGGVCGRGRGRGRGRGRGR GRGRGRGRPPSGGSGLGGDGGGCGGGGSGGGGAPRREPVPFPSGSA GPGPRGPRATESGKRMDCPALPPGWKKEEVIRKSGLSAGKSDVYYF SPSGKKFRSKPQLARYLGNTVDLSSFDFRTGKMMPSKLQKNKQRLR NDPLNQNKGKPDLNTTLPIRQTASIFKQPVTKVTNHPSNKVKSDPQR MNEQPRQLFWEKRLQGLSASDVTEQIIKTMELPKGLQGVGPGSNDE TLLSAVASALHTSSAPITGQVSAAVEKNPAVWLNTSQPLCKAFIVTD EDIRKQEERVQQVRKKLEEALMADILSRAADTEEMDIEMDSGDEA
1101	Cas-MBD3	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ

		LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMERKRWECPALPQGWEREEVPRRSGLSAGHRDVFYYSPSGKKFR SKPQLARYLGGSMDLSTFDFRTGKMLMSKMNKSRQRVRYDSSNQV KGKPDLNTALPVRQTASIFKQPVTKITNHPSNKVKSDPQKAVDQPRQ LFWEKKLSGLNAFDIAEELVKTMDLPKGLQGVGPGCTDETLLSAIAS ALHTSTMPITGQLSAAVEKNPGVWLNTTQPLCKAFMVTDEDIRKQE ELVQQVRKRLEEALMADMLAHVEELARDGEAPLDKACAEDDDEED EEEEEEEPDPDPEMEHV
1102	Cas-MBD4	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGTTGLESLSLGDRGAAPTVTSSERLVPDPPNDLRKEDVAMELE RVGEDEEQMMIKRSSECNPLLQEPIASAQFGATAGTECRKSVPCGWE RVVKQRLFGKTAGRFDVYFISPQGLKFRSKSSLANYLHKNGETSLKP EDFDFTVLSKRGIKSRYKDCSMAALTSHLQNQSNNSNWNLRTRSKC KKDVFMPPSSSSELQESRGLSNFTSTHLLLKEDEGVDDVNFRKVRKP

		KGKVTILKGIPIKKTKKGCRKSCSGFVQSDSKRESVCNKADAESEPV AQKSQLDRTVCISDAGACGETLSVTSEENSLVKKKERSLSSGSNFCSE QKTSGIINKFCSAKDSEHNEKYEDTFLESEEIGTKVEVVERKEHLHTD ILKRGSEMDNNCSPTRKDFTGEKIFQEDTIPRTQIERRKTSLYFSSKYN KEALSPPRRKAFKKWTPPRSPFNLVQETLFHDPWKLLIATIFLNRTSG KMAIPVLWKFLEKYPSAEVARTADWRDVSELLKPLGLYDLRAKTIV KFSDEYLTKQWKYPIELHGIGKYGNDSYRIFCVNEWKQVHPEDHKL NKYHDWLWENHEKLSLS
1103	Cas-MeCP2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMVAGMLGLREEKSEDQDLQGLKDKPLKFKKVKKDKKEEKEGK HEPVQPSAHHSAEPAEAGKAETSEGSGSAPAVPEASASPKQRRSIIRD RGPMYDDPTLPEGWTRKLKQRKSGRSAGKYDVYLINPQGKAFRSK VELIAYFEKVGDTSLDPNDFDFTVTGRGSPSRREQKPPKKPKSPKAPG TGRGRGRPKGSGTTRPKAATSEGVQVKRVLEKSPGKLLVKMPFQTS PGGKAEGGGATTSTQVMVIKRPGRKRKAEADPQAIPKKRGRKPGSV VAAAAAEAKKKAVKESSIRSVQETVLPIKKRKTRETVSIEVKEVVKP LLVSTLGEKSGKGLKTCKSPGRKSKESSPKGRSSSASSPPKKEHHHH HHHSESPKAPVPLLPPLPPPPPEPESSEDPTSPPEPQDLSSSVCKEEKMP RGGSLESDGCPKEPAKTQPAVATAATAAEKYKHRGEGERKDIVSSS MPRPNREEPVDSRTPVTERVS
1104	Cas-Kap1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA

		QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAASAAAASAAAASAASGSPGPGEGSAGGEKRSTAPSAAASASA SAAASSPAGGGAEALELLEHCGVCRERLRPEREPRLLPCLHSACSAC LGPAAPAAANSSGDGGAAGDGTVVDCPVCKQQCFSKDIVENYFMR DSGSKAATDAQDANQCCTSCEDNAPATSYCVECSEPLCETCVEAHQ RVKYTKDHTVRSTGPAKSRDGERTVYCNVHKHEPLVLFCESCDTLT CRDCQLNAHKDHQYQFLEDAVRNQRKLLASLVKRLGDKHATLQKS TKEVRSSIRQVSDVQKRVQVDVKMAILQIMKELNKRGRVLVNDAQ KVTEGQQERLERQHWTMTKIQKHQEHILRFASWALESDNNTALLLS KKLIYFQLHRALKMIVDPVEPHGEMKFQWDLNAWTKSAEAFGKIVA ERPGTNSTGPAPMAPPRAPGPLSKQGSGSSQPMEVQEGYGFGSGDDP YSSAEPHVSGVKRSRSGEGEVSGLMRKVPRVSLERLDLDLTADSQPP VFKVFPGSTTEDYNLIVIERGAAAAATGQPGTAPAGTPGAPPLAGMA IVKEEETEAAIGAPPTATEGPETKPVLMALAEGPGAEGPRLASPSGST SSGLEVVAPEGTSAPGGGPGTLDDSATICRVCQKPGDLVMCNQCEFC FHLDCHLPALQDVPGEEWSCSLCHVLPDLKEEDGSLSLDGADSTGV VAKLSPANQRKCERVLLALFCHEPCRPLHQLATDSTFSLDQPGGTLD LTLIRARLQEKLSPPYSSPQEFAQDVGRMFKQFNKLTEDKADVQSIIG LQRFFETRMNEAFGDTKFSAVLVEPPPMSLPGAGLSSQELSGGPGDG P
1105	Cas-HP1a	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW

		MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGKKTKRTADSSSSEDEEEYVVEKVLDRRVVKGQVEYLLKWKG FSEEHNTWEPEKNLDCPELISEFMKKYKKMKEGENNKPREKSESNK RKSNFSNSADDIKSKKKREQSNDIARGFERGLEPEKIIGATDSCGDLM FLMKWKGTDEADLVLAKEANVKCPQIVIAFYEERLTWHAYPEDAE NKEKETAKS
1106	Cas-HP1b	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE

		TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGKKQNKKKVEEVLEEEEEEYVVEKVLDRRVVKGKVEYLLKW KGFSDEDNTWEPEENLDCPDLIAEFLQSQKTAHETDKSEGGKRKADS DSEDKGEESKPKKKKEESEKPRGFARGLEPERIIGATDSSGELMFLM KWKNSDEADLVPAKEANVKCPQVVISFYEERLTWHSYPSEDDDKK DDKN
1107	Cas-HP1g	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMASNKTTLQKMGKKQNGKSKKVEEAEPEEFVVEKVLDRRVVNG KVEYFLKWKGFTDADNTWEPEENLDCPELIEAFLNSQKAGKEKDGT KRKSLSDSESDDSKSKKKRDAADKPRGFARGLDPERIIGATDSSGEL MFLMKWKDSDEADLVLAKEANMKCPQIVIAFYEERLTWHSCPEDE AQ
1108	Cas-SetDB2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR

		KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGEKNGDAKTFWMELEDDGKVDFIFEQVQNVLQSLKQKIKDGS ATNKEYIQAMILVNEATIINSSTSIKGASQKEVNAQSSDPMPVTQKEQ ENKSNAFPSTSCENSFPEDCTFLTTENKEILSLEDKVVDFREKDSSSNL SYQSHDCSGACLMKMPLNLKGENPLQLPIKCHFQRRHAKTNSHSSA LHVSYKTPCGRSLRNVEEVFRYLLETECNFLFTDNFSFNTYVQLARN YPKQKEVVSDVDISNGVESVPISFCNEIDSRKLPQFKYRKTVWPRAY NLTNFSSMFTDSCDCSEGCIDITKCACLQLTARNAKTSPLSSDKITTG YKYKRLQRQIPTGIYECSLLCKCNRQLCQNRVVQHGPQVRLQVFKT EQKGWGVRCLDDIDRGTFVCIYSGRLLSRANTEKSYGIDENGRDENT MKNIFSKKRKLEVACSDCEVEVLPLGLETHPRTAKTEKCPPKFSNNP KELTVETKYDNISRIQYHSVIRDPESKTAIFQHNGKKMEFVSSESVTP EDNDGFKPPREHLNSKTKGAQKDSSSNHVDEFEDNLLIESDVIDITKY REETPPRSRCNQATTLDNQNIKKAIEVQIQKPQEGRSTACQRQQVFC DEELLSETKNTSSDSLTKFNKGNVFLLDATKEGNVGRFLNHSCCPNL LVQNVFVETHNRNFPLVAFFTNRYVKARTELTWDYGYEAGTVPEKE IFCQCGVNKCRKKIL
1109	Cas-SUV39H1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA

		QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAENLKGCSVCCKSSWNQLQDLCRLAKLSCPALGISKRNLYDFE VEYLCDYKKIREQEYYLVKWRGYPDSESTWEPRQNLKCVRILKQFH KDLERELLRRHHRSKTPRHLDPSLANYLVQKAKQRRALRRWEQELN AKRSHLGRITVENEVDLDGPPRAFVYINEYRVGEGITLNQVAVGCEC QDCLWAPTGGCCPGASLHKFAYNDQGQVRLRAGLPIYECNSRCRCG YDCPNRVVQKGIRYDLCIFRTDDGRGWGVRTLEKIRKNSFVMEYVG EIITSEEAERRGQIYDRQGATYLFDLDYVEDVYTVDAAYYGNISHFV NHSCDPNLQVYNVFIDNLDERLPRIAFFATRTIRAGEELTFDYNMQV DPVDMESTRMDSNFGLAGLPGSPKKRVRIECKCGTESCRKYLF
1110	Cas-SUV39H1[H320R]	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAENLKGCSVCCKSSWNQLQDLCRLAKLSCPALGISKRNLYDFE VEYLCDYKKIREQEYYLVKWRGYPDSESTWEPRQNLKCVRILKQFH KDLERELLRRHHRSKTPRHLDPSLANYLVQKAKQRRALRRWEQELN AKRSHLGRITVENEVDLDGPPRAFVYINEYRVGEGITLNQVAVGCEC

		QDCLWAPTGGCCPGASLHKFAYNDQGQVRLRAGLPIYECNSRCRCG YDCPNRVVQKGIRYDLCIFRTDDGRGWGVRTLEKIRKNSFVMEYVG EIITSEEAERRGQIYDRQGATYLFDLDYVEDVYTVDAAYYGNISRFV NHSCDPNLQVYNVFIDNLDERLPRIAFFATRTIRAGEELTFDYNMQV DPVDMESTRMDSNFGLAGLPGSPKKRVRIECKCGTESCRKYLF
1111	Cas-SUV39H2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAAVGAEARGAWCVPCLVSLDTLQELCRKEKLTCKSIGITKRNL NNYEVEYLCDYKVVKDMEYYLVKWKGWPDSTNTWEPLQNLKCPL LLQQFSNDKHNYLSQVKKGKAITPKDNNKTLKPAIAEYIVKKAKQRI ALQRWQDELNRRKNHKGMIFVENTVDLEGPPSDFYYINEYKPAPGIS LVNEATFGCSCTDCFFQKCCPAEAGVLLAYNKNQQIKIPPGTPIYECN SRCQCGPDCPNRIVQKGTQYSLCIFRTSNGRGWGVKTLVKIKRMSFV MEYVGEVITSEEAERRGQFYDNKGITYLFDLDYESDEFTVDAARYG NVSHFVNHSCDPNLQVFNVFIDNLDTRLPRIALFSTRTINAGEELTFD YQMKGSGDISSDSIDHSPAKKRVRTVCKCGAVTCRGYLN
1112	Cas-SUV420H1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW

		MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMKWLGESKNMVVNGRRNGGKLSNDHQQNQSKLQHTGKDTLKA GKNAVERRSNRCNGNSGFEGQSRYVPSSGMSAKELCENDDLATSLV LDPYLGFQTHKMNTSAFPSRSSRHFSKSDSFSHNNPVRFRPIKGRQEE LKEVIERFKKDEHLEKAFKCLTSGEWARHYFLNKNKMQEKLFKEHV FIYLRMFATDSGFEILPCNRYSSEQNGAKIVATKEWKRNDKIELLVG CIAELSEIEENMLLRHGENDFSVMYSTRKNCAQLWLGPAAFINHDCR PNCKFVSTGRDTACVKALRDIEPGEEISCYYGDGFFGENNEFCECYT CERRGTGAFKSRVGLPAPAPVINSKYGLRETDKRLNRLKKLGDSSKN SDSQSVSSNTDADTTQEKNNATSNRKSSVGVKKNSKSRTLTRQSMS RIPASSNSTSSKLTHINNSRVPKKLKKPAKPLLSKIKLRNHCKRLEQK NASRKLEMGNLVLKEPKVVLYKNLPIKKDKEPEGPAQAAVASGCLT RHAAREHRQNPVRGAHSQGESSPCTYITRRSVRTRTNLKEASDIKLE PNTLNGYKSSVTEPCPDSGEQLQPAPVLQEEELAHETAQKGEAKCH KSDTGMSKKKSRQGKLVKQFAKIEESTPVHDSPGKDDAVPDLMGPH SDQGEHSGTVGVPVSYTDCAPSPVGCSVVTSDSFKTKDSFRTAKSKK KRRITRYDAQLILENNSGIPKLTLRRRHDSSSKTNDQENDGMNSSKIS IKLSKDHDNDNNLYVAKLNNGFNSGSGSSSTKLKIQLKRDEENRGSY TEGLHENGVCCSDPLSLLESRMEVDDYSQYEEESTDDSSSSEGDEEE DDYDDDFEDDFIPLPPAKRLRLIVGKDSIDIDISSRRREDQSLRLNA
1113	Cas-SUV420H2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD

		DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGPDRVTARELCENDDLATSLVLDPYLGFRTHKMNVSPVPPLRR QQHLRSALETFLRQRDLEAAYRALTLGGWTARYFQSRGPRQEAALK THVYRYLRAFLPESGFTILPCTRYSMETNGAKIVSTRAWKKNEKLEL LVGCIAELREADEGLLRAGENDFSIMYSTRKRSAQLWLGPAAFINHD CKPNCKFVPADGNAACVKVLRDIEPGDEVTCFYGEGFFGEKNEHCE CHTCERKGEGAFRTRPREPALPPRPLDKYQLRETKRRLQQGLDSGSR QGLLGPRACVHPSPLRRDPFCAACQPLRLPACSARPDTSPLWLQWLP QPQPRVRPRKRRRPRPRRAPVLSTHHAARVSLHRWGGCGPHCRLRG EALVALGQPPHARWAPQQDWHWARRYGLPYVVRVDLRRLAPAPP ATPAPAGTPGPILIPKQALAFAPFSPPKRLRLVVSHGSIDLDVGGEEL
1114	Cas-EZH1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE

		TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMEIPNPPTSKCITYWKRKVKSEYMRLRQLKRLQANMGAKALYV ANFAKVQEKTQILNEEWKKLRVQPVQSMKPVSGHPFLKKCTIESIFP GFASQHMLMRSLNTVALVPIMYSWSPLQQNFMVEDETVLCNIPYMG DEVKEEDETFIEELINNYDGKVHGEEEMIPGSVLISDAVFLELVDALN QYSDEEEEGHNDTSDGKQDDSKEDLPVTRKRKRHAIEGNKKSSKKQ FPNDMIFSAIASMFPENGVPDDMKERYRELTEMSDPNALPPQCTPNI DGPNAKSVQREQSLHSFHTLFCRRCFKYDCFLHPFHATPNVYKRKN KEIKIEPEPCGTDCFLLLEGAKEYAMLHNPRSKCSGRRRRRHHIVSAS CSNASASAVAETKEGDSDRDTGNDWASSSSEANSRCQTPTKQKASP APPQLCVVEAPSEPVEWTGAEESLFRVFHGTYFNNFCSIARLLGTKT CKQVFQFAVKESLILKLPTDELMNPSQKKKRKHRLWAAHCRKIQLK KDNSSTQVYNYQPCDHPDRPCDSTCPCIMTQNFCEKFCQCNPDCQN RFPGCRCKTQCNTKQCPCYLAVRECDPDLCLTCGASEHWDCKVVSC KNCSIQRGLKKHLLLAPSDVAGWGTFIKESVQKNEFISEYCGELISQD EADRRGKVYDKYMSSFLFNLNNDFVVDATRKGNKIRFANHSVNPN CYAKVVMVNGDHRIGIFAKRAIQAGEELFFDYRYSQADALKYVGIE RETDVL
1115	Cas-EZH2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGQTGKKSEKGPVCWRKRVKSEYMRLRQLKRFRRADEVKSMFS SNRQKILERTEILNQEWKQRRIQPVHILTSVSSLRGTRECSVTSDLDFP TQVIPLKTLNAVASVPIMYSWSPLQQNFMVEDETVLHNIPYMGDEV LDQDGTFIEELIKNYDGKVHGDRECGFINDEIFVELVNALGQYNDDD DDDDGDDPEEREEKQKDLEDHRDDKESRPPRKFPSDKIFEAISSMFP

		DKGTAEELKEKYKELTEQQLPGALPPECTPNIDGPNAKSVQREQSLH SFHTLFCRRCFKYDCFLHPFHATPNTYKRKNTETALDNKPCGPQCYQ HLEGAKEFAAALTAERIKTPPKRPGGRRRGRLPNNSSRPSTPTINVLE SKDTDSDREAGTETGGENNDKEEEEKKDETSSSSEANSRCQTPIKMK PNIEPPENVEWSGAEASMFRVLIGTYYDNFCAIARLIGTKTCRQVYEF RVKESSIIAPAPAEDVDTPPRKKKRKHRLWAAHCRKIQLKKDGSSNH VYNYQPCDHPRQPCDSSCPCVIAQNFCEKFCQCSSECQNRFPGCRCK AQCNTKQCPCYLAVRECDPDLCLTCGAADHWDSKNVSCKNCSIQR GSKKHLLLAPSDVAGWGIFIKDPVQKNEFISEYCGEIISQDEADRRGK VYDKYMCSFLFNLNNDFVVDATRKGNKIRFANHSVNPNCYAKVMM VNGDHRIGIFAKRAIQTGEELFFDYRYSQADALKYVGIEREMEIP
1116	Cas-EZH2[S21A]	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMGQTGKKSEKGPVCWRKRVKAEYMRLRQLKRFRRADEVKSMF SSNRQKILERTEILNQEWKQRRIQPVHILTSVSSLRGTRECSVTSDLDF PTQVIPLKTLNAVASVPIMYSWSPLQQNFMVEDETVLHNIPYMGDEV LDQDGTFIEELIKNYDGKVHGDRECGFINDEIFVELVNALGQYNDDD DDDDGDDPEEREEKQKDLEDHRDDKESRPPRKFPSDKIFEAISSMFP DKGTAEELKEKYKELTEQQLPGALPPECTPNIDGPNAKSVQREQSLH SFHTLFCRRCFKYDCFLHPFHATPNTYKRKNTETALDNKPCGPQCYQ HLEGAKEFAAALTAERIKTPPKRPGGRRRGRLPNNSSRPSTPTINVLE SKDTDSDREAGTETGGENNDKEEEEKKDETSSSSEANSRCQTPIKMK PNIEPPENVEWSGAEASMFRVLIGTYYDNFCAIARLIGTKTCRQVYEF RVKESSIIAPAPAEDVDTPPRKKKRKHRLWAAHCRKIQLKKDGSSNH VYNYQPCDHPRQPCDSSCPCVIAQNFCEKFCQCSSECQNRFPGCRCK AQCNTKQCPCYLAVRECDPDLCLTCGAADHWDSKNVSCKNCSIQR

		GSKKHLLLAPSDVAGWGIFIKDPVQKNEFISEYCGEIISQDEADRRGK VYDKYMCSFLFNLNNDFVVDATRKGNKIRFANHSVNPNCYAKVMM VNGDHRIGIFAKRAIQTGEELFFDYRYSQADALKYVGIEREMEIP
1117	Cas-EHMT1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAAADAEAVPARGEPQQDCCVKTELLGEETPMAADEGSAEKQA GEAHMAADGETNGSCENSDASSHANAAKHTQDSARVNPQDGTNTL TRIAENGVSERDSEAAKQNHVTADDFVQTSVIGSNGYILNKPALQAQ PLRTTSTLASSLPGHAAKTLPGGAGKGRTPSAFPQTPAAPPATLGEGS ADTEDRKLPAPGADVKVHRARKTMPKSVVGLHAASKDPREVREAR DHKEPKEEINKNISDFGRQQLLPPFPSLHQSLPQNQCYMATTKSQTA CLPFVLAAAVSRKKKRRMGTYSLVPKKKTKVLKQRTVIEMFKSITHS TVGSKGEKDLGASSLHVNGESLEMDSDEDDSEELEEDDGHGAEQAA AFPTEDSRTSKESMSEADRAQKMDGESEEEQESVDTGEEEEGGDESD LSSESSIKKKFLKRKGKTDSPWIKPARKRRRSRKKPSGALGSESYKS SAGSAEQTAPGDSTGYMEVSLDSLDLRVKGILSSQAEGLANGPDVLE TDGLQEVPLCSCRMETPKSREITTLANNQCMATESVDHELGRCTNSV VKYELMRPSNKAPLLVLCEDHRGRMVKHQCCPGCGYFCTAGNFME CQPESSISHRFHKDCASRVNNASYCPHCGEESSKAKEVTIAKADTTST VTPVPGQEKGSALEGRADTTTGSAAGPPLSEDDKLQGAASHVPEGF DPTGPAGLGRPTPGLSQGPGKETLESALIALDSEKPKKLRFHPKQLYF SARQGELQKVLLMLVDGIDPNFKMEHQNKRSPLHAAAEAGHVDICH MLVQAGANIDTCSEDQRTPLMEAAENNHLEAVKYLIKAGALVDPK DAEGSTCLHLAAKKGHYEVVQYLLSNGQMDVNCQDDGGWTPMIW ATEYKHVDLVKLLLSKGSDINIRDNEENICLHWAAFSGCVDIAEILLA AKCDLHAVNIHGDSPLHIAARENRYDCVVLFLSRDSDVTLKNKEGE

		TPLQCASLNSQVWSALQMSKALQDSAPDRPSPVERIVSRDIARGYER IPIPCVNAVDSEPCPSNYKYVSQNCVTSPMNIDRNITHLQYCVCIDDC SSSNCMCGQLSMRCWYDKDGRLLPEFNMAEPPLIFECNHACSCWRN CRNRVVQNGLRARLQLYRTRDMGWGVRSLQDIPPGTFVCEYVGELI SDSEADVREEDSYLFDLDNKDGEVYCIDARFYGNVSRFINHHCEPNL VPVRVFMAHQDLRFPRIAFFSTRLIEAGEQLGFDYGERFWDIKGKLFS CRCGSPKCRHSSAALAQRQASAAQEAQEDGLPDTSSAAAADPL
1118	Cas-EHMT2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAAAAGAAAAAAAEGEAPAEMGALLLEKETRGATERVHGSLG DTPRSEETLPKATPDSLEPAGPSSPASVTVTVGDEGADTPVGATPLIG DESENLEGDGDLRGGRILLGHATKSFPSSPSKGGSCPSRAKMSMTGA GKSPPSVQSLAMRLLSMPGAQGAAAAGSEPPPATTSPEGQPKVHRA RKTMSKPGNGQPPVPEKRPPEIQHFRMSDDVHSLGKVTSDLAKRRK LNSGGGLSEELGSARRSGEVTLTKGDPGSLEEWETVVGDDFSLYYDS YSVDERVDSDSKSEVEALTEQLSEEEEEEEEEEEEEEEEEEEEEED EESGNQSDRSGSSGRRKAKKKWRKDSPWVKPSRKRRKREPPRAKEP RGVNGVGSSGPSEYMEVPLGSLELPSEGTLSPNHAGVSNDTSSLETE RGFEELPLCSCRMEAPKIDRISERAGHKCMATESVDGELSGCNAAIL KRETMRPSSRVALMVLCETHRARMVKHHCCPGCGYFCTAGTFLEC HPDFRVAHRFHKACVSQLNGMVFCPHCGEDASEAQEVTIPRGDGVT PPAGTAAPAPPPLSQDVPGRADTSQPSARMRGHGEPRRPPCDPLADT IDSSGPSLTLPNGGCLSAVGLPLGPGREALEKALVIQESERRKKLRFH PRQLYLSVKQGELQKVILMLLDNLDPNFQSDQQSKRTPLHAAAQKG SVEICHVLLQAGANINAVDKQQRTPLMEAVVNNHLEVARYMVQRG GCVYSKEEDGSTCLHHAAKIGNLEMVSLLLSTGQVDVNAQDSGGW

		TPIIWAAEHKHIEVIRMLLTRGADVTLTDNEENICLHWASFTGSAAIA EVLLNARCDLHAVNYHGDTPLHIAARESYHDCVLLFLSRGANPELR NKEGDTAWDLTPERSDVWFALQLNRKLRLGVGNRAIRTEKIICRDV ARGYENVPIPCVNGVDGEPCPEDYKYISENCETSTMNIDRNITHLQH CTCVDDCSSSNCLCGQLSIRCWYDKDGRLLQEFNKIEPPLIFECNQAC SCWRNCKNRVVQSGIKVRLQLYRTAKMGWGVRALQTIPQGTFICEY VGELISDAEADVREDDSYLFDLDNKDGEVYCIDARYYGNISRFINHL CDPNIIPVRVFMLHQDLRFPRIAFFSSRDIRTGEELGFDYGDRFWDIKS KYFTCQCGSEKCKHSAEAIALEQSRLARLDPHPELLPELGSLPPVNT
1119	Cas-LSD1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMLSGKKAAAAAAAAAAAATGTEAGPGTAGGSENGSEVAAQPA GLSGPAEVGPGAVGERTPRKKEPPRASPPGGLAEPPGSAGPQAGPTV VPGSATPMETGIAETPEGRRTSRRKRAKVEYREMDESLANLSEDEYY SEEERNAKAEKEKKLPPPPPQAPPEEENESEPEEPSGVEGAAFQSRLP HDRMTSQEAACFPDIISGPQQTQKVFLFIRNRTLQLWLDNPKIQLTFE ATLQQLEAPYNSDTVLVHRVHSYLERHGLINFGIYKRIKPLPTKKTG KVIIIGSGVSGLAAARQLQSFGMDVTLLEARDRVGGRVATFRKGNY VADLGAMVVTGLGGNPMAVVSKQVNMELAKIKQKCPLYEANGQA VPKEKDEMVEQEFNRLLEATSYLSHQLDFNVLNNKPVSLGQALEVV IQLQEKHVKDEQIEHWKKIVKTQEELKELLNKMVNLKEKIKELHQQ YKEASEVKPPRDITAEFLVKSKHRDLTALCKEYDELAETQGKLEEKL QELEANPPSDVYLSSRDRQILDWHFANLEFANATPLSTLSLKHWDQD DDFEFTGSHLTVRNGYSCVPVALAEGLDIKLNTAVRQVRYTASGCE VIAVNTRSTSQTFIYKCDAVLCTLPLGVLKQQPPAVQFVPPLPEWKTS AVQRMGFGNLNKVVLCFDRVFWDPSVNLFGHVGSTTASRGELFLF

		WNLYKAPILLALVAGEAAGIMENISDDVIVGRCLAILKGIFGSSAVPQ PKETVVSRWRADPWARGSYSYVAAGSSGNDYDLMAQPITPGPSIPG APQPIPRLFFAGEHTIRNYPATVHGALLSGLREAGRIADQFLGAMYTL PRQATPGVPAQQSPSM
1120	Cas-SUZ12	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMAPQKHGGGGGGGSGPSAGSGGGGFGGSAAVAAATASGGKSG GGSCGGGGSYSASSSSSAAAAAGAAVLPVKKPKMEHVQADHELFL QAFEKPTQIYRFLRTRNLIAPIFLHRTLTYMSHRNSRTNIKRKTFKVD DMLSKVEKMKGEQESHSLSAHLQLTFTGFFHKNDKPSPNSENEQNS VTLEVLLVKVCHKKRKDVSCPIRQVPTGKKQVPLNPDLNQTKPGNF PSLAVSSNEFEPSNSHMVKSYSLLFRVTRPGRREFNGMINGETNENID VNEELPARRKRNREDGEKTFVAQMTVFDKNRRLQLLDGEYEVAMQ EMEECPISKKRATWETILDGKRLPPFETFSQGPTLQFTLRWTGETNDK STAPIAKPLATRNSESLHQENKPGSVKPTQTIAVKESLTTDLQTRKEK DTPNENRQKLRIFYQFLYNNNTRQQTEARDDLHCPWCTLNCRKLYS LLKHLKLCHSRFIFNYVYHPKGARIDVSINECYDGSYAGNPQDIHRQ PGFAFSRNGPVKRTPITHILVCRPKRTKASMSEFLESEDGEVEQQRTY SSGHNRLYFHSDTCLPLRPQEMEVDSEDEKDPEWLREKTITQIEEFSD VNEGEKEVMKLWNLHVMKHGFIADNQMNHACMLFVENYGQKIIK KNLCRNFMLHLVSMHDFNLISIMSIDKAVTKLREMQQKLEKGESASP ANEEITEEQNGTANGFSEINSKEKALETDSVSGVSKQSKKQKL
1121	Cas-EED	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS

		DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMSEREVSTAPAGTDMPAAKKQKLSSDENSNPDLSGDENDDAVSI ESGTNTERPDTPTNTPNAPGRKSWGKGKWKSKKCKYSFKCVNSLKE DHNQPLFGVQFNWHSKEGDPLVFATVGSNRVTLYECHSQGEIRLLQ SYVDADADENFYTCAWTYDSNTSHPLLAVAGSRGIIRIINPITMQCIK HYVGHGNAINELKFHPRDPNLLLSVSKDHALRLWNIQTDTLVAIFGG VEGHRDEVLSADYDLLGEKIMSCGMDHSLKLWRINSKRMMNAIKES YDYNPNKTNRPFISQKIHFPDFSTRDIHRNYVDCVRWLGDLILSKSCE NAIVCWKPGKMEDDIDKIKPSESNVTILGRFDYSQCDIWYMRFSMDF WQKMLALGNQVGKLYVWDLEVEDPHKAKCTTLTHHKCGAAIRQT SFSRDSSILIAVCDDASIWRWDRLR
1122	Cas-RING1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD

		AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMTTPANAQNASKTWELSLYELHRTPQEAIMDGTEIAVSPRSLHSE LMCPICLDMLKNTMTTKECLHRFCSDCIVTALRSGNKECPTCRKKLV SKRSLRPDPNFDALISKIYPSREEYEAHQDRVLIRLSRLHNQQALSSSI EEGLRMQAMHRAQRVRRPIPGSDQTTTMSGGEGEPGEGEGDGEDVS SDSAPDSAPGPAPKRPRGGGAGGSSVGTGGGGTGGVGGGAGSEDSG DRGGTLGGGTLGPPSPPGAPSPPEPGGEIELVFRPHPLLVEKGEYCQT RYVKTTGNATVDHLSKYLALRIALERRQQQEAGEPGGPGGGASDTG GPDGCGGEGGGAGGGDGPEEPALPSLEGVSEKQYTIYIAPGGGAFTT LNGSLTLELVNEKFWKVSRPLELCYAPTKDPK
1123	Cas-RING2	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMSQAVQTNGTQPLSKTWELSLYELQRTPQEAITDGLEIVVSPRSL HSELMCPICLDMLKNTMTTKECLHRFCADCIITALRSGNKECPTCRK

		KLVSKRSLRPDPNFDALISKIYPSRDEYEAHQERVLARINKHNNQQA LSHSIEEGLKIQAMNRLQRGKKQQIENGSGAEDNGDSSHCSNASTHS NQEAGPSNKRTKTSDDSGLELDNNNAAMAIDPVMDGASEIELVFRP HPTLMEKDDSAQTRYIKTSGNATVDHLSKYLAVRLALEELRSKGES NQMNLDTASEKQYTIYIATASGQFTVLNGSFSLELVSEKYWKVNKP MELYYAPTKEHK
1124	Cas-PHC1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMETESEQNSNSTNGSSSSGGSSRPQIAQMSLYERQAVQALQALQR QPNAAQYFHQFMLQQQLSNAQLHSLAAVQQATIAASRQASSPNTST TQQQTTTTQASINLATTSAAQLISRSQSVSSPSATTLTQSVLLGNTTSP PLNQSQAQMYLRPQLGNLLQVNRTLGRNVPLASQLILMPNGAVAAV QQEVPSAQSPGVHADADQVQNLAVRNQQASAQGPQMQGSTQKAIP PGASPVSSLSQASSQALAVAQASSGATNQSLNLSQAGGGSGNSIPGS MGPGGGGQAHGGLGQLPSSGMGGGSCPRKGTGVVQPLPAAQTVTV SQGSQTEAESAAAKKAEADGSGQQNVGMNLTRTATPAPSQTLISSA TYTQIQPHSLIQQQQQIHLQQKQVVIQQQIAIHHQQQFQHRQSQLLHT ATHLQLAQQQQQQQQQQQQQQPQATTLTAPQPPQVPPTQQVPPSQ SQQQAQTLVVQPMLQSSPLSLPPDAAPKPPIPIQSKPPVAPIKPPQLGA AKMSAAQQPPPHIPVQVVGTRQPGTAQAQALGLAQLAAAVPTSRG MPGTVQSGQAHLASSPPSSQAPGALQECPPTLAPGMTLAPVQGTAH VVKGGATTSSPVVAQVPAAFYMQSVHLPGKPQTLAVKRKADSEEER DDVSTLGSMLPAKASPVAESPKVMDEKSSLGEKAESVANVNANTPS SELVALTPAPSVPPPTLAMVSRQMGDSKPPQAIVKPQILTHIIEGFVIQ EGAEPFPVGCSQLLKESEKPLQTGLPTGLTENQSGGPLGVDSPSAELD KKANLLKCEYCGKYAPAEQFRGSKRFCSMTCAKRYNVSCSHQFRLK

		RKKMKEFQEANYARVRRRGPRRSSSDIARAKIQGKCHRGQEDSSRG SDNSSYDEALSPTSPGPLSVRAGHGERDLGNPNTAPPTPELHGINPVF LSSNPSRWSVEEVYEFIASLQGCQEIAEEFRSQEIDGQALLLLKEEHL MSAMNIKLGPALKICAKINVLKET
1125	Cas-BMI1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMHRTTRIKITELNPHLMCVLCGGYFIDATTIIECLHSFCKTCIVRYL ETSKYCPICDVQVHKTRPLLNIRSDKTLQDIVYKLVPGLFKNEMKRR RDFYAAHPSADAANGSNEDRGEVADEDKRIITDDEIISLSIEFFDQNR LDRKVNKDKEKSKEEVNDKRYLRCPAAMTVMHLRKFLRSKMDIPN TFQIDVMYEEEPLKDYYTLMDIAYIYTWRRNGPLPLKYRVRPTCKR MKISHQRDGLTNAGELESDSGSDKANSPAGGIPSTSSCLPSPSTPVQS PHPQFPHISSTMNGTSNSPSGNHQSSFANRPRKSSVNGSSATSSG
1126	Cas-RBBP4	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD

		FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMADKEAAFDDAVEERVINEEYKIWKKNTPFLYDLVMTHALEWP SLTAQWLPDVTRPEGKDFSIHRLVLGTHTSDEQNHLVIASVQLPNDD AQFDASHYDSEKGEFGGFGSVSGKIEIEIKINHEGEVNRARYMPQNP CIIATKTPSSDVLVFDYTKHPSKPDPSGECNPDLRLRGHQKEGYGLS WNPNLSGHLLSASDDHTICLWDISAVPKEGKVVDAKTIFTGHTAVVE DVSWHLLHESLFGSVADDQKLMIWDTRSNNTSKPSHSVDAHTAEVN CLSFNPYSEFILATGSADKTVALWDLRNLKLKLHSFESHKDEIFQVQ WSPHNETILASSGTDRRLNVWDLSKIGEEQSPEDAEDGPPELLFIHGG HTAKISDFSWNPNEPWVICSVSEDNIMQVWQMAENIYNDEDPEGSV DPEGQGS
1127	Cas-RBBP7	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE

		QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMASKEMFEDTVEERVINEEYKIWKKNTPFLYDLVMTHALQWPSL TVQWLPEVTKPEGKDYALHWLVLGTHTSDEQNHLVVARVHIPNDD AQFDASHCDSDKGEFGGFGSVTGKIECEIKINHEGEVNRARYMPQNP HIIATKTPSSDVLVFDYTKHPAKPDPSGECNPDLRLRGHQKEGYGLS WNSNLSGHLLSASDDHTVCLWDINAGPKEGKIVDAKAIFTGHSAVV EDVAWHLLHESLFGSVADDQKLMIWDTRSNTTSKPSHLVDAHTAEV NCLSFNPYSEFILATGSADKTVALWDLRNLKLKLHTFESHKDEIFQV HWSPHNETILASSGTDRRLNVWDLSKIGEEQSAEDAEDGPPELLFIHG GHTAKISDFSWNPNEPWVICSVSEDNIMQIWQMAENIYNDEESDVTT SELEGQGS
1128	Cas-REST	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMATQVMGQSSGGGGLFTSSGNIGMALPNDMYDLHDLSKAELAA PQLIMLANVALTGEVNGSCCDYLVGEERQMAELMPVGDNNFSDSEE GEGLEESADIKGEPHGLENMELRSLELSVVEPQPVFEASGAPDIYSSN KDLPPETPGAEDKGKSSKTKPFRCKPCQYEAESEEQFVHHIRVHSAK KFFVEESAEKQAKARESGSSTAEEGDFSKGPIRCDRCGYNTNRYDHY TAHLKHHTRAGDNERVYKCIICTYTTVSEYHWRKHLRNHFPRKVYT CGKCNYFSDRKNNYVQHVRTHTGERPYKCELCPYSSSQKTHLTRHM RTHSGEKPFKCDQCSYVASNQHEVTRHARQVHNGPKPLNCPHCDY KTADRSNFKKHVELHVNPRQFNCPVCDYAASKKCNLQYHFKSKHPT CPNKTMDVSKVKLKKTKKREADLPDNITNEKTEIEQTKIKGDVAGK

		KNEKSVKAEKRDVSKEKKPSNNVSVIQVTTRTRKSVTEVKEMDVHT GSNSEKFSKTKKSKRKLEVDSHSLHGPVNDEESSTKKKKKVESKSKN NSQEVPKGDSKVEENKKQNTCMKKSTKKKTLKNKSSKKSSKPPQKE PVEKGSAQMDPPQMGPAPTEAVQKGPVQVEPPPPMEHAQMEGAQI RPAPDEPVQMEVVQEGPAQKELLPPVEPAQMVGAQIVLAHMELPPP METAQTEVAQMGPAPMEPAQMEVAQVESAPMQVVQKEPVQMELS PPMEVVQKEPVQIELSPPMEVVQKEPVKIELSPPIEVVQKEPVQMELS PPMGVVQKEPAQREPPPPREPPLHMEPISKKPPLRKDKKEKSNMQSE RARKEQVLIEVGLVPVKDSWLLKESVSTEDLSPPSPPLPKENLREEAS GDQKLLNTGEGNKEAPLQKVGAEEADESLPGLAANINESTHISSSGQ NLNTPEGETLNGKHQTDSIVCEMKMDTDQNTRENLTGINSTVEEPVS PMLPPSAVEEREAVSKTALASPPATMAANESQEIDEDEGIHSHEGSDL SDNMSEGSDDSGLHGARPVPQESSRKNAKEALAVKAAKGDFVCIFC DRSFRKGKDYSKHLNRHLVNVYYLEEAAQGQE
1129	Cas-RCOR1	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMPAMVEKGPEVSGKRRGRNNAAASASAAAASAAASAACASPAA TAASGAAASSASAAAASAAAAPNNGQNKSLAAAAPNGNSSSNSWE EGSSGSSSDEEHGGGGMRVGPQYQAVVPDFDPAKLARRSQERDNLG MLVWSPNQNLSEAKLDEYIAIAKEKHGYNMEQALGMLFWHKHNIE KSLADLPNFTPFPDEWTVEDKVLFEQAFSFHGKTFHRIQQMLPDKSI ASLVKFYYSWKKTRTKTSVMDRHARKQKREREESEDELEEANGNN PIDIEVDQNKESKKEVPPTETVPQVKKEKHSTQAKNRAKRKPPKGMF LSQEDVEAVSANATAATTVLRQLDMELVSVKRQIQNIKQTNSALKE KLDGGIEPYRLPEVIQKCNARWTTEEQLLAVQAIRKYGRDFQAISDVI

		GNKSVVQVKNFFVNYRRRFNIDEVLQEWEAEHGKEETNGPSNQKPV KSPDNSIKMPEEEDEAPVLDVRYASAS
1130	Cas-SIN3A	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMKRRLDDQESPVYAAQQRRIPGSTEAFPHQHRVLAPAPPVYEAV SETMQSATGIQYSVTPSYQVSAMPQSSGSHGPAIAAVHSSHHHPTAV QPHGGQVVQSHAHPAPPVAPVQGQQQFQRLKVEDALSYLDQVKLQ FGSQPQVYNDFLDIMKEFKSQSIDTPGVISRVSQLFKGHPDLIMGFNT FLPPGYKIEVQTNDMVNVTTPGQVHQIPTHGIQPQPQPPPQHPSQPSA QSAPAPAQPAPQPPPAKVSKPSQLQAHTPASQQTPPLPPYASPRSPPV QPHTPVTISLGTAPSLQNNQPVEFNHAINYVNKIKNRFQGQPDIYKAF LEILHTYQKEQRNAKEAGGNYTPALTEQEVYAQVARLFKNQEDLLS EFGQFLPDANSSVLLSKTTAEKVDSVRNDHGGTVKKPQLNNKPQRP SQNGCQIRRHPTGTTPPVKKKPKLLNLKDSSMADASKHGGGTESLFF DKVRKALRSAEAYENFLRCLVIFNQEVISRAELVQLVSPFLGKFPELF NWFKNFLGYKESVHLETYPKERATEGIAMEIDYASCKRLGSSYRALP KSYQQPKCTGRTPLCKEVLNDTWVSFPSWSEDSTFVSSKKTQYEEHI YRCEDERFELDVVLETNLATIRVLEAIQKKLSRLSAEEQAKFRLDNTL GGTSEVIHRKALQRIYADKAADIIDGLRKNPSIAVPIVLKRLKMKEEE WREAQRGFNKVWREQNEKYYLKSLDHQGINFKQNDTKVLRSKSLL NEIESIYDERQEQATEENAGVPVGPHLSLAYEDKQILEDAAALIIHHV KRQTGIQKEDKYKIKQIMHHFIPDLLFAQRGDLSDVEEEEEEEMDVD EATGAVKKHNGVGGSPPKSKLLFSNTAAQKLRGMDEVYNLFYVNN NWYIFMRLHQILCLRLLRICSQAERQIEEENREREWEREVLGIKRDKS DSPAIQLRLKEPMDVDVEDYYPAFLDMVRSLLDGNIDSSQYEDSLRE MFTIHAYIAFTMDKLIQSIVRQLQHIVSDEICVQVTDLYLAENNNGAT

		GGQLNTQNSRSLLESTYQRKAEQLMSDENCFKLMFIQSQGQVQLTIE LLDTEEENSDDPVEAERWSDYVERYMNSDTTSPELREHLAQKPVFLP RNLRRIRKCQRGREQQEKEGKEGNSKKTMENVDSLDKLECRFKLNS YKMVYVIKSEDYMYRRTALLRAHQSHERVSKRLHQRFQAWVDKW TKEHVPREMAAETSKWLMGEGLEGLVPCTTTCDTETLHFVSINKYR VKYGTVFKAP
1131	Cas-HDAC5	MGTPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKVGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE TGMNSPNESDGMSGREPSLEILPRTSLHSIPVTVEVKPVLPRAMPSSM GGGGGGSPSPVELRGALVGSVDPTLREQQLQQELLALKQQQQLQKQ LLFAEFQKQHDHLTRQHEVQLQKHLKQQQEMLAAKQQQEMLAAK RQQELEQQRQREQQRQEELEKQRLEQQLLILRNKEKSKESAIASTEV KLRLQEFLLSKSKEPTPGGLNHSLPQHPKCWGAHHASLDQSSPPQSG PPGTPPSYKLPLPGPYDSRDDFPLRKTASEPNLKVRSRLKQKVAERRS SPLLRRKDGTVISTFKKRAVEITGAGPGASSVCNSAPGSGPSSPNSSHS TIAENGFTGSVPNIPTEMLPQHRALPLDSSPNQFSLYTSPSLPNISLGL QATVTVTNSHLTASPKLSTQQEAERQALQSLRQGGTLTGKFMSTSSI PGCLLGVALEGDGSPHGHASLLQHVLLLEQARQQSTLIAVPLHGQSP LVTGERVATSMRTVGKLPRHRPLSRTQSSPLPQSPQALQQLVMQQQ HQQFLEKQKQQQLQLGKILTKTGELPRQPTTHPEETEEELTEQQEVL LGEGALTMPREGSTESESTQEDLEEEDEEDDGEEEEDCIQVKDEEGE SGAEEGPDLEEPGAGYKKLFSDAQPLQPLQVYQAPLSLATVPHQAL GRTQSSPAAPGGMKSPPDQPVKHLFTTGVVYDTFMLKHQCMCGNT HVHPEHAGRIQSIWSRLQETGLLSKCERIRGRKATLDEIQTVHSEYHT LLYGTSPLNRQKLDSKKLLGPISQKMYAVLPCGGIGVDSDTVWNEM HSSSAVRMAVGCLLELAFKVAAGELKNGFAIIRPPGHHAEESTAMGF

		CFFNSVAITAKLLQQKLNVGKVLIVDWDIHHGNGTQQAFYNDPSVL YISLHRYDNGNFFPGSGAPEEVGGGPGVGYNVNVAWTGGVDPPIGD VEYLTAFRTVVMPIAHEFSPDVVLVSAGFDAVEGHLSPLGGYSVTAR CFGHLTRQLMTLAGGRVVLALEGGHDLTAICDASEACVSALLSVEL QPLDEAVLQQKPNINAVATLEKVIEIQSKHWSCVQKFAAGLGRSLRE AQAGETEEAETVSAMALLSVGAEQAQAAAAAREHSPRPAEEPMEQEP AL
1132	(Hs)DNMT 1-Cas	MPARTAPARVPTLAVPAISLPDDVRRRLKDLERDSLTEKECVKEKLN LLHEFLQTEIKNQLCDLETKLRKEELSEEGYLAKVKSLLNKDLSLEN GAHAYNREVNGRLENGNQARSEARRVGMADANSPPKPLSKPRTPR RSKSDGEAKPEPSPSPRITRKSTRQTTITSHFAKGPAKRKPQEESERAK SDESIKEEDKDQDEKRRRVTSRERVARPLPAEEPERAKSGTRTEKEEE RDEKEEKRLRSQTKEPTPKQKLKEEPDREARAGVQADEDEDGDEKD EKKHRSQPKDLAAKRRPEEKEPEKVNPQISDEKDEDEKEEKRRKTTP KEPTEKKMARAKTVMNSKTHPPKCIQCGQYLDDPLKYGQHPPDAV DEPQMLTNEKLSIFDANESGFESYEALPQHKLTCFSVYCKHGHLCPID TGLIEKNIELFFSGSAKPIYDDDPSLEGGVNGKNLGPINEWWITGFDG GEKALIGFSTSFAEYILMDPSPEYAPIFGLMQEKIYISKIVVEFLQSNSD STYEDLINKIETTVPPSGLNLNRFTEDSLLRHAQFVVEQVESYDEAGD SDEQPIFLTPCMRDLIKLAGVTLGQRRAQARRQTIRHSTREKDRGPT KATTTKLVYQIFDTFFAEQIEKDDREDKENAFKRRRCGVCEVCQQPE CGKCKACKDMVKFGGSGRSKQACQERRCPNMAMKEADDDEEVDD NIPEMPSPKKMHQGKKKKQNKNRISWVGEAVKTDGKKSYYKKVCI DAETLEVGDCVSVIPDDSSKPLYLARVTALWEDSSNGQMFHAHWFC AGTDTVLGATSDPLELFLVDECEDMQLSYIHSKVKVIYKAPSENWA MEGGMDPESLLEGDDGKTYFYQLWYDQDYARFESPPKTQPTEDNK FKFCVSCARLAEMRQKEIPRVLEQLEDLDSRVLYYSATKNGILYRVG DGVYLPPEAFTFNIKLSSPVKRPRKEPVDEDLYPEHYRKYSDYIKGSN LDAPEPYRIGRIKEIFCPKKSNGRPNETDIKIRVNKFYRPENTHKSTPA SYHADINLLYWSDEEAVVDFKAVQGRCTVEYGEDLPECVQVYSMG GPNRFYFLEAYNAKSKSFEDPPNHARSPGNKGKGKGKGKGKPKSQA CEPSEPEIEIKLPKLRTLDVFSGCGGLSEGFHQAGISDTLWAIEMWDP AAQAFRLNNPGSTVFTEDCNILLKLVMAGETTNSRGQRLPQKGDVE MLCGGPPCQGFSGMNRFNSRTYSKFKNSLVVSFLSYCDYYRPRFFLL ENVRNFVSFKRSMVLKLTLRCLVRMGYQCTFGVLQAGQYGVAQTR RRAIILAAAPGEKLPLFPEPLHVFAPRACQLSVVVDDKKFVSNITRLS SGPFRTITVRDTMSDLPEVRNGASALEISYNGEPQSWFQRQLRGAQY QPILRDHICKDMSALVAARMRHIPLAPGSDWRDLPNIEVRLSDGTMA RKLRYTHHDRKNGRSSSGALRGVCSCVEAGKACDPAARQFNTLIPW CLPHTGNRHNHWAGLYGRLEWDGFFSTTVTNPEPMGKQGRVLHPE QHRVVSVRECARSQGFPDTYRLFGNILDKHRQVGNAVPPPLAKAIGL EIKLCMLAKARESASAKIKEEEAAKDGGPSSGAPPPSGGSPAGSPTST EEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAP GTSTEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKF KVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRI CYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVA YHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDL NPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRL ENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDT YDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLS ASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYID GGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPH QIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSR FAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKV LPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLF

		KTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKII KDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVM KQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFM QLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVK VVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGI KELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLS DYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMK NYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQI TKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYK VREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVR KMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGE TGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRN SDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSV KELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENG RKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQK QLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIR EQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSI TGLYETRIDLSQLGGDPKKKRKV
1133	(Hs)DNMT3A-Cas	MPAMPSSGPGDTSSSAAEREEDRKDGEEQEEPRGKEERQEPSTTARK VGRPGRKRKHPPVESGDTPKDPAVISKSPSMAQDSGASELLPNGDLE KRSEPQPEEGSPAGGQKGGAPAEGEGAAETLPEASRAVENGCCTPKE GRGAPAEAGKEQKETNIESMKMEGSRGRLRGGLGWESSLRQRPMPR LTFQAGDPYYISKRKRDEWLARWKREAEKKAKVIAGMNAVEENQG PGESQKVEEASPPAVQQPTDPASPTVATTPEPVGSDAGDKNATKAG DDEPEYEDGRGFGIGELVWGKLRGFSWWPGRIVSWWMTGRSRAAE GTRWVMWFGDGKFSVVCVEKLMPLSSFCSAFHQATYNKQPMYRK AIYEVLQVASSRAGKLFPVCHDSDESDTAKAVEVQNKPMIEWALGG FQPSGPKGLEPPEEEKNPYKEVYTDMWVEPEAAAYAPPPPAKKPRK STAEKPKVKEIIDERTRERLVYEVRQKCRNIEDICISCGSLNVTLEHPL FVGGMCQNCKNCFLECAYQYDDDGYQSYCTICCGGREVLMCGNNN CCRCFCVECVDLLVGPGAAQAAIKEDPWNCYMCGHKGTYGLLRRR EDWPSRLQMFFANNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIAT GLLVLKDLGIQVDRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVT QKHIQEWGPFDLVIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLH DARPKEGDDRPFFWLFENVVAMGVSDKRDISRFLESNPVMIDAKEV SAAHRARYFWGNLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVR TITTRSNSIKQGKDQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVS NMSRLARQRLLGRSWSVPVIRHLFAPLKEYFACVGGPSSGAPPPSGG SPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTS TEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITD EYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARR RYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPI FGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFR GHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILS ARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAED AKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRV NTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQS KNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQ RTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYY VGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTN FDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGE QKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNAS LGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTY AHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKS DGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPA

		IKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSR ERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYV DQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVP SEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFI KRQLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVS DFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVY GDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEI RKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTG GFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKV EKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIK LPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYE KLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVL SAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTST KEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1134	(Hs)DNMT3A(CD)-Cas	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVGGPSSGAPPPSGGSPAGSPTSTEEGT SESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTST EPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVL GNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYL QEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHE KYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPD NSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENL IAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYD DDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSAS MIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGG ASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQI HLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRF AWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVL PKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFK TNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIK DKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMK QLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQL IHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVV DELVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKE LGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDY DVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNY WRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITK HVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVR EINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMI AKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGE IVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDK LIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKEL LGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKR MLASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLF VEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQA ENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGL YETRIDLSQLGGDPKKKRKV
1135	(Hs/Hs)DNMT3A(CD	MNHDQEFDPPKVYPPVPAEKRKPIRVLSLFDGIATGLLVLKDLGIQV DRYIASEVCEDSITVGMVRHQGKIMYVGDVRSVTQKHIQEWGPFDL VIGGSPCNDLSIVNPARKGLYEGTGRLFFEFYRLLHDARPKEGDDRPF

	)/L(CD)-Cas	FWLFENVVAMGVSDKRDISRFLESNPVMIDAKEVSAAHRARYFWG NLPGMNRPLASTVNDKLELQECLEHGRIAKFSKVRTITTRSNSIKQGK DQHFPVFMNEKEDILWCTEMERVFGFPVHYTDVSNMSRLARQRLLG RSWSVPVIRHLFAPLKEYFACVSSGNSNANSRGPSFSSGLVPLSLRGS HNPLEMFETVPVWRRQPVRVLSLFEDIKKELTSLGFLESGSDPGQLK HVVDVTDTVRKDVEEWGPFDLVYGATPPLGHTCDRPPSWYLFQFH RLLQYARPKPGSPRPFFWMFVDNLVLNKEDLDVASRFLEMEPVTIPD VHGGSLQNAVRVWSNIPAIRSRHWALVSEEELSLLAQNKQSSKLAA KWPTKLVKNCFLPLREYFKYFSTELTSSLGGPSSGAPPPSGGSPAGSP TSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEG SAPGTSTEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPS KKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRK NRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVD EVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIE GDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSK SRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQL SKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEIT KAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGY AGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFD NGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPL ARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKN LPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKA IVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTY HDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLF DDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFA NRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGI LQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMK RIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELD INRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVV KKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLV ETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKD FQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKV YDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLI ETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESI LPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSK KLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLF ELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPE DNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKH RDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDA TLIHQSITGLYETRIDLSQLGGDPKKKRKV
1136	(Hs)DNMT3B-Cas	MKGDTRHLNGEEDAGGREDSILVNGACSDQSSDSPPILEAIRTPEIRG RRSSSRLSKREVSSLLSYTQDLTGDGDGEDGDGSDTPVMPKLFRETR TRSESPAVRTRNNNSVSSRERHRPSPRSTRGRQGRNHVDESPVEFPAT RSLRRRATASAGTPWPSPPSSYLTIDLTDDTEDTHGTPQSSSTPYARL AQDSQQGGMESPQVEADSGDGDSSEYQDGKEFGIGDLVWGKIKGFS WWPAMVVSWKATSKRQAMSGMRWVQWFGDGKFSEVSADKLVAL GLFSQHFNLATFNKLVSYRKAMYHALEKARVRAGKTFPSSPGDSLE DQLKPMLEWAHGGFKPTGIEGLKPNNTQPVVNKSKVRRAGSRKLES RKYENKTRRRTADDSATSDYCPAPKRLKTNCYNNGKDRGDEDQSR EQMASDVANNKSSLEDGCLSCGRKNPVSFHPLFEGGLCQTCRDRFL ELFYMYDDDGYQSYCTVCCEGRELLLCSNTSCCRCFCVECLEVLVG TGTAAEAKLQEPWSCYMCLPQRCHGVLRRRKDWNVRLQAFFTSDT GLEYEAPKLYPAIPAARRRPIRVLSLFDGIATGYLVLKELGIKVGKYV ASEVCEESIAVGTVKHEGNIKYVNDVRNITKKNIEEWGPFDLVIGGSP CNDLSNVNPARKGLYEGTGRLFFEFYHLLNYSRPKEGDDRPFFWMF

		ENVVAMKVGDKRDISRFLECNPVMIDAIKVSAAHRARYFWGNLPG MNRPVIASKNDKLELQDCLEYNRIAKLKKVQTITTKSNSIKQGKNQL FPVVMNGKEDVLWCTELERIFGFPVHYTDVSNMGRGARQKLLGRS WSVPVIRHLFAPLKDYFACEGGPSSGAPPPSGGSPAGSPTSTEEGTSE SATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEP SEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGN TDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEI FSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKY PTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSD VDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQ LPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDL DNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIK RYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQ EEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLG ELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWM TRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHS LLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRK VTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDF LDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKR RRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDD SLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDEL VKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKV
1137	(Hs)DNMT3B(CD)-Cas	MIRVLSLFDGIATGYLVLKELGIKVGKYVASEVCEESIAVGTVKHEG NIKYVNDVRNITKKNIEEWGPFDLVIGGSPCNDLSNVNPARKGLYEG TGRLFFEFYHLLNYSRPKEGDDRPFFWMFENVVAMKVGDKRDISRF LECNPVMIDAIKVSAAHRARYFWGNLPGMNRPVIASKNDKLELQDC LEYNRIAKLKKVQTITTKSNSIKQGKNQLFPVVMNGKEDVLWCTEL ERIFGFPVHYTDVSNMGRGARQKLLGRSWSVPVIRHLFAPLKDYFAC EGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAP GSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGL AIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSG ETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEE SFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKAD LRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEE NPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSL GLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLA AKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVR QQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEE LLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDN REKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDK GASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVT EGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSV

		EISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFE DREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDK QSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSL HEHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMAREN QTTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLY YLQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRS DKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAER GGLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIRE VKVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALI KKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMN FFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQV NIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTV AYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKG YKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYV NFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVI LADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFD TTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1138	(Hs/Mm)DNMT3B(CD)/L(CD)-Cas	MIRVLSLFDGIATGYLVLKELGIKVGKYVASEVCEESIAVGTVKHEG NIKYVNDVRNITKKNIEEWGPFDLVIGGSPCNDLSNVNPARKGLYEG TGRLFFEFYHLLNYSRPKEGDDRPFFWMFENVVAMKVGDKRDISRF LECNPVMIDAIKVSAAHRARYFWGNLPGMNRPVIASKNDKLELQDC LEYNRIAKLKKVQTITTKSNSIKQGKNQLFPVVMNGKEDVLWCTEL ERIFGFPVHYTDVSNMGRGARQKLLGRSWSVPVIRHLFAPLKDYFAC ESSGNSNANSRGPSFSSGLVPLSLRGSHMGPMEIYKTVSAWKRQPVR VLSLFRNIDKVLKSLGFLESGSGSGGGTLKYVEDVTNVVRRDVEKW GPFDLVYGSTQPLGSSCDRCPGWYMFQFHRILQYALPRQESQRPFFW IFMDNLLLTEDDQETTTRFLQTEAVTLQDVRGRDYQNAMRVWSNIP GLKSKHAPLTPKEEEYLQAQVRSRSKLDAPKVDLLVKNCLLPLREYF KYFSQNSLPLGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTS TEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVM DKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLI GALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDD SFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLV DSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQ TYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLF GNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQ YADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLT LLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILE KMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQED FYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPW NFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNE LTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILED IVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSR KLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQ VSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENI VIEMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQL QNEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSI DNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKF DNLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYD ENDKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNA VVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYF FYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRK VLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKY GGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPI

		DFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNEL ALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQIS EFSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPA AFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPK KKRKV
1139	(Hs)DNMT3-Cas	MAAIPALDPEAEPSMDVILVGSSELSSSVSPGTGRDLIAYEVKANQRN IEDICICCGSLQVHTQHPLFEGGICAPCKDKFLDALFLYDDDGYQSYC SICCSGETLLICGNPDCTRCYCFECVDSLVGPGTSGKVHAMSNWVCY LCLPSSRSGLLQRRRKWRSQLKAFYDRESENPLEMFETVPVWRRQP VRVLSLFEDIKKELTSLGFLESGSDPGQLKHVVDVTDTVRKDVEEW GPFDLVYGATPPLGHTCDRPPSWYLFQFHRLLQYARPKPGSPRPFFW MFVDNLVLNKEDLDVASRFLEMEPVTIPDVHGGSLQNAVRVWSNIP AIRSSRHWALVSEEELSLLAQNKQSSKLAAKWPTKLVKNCFLPLREY FKYFSTELTSSLGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGT STEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKV MDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKN LIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKV DDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKK LVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQL VQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKN GLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQI GDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHH QDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFI KPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILR RQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEET ITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFT VYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLK EDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENE DILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGW GRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKED IQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGR HKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPV ENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFL KDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLI TQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRM NTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHD AYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKA TAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDF ATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDW DPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSS FEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGEL QKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYL DEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLT NLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQ LGGDPKKKRKV
1140	(Mm)DNMT3L-Cas	MGSRETPSSCSKTLETLDLETSDSSSPDADSPLEEQWLKSSPALKEDS VDVVLEDCKEPLSPSSPPTGREMIRYEVKVNRRSIEDICLCCGTLQVY TRHPLFEGGLCAPCKDKFLESLFLYDDDGHQSYCTICCSGGTLFICES PDCTRCYCFECVDILVGPGTSERINAMACWVCFLCLPFSRSGLLQRR KRWRHQLKAFHDQEGAGPMEIYKTVSAWKRQPVRVLSLFRNIDKV LKSLGFLESGSGSGGGTLKYVEDVTNVVRRDVEKWGPFDLVYGSTQ PLGSSCDRCPGWYMFQFHRILQYALPRQESQRPFFWIFMDNLLLTED DQETTTRFLQTEAVTLQDVRGRDYQNAMRVWSNIPGLKSKHAPLTP KEEEYLQAQVRSRSKLDAPKVDLLVKNCLLPLREYFKYFSQNSLPLG GPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSP

		AGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIG TNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETA EATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFL VEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRL IYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPI NASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLT PNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKN LSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQL PEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLV KLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREK IEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGAS AQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEG MRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEI SGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFED REMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQ SGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLH EHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQ TTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYY LQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSD KNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERG GLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIREV KVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIK KYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFF KTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNI VKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVA YSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGY KEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVN FLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVIL ADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDT TIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1141	(Mm)DNMT3C-Cas	MRGGSRHLSNEEDVSGCEDCIIISGTCSDQSSDPKTVPLTQVLEAVCT VENRGCRTSSQPSKRKASSLISYVQDLTGDGDEDRDGEVGGSSGSGT PVMPQLFCETRIPSKTPAPLSWQANTSASTPWLSPASPYPIIDLTDEDV IPQSISTPSVDWSQDSHQEGMDTTQVDAESRDGGNIEYQVSADKLLL SQSCILAAFYKLVPYRESIYRTLEKARVRAGKACPSSPGESLEDQLKP MLEWAHGGFKPTGIEGLKPNKKQPENKSRRRTTNDPAASESSPPKRL KTNSYGGKDRGEDEESREQMASDVTNNKGNLEDHCLSCGRKDPVS FHPLFEGGLCQSCRDRFLELFYMYDEDGYQSYCTVCCEGRELLLCSN TSCCRCFCVECLEVLVGAGTAEDVKLQEPWSCYMCLPQRCHGVLRR RKDWNMRLQDFFTTDPDLEEFEPPKLYPAIPAAKRRPIRVLSLFDGIA TGYLVLKELGIKVEKYIASEVCAESIAVGTVKHEGQIKYVDDIRNITK EHIDEWGPFDLVIGGSPCNDLSCVNPVRKGLFEGTGRLFFEFYRLLN YSCPEEEDDRPFFWMFENVVAMEVGDKRDISRFLECNPVMIDAIKVS AAHRARYFWGNLPGMNRPVMASKNDKLELQDCLEFSRTAKLKKVQ TITTKSNSIRQGKNQLFPVVMNGKDDVLWCTELERIFGFPEHYTDVS NMGRGARQKLLGRSWSVPVIRHLFAPLKDHFACEGGPSSGAPPPSG GSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGT STEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITD EYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARR RYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPI FGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFR GHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILS ARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAED AKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRV NTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQS

		KNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQ RTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYY VGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTN FDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGE QKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNAS LGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTY AHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKS DGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPA IKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSR ERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYV DQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVP SEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFI KRQLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVS DFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVY GDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEI RKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTG GFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKV EKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIK LPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYE KLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVL SAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTST KEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1142	(Mm)DNMT3C(CD)-Cas	MIRVLSLFDGIATGYLVLKELGIKVEKYIASEVCAESIAVGTVKHEGQ IKYVDDIRNITKEHIDEWGPFDLVIGGSPCNDLSCVNPVRKGLFEGTG RLFFEFYRLLNYSCPEEEDDRPFFWMFENVVAMEVGDKRDISRFLEC NPVMIDAIKVSAAHRARYFWGNLPGMNRPVMASKNDKLELQDCLE FSRTAKLKKVQTITTKSNSIRQGKNQLFPVVMNGKDDVLWCTELERI FGFPEHYTDVSNMGRGARQKLLGRSWSVPVIRHLFAPLKDHFACEG GPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSP AGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIG TNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETA EATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFL VEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRL IYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPI NASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLT PNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKN LSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQL PEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLV KLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREK IEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGAS AQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEG MRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEI SGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFED REMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQ SGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLH EHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQ TTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYY LQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSD KNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERG GLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIREV KVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIK KYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFF KTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNI VKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVA YSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGY

		KEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVN FLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVIL ADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDT TIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1143	(Mm/Mm) DNMT3C(CD)/L(CD) -Cas	MIRVLSLFDGIATGYLVLKELGIKVEKYIASEVCAESIAVGTVKHEGQ IKYVDDIRNITKEHIDEWGPFDLVIGGSPCNDLSCVNPVRKGLFEGTG RLFFEFYRLLNYSCPEEEDDRPFFWMFENVVAMEVGDKRDISRFLEC NPVMIDAIKVSAAHRARYFWGNLPGMNRPVMASKNDKLELQDCLE FSRTAKLKKVQTITTKSNSIRQGKNQLFPVVMNGKDDVLWCTELERI FGFPEHYTDVSNMGRGARQKLLGRSWSVPVIRHLFAPLKDHFACESS GNSNANSRGPSFSSGLVPLSLRGSHMGPMEIYKTVSAWKRQPVRVLS LFRNIDKVLKSLGFLESGSGSGGGTLKYVEDVTNVVRRDVEKWGPF DLVYGSTQPLGSSCDRCPGWYMFQFHRILQYALPRQESQRPFFWIFM DNLLLTEDDQETTTRFLQTEAVTLQDVRGRDYQNAMRVWSNIPGLK SKHAPLTPKEEEYLQAQVRSRSKLDAPKVDLLVKNCLLPLREYFKYF SQNSLPLGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPS EGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKK YSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALL FDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFH RLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTD KADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQ LFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLI ALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYAD LFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLK ALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKM DGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYP FLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFE EVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTK VKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKI ECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVL TLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLI NGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVS GQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVI EMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQN EKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDN KVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDN LTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDEN DKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAV VGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFF YSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKV LSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYG GFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPID FLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELA LPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISE FSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAA FKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKK KRKV
1144	(Mp)M.Mp eI-Cas	MNSNKDKIKVIKVFEAFAGIGSQFKALKNIARSKNWEIQHSGMVEW FVDAIVSYVAIHSKNFNPKIEQLDKDILSISNDSKMPISEYGIKKINNTI KASYLNYAKKHFNNLFDIKKVNKDNFPKNIDIFTYSFPCQDLSVQGL QKGIDKELNTRSGLLWEIERILEEIKNSFSKEEMPKYLLMENVKNLLS HKNKKNYNTWLKQLEKFGYKSKTYLLNSKNFDNCQNRERVFCLSIR DDYLEKTGFKFKELEKVKNPPKKIKDILVDSSNYKYLNLNKYETTTF RETKSNIISRSLKNYTTFNSENYVYNINGIGPTLTASGANSRIKIETQQ GVRYLTPLECFKYMQFDVNDFKKVQSTNLISENKMIYIAGNSIPVKIL

		EAIFNTLEFVNNEEGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESG PGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKK RKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSI KKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEM AKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHL RKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLF IQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEK KNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLA QIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEH HQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKF IKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAIL RRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEE TITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYF TVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQL KEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEEN EDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGW GRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKED IQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGR HKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPV ENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFL KDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLI TQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRM NTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHD AYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKA TAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDF ATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDW DPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSS FEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGEL QKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYL DEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLT NLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQ LGGDPKKKRKV
1145	(Sm)M.Sss I-Cas	MSKVENKTKKLRVFEAFAGIGAQRKALEKVRKDEYEIVGLAEWYVP AIVMYQAIHNNFHTKLEYKSVSREEMIDYLENKTLSWNSKNPVSNG YWKRKKDDELKIIYNAIKLSEKEGNIFDIRDLYKRTLKNIDLLTYSFP CQDLSQQGIQKGMKRGSGTRSGLLWEIERALDSTEKNDLPKYLLME NVGALLHKKNEEELNQWKQKLESLGYQNSIEVLNAADFGSSQARRR VFMISTLNEFVELPKGDKKPKSIKKVLNKIVSEKDILNNLLKYNLTEF KKTKSNINKASLIGYSKFNSEGYVYDPEFTGPTLTASGANSRIKIKDG SNIRKMNSDETFLYIGFDSQDGKRVNEIEFLTENQKIFVCGNSISVEVL EAIIDKIGGGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTE PSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMD KKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIG ALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDS FFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVD STDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQT YNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFG NLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQY ADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTL LKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEK MDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDF YPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWN FEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNEL TKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFK KIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDI

		VLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRK LINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQV SGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIV IEMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQ NEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSID NKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFD NLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDE NDKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNA VVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYF FYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRK VLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKY GGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPI DFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNEL ALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQIS EFSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPA AFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPK KKRKV
1146	(Hp)M.HpaII-Cas	MKDVLDDNLLEEPAAQYSLFEPESNPNLREKFTFIDLFAGIGGFRIAM QNLGGKCIFSSEWDEQAQKTYEANFGDLPYGDITLEETKAFIPEKFDI LCAGFPCQAFSIAGKRGGFEDTRGTLFFDVAEIIRRHQPKAFFLENVK GLKNHDKGRTLKTILNVLREDLGYFVPEPAIVNAKNFGVPQNRERIY IVGFHKSTGVNSFSYPEPLDKIVTFADIREEKTVPTKYYLSTQYIDTLR KHKERHESKGNGFGYEIIPDDGIANAIVVGGMGRERNLVIDHRITDFT PTTNIKGEVNREGIRKMTPREWARLQGFPDSYVIPVSDASAYKQFGN SVAVPAIQATGKKILEKLGNLYDGGPSSGAPPPSGGSPAGSPTSTEEG TSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTS TEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVL GNTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYL QEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHE KYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPD NSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENL IAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYD DDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSAS MIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGG ASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQI HLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRF AWMTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVL PKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFK TNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIK DKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMK QLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQL IHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVV DELVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKE LGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDY DVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNY WRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITK HVAQILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVR EINNYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMI AKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGE IVWDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDK LIARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKEL LGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKR MLASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLF VEQHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQA

		ENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGL YETRIDLSQLGGDPKKKRKV
1147	(Al)M.AluI -Cas	MSKANAKYSFVDLFAGIGGFHAALAATGGVCEYAVEIDREAAAVYE RNWNKPALGDITDDANDEGVTLRGYDGPIDVLTGGFPCQPFSKSGA QHGMAETRGTLFWNIARIIEEREPTVLILENVRNLVGPRHRHEWLTII ETLRFFGYEVSGAPAIFSPHLLPAWMGGTPQVRERVFITATLVPERM RDERIPRTETGEIDAEAIGPKPVATMNDRFPIKKGGTELFHPGDRKSG WNLLTSGIIREGDPEPSNVDLRLTETETLWIDAWDDLESTIRRATGRP LEGFPYWADSWTDFRELSRLVVIRGFQAPEREVVGDRKRYVARTDM PEGFVPASVTRPAIDETLPAWKQSHLRRNYDFFERHFAEVVAWAYR WGVYTDLFPASRRKLEWQAQDAPRLWDTVMHFRPSGIRAKRPTYL PALVAITQTSIVGPLERRLSPRETARLQGLPEWFDFGEQRAAATYKQ MGNGVNVGVVRHILREHVRRDRALLKLTPAGQRIINAVLADEPDAT VGALGAAEGGPSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTST EPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVM DKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLI GALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDD SFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLV DSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQ TYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLF GNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQ YADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLT LLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILE KMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQED FYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPW NFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNE LTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYF KKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILED IVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSR KLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQ VSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENI VIEMARENQTTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQL QNEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSI DNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKF DNLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYD ENDKLIREVKVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNA VVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYF FYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRK VLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKY GGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPI DFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNEL ALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQIS EFSKRVILADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPA AFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPK KKRKV
1148	(Al)M.AluI -del82-Cas	MSKANAKYSFVDLFAGIGGFHAALAATGGVCEYAVEIDREAAAVYE RNWNKPALGDITDDANDEGVTLRGYDGPIDVLTGGFPCQPFSKSGA QHGMAETRGTLFWNIARIIEEREPTVLILENVRNLVGPRHRHEWLTII ETLRFFGYEVSGAPAIFSPHLLPAWMGGTPQVRERVFITATLVPERM RDERSTIRRATGRPLEGFPYWADSWTDFRELSRLVVIRGFQAPEREV VGDRKRYVARTDMPEGFVPASVTRPAIDETLPAWKQSHLRRNYDFF ERHFAEVVAWAYRWGVYTDLFPASRRKLEWQAQDAPRLWDTVMH FRPSGIRAKRPTYLPALVAITQTSIVGPLERRLSPRETARLQGLPEWFD FGEQRAAATYKQMGNGVNVGVVRHILREHVRRDRALLKLTPAGQR IINAVLADEPDATVGALGAAEGGPSSGAPPPSGGSPAGSPTSTEEGTS

		ESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTE PSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLG NTDRHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQ EIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEK YPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNS DVDKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIA QLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDD LDNLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMI KRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGAS QEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHL GELHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAW MTRKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKH SLLYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNR KVTVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKD FLDNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLK RRRYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHD DSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDE LVKVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGS QILKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVD AIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQ LLNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVA QILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREIN NYHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAK SEQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIV WDKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLI ARKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLG ITIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKV
1149	(Ha)M.Hae III-Cas	MNLISLFSGAGGLDLGFQKAGFRIICANEYDKSIWKTYESNHSAKLIK GDISKISSDEFPKCDGIIGGPPCQSWSEGGSLRGIDDPRGKLFYEYIRIL KQKKPIFFLAENVKGMMAQRHNKAVQEFIQEFDNAGYDVHIILLNA NDYGVAQDRKRVFYIGFRKELNINYLPPIPHLIKPTFKDVIWDLKDNP IPALDKNKTNGNKCIYPNHEYFIGSYSTIFMSRNRVRQWNEPAFTVQ ASGRQCQLHPQAPVMLKVSKNLNKFVEGKEHLYRRLTVRECARVQ GFPDDFIFHYESLNDGYKMIGNAVPVNLAYEIAKTIKSALEICKGNGG PSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPA GSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGT NSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAE ATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLV EEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLI YLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPIN ASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTP NFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNL SDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLP EKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVK LNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKI EKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGAS AQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEG MRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEI SGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFED REMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQ SGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLH

		EHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQ TTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYY LQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSD KNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERG GLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIREV KVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIK KYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFF KTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNI VKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVA YSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGY KEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVN FLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVIL ADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDT TIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1150	(Ha)M.HaeIII-T29-Cas	MNLISLFSGAGGLDLGFQKAGFRIICANEYDKSIWKTYESNHSAKLIK GDISKISSDEFPKCDGIIGGPPCQSWSEGGSLRGIDDPRGKLFYEYIRIL KQKKPIFFLAENVKGMMAQRHNKAVQEFIQEFDNAGYDVHIILLNA NDYGVAQDRKRVFYIGFRKELNINYLPPIPHLIKPTFKDVIWDLKDNP IPALDKNKTNGNKCIYPNHEYFIGSYSTIFMSANRVRQWNEPAFTVQ ASGRQCQLHPQAPVMLKVSKLMWKFVEGKEHLYRRLTVRECARVQ GFPDDFIFHYESLNDGYKMIGNAVPVNLAYEIAKTIKSALEICKGNGG PSSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPA GSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGT NSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAE ATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLV EEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLI YLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPIN ASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTP NFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNL SDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLP EKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVK LNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKI EKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGAS AQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEG MRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEI SGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFED REMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQ SGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLH EHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQ TTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYY LQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSD KNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERG GLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIREV KVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIK KYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFF KTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNI VKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVA YSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGY KEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVN FLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVIL ADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDT TIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1151	(Hh)M.HhaI-Cas	MIEIKDKQLTGLRFIDLFAGLGGFRLALESCGAECVYSNEWDKYAQE VYEMNFGEKPEGDITQVNEKTIPDHDILCAGFPCQAFSISGKQKGFED SRGTLFFDIARIVREKKPKVVFMENVKNFASHDNGNTLEVVKNTMN

		ELDYSFHAKVLNALDYGIPQKRERIYMICFRNDLNIQNFQFPKPFELN TFVKDLLLPDSEVEHLVIDRKDLVMTNQEIEQTTPKTVRLGIVGKGG QGERIYSTRGIAITLSAYGGGIFAKTGGYLVNGKTRKLHPRECARVM GYPDSYKVHPSTSQAYKQFGNSVVINVLQYIAYNIGSSLNFKPYGGP SSGAPPPSGGSPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPA GSPTSTEEGTSTEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGT NSVGWAVITDEYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAE ATRLKRTARRRYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLV EEDKKHERHPIFGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLI YLALAHMIKFRGHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPIN ASGVDAKAILSARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTP NFKSNFDLAEDAKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNL SDAILLSDILRVNTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLP EKYKEIFFDQSKNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVK LNREDLLRKQRTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKI EKILTFRIPYYVGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGAS AQSFIERMTNFDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEG MRKPAFLSGEQKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEI SGVEDRFNASLGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFED REMIEERLKTYAHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQ SGKTILDFLKSDGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLH EHIANLAGSPAIKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQ TTQKGQKNSRERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYY LQNGRDMYVDQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSD KNRGKSDNVPSEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERG GLSELDKAGFIKRQLVETRQITKHVAQILDSRMNTKYDENDKLIREV KVITLKSKLVSDFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIK KYPKLESEFVYGDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFF KTEITLANGEIRKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNI VKKTEVQTGGFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVA YSVLVVAKVEKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGY KEVKKDLIIKLPKYSLFELENGRKRMLASAGELQKGNELALPSKYVN FLYLASHYEKLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVIL ADANLDKVLSAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDT TIDRKRYTSTKEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1152	(Ms)M.MspI-Cas	MKPEILKLIRSKLDLTQKQASEIIEVSDKTWQQWESGKTEMHPAYYS FLQEKLKDKINFEELSAQKTLQKKIFDKYNQNQITKNAEELAEITHIE ERKDAYSSDFKFIDLFSGIGGIRQSFEVNGGKCVFSSEIDPFAKFTYYT NFGVVPFGDITKVEATTIPQHDILCAGFPCQPFSHIGKREGFEHPTQGT MFHEIVRIIETKKTPVLFLENVPGLINHDDGNTLKVIIETLEDMGYKV HHTVLDASHFGIPQKRKRFYLVAFLNQNIHFEFPKPPMISKDIGEVLE SDVTGYSISEHLQKSYLFKKDDGKPSLIDKNTTGAVKTLVSTYHKIQ RLTGTFVKDGETGIRLLTTNECKAIMGFPKDFVIPVSRTQMYRQMGN SVVVPVVTKIAEQISLALKTVNQQSPQENFELELVGGPSSGAPPPSGG SPAGSPTSTEEGTSESATPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTS TEPSEGSAPGTSTEPSEPKKKRKVMDKKYSIGLAIGTNSVGWAVITD EYKVPSKKFKVLGNTDRHSIKKNLIGALLFDSGETAEATRLKRTARR RYTRRKNRICYLQEIFSNEMAKVDDSFFHRLEESFLVEEDKKHERHPI FGNIVDEVAYHEKYPTIYHLRKKLVDSTDKADLRLIYLALAHMIKFR GHFLIEGDLNPDNSDVDKLFIQLVQTYNQLFEENPINASGVDAKAILS ARLSKSRRLENLIAQLPGEKKNGLFGNLIALSLGLTPNFKSNFDLAED AKLQLSKDTYDDDLDNLLAQIGDQYADLFLAAKNLSDAILLSDILRV NTEITKAPLSASMIKRYDEHHQDLTLLKALVRQQLPEKYKEIFFDQS KNGYAGYIDGGASQEEFYKFIKPILEKMDGTEELLVKLNREDLLRKQ RTFDNGSIPHQIHLGELHAILRRQEDFYPFLKDNREKIEKILTFRIPYY

		VGPLARGNSRFAWMTRKSEETITPWNFEEVVDKGASAQSFIERMTN FDKNLPNEKVLPKHSLLYEYFTVYNELTKVKYVTEGMRKPAFLSGE QKKAIVDLLFKTNRKVTVKQLKEDYFKKIECFDSVEISGVEDRFNAS LGTYHDLLKIIKDKDFLDNEENEDILEDIVLTLTLFEDREMIEERLKTY AHLFDDKVMKQLKRRRYTGWGRLSRKLINGIRDKQSGKTILDFLKS DGFANRNFMQLIHDDSLTFKEDIQKAQVSGQGDSLHEHIANLAGSPA IKKGILQTVKVVDELVKVMGRHKPENIVIEMARENQTTQKGQKNSR ERMKRIEEGIKELGSQILKEHPVENTQLQNEKLYLYYLQNGRDMYV DQELDINRLSDYDVDAIVPQSFLKDDSIDNKVLTRSDKNRGKSDNVP SEEVVKKMKNYWRQLLNAKLITQRKFDNLTKAERGGLSELDKAGFI KRQLVETRQITKHVAQILDSRMNTKYDENDKLIREVKVITLKSKLVS DFRKDFQFYKVREINNYHHAHDAYLNAVVGTALIKKYPKLESEFVY GDYKVYDVRKMIAKSEQEIGKATAKYFFYSNIMNFFKTEITLANGEI RKRPLIETNGETGEIVWDKGRDFATVRKVLSMPQVNIVKKTEVQTG GFSKESILPKRNSDKLIARKKDWDPKKYGGFDSPTVAYSVLVVAKV EKGKSKKLKSVKELLGITIMERSSFEKNPIDFLEAKGYKEVKKDLIIK LPKYSLFELENGRKRMLASAGELQKGNELALPSKYVNFLYLASHYE KLKGSPEDNEQKQLFVEQHKHYLDEIIEQISEFSKRVILADANLDKVL SAYNKHRDKPIREQAENIIHLFTLTNLGAPAAFKYFDTTIDRKRYTST KEVLDATLIHQSITGLYETRIDLSQLGGDPKKKRKV
1153	(Ai)Masc1-Cas	MSERRYEAGMTVALHEGSFLKIQRVYIRQYHADNRREHMLVGPLFR RTKYLKALSKKVNEVAIVHESIHVPVQDVIGVRELIITNRPFPECRKG DEHTGRLVCRWVYNLDERAKGREYKKQRYIRRITEAEADPEYRVED RVLRRRWFQEGYIGDEISYKEHGNGDIVDIRSESPLQVLDGWGGDLV DLENGEETSIPGPCRSASSYGRLMKPPLAQAADSNTSRKYTFGDTFC GGGGVSLGARQAGLEVKWAFDMNPNAGANYRRNFPNTDFFLAEAE QFIQLSVGISQHVDILHLSPPCQTFSRAHTIAGKNDENNEASFFAVVN LIKAVRPRLFTVEETDGIMDRQSRQFIDTALMGITELGYSFRICVLNAI EYGVCQNRKRLIIIGAAPGEELPPFPLPTHQDFFSKDPRRDLLPAVTLD DALSTITPESTDHHLNHVWQPAEWKTPYDAHRPFKNAIRAGGGEYD IYPDGRRKFTVRELACIQGFPDEYEFVGTLTDKRRIIGNAVPPPLSAAI MSTLRQWMTEKDFERMEGGPSSGAPPPSGGSPAGSPTSTEEGTSESA TPESGPGTSTEPSEGSAPGSPAGSPTSTEEGTSTEPSEGSAPGTSTEPSE PKKKRKVMDKKYSIGLAIGTNSVGWAVITDEYKVPSKKFKVLGNTD RHSIKKNLIGALLFDSGETAEATRLKRTARRRYTRRKNRICYLQEIFS NEMAKVDDSFFHRLEESFLVEEDKKHERHPIFGNIVDEVAYHEKYPT IYHLRKKLVDSTDKADLRLIYLALAHMIKFRGHFLIEGDLNPDNSDV DKLFIQLVQTYNQLFEENPINASGVDAKAILSARLSKSRRLENLIAQL PGEKKNGLFGNLIALSLGLTPNFKSNFDLAEDAKLQLSKDTYDDDLD NLLAQIGDQYADLFLAAKNLSDAILLSDILRVNTEITKAPLSASMIKR YDEHHQDLTLLKALVRQQLPEKYKEIFFDQSKNGYAGYIDGGASQE EFYKFIKPILEKMDGTEELLVKLNREDLLRKQRTFDNGSIPHQIHLGE LHAILRRQEDFYPFLKDNREKIEKILTFRIPYYVGPLARGNSRFAWMT RKSEETITPWNFEEVVDKGASAQSFIERMTNFDKNLPNEKVLPKHSL LYEYFTVYNELTKVKYVTEGMRKPAFLSGEQKKAIVDLLFKTNRKV TVKQLKEDYFKKIECFDSVEISGVEDRFNASLGTYHDLLKIIKDKDFL DNEENEDILEDIVLTLTLFEDREMIEERLKTYAHLFDDKVMKQLKRR RYTGWGRLSRKLINGIRDKQSGKTILDFLKSDGFANRNFMQLIHDDS LTFKEDIQKAQVSGQGDSLHEHIANLAGSPAIKKGILQTVKVVDELV KVMGRHKPENIVIEMARENQTTQKGQKNSRERMKRIEEGIKELGSQI LKEHPVENTQLQNEKLYLYYLQNGRDMYVDQELDINRLSDYDVDAI VPQSFLKDDSIDNKVLTRSDKNRGKSDNVPSEEVVKKMKNYWRQL LNAKLITQRKFDNLTKAERGGLSELDKAGFIKRQLVETRQITKHVAQ ILDSRMNTKYDENDKLIREVKVITLKSKLVSDFRKDFQFYKVREINN YHHAHDAYLNAVVGTALIKKYPKLESEFVYGDYKVYDVRKMIAKS

		EQEIGKATAKYFFYSNIMNFFKTEITLANGEIRKRPLIETNGETGEIVW DKGRDFATVRKVLSMPQVNIVKKTEVQTGGFSKESILPKRNSDKLIA RKKDWDPKKYGGFDSPTVAYSVLVVAKVEKGKSKKLKSVKELLGI TIMERSSFEKNPIDFLEAKGYKEVKKDLIIKLPKYSLFELENGRKRML ASAGELQKGNELALPSKYVNFLYLASHYEKLKGSPEDNEQKQLFVE QHKHYLDEIIEQISEFSKRVILADANLDKVLSAYNKHRDKPIREQAEN IIHLFTLTNLGAPAAFKYFDTTIDRKRYTSTKEVLDATLIHQSITGLYE TRIDLSQLGGDPKKKRKV
1157	Zinc Finger Array	SRPGERPFQCRICMRNFSNNNNNNNHTRTHTGEKPFQCRICMRNFSN NNNNNNHLRTH[linker]FQCRICMRNFSNNNNNNNHTRTHTGEKPFQ CRICMRNFSNNNNNNNHLRTH[linker]FQCRICMRNFSNNNNNNNHT RTHTGEKPFQCRICMRNFSNNNNNNNHLRTHLRGS

CLAIMS

WHAT IS CLAIMED IS:

1. An epigenetic editor comprising a fusion protein, wherein the fusion protein comprises:
   (a) a first DNMT domain;
   (b) a DNA binding domain;
   (c) a first repressor domain; and
   (d) a second repressor domain.
2. The epigenetic editor of claim 1, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene.
3. The epigenetic editor of claim 1, wherein the repressor domain specifically binds to an epigenetic effector protein in a cell comprising a target gene and directs the epigenetic editor to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene.
4. The epigenetic editor of claim 1, wherein the fusion protein further comprises a second DNMT domain.
5. The epigenetic editor of claim 1, wherein the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain.
6. The epigenetic editor of claim 5, wherein the first DNMT domain is the DNMT3A domain.
7. The epigenetic editor of claim 5, wherein the first DNMT domain is the DNMT3L domain.
8. The epigenetic editor of claim 1, wherein the first DNMT domain is a human DNMT domain.
9. The epigenetic editor of claim 8, wherein the human DNMT domain is a human DNMT3A domain.
10. The epigenetic editor of claim 8, wherein the human DNMT domain is a human DNMT3L domain.
11. The epigenetic editor of claim 1, wherein the first DNMT domain is a mouse DNMT domain.
12. The epigenetic editor of claim 11, wherein the mouse DNMT domain is a mouse DNMT3A domain.
13. The epigenetic editor of claim 11, wherein the mouse DNMT domain is a mouse DNMT3L domain.
14. The epigenetic editor of claim 4, wherein the first DNMT domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain.

15. The epigenetic editor of claim 14, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain.
16. The epigenetic editor of claim 14, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.
17. The epigenetic editor of claim 14, wherein the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain.
18. The epigenetic editor of claim 14, wherein the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.
19. The epigenetic editor of claim 1, wherein the first DNMT domain is a catalytic portion of a DNMT domain.
20. The epigenetic editor of claim 1, wherein the second DNMT domain is a catalytic portion of a DNMT domain.
21. The epigenetic editor of claim 1, wherein the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.
22. The epigenetic editor of claim 1, wherein at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320, ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607, ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100, ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677,

ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234, ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2, ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1, RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1, ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2, and fragments thereof.

23. The epigenetic editor of claim 22, wherein at least one of the repressor domains is selected from the group consisting of: SEQ ID NO: 67-595.

24. The epigenetic editor of claim 22, wherein at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.
25. The epigenetic editor of claim 1, wherein one of the repressor domains is a KRAB domain.
26. The epigenetic editor of claim 25, wherein the KRAB domain is a KOX1 KRAB domain.
27. The epigenetic editor of claim 1, wherein the DNA binding domain comprises a zinc finger motif.
28. The epigenetic editor of claim 1, wherein the DNA binding domain comprises a zinc finger array.
29. The epigenetic editor of claim 1, wherein the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide.
30. The epigenetic editor of claim 29, wherein the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide.
31. The epigenetic editor of claim 29, wherein the guide polynucleotide hybridizes with a target sequence.
32. The epigenetic editor of claim 30 or 31, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9).
33. The epigenetic editor of claim 32, wherein the dCas9 is a dSpCas9.
34. The epigenetic editor of claim 32, wherein the dSpCas9 is defined as SEQ ID NO: 3.
35. The epigenetic editor of claim 30 or 31, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a).
36. The epigenetic editor of claim 30 or 31, wherein the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).
37. The epigenetic editor of claim 1, wherein the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-dSpCas9-KOX1KRAB-the second repressor domain.
38. The epigenetic editor of claim 1, wherein a linker connects the domains of the fusion protein.
39. The epigenetic editor of claim 38, wherein the linker is an XTEN linker.
40. The epigenetic editor of claim 39, wherein the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80.
41. The epigenetic editor of claim 37, wherein the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-KOX1KRAB- XTEN18-the second repressor domain.
42. An epigenetic editor comprising a fusion protein, wherein the fusion protein comprises:

(a) a first DNMT domain;

(b) a DNA binding domain; and

(c) a repressor domain,

wherein the repressor domain is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320, ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607, ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100, ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677, ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234, ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2,

ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1, RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1, ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2 and fragments thereof.

43. The epigenetic editor of claim 42, wherein at least one of the repressor domains is selected from the group consisting of: SEQ ID NO: 67-595.

44. The epigenetic editor of claim 42, wherein the DNA binding domain binds to a target sequence in a target chromosome comprising a target gene.

45. The epigenetic editor of claim 42, wherein the repressor domain specifically binds to an epigenetic effector protein in a cell comprising a target gene and directs the epigenetic editor to the target gene to effect an epigenetic modification in a nucleotide in the target gene or a histone bound to the target gene.

46. The epigenetic editor of claim 42, wherein the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.

47. The epigenetic editor of claim 42, wherein the fusion protein further comprises a second DNMT domain.

48. The epigenetic editor of claim 42, wherein the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain.
49. The epigenetic editor of claim 48, wherein the first DNMT domain is the DNMT3A domain.
50. The epigenetic editor of claim 48, wherein the first DNMT domain is the DNMT3L domain.
51. The epigenetic editor of claim 42, wherein the first DNMT domain is a human DNMT domain.
52. The epigenetic editor of claim 51, wherein the first human DNMT domain is a human DNMT3A domain.
53. The epigenetic editor of claim 52, wherein the human DNMT domain is a human DNMT3L domain.
54. The epigenetic editor of claim 42, wherein the first DNMT domain is a mouse DNMT domain.
55. The epigenetic editor of claim 54, wherein the mouse DNMT domain is a mouse DNMT3A domain.
56. The epigenetic editor of claim 54, wherein the mouse DNMT domain is a mouse DNMT3L domain.
57. The epigenetic editor of claim 44, wherein the first DNMT domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain.
58. The epigenetic editor of claim 57, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain.
59. The epigenetic editor of claim 57, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.
60. The epigenetic editor of claim 57, wherein the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain.
61. The epigenetic editor of claim 57, wherein the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.
62. The epigenetic editor of claim 42, wherein the first DNMT domain is a catalytic portion of the DNMT domain.
63. The epigenetic editor of claim 42, wherein the second DNMT domain is a catalytic portion of a DNMT domain.
64. The epigenetic editor of claim 42, wherein the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.

65. The epigenetic editor of claim 42, wherein the DNA binding domain comprises a zinc finger motif.
66. The epigenetic editor of claim 42, wherein the DNA binding domain comprises a zinc finger array.
67. The epigenetic editor of claim 42, wherein the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide.
68. The epigenetic editor of claim 67, wherein the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide.
69. The epigenetic editor of claim 67, wherein the guide polynucleotide hybridizes with a target sequence.
70. The epigenetic editor of claim 68 or 69, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9).
71. The epigenetic editor of claim 70, wherein the dCas9 is a dSpCas9.
72. The epigenetic editor of claim 71, wherein the dSpCas9 is defined as SEQ ID NO: 3.
73. The epigenetic editor of claim 68 or 69, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a).
74. The epigenetic editor of claim 68 or 69, wherein the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).
75. The epigenetic editor of claim 42, wherein the fusion protein domain comprises from N-terminus to C-terminus DNMT3A-DNMT3L-dSpCas9-the repressor domain.
76. The epigenetic editor of claim 42, wherein a linker connects the domains of the fusion protein.
77. The epigenetic editor of claim 76, wherein the linker is an XTEN linker.
78. The epigenetic editor of claim 77, wherein the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80.
79. The epigenetic editor of claim 75, wherein the fusion protein comprises from N-terminus to C-terminus: DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-the repressor domain.
80. An epigenetic editor comprising a fusion protein, wherein the fusion protein comprises:
    (a) a demethylase domain;
    (b) a DNA binding domain; and
    (c) an activator domain.
81. The epigenetic editor of claim 74, wherein there is increased expression of the target gene when contacted with the epigenetic editor of any of the preceding claims as compared to the target gene not contacted with the epigenetic editor.

82. An epigenetic editor comprising a fusion protein, wherein the fusion protein comprises:
    (a) a DNA binding domain;
    (b) a repressor domain;
    (c) a first catalytic domain wherein the catalytic domain is selected from the group consisting of a DNMT3A catalytic domain and a DNMT3L catalytic domain; and
    (d) a second catalytic domain wherein the catalytic domain is selected from the group consisting of a DNMT3A catalytic domain and a DNMT3L catalytic domain,
    wherein the first catalytic domain has less than 380 amino acids or
    wherein the second catalytic domain has less than 380 amino acids.
83. A method of modifying an epigenetic state of a target gene in a target chromosome, the method comprising contacting the target chromosome with an epigenetic editor, wherein the epigenetic editor comprises:
    (a) a first DNMT domain;
    (b) a DNA binding domain;
    (c) a first repressor domain; and
    (d) a second repressor domain, and
    wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby modifying the epigenetic state of the target gene.
84. A method of modulating expression of a target gene in a target chromosome, the method comprising contacting the target chromosome with an epigenetic editor, wherein the epigenetic editor comprises:
    (a) a first DNMT domain;
    (b) a DNA binding domain;
    (c) a first repressor domain; and
    (d) a second repressor domain, and
    wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby modulating the epigenetic state of the target gene.
85. A method of treating a disease in a subject in need thereof, the method comprising administering to the subject an epigenetic editor, wherein the epigenetic editor comprises:
    (a) a first DNMT domain;

(b) a DNA binding domain;

(c) a first repressor domain; and

(d) a second repressor domain,

wherein the DNA binding domain binds to a target sequence in the target chromosome and directs the epigenetic effector domain to effect a site-specific epigenetic modification in the target gene or a histone bound to the target gene in the target chromosome, thereby treating the disease,

wherein the target gene is associated with disease, and

wherein the site-specific epigenetic modification modulates expression of the target gene, thereby treating the disease.

86. The method of any one of claims 83-85, wherein the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of the target sequence.

87. The method of claim 86, wherein the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the target sequence.

88. The method of any one of claims 83-87, wherein the site-specific epigenetic modification is within 3000 base pairs upstream or downstream of an expression regulatory sequence.

89. The method of claim 88, wherein the site-specific epigenetic modification is within 2000 base pairs upstream or downstream of the expression regulatory sequence.

90. The method of claim 89, wherein the site-specific epigenetic modification is within 1000 base pairs upstream or downstream of the expression regulatory sequence.

91. The method of any one of claims 83-90, comprising administering to the subject a cell comprising the epigenetic editor.

92. The method of claim 91, wherein the cell is an allogeneic cell.

93. The method of claim 92, wherein the cell is an autologous cell.

94. The method of any one of claims 83-93, wherein the epigenetic modification is within a coding region of the target gene.

95. The method of any one of claims 83-94, wherein the target gene comprises an allele associated with a disease.

96. The method of any one of claims 83-95, wherein the fusion protein further comprises a second DNMT domain.

97. The method of any one of claims 83-96, wherein the first DNMT domain is selected from the group consisting of a DNMT3A domain, a DNMT3B domain, a DNMT3C domain, and a DNMT3L domain.

98. The method of any one of claims 83-97, wherein the first DNMT domain is the DNMT3A domain.

99. The method of any one of claims 83-98, wherein the first DNMT domain is the DNMT3L domain.

100. The method of any one of claims 83-99, wherein the first DNMT domain is a human DNMT domain.

101. The method of claim 100, wherein the human DNMT domain is a human DNMT3A domain.

102. The method of claim 100, wherein the human DNMT domain is a human DNMT3L domain.

103. The method of any one of claims 83-102, wherein the first DNMT domain is a mouse DNMT domain.

104. The method of claim 103, wherein the mouse DNMT domain is a mouse DNMT3A domain.

105. The method of claim 103, wherein the mouse DNMT domain is a mouse DNMT3L domain.

106. The method of any one of claims 83-105, wherein the first DNMT domain is a DNMT3A domain and the second DNMT domain is a DNMT3L domain.

107. The method of claim 96, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a human DNMT3L domain.

108. The method of claim 96, wherein the first DNMT domain is a human DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.

109. The method of claim 96, wherein the first DNMT domain is the mouse DNMT3A domain and the second DNMT domain is a human DNMT3L domain.

110. The method of claim 96, wherein the first DNMT domain is a mouse DNMT3A domain and the second DNMT domain is a mouse DNMT3L domain.

111. The method of any one of claims 83-110, wherein the first DNMT domain is a catalytic portion of a DNMT domain.

112. The method of any one of claims 83-111, wherein the second DNMT domain is a catalytic portion of a DNMT domain.

113. The method of any one of claims 83-112, wherein the first DNMT domain and the second DNMT domain are selected from the group consisting of SEQ ID NO: 32-66.

114. The method of any one of claims 83-113, wherein at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF436, ZNF257, ZNF675, ZNF490, ZNF320,

ZNF331, ZNF816, ZNF680, ZNF41, ZNF189, ZNF528, ZNF543, ZNF554, ZNF140, ZNF610, ZNF264, ZNF350, ZNF8, ZNF582, ZNF30, ZNF324, ZNF98, ZNF669, ZNF677, ZNF596, ZNF214, ZNF37A, ZNF34, ZNF250, ZNF547, ZNF273, ZNF354A, ZFP82, ZNF224, ZNF33A, ZNF45, ZNF175, ZNF595, ZNF184, ZNF419, ZFP28-1, ZFP28-2, ZNF18, ZNF213, ZNF394, ZFP1, ZFP14, ZNF416, ZNF557, ZNF566, ZNF729, ZIM2, ZNF254, ZNF764, ZNF785, ZNF10, CBX5, RYBP, YAF2, MGA, CBX1, SCMH1, MPP8, SUMO3, HERC2, BIN1, PCGF2, TOX, FOXA1, FOXA2, IRF2BP1, IRF2BP2, IRF2BPL IRF-2BP1_2 N-terminal domain, HOXA13, HOXB13, HOXC13, HOXA11, HOXC11, HOXC10, HOXA10, HOXB9, HOXA9, ZFP28, ZN334, ZN568, ZN37A, ZN181, ZN510, ZN862, ZN140, ZN208, ZN248, ZN571, ZN699, ZN726, ZIK1, ZNF2, Z705F, ZNF14, ZN471, ZN624, ZNF84, ZNF7, ZN891, ZN337, Z705G, ZN529, ZN729, ZN419, Z705A, ZNF45, ZN302, ZN486, ZN621, ZN688, ZN33A, ZN554, ZN878, ZN772, ZN224, ZN184, ZN544, ZNF57, ZN283, ZN549, ZN211, ZN615, ZN253, ZN226, ZN730, Z585A, ZN732, ZN681, ZN667, ZN649, ZN470, ZN484, ZN431, ZN382, ZN254, ZN124, ZN607, ZN317, ZN620, ZN141, ZN584, ZN540, ZN75D, ZN555, ZN658, ZN684, RBAK, ZN829, ZN582, ZN112, ZN716, HKR1, ZN350, ZN480, ZN416, ZNF92, ZN100, ZN736, ZNF74, CBX1, ZN443, ZN195, ZN530, ZN782, ZN791, ZN331, Z354C, ZN157, ZN727, ZN550, ZN793, ZN235, ZNF8, ZN724, ZN573, ZN577, ZN789, ZN718, ZN300, ZN383, ZN429, ZN677, ZN850, ZN454, ZN257, ZN264, ZFP82, ZFP14, ZN485, ZN737, ZNF44, ZN596, ZN565, ZN543, ZFP69, SUMO1, ZNF12, ZN169, ZN433, SUMO3, ZNF98, ZN175, ZN347, ZNF25, ZN519, Z585B, ZIM3, ZN517, ZN846, ZN230, ZNF66, ZFP1, ZN713, ZN816, ZN426, ZN674, ZN627, ZNF20, Z587B, ZN316, ZN233, ZN611, ZN556, ZN234, ZN560, ZNF77, ZN682, ZN614, ZN785, ZN445, ZFP30, ZN225, ZN551, ZN610, ZN528, ZN284, ZN418, MPP8, ZN490, ZN805, Z780B, ZN763, ZN285, ZNF85, ZN223, ZNF90, ZN557, ZN425, ZN229, ZN606, ZN155, ZN222, ZN442, ZNF91, ZN135, ZN778, RYBP, ZN534, ZN586, ZN567, ZN440, ZN583, ZN441, ZNF43, CBX5, ZN589, ZNF10, ZN563, ZN561, ZN136, ZN630, ZN527, ZN333, Z324B, ZN786, ZN709, ZN792, ZN599, ZN613, ZF69B, ZN799, ZN569, ZN564, ZN546, ZFP92,YAF2, ZN723, ZNF34, ZN439, ZFP57, ZNF19, ZN404, ZN274, CBX3, ZNF30, ZN250, ZN570, ZN675, ZN695, ZN548, ZN132, ZN738, ZN420, ZN626, ZN559, ZN460, ZN268, ZN304, ZIM2, ZN605, ZN844, SUMO5, ZN101, ZN783, ZN417, ZN182, ZN823, ZN177, ZN197, ZN717, ZN669, ZN256, ZN251, CBX4, PCGF2, CDY2, CDYL2, HERC2, ZN562, ZN461, Z324A, ZN766, ID2, TOX, ZN274, SCMH1, ZN214, CBX7, ID1, CREM, SCX, ASCL1, ZN764, SCML2, TWST1, CREB1, TERF1, ID3, CBX8, CBX4, GSX1, NKX22, ATF1, TWST2, ZNF17, TOX3, TOX4, ZMYM3, I2BP1,

RHXF1, SSX2, I2BPL, ZN680, CBX1, TRI68, HXA13, PHC3, TCF24, CBX3, HXB13, HEY1, PHC2, ZNF81, FIGLA, SAM11, KMT2B, HEY2,JDP2, HXC13, ASCL4, HHEX, HERC2, GSX2, BIN1, ETV7, ASCL3, PHC1, OTP, I2BP2, VGLL2, HXA11, PDLI4, ASCL2, CDX4, ZN860, LMBL4, PDIP3, NKX25, CEBPB, ISL1, CDX2, PROP1, SIN3B, SMBT1, HXC11, HXC10, PRS6A, VSX1, NKX23, MTG16, HMX3, HMX1, KIF22, CSTF2, CEBPE, DLX2, ZMYM3, PPARG, PRIC1,UNC4, BARX2, ALX3, TCF15, TERA, VSX2, HXD12, CDX1, TCF23, ALX1, HXA10, RX, CXXC5, SCML1, NFIL3, DLX6, MTG8, CBX8, CEBPD, SEC13, FIP1, ALX4, LHX3, PRIC2, MAGI3, NELL1, PRRX1, MTG8R, RAX2, DLX3, DLX1, NKX26, NAB1, SAMD7, PITX3,WDR5, MEOX2, NAB2, DHX8, FOXA2, CBX6, EMX2, CPSF6, HXC12, KDM4B, LMBL3, PHX2A, EMX1, NC2B, DLX4, SRY, ZN777, NELL1, ZN398, GATA3, BSH, SF3B4, TEAD1, TEAD3, RGAP1, PHF1, FOXA1, GATA2, FOXO3, ZN212, IRX4, ZBED6, LHX4, SIN3A, RBBP7, NKX61, TRI68, R51A1, MB3L1, DLX5, NOTC1, TERF2, ZN282, RGS12, ZN840, SPI2B, PAX7, NKX62, ASXL2, FOXO1, GATA3, GATA1, ZMYM5, ZN783, SPI2B, LRP1, MIXL1, SGT1, LMCD1, CEBPA, GATA2, SOX14,WTIP, PRP19, CBX6, NKX11, RBBP4, DMRT2, SMCA2 and fragments thereof.

115. The method of claim 114 wherein at least one of the repressor domains is selected from the group consisting of: SEQ ID NO: 67-595.

116. The method of claim 114, wherein at least one of the repressor domains is selected from the group consisting of: ZIM3, ZNF264, ZN577, ZN793, ZFP28, ZN627, RYBP, TOX, TOX3, TOX4, I2BP1, SCMH1, SCML2, CDYL2, CBX8, CBX5, and CBX1, and fragments thereof.

117. The method of any one of claims 83-116, wherein one of the repressor domains is a KRAB domain.

118. The method of claim 117, wherein the KRAB domain is a KOX1 KRAB domain.

119. The method of any one of claims 83-118, wherein the DNA binding domain comprises a zinc finger motif.

120. The method of any one of claims 83-119, wherein the DNA binding domain comprises a zinc finger array.

121. The method of any one of claims 83-120, wherein the DNA binding domain comprises a nucleic acid guided DNA binding domain bound to a guide polynucleotide.

122. The method of claim 121, wherein the DNA binding domain comprises CRISPR-Cas protein bound to the guide polynucleotide.

123. The method of claim 121, wherein the guide polynucleotide hybridizes with a target sequence.

124. The method of claim 122 or 123, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas9 (dCas9).

125. The method of claim 124, wherein the dCas9 is a dSpCas9.

126. The method of claim 122 or 123, wherein the CRISPR-Cas protein comprises a nuclease inactive Cas12a (dCas12a).

127. The epigenetic editor of claim 32, wherein the dSpCas9 is defined as SEQ ID NO: 3.

128. The method of claim 122 or 123, wherein the CRISPR-Cas protein comprises a nuclease inactive CasX (dCasX).

129. The method of any one of claims 83-128, wherein the fusion protein comprises from N-terminus to C-terminus DNMT3A-DNMT3L-dSpCas9-KOX1KRAB-the second repressor domain.

130. The method of any one of claims 83-129, wherein a linker connects the domains of the fusion protein.

131. The method of claim 130, wherein the linker is an XTEN linker.

132. The method of claim 131, wherein the XTEN linker is selected from the group consisting of: XTEN-16, XTEN-18, and XTEN-80.

133. The method of claim 129, wherein the fusion protein comprises from N-terminus to C-terminus DNMT3A-DNMT3L-XTEN80-dSpCas9- XTEN16-KOX1KRAB- XTEN18-the second repressor domain.

134. A composition for use in the treatment of a subject, the composition comprising a fusion protein, wherein the fusion protein comprises:

(a) a first DNMT domain;

(b) a DNA binding domain;

(c) a first repressor domain; and

(d) a second repressor domain.

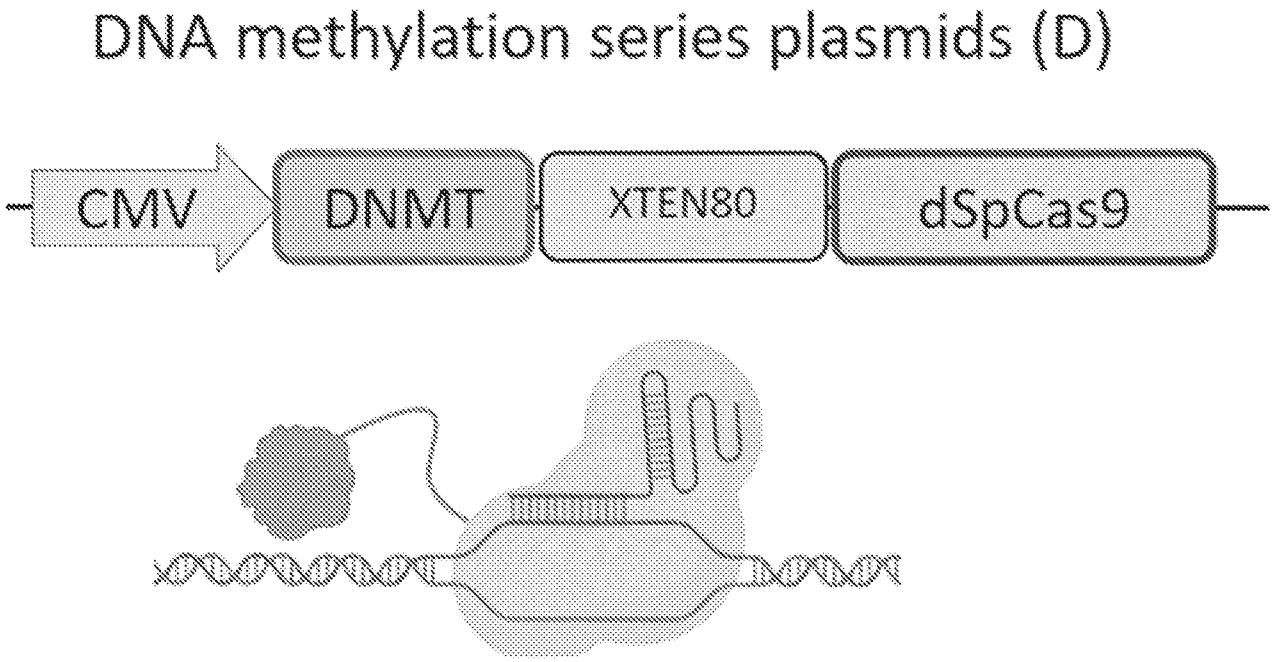

Fig. 1

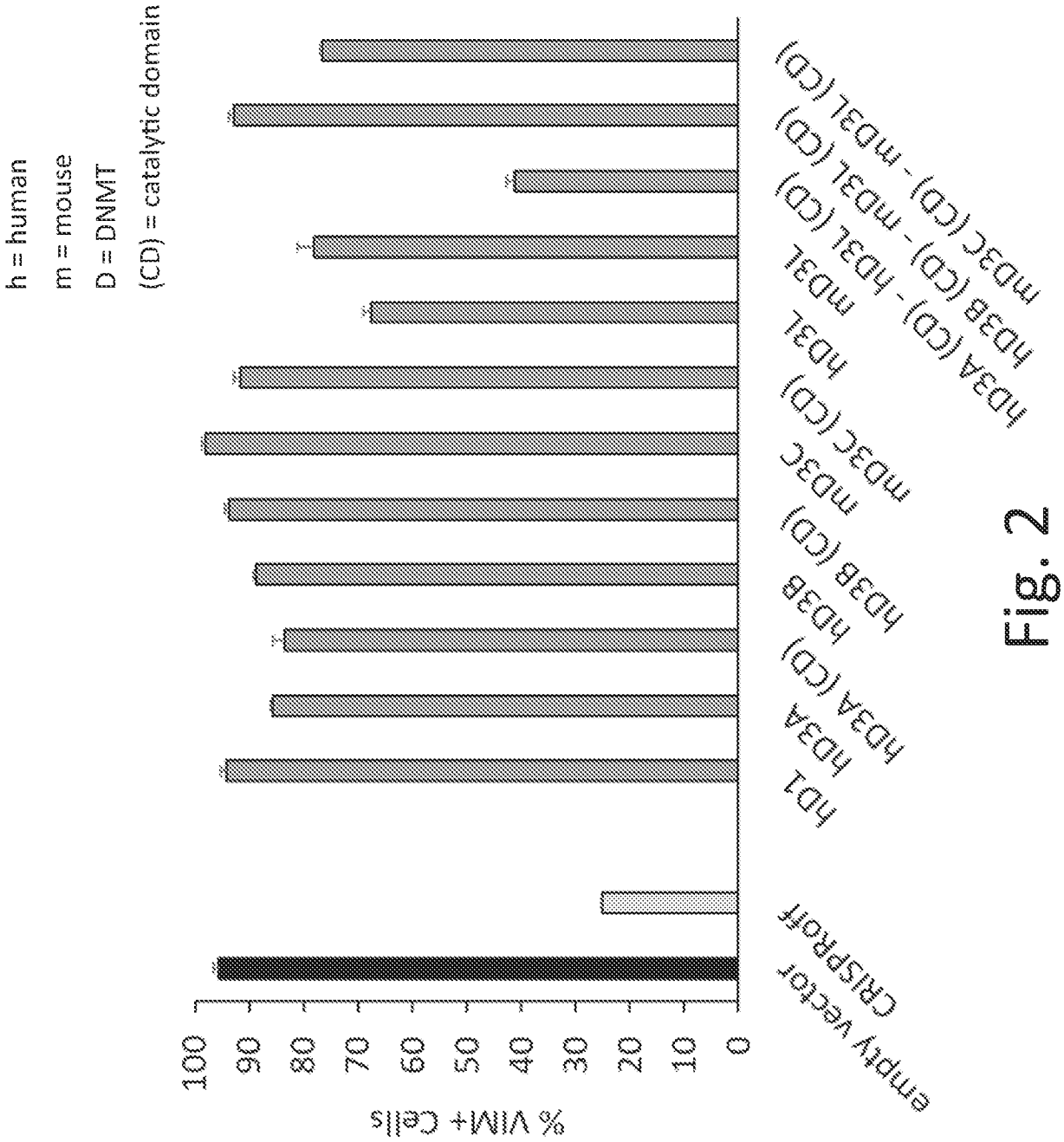
h = human
m = mouse
D = DNMT
(CD) = catalytic domain
% VIM+ Cells
100
90
80
70
60
50
40
30
20
10
0
empty vector
CRISPRoff
hD1
hD3A
hD3A (CD)
hD3B
hD3B (CD)
mD3C
mD3C (CD)
hD3L
mD3L
hD3A (CD) - hD3L (CD)
hD3B (CD) - hD3L (CD)
mD3C (CD) - mD3L (CD)

Fig. 2

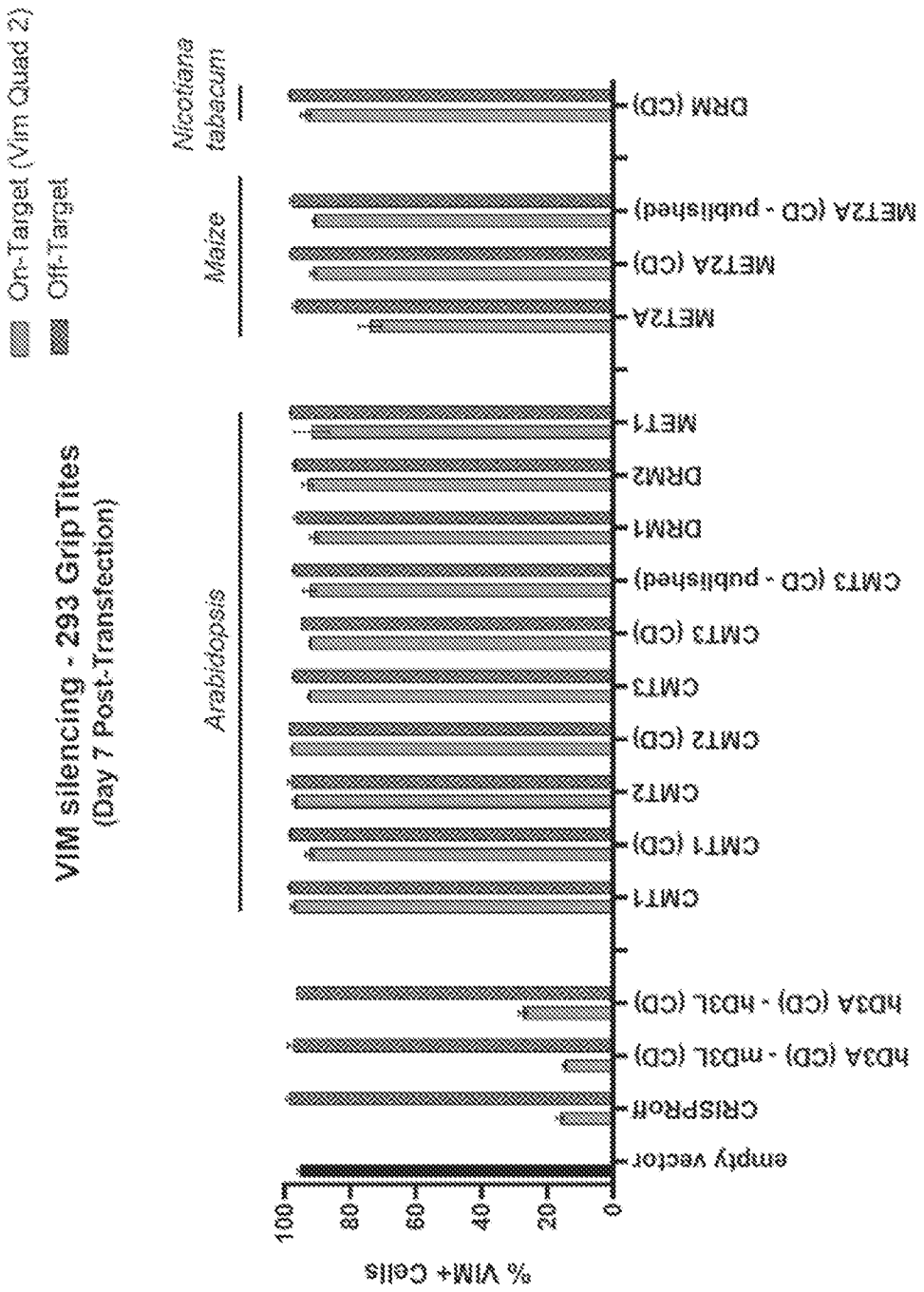
On-Target (Vim Quad 2)
Off-Target
VIM silencing - 293 GripTites
(Day 7 Post-Transfection)
% VIM+ cells
100
80
60
40
20
0
Arabidopsis
Maize
Nicotiana tabacum
empty vector
CRISPRoff
hD3A (CD) - mD3L (CD)
hD3A (CD) - hD3L (CD)
CMT1
CMT1 (CD)
CMT2
CMT2 (CD)
CMT3
CMT3 (CD)
CMT3 (CD - published)
DRM1
DRM2
MET1
MET2A
MET2A (CD)
MET2A (CD - published)
DRM (CD)

Fig. 3A

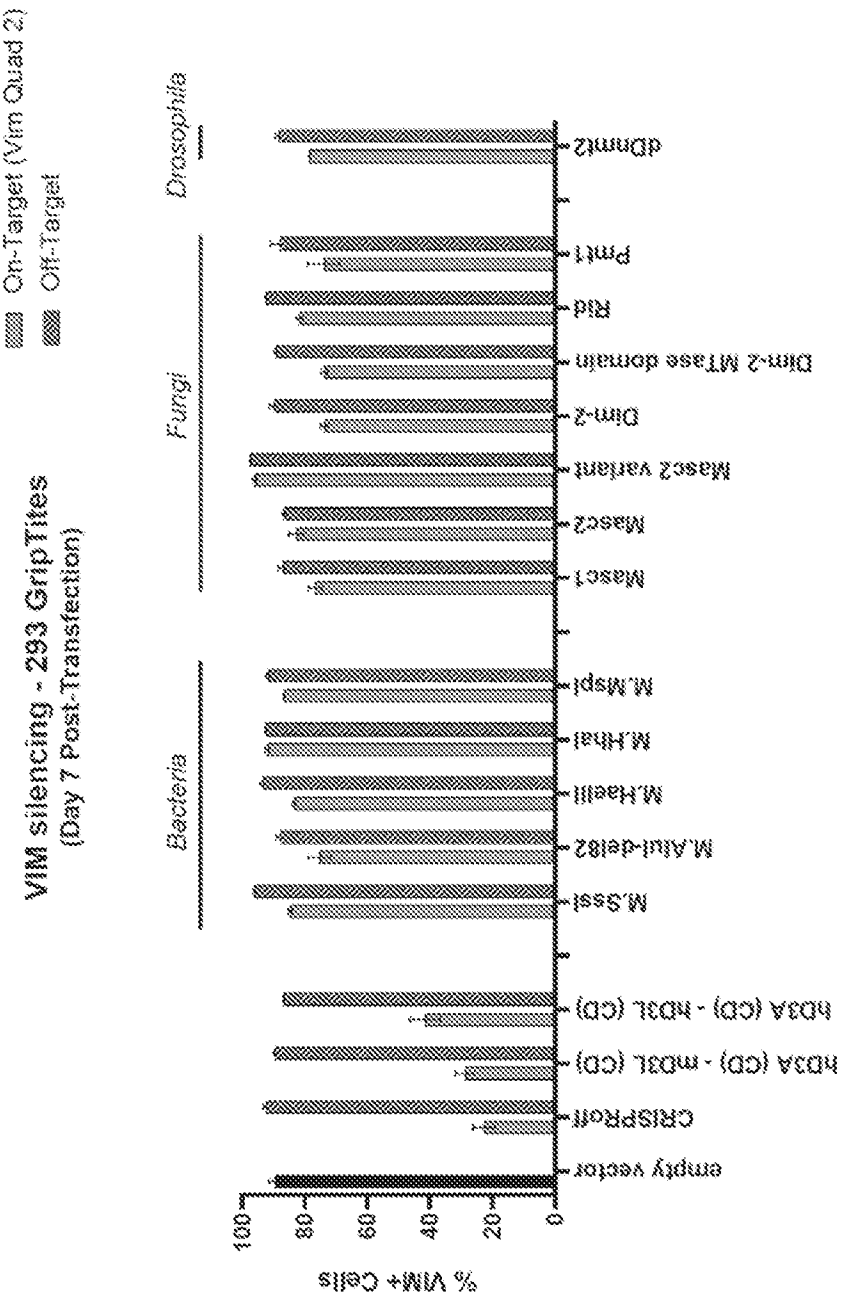
VIM silencing - 293 GripTites
(Day 7 Post-Transfection)
On-Target (Vim Quad 2)
Off-Target
% VIM+ Cells
100
80
60
40
20
0
Bacteria
Fungi
Drosophila
empty vector
CRISPRoff
hD3A (CD) - mD3L (CD)
hD3A (CD) - hD3L (CD)
M.Sssl
M.Alul-dal82
M.Haelll
M.Hhal
M.Mspl
Masc1
Masc2
Masc2 variant
Dim-2
Dim-2 MTase domain
Rid
Pmt1
dDnmt2

Fig. 3B

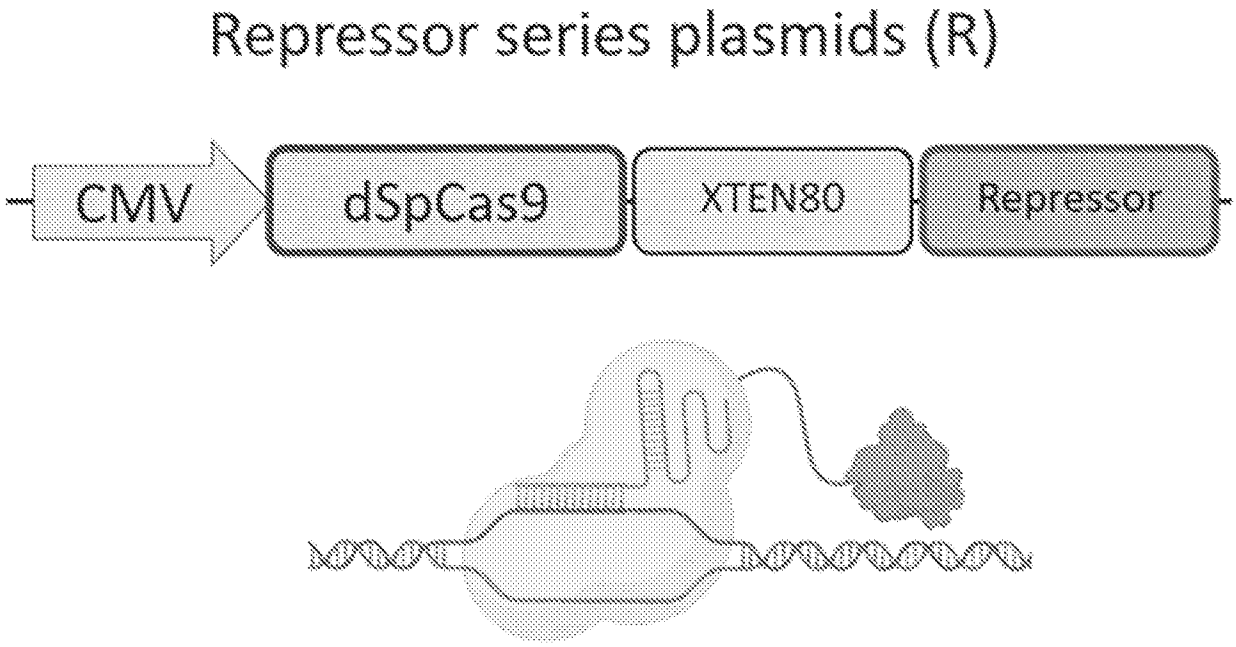

Fig. 4

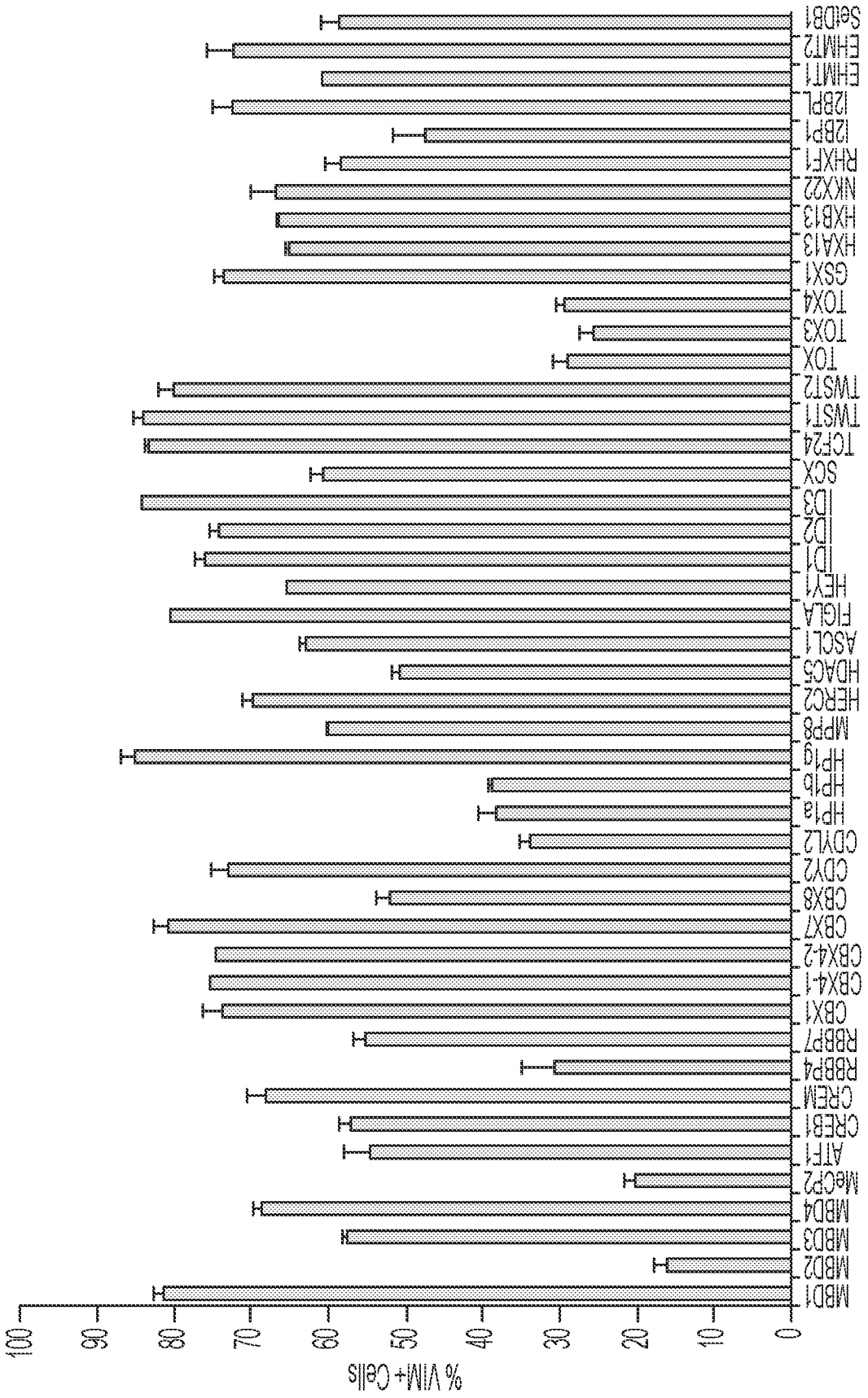
% VIM+ Cells
0
10
20
30
40
50
60
70
80
90
100
MBD1
MBD2
MBD3
MBD4
MeCP2
ATF1
CREB1
CREM
RBBP4
RBBP7
CBX1
CBX4-1
CBX4-2
CBX7
CBX8
CDY2
CDYL2
HP1a
HP1b
HP1g
MPP8
HER02
HDAC5
ASCL1
FIGLA
HEY1
ID1
ID2
ID3
SCX
TCF24
TWIST1
TWIST2
TOX
TOX3
TOX4
GSX1
HXA13
HXB13
NKX22
RHXF1
I2BP1
I2BPL
EHMT1
EHMT2
SetDB1

Fig. 5

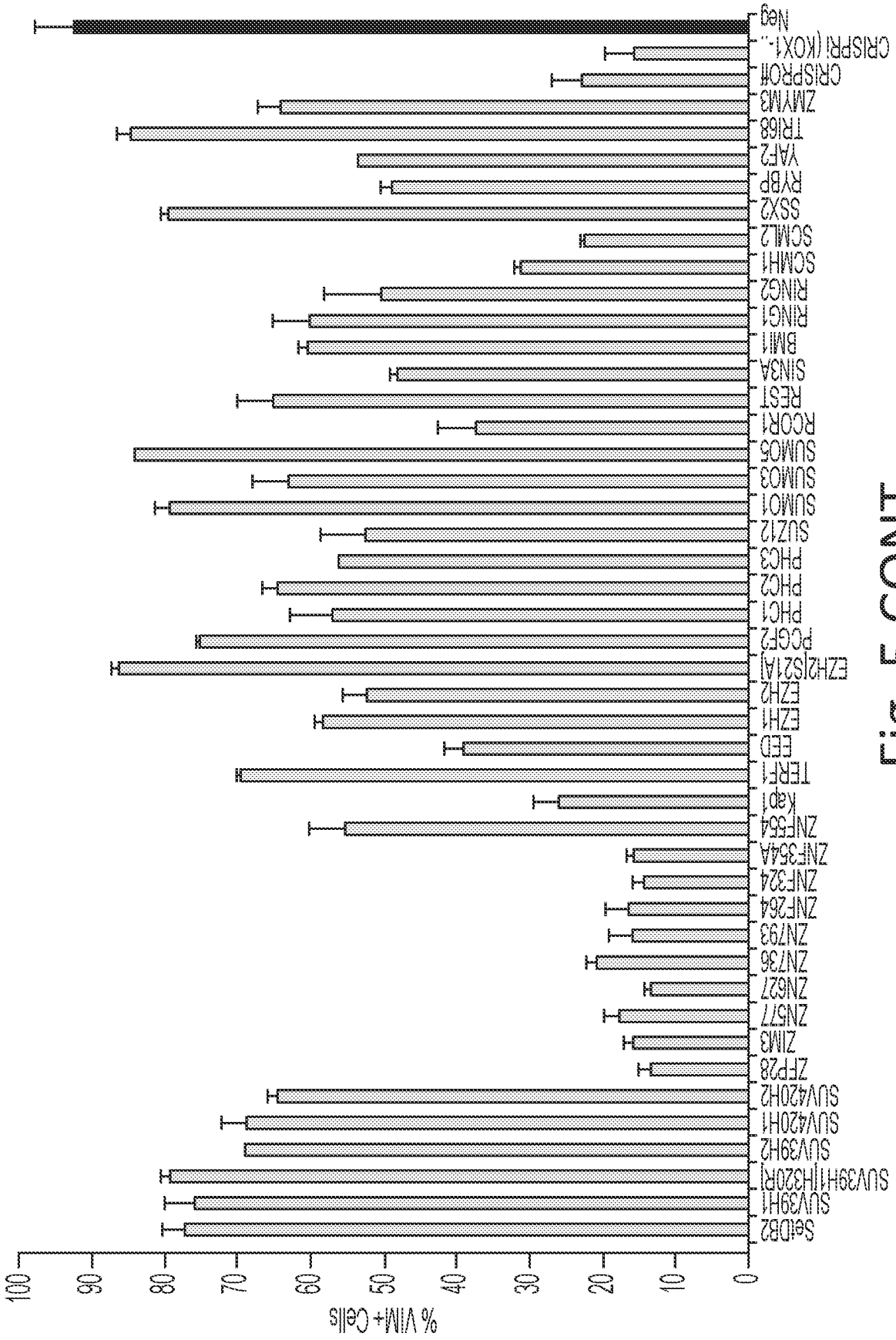
% VIM+ Cells
100
90
80
70
60
50
40
30
20
10
0
SelDB2
SUV39H1
SUV39H1|H320R|
SUV39H2
SUV420H1
SUV420H2
ZFP28
ZIM3
ZN577
ZN627
ZN736
ZN793
ZNF264
ZNF324
ZNF354A
ZNF554
kap1
TERF1
EED
EZH1
EZH2
EZH2|S21A|
PCGF2
PHC1
PHC2
PHC3
SUZ12
SUMO1
SUMO3
SUMO5
RCOR1
REST
SIN3A
BMI1
RING1
RING2
SCMH1
SCML2
SSX2
RYBP
YAF2
TRI68
ZMYM3
CRISPROff
CRISPRi (KOX1-...
Neg

Fig. 5 CONT.

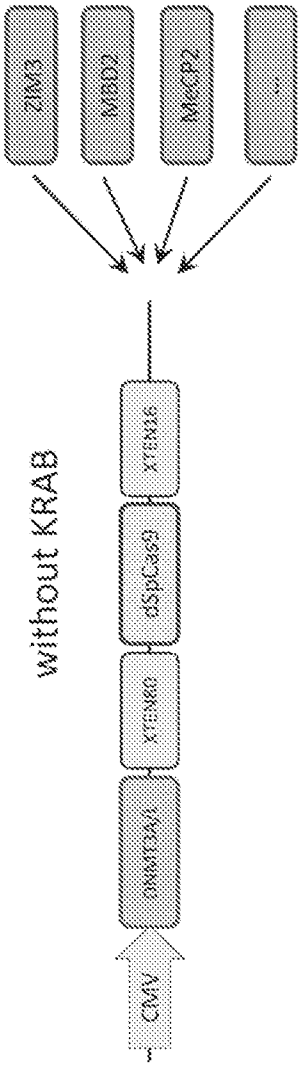
ZIM3
MBD2
MeCP2
...
without KRAB
CMV
XTEN16

Fig. 6A

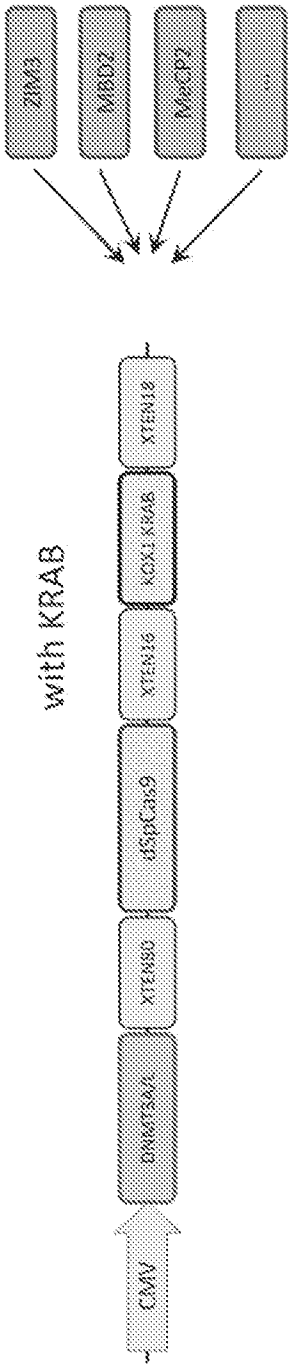
ZIM3
MBD2
MeCP2
...
with KRAB
CMV
XTEN16

Fig. 6B

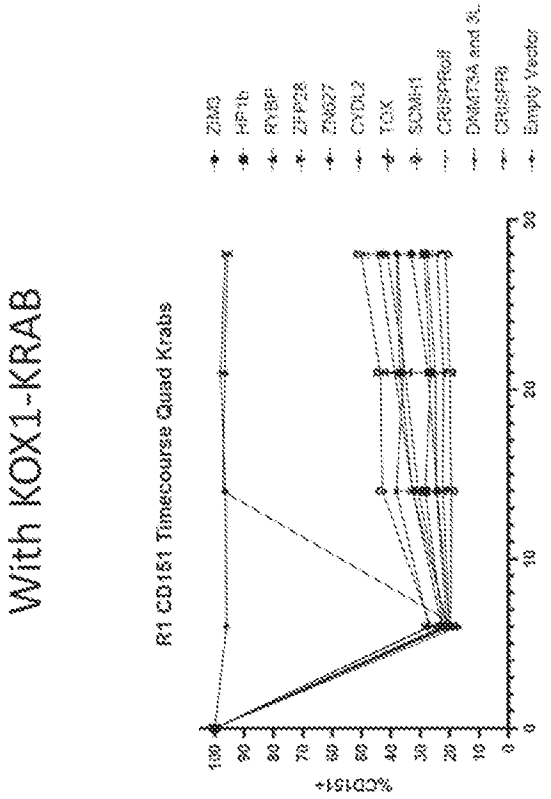
With KOX1-KRAB
R1 CD151 Timecourse Quad Krabs
%CD151+
Day
ZIM3
HP1b
RYBP
ZFP28
ZNF27
CYDL2
KOX
SOX41
CRISPRoff
DNMT3A and 3L
CRISPRi
Empty Vector

Fig. 7B

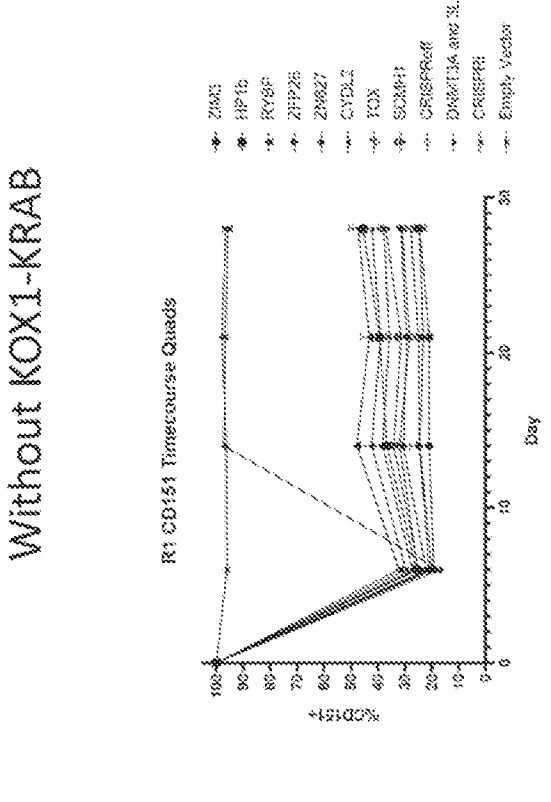
Without KOX1-KRAB
R1 CD151 Timecourse Quads
%CD151+
Day
ZIM3
HP1b
RYBP
ZFP28
ZNF27
CYDL2
KOX
SOX41
CRISPRoff
DNMT3A and 3L
CRISPRi
Empty Vector

Fig. 7A

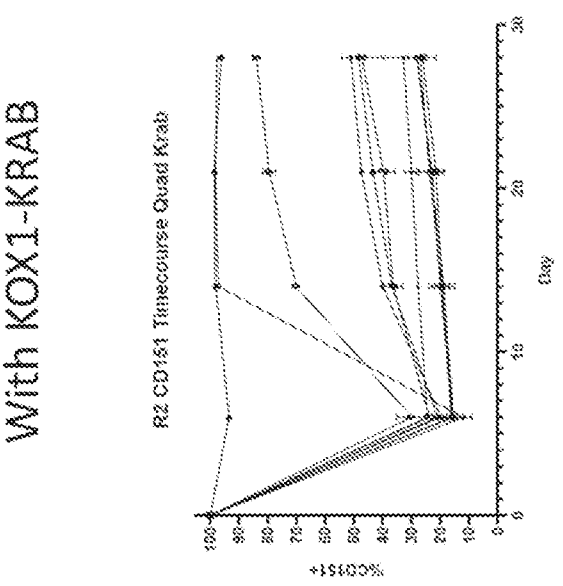
Without KOX1-KRAB
R2 CD151 Timecourse Quads
%CD151+
Day
SCML2
CBX8
TOX3
TOX4
IZBP1
MBD2
Set7/9.1
CRISPRoff
DNMT3A and 3L
CRISPR
Empty Vector

Fig. 7C

With KOX1-KRAB
R2 CD151 Timecourse Quad Krab
%CD151+
Day
SCML2
CBX8
TOX3
TOX4
IZBP1
MBD2
Set7/9.1
CRISPRoff
DNMT3A and 3L
CRISPR
Empty Vector

Fig. 7D