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(54) **Titre :** Targeted chromosomal genomic alterations with modified single stranded oligonucleotides.

(57) **Abrégé :** Presented are methods and compositions for targeted chromosomal genomic alterations using modified single-stranded oligonucleotides. The oligonucleotides of the invention have at least one modified nuclease-resistant terminal region comprising phosphorothioate linkages, LNA analogs or 2'-O-Me base analogs.

TARGETED CHROMOSOMAL GENOMIC ALTERATIONS WITH MODIFIED SINGLE STRANDED OLIGONUCLEOTIDES

Field Of The Invention

The technical field of the invention is oligonucleotide-directed repair or alteration of genetic information using novel chemically modified oligonucleotides. Such genetic information is preferably from a eukaryotic organism, i.e. a plant, animal or fungus.

Background Of The Invention

A number of methods have been developed specifically to alter the sequence of an isolated DNA in addition to methods to alter directly the genomic information of various plants, fungi and animals, including humans ("gene therapy"). The latter methods generally include the use of viral or plasmid vectors carrying nucleic acid sequences encoding partial or complete portions of a particular protein which is expressed in a cell or tissue to effect the alteration. The expression of the particular protein then results in the desired phenotype. For example, retroviral vectors containing a transgenic DNA sequence allowing for the production of a normal CFTR protein when administered to defective cells are described in U.S. Patent 5,240,846. Others have developed different "gene therapy vectors" which include, for example, portions of adenovirus (Ad) or adeno-associated virus (AAV), or other viruses. The virus portions used are often long terminal repeat sequences which are added to the ends of a transgene of choice along with other necessary control sequences which allow expression of the transgene. See U.S. Patents 5,700,470 and 5,139,941. Similar methods have been developed for use in plants. See, for example, U.S. Patent 4,459,355 which describes a method for transforming plants with a DNA vector and U.S. Patent 5,188,642 which describes cloning or expression vectors containing a transgenic DNA sequence which when expressed in plants confers resistance to the herbicide glyphosate. The use of such transgene vectors in any eukaryotic organism adds one or more exogenous copies of a gene, which gene may be foreign to the host, in a usually random fashion at one or more integration sites of the organism's genome at some frequency. The gene which was originally present in the genome, which may be a normal allelic variant, mutated, defective, and/or functional, is retained in the genome of the host.

These methods of gene correction are problematic in that complications which can compromise the health of the recipient, or even lead to death, may result. One such problem is that insertion of exogenous nucleic acid at random location(s) in the genome can have deleterious effects. Another problem with such systems includes the addition of unnecessary and unwanted genetic material to the genome of the recipient, including, for example, viral or other vector remnants, control sequences required to allow production of the transgene protein, and reporter genes or resistance markers. Such remnants and added sequences may have presently unrecognized consequences, for example, involving genetic rearrangements of the recipient genomes. Other problems associated with these types of traditional gene therapy methods include autoimmune suppression of cells expressing an inserted gene due to the presence of foreign antigens. Concerns have also been raised with consumption, especially by humans, of plants containing exogenous genetic material.

More recently, simpler systems involving poly- or oligo- nucleotides have been described for use in the alteration of genomic DNA. These chimeric RNA-DNA oligonucleotides, requiring contiguous RNA and DNA bases in a double-stranded molecule folded by complementarity into a double hairpin conformation, have been shown to effect single basepair or frameshift alterations, for example, for mutation or repair of plant or animal genomes. See, for example, WO 99/07865 and U.S. Patent 5,565,350. In the chimeric RNA-DNA oligonucleotide, an uninterrupted stretch of DNA bases within the molecule is required for sequence alteration of the targeted genome while the obligate RNA residues are involved in complex stability. Due to the length, backbone composition, and structural configuration of these chimeric RNA-DNA molecules, they are expensive to synthesize and difficult to purify. Moreover, if the RNA-containing strand of the chimeric RNA-DNA oligonucleotide is designed so as to direct gene conversion, a series of mutagenic reactions resulting in nonspecific base alteration can result. Such a result compromises the utility of such a molecule in methods designed to alter the genomes of plants and animals, including in human gene therapy applications.

Alternatively, other oligo- or poly- nucleotides have been used which require a triplex forming, usually polypurine or polypyrimidine, structural domain which binds to a DNA helical duplex through Hoogsteen interactions between the major groove of the DNA duplex and the oligonucleotide. Such oligonucleotides may have an additional DNA reactive moiety, such as psoralen, covalently linked to the oligonucleotide. These reactive moieties function as effective intercalation agents, stabilize the formation of a triplex and can be mutagenic. Such agents may be required in order to stabilize the triplex forming domain of the oligonucleotide with the DNA double helix if the Hoogsteen interactions from the oligonucleotide/target base composition are insufficient. See, e.g., U.S. Patent 5,422,251. The utility of these oligonucleotides for directing gene conversion is compromised by a high frequency of nonspecific base changes.

In more recent work, the domain for altering a genome is linked or tethered to the triplex forming domain of the bi-functional oligonucleotide, adding an additional linking or tethering functional domain to the oligonucleotide. See, e.g., Culver et al., Nature Biotechnology 17: 989-93 (1999). Such chimeric or triplex forming molecules have distinct structural requirements for each of the different domains of the complete poly- or oligo-nucleotide in order to effect the desired genomic alteration in either episomal or chromosomal targets.

Other genes, e.g. CFTR, have been targeted by homologous recombination using duplex fragments having several hundred basepairs. See, e.g., Kunzelmann et al., Gene Ther. 3:859-867 (1996). Early experiments to mutagenize an antibiotic resistance indicator gene by homologous recombination used an unmodified DNA oligonucleotide with no functional domains other than a region of complementary sequence to the target. See Campbell et al., New Biologist 1: 223-227 (1989). These experiments required large concentrations of the oligonucleotide, exhibited a very low frequency of episomal modification of a targeted exogenous plasmid gene not normally found in the cell and have not been reproduced. However, as shown in the examples herein, we have observed that an unmodified DNA oligonucleotide can convert a base at low frequency which is detectable using the assay systems described herein.

Artificial chromosomes can be useful for the screening purposed identified herein. These molecules are man-made linear or circular DNA molecules constructed from essential cis-acting DNA sequence elements that are responsible for the proper replication and partitioning of natural chromosomes (Murray et al., 1983). The essential elements are: (1) Autonomous Replication Sequences (ARS), (2) Centromeres, and (3) Telomeres.

Yeast artificial chromosomes (YACs) allow large genomic DNA to be modified and used for generating transgenic animals [Burke et al., Science 236:806; Peterson et al., Trends Genet. 13:61 (1997); Choi, et al., Nat. Genet., 4:117-223 (1993), Davies, et al., Biotechnology 11:911-914 (1993), Matsuura, et al., Hum. Mol. Genet., 5:451-459 (1996), Peterson et al., Proc. Natl. Acad. Sci., 93:6605-6609 (1996); and Schedl, et al., Cell, 86:71-82 (1996)]. Other vectors also have been developed for the cloning of large segments of mammalian DNA, including cosmids, and bacteriophage P1 [Sternberg et al., Proc. Natl. Acad. Sci. U.S.A., 87:103-107 (1990)]. YACs have certain advantages over these alternative large capacity cloning vectors [Burke et al., Science, 236:806-812 (1987)]. The maximum insert size is 35-30 kb for cosmids, and 100 kb for bacteriophage P1, both of which are much smaller than the maximal insert for a YAC.

An alternative to YACs are E. coli based cloning systems based on the E. coli fertility factor that have been developed to construct large genomic DNA insert libraries. They are bacterial artificial chromosomes (BACs) and P-1 derived artificial chromosomes (PACs) [Mejia et al., Genome Res. 7:179-186 (1997); Shizuya

et al., Proc. Natl. Acad. Sci. 89:8794-8797 (1992); Ioannou et al., Nat. Genet., 6:84-89 (1994); Hosoda et al., Nucleic Acids Res. 18:3863 (1990)]. BACs are based on the E. coli fertility plasmid (F factor); and PACs are based on the bacteriophage P1. These vectors propagate at a very low copy number (1-2 per cell) enabling genomic inserts up to 300 kb in size to be stably maintained in recombination deficient hosts. Furthermore, the PACs and BACs are circular DNA molecules that are readily isolated from the host genomic background by classical alkaline lysis [Birnboim et al., Nucleic Acids Res. 7:1513-1523 (1979].

Oligonucleotides designed for use in the alteration of genetic information are significantly different from oligonucleotides designed for antisense approaches. For example, antisense oligonucleotides are perfectly complementary to and bind an mRNA strand in order to modify expression of a targeted mRNA and are used at high concentration. As a consequence, they are unable to produce a gene conversion event by either mutagenesis or repair of a defect in the chromosomal DNA of a host genome. Furthermore, the backbone chemical composition used in most oligonucleotides designed for use in antisense approaches renders them inactive as substrates for homologous pairing or mismatch repair enzymes and the high concentrations of oligonucleotide required for antisense applications can be toxic with some types of nucleotide modifications. In addition, antisense oligonucleotides must be complementary to the mRNA and therefore, may not be complementary to the other DNA strand or to genomic sequences that span the junction between intron sequence and exon sequence.

A need exists for simple, inexpensive oligonucleotides capable of producing targeted alteration of genetic material such as those described herein as well as methods to identify optimal oligonucleotides that accurately and efficiently alter target DNA.

Summary Of The Invention

Novel, modified single-stranded nucleic acid molecules that direct gene alteration in plants, fungi and animals are identified and the efficiency of alteration is analyzed both in vitro using a cell-free extract assay and in vivo using a yeast cell system. The alteration in an oligonucleotide of the invention may comprise an insertion, deletion, substitution, as well as any combination of these. Site specific alteration of DNA is not only useful for studying function of proteins in vivo, but it is also useful for creating animal models for human disease, and in gene therapy. As described herein, oligonucleotides of the invention target directed specific gene alterations in genomic double-stranded DNA cells. The target DNA can be normal, cellular chromosomal DNA, extrachromosomal DNA present in cells in different forms including, e.g., mammalian artificial chromosomes (MACs), PACs from P-1 vectors, yeast artificial chromosomes (YACs), bacterial artificial chromosomes (BACs), plant artificial chromosomes (PLACs), as well as episomal DNA, including

episomal DNA from an exogenous source such as a plasmid or recombinant vector. Many of these artificial chromosome constructs containing human DNA can be obtained from a variety of sources, including, e.g., the Whitehead Institute, and are described, e.g., in Cohen et al., Nature 336:698-701 (1993) and Chumakov, et al., Nature 377:174-297 (1995). The target DNA may be transcriptionally silent or active. In a preferred embodiment, the target DNA to be altered is the non-transcribed strand of a genomic DNA duplex.

The low efficiency of gene alteration obtained using unmodified DNA oligonucleotides is believed to be largely the result of degradation by nucleases present in the reaction mixture or the target cell.. Although different modifications are known to have different effects on the nuclease resistance of oligonucleotides or stability of duplexes formed by such oligonucleotides (see, e.g., Koshkin et al., J. Am. Chem. Soc., 120:13252-3), we have found that it is not possible to predict which of any particular known modification would be most useful for any given alteration event, including for the construction of gene conversion oligonucleotides, because of the interaction of different as yet unidentified proteins during the gene alteration event. Herein, a variety of nucleic acid analogs have been developed that increase the nuclease resistance of oligonucleotides that contain them, including, e.g., nucleotides containing phosphorothioate linkages or 2'-O-methyl analogs. We recently discovered that single-stranded DNA oligonucleotides modified to contain 2'-O-methyl RNA nucleotides or phosphorothioate linkages can enable specific alteration of genetic information at a higher level than either unmodified single-stranded DNA or a chimeric RNA/DNA molecule. See priority applications incorporated herein in their entirety; see also Gamper et al., Nucleic Acids Research 28: 4332-4339 (2000). We also found that additional nucleic acid analogs which increase the nuclease resistance of oligonucleotides that contain them, including, e.g., "locked nucleic acids" or "LNAs", xylo-LNAs and L-ribo-LNAs; see, for example, Wengel & Nielsen, WO 99/14226; Wengel, WO 00/56748 and Wengel, WO 00/66604; also allow specific targeted alteration of genetic information.

The assay allows for determining the optimum length of the oligonucleotide, optimum sequence of the oligonucleotide, optimum position of the mismatched base or bases, optimum chemical modification or modifications, optimum strand targeted for identifying and selecting the most efficient oligonucleotide for a particular gene alteration event by comparing to a control oligonucleotide. Control oligonucleotides may include a chimeric RNA-DNA double hairpin oligonucleotide directing the same gene alteration event, an oligonucleotide that matches its target completely, an oligonucleotide in which all linkages are phosphorothiolated, an oligonucleotide fully substituted with 2'-O-methyl analogs or an RNA oligonucleotide. Such control oligonucleotides either fail to direct a targeted alteration or do so at a lower efficiency as compared to the oligonucleotides of the invention. The assay further allows for determining the optimum position of a gene alteration event within an oligonucleotide, optimum concentration of the selected

oligonucleotide for maximum alteration efficiency by systematically testing a range of concentrations, as well as optimization of either the source of cell extract by testing different organisms or strains, or testing cells derived from different organisms or strains, or cell lines. Using a series of single-stranded oligonucleotides, comprising all RNA or DNA residues and various mixtures of the two, several new structures are identified as viable molecules in nucleotide conversion to direct or repair a genomic mutagenic event. When extracts from mammalian, plant and fungal cells are used and are analyzed using a genetic readout assay in bacteria, single-stranded oligonucleotides having one of several modifications are found to be more active than a control RNA-DNA double hairpin chimera structure when evaluated using an in vitro gene repair assay. Similar results are also observed in vivo using yeast, mammalian, rodent, monkey, human and embryonic cells, including stem cells. Molecules containing various lengths of modified bases were found to possess greater activity than unmodified single-stranded DNA molecules.

Detailed Description Of The Invention

The present invention provides oligonucleotides having chemically modified, nuclease resistant residues, preferably at or near the termini of the oligonucleotides, and methods for their identification and use in targeted alteration of genetic material, including gene mutation, targeted gene repair and gene knockout. The oligonucleotides are preferably used for mismatch repair or alteration by changing at least one nucleic acid base, or for frameshift repair or alteration by addition or deletion of at least one nucleic acid base. The oligonucleotides of the invention direct any such alteration, including gene correction, gene repair or gene mutation and can be used, for example, to introduce a polymorphism or haplotype or to eliminate ("knockout") a particular protein activity.

The oligonucleotides of the invention are designed as substrates for homologous pairing and repair enzymes and as such have a unique backbone composition that differs from chimeric RNA-DNA double hairpin oligonucleotides, antisense oligonucleotides, and/or other poly- or oligo-nucleotides used for altering genomic DNA, such as triplex forming oligonucleotides. The single-stranded oligonucleotides described herein are inexpensive to synthesize and easy to purify. In side-by-side comparisons, an optimized single-stranded oligonucleotide comprising modified residues as described herein is significantly more efficient than a chimeric RNA-DNA double hairpin oligonucleotide in directing a base substitution or frameshift mutation in a cell-free extract assay.

We have discovered that single-stranded oligonucleotides having a DNA domain surrounding the targeted base, with the domain preferably central to the poly- or oligo-nucleotide, and having at least one modified end, preferably at the 3' terminal region are able to alter a target genetic sequence and with an

efficiency that is higher than chimeric RNA-DNA double hairpin oligonucleotides disclosed in US Patent 5,565,350. Oligonucleotides of the invention can efficiently be used to introduce targeted alterations in a genetic sequence of DNA in the presence of human, animal, plant, fungal (including yeast) proteins and in cultured cells of human liver, lung, colon, cervix, kidney, epethelium and cancer cells and in monkey, hamster, rat and mouse cells of different types, as well as embryonic stem cells. Cells for use in the invention include, e.g., fungi including *S. cerevisiae*, *Ustillago maydis* and *Candida albicans*, mammalian, mouse, hamster, rat, monkey, human and embryonic cells including stem cells. The DNA domain is preferably fully complementary to one strand of the gene target, except for the mismatch base or bases responsible for the gene alteration or conversion events. On either side of the preferably central DNA domain, the contiguous bases may be either RNA bases or, preferably, are primarily DNA bases. The central DNA domain is generally at least 8 nucleotides in length. The base(s) targeted for alteration in the most preferred embodiments are at least about 8, 9 or 10 bases from one end of the oligonucleotide.

According to certain embodiments, the termini of the oligonucleotides of the present invention comprise phosphorothioate modifications, LNA backbone modifications, or 2'-O-methyl base analogs, or any combination of these modifications. Oligonucleotides comprising 2'-O-methyl or LNA analogs are a mixed DNA/RNA polymer. These oligonucleotides are, however, single-stranded and are not designed to form a stable internal duplex structure within the oligonucleotide. The efficiency of gene alteration is surprisingly increased with oligonucleotides having internal complementary sequence comprising phosphorothioate modified bases as compared to 2'-O-methyl modifications. This result indicates that specific chemical interactions are involved between the converting oligonucleotide and the proteins involved in the conversion. The effect of other such chemical interactions to produce nuclease resistant termini using modifications other than LNA, phosphorothioate linkages, or 2'-O-methyl analog incorporation into an oligonucleotide can not yet be predicted because the proteins involved in the alteration process and their particular chemical interaction with the oligonucleotide substituents are not yet known and cannot be predicted.

In the examples, correcting oligonucleotides of defined sequence are provided for correction of genes mutated in human diseases. In the tables of these examples, the oligonucleotides of the invention are not limited to the particular sequences disclosed. The oligonucleotides of the invention include extensions of the appropriate sequence of the longer 120 base oligonucleotides which can be added base by base to the smallest disclosed oligonucleotides of 17 bases. Thus the oligonucleotides of the invention include for each correcting change, oligonucleotides of length 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, 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, or 120 with further single-nucleotide additions up to the longest sequence disclosed. Moreover, the oligonucleotides of the invention do not require a symmetrical extension on either side of the central DNA domain. Similarly, the oligonucleotides of the invention as disclosed in the various tables for correction of human diseases contain phosphorothioate linkages, 2'-O-methyl analogs or LNAs or any combination of these modifications just as the assay oligonucleotides do.

The present invention, however, is not limited to oligonucleotides that contain any particular nuclease resistant modification. Oligonucleotides of the invention may be altered with any combination of additional LNAs, phosphorothioate linkages or 2'-O-methyl analogs to maximize conversion efficiency. For oligonucleotides of the invention that are longer than about 17 to about 25 bases in length, internal as well as terminal region segments of the backbone may be altered. Alternatively, simple fold-back structures at each end of a oligonucleotide or appended end groups may be used in addition to a modified backbone for conferring additional nuclease resistance.

The different oligonucleotides of the present invention preferably contain more than one of the aforementioned backbone modifications at each end. In some embodiments, the backbone modifications are adjacent to one another. However, the optimal number and placement of backbone modifications for any individual oligonucleotide will vary with the length of the oligonucleotide and the particular type of backbone modification(s) that are used. If constructs of identical sequence having phosphorothioate linkages are compared, 2, 3, 4, 5, or 6 phosphorothioate linkages at each end are preferred. If constructs of identical sequence having 2'-O-methyl base analogs are compared, 1, 2, 3 or 4 analogs are preferred. The optimal number and type of backbone modifications for any particular oligonucleotide useful for altering target DNA may be determined empirically by comparing the alteration efficiency of the oligonucleotide comprising any combination of the modifications to a control molecule of comparable sequence using any of the assays described herein. The optimal position(s) for oligonucleotide modifications for a maximally efficient altering oligonucleotide can be determined by testing the various modifications as compared to control molecule of comparable sequence in one of the assays disclosed herein. In such assays, a control molecule includes, e.g., a completely 2'-O-methyl substituted molecule, a completely complementary oligonucleotide, or a chimeric RNA-DNA double hairpin.

Increasing the number of phosphorothioate linkages, LNAs or 2'-O-methyl bases beyond the preferred number generally decreases the gene repair activity of a 25 nucleotide long oligonucleotide. Based on analysis of the concentration of oligonucleotide present in the extract after different time periods of incubation, it is believed that the terminal modifications impart nuclease resistance to the oligonucleotide thereby allowing

it to survive within the cellular environment. However, this may not be the only possible mechanism by which such modifications confer greater efficiency of conversion. For example, as disclosed herein, certain modifications to oligonucleotides confer a greater improvement to the efficiency of conversion than other modifications.

Efficiency of conversion is defined herein as the percentage of recovered substate molecules that have undergone a conversion event. Depending on the nature of the target genetic material, e.g. the genome of a cell, efficiency could be represented as the proportion of cells or clones containing an extrachromosomal element that exhibit a particular phenotype. Alternatively, representative samples of the target genetic material can be sequenced to determine the percentage that have acquired the desire change. The oligonucleotides of the invention in different embodiments can alter DNA one, two, three, four, five, six, seven, eight, nine, ten, twelve, fifteen, twenty, thirty, and fifty or more fold more than control oligonucleotides. Such control oligonucleotides are oligonucleotides with fully phosphorothiolated linkages, oligonucleotides that are fully substituted with 2'-O-methyl analogs, a perfectly matched oligonucleotide that is fully complementary to a target sequence or a chimeric DNA-RNA double hairpin oligonucleotide such as disclosed in US Patent 5,565,350.

In addition, for a given oligonucleotide length, additional modifications interfere with the ability of the oligonucleotide to act in concert with the cellular recombination or repair enzyme machinery which is necessary and required to mediate a targeted substitution, addition or deletion event in DNA. For example, fully phosphorothiolated or fully 2-O-methylated molecules are inefficient in targeted gene alteration.

The oligonucleotides of the invention as optimized for the purpose of targeted alteration of genetic material, including gene knockout or repair, are different in structure from antisense oligonucleotides that may possess a similar mixed chemical composition backbone. The oligonucleotides of the invention differ from such antisense oligonucleotides in chemical composition, structure, sequence, and in their ability to alter genomic DNA. Significantly, antisense oligonucleotides fail to direct targeted gene alteration. The oligonucleotides of the invention may target either the Watson or the Crick strand of DNA and can include any component of the genome including, for example, intron and exon sequences. The preferred embodiment of the invention is a modified oligonucleotide that binds to the non-transcribed strand of a genomic DNA duplex. In other words, the preferred oligonucleotides of the invention target the sense strand of the DNA, i.e. the oligonucleotides of the invention are complementary to the non-transcribed strand of the target duplex DNA. The sequence of the non-transcribed strand of a DNA duplex is found in the mRNA produced from that duplex, given that mRNA uses uracil-containing nucleotides in place of thymine-containing nucleotides.

Moreover, the initial observation that single-stranded oligonucleotides comprising these modifications and lacking any particular triplex forming domain have reproducibly enhanced gene repair activity in a variety of assay systems as compared to a chimeric RNA-DNA double-stranded hairpin control or single-stranded oligonucleotides comprising other backbone modifications was surprising. The single-stranded molecules of the invention totally lack the complementary RNA binding structure that stabilizes a normal chimeric double-stranded hairpin of the type disclosed in U.S. Patent 5,565,350 yet is more effective in producing targeted base conversion as compared to such a chimeric RNA-DNA double-stranded hairpin. In addition, the molecules of the invention lack any particular triplex forming domain involved in Hoogsteen interactions with the DNA double helix and required by other known oligonucleotides in other oligonucleotide dependant gene conversion systems. Although the lack of these functional domains was expected to decrease the efficiency of an alteration in a sequence, just the opposite occurs: the efficiency of sequence alteration using the modified oligonucleotides of the invention is higher than the efficiency of sequence alteration using a chimeric RNA-DNA hairpin targeting the same sequence alteration. Moreover, the efficiency of sequence alteration or gene conversion directed by an unmodified oligonucleotide is many times lower as compared to a control chimeric RNA-DNA molecule or the modified oligonucleotides of the invention targeting the same sequence alteration. Similarly, molecules containing at least 3 2'-O-methyl base analogs are about four to five fold less efficient as compared to an oligonucleotide having the same number of phosphorothioate linkages.

The oligonucleotides of the present invention for alteration of a single base are about 17 to about 121 nucleotides in length, preferably about 17 to about 74 nucleotides in length. Most preferably, however, the oligonucleotides of the present invention are at least about 25 bases in length, unless there are self-dimerization structures within the oligonucleotide. If the oligonucleotide has such an unfavorable structure, lengths longer than 35 bases are preferred. Oligonucleotides with modified ends both shorter and longer than certain of the exemplified, modified oligonucleotides herein function as gene repair or gene knockout agents and are within the scope of the present invention.

Once an oligomer is chosen, it can be tested for its tendency to self-dimerize, since self-dimerization may result in reduced efficiency of alteration of genetic information. Checking for self-dimerization tendency can be accomplished manually or, more preferably, by using a software program. One such program is Oligo Analyzer 2.0, available through Integrated DNA Technologies (Coralville, IA 52241) (http://www.idtdna.com); this program is available for use on the world wide web at

http://www.idtdna.com/program/oligoanalyzer/

oligoanalyzer.asp.

For each oligonucleotide sequence input into the program, Oligo Analyzer 2.0 reports possible self-dimerized duplex forms, which are usually only partially duplexed, along with the free energy change associated with such self-dimerization. Delta G-values that are negative and large in magnitude, indicating strong self-dimerization potential, are automatically flagged by the software as "bad". Another software program that analyzes oligomers for pair dimer formation is Primer Select from DNASTAR, Inc., 1228 S. Park St., Madison, WI 53715, Phone: (608) 258-7420

(http://www.dnastar.com/products/PrimerSelect.html).

If the sequence is subject to significant self-dimerization, the addition of further sequence flanking the "repair" nucleotide can improve gene correction frequency.

Generally, the oligonucleotides of the present invention are identical in sequence to one strand of the target DNA, which can be either strand of the target DNA, with the exception of one or more targeted bases positioned within the DNA domain of the oligonucleotide, and preferably toward the middle between the modified terminal regions. Preferably, the difference in sequence of the oligonucleotide as compared to the targeted genomic DNA is located at about the middle of the oligonucleotide sequence. In a preferred embodiment, the oligonucleotides of the invention are complementary to the non-transcribed strand of a duplex. In other words, the preferred oligonucleotides target the sense strand of the DNA, i.e. the oligonucleotides of the invention are preferably complementary to the strand of the target DNA the sequence of which is found in the mRNA.

The oligonucleotides of the invention can include more than a single base change. In an oligonucleotide that is about a 70-mer, with at least one modified residue incorporated on the ends, as disclosed herein, multiple bases can be simultaneously targeted for change. The target bases may be up to 27 nucleotides apart and may not be changed together in all resultant plasmids in all cases. There is a frequency distribution such that the closer the target bases are to each other in the central DNA domain within the oligonucleotides of the invention, the higher the frequency of change in a given cell. Target bases only two nucleotides apart are changed together in every case that has been analyzed. The farther apart the two target bases are, the less frequent the simultaneous change. Thus, oligonucleotides of the invention may be used to repair or alter multiple bases rather than just one single base. For example, in a 74-mer oligonucleotide having a central base targeted for change, a base change event up to about 27 nucleotides away can also be effected. The positions of the altering bases within the oligonucleotide can be optimized using any one of the assays described herein. Preferably, the altering bases are at least about 8 nucleotides from one end of the oligonucleotide.

The oligonucleotides of the present invention can be introduced into cells by any suitable means. According to certain preferred embodiments, the modified oligonucleotides may be used alone. Suitable means, however, include the use of polycations, cationic lipids, liposomes, polyethylenimine (PEI), electroporation, biolistics, microinjection and other methods known in the art to facilitate cellular uptake. According to certain preferred embodiments of the present invention, the isolated cells are treated in culture according to the methods of the invention, to mutate or repair a target gene. Modified cells may then be reintroduced into the organism as, for example, in bone marrow having a targeted gene. Alternatively, modified cells may be used to regenerate the whole organism as, for example, in a plant having a desired targeted genomic change. In other instances, targeted genomic alteration, including repair or mutagenesis, may take place in vivo following direct administration of the modified, single-stranded oligonucleotides of the invention to a subject.

The single-stranded, modified oligonucleotides of the present invention have numerous applications as gene repair, gene modification, or gene knockout agents. Such oligonucleotides may be advantageously used, for example, to introduce or correct multiple point mutations. Each mutation leads to the addition, deletion or substitution of at least one base pair. The methods of the present invention offer distinct advantages over other methods of altering the genetic makeup of an organism, in that only the individually targeted bases are altered. No additional foreign DNA sequences are added to the genetic complement of the organism. Such agents may, for example, be used to develop plants or animals with improved traits by rationally changing the sequence of selected genes in cultured cells. Modified cells are then cloned into whole plants or animals having the altered gene. See, e.g., U.S. Patent 6,046,380 and U.S. Patent 5,905,185 incorporated hererin by reference. Such plants or animals produced using the compositions of the invention lack additional undesirable selectable markers or other foreign DNA sequences. Targeted base pair substitution or frameshift mutations introduced by an oligonucleotide in the presence of a cell-free extract also provides a way to modify the sequence of extrachromosomal elements, including, for example, plasmids, cosmids and artificial chromosomes. The oligonucleotides of the invention also simplify the production of transgenic animals having particular modified or inactivated genes. Altered animal or plant model systems such as those produced using the methods and oligonucleotides of the invention are invaluable in determining the function of a gene and in evaluating drugs. The oligonucleotides and methods of the present invention may also be used for gene therapy to correct mutations causative of human diseases.

The purified oligonucleotide compositions may be formulated in accordance with routine procedures as a pharmaceutical composition adapted for bathing cells in culture, for microinjection into cells in culture, and for intravenous administration to human beings or animals. Typically, compositions for cellular

administration or for intravenous administration into animals, including humans, are solutions in sterile isotonic aqueous buffer. Where necessary, the composition may also include a solubilizing agent and a local anaesthetic such as lignocaine to ease pain at the site of the injection. Generally, the ingredients will be supplied either separately or mixed together in unit dosage form, for example, as a dry, lyophilized powder or water-free concentrate. The composition may be stored in a hermetically sealed container such as an ampule or sachette indicating the quantity of active agent in activity units. Where the composition is administered by infusion, it can be dispensed with an infusion bottle containing sterile pharmaceutical grade "water for injection" or saline. Where the composition is to be administered by injection, an ampule of sterile water for injection or saline may be provided so that the ingredients may be mixed prior to administration.

Pharmaceutical compositions of this invention comprise the compounds of the present invention and pharmaceutically acceptable salts thereof, with any pharmaceutically acceptable ingredient, excipient, carrier, adjuvant or vehicle.

The oligonucleotides of the invention are preferably administered to the subject in the form of an injectable composition. The composition is preferably administered parenterally, meaning intravenously, intraarterially, intrathecally, interstitially or intracavitarilly. Pharmaceutical compositions of this invention can be administered to mammals including humans in a manner similar to other diagnostic or therapeutic agents. The dosage to be administered, and the mode of administration will depend on a variety of factors including age, weight, sex, condition of the subject and genetic factors, and will ultimately be decided by medical personnel subsequent to experimental determinations of varying dosage as described herein. In general, dosage required for correction and therapeutic efficacy will range from about 0.001 to 50,000 μg/kg, preferably between 1 to 250 μg/kg of host cell or body mass, and most preferably at a concentration of between 30 and 60 micromolar.

For cell administration, direct injection into the nucleus, biolistic bombardment, electroporation, liposome transfer and calcium phosphate precipitation may be used. In yeast, lithium acetate or spheroplast transformation may also be used. In a preferred method, the administration is performed with a liposomal transfer compound, e.g., DOTAP (Boehringer-Mannheim) or an equivalent such as lipofectin. The amount of the oligonucleotide used is about 500 nanograms in 3 micrograms of DOTAP per 100,000 cells. For electroporation, between 20 and 2000 nanograms of oligonucleotide per million cells to be electroporated is an appropriate range of dosages which can be increased to improve efficiency of genetic alteration upon review of the appropriate sequence according to the methods described herein.

Another aspect of the invention is a kit comprising at least one oligonucleotide of the invention. The kit may comprise an addition reagent or article of manufacture. The additional reagent or article of

manufacture may comprise a cell extract, a cell, or a plasmid, such as one of those disclosed in the Figures herein, for use in an assay of the invention.

Brief Description Of The Drawings

Figure 1. Flow diagram for the generation of modified single-stranded oligonucleotides. The upper strands of chimeric oligonucleotides I and II are separated into pathways resulting in the generation of single-stranded oligonucleotides that contain (A) 2'-O-methyl RNA nucleotides or (B) phosphorothioate linkages. Fold changes in repair activity for correction of kan^s in the HUH7 cell-free extract are presented in parenthesis. HUH7 cells are described in Nakabayashi et al., Cancer Research 42: 3858-3863 (1982). Each single-stranded oligonucleotide is 25 bases in length and contains a G residue mismatched to the complementary sequence of the kan^s gene. The numbers 3, 6, 8, 10, 12 and 12.5 respectively indicate how many phosphorothioate linkages (S) or 2'-O-methyl RNA nucleotides (R) are at each end of the molecule. Hence oligo 12S/25G contains an all phosphorothioate backbone, displayed as a dotted line. Smooth lines indicate DNA residues, wavy lines indicate 2'-O-methyl RNA residues and the carat indicates the mismatched base site (G). Figure 1(C) provides a schematic plasmid indicating the sequence of the kan chimeric double-stranded hairpin oligonucleotide (left; SEQ ID NO: 4341) and the sequence the tet chimeric double-stranded hairpin oligonucleotide used in other experiments (SEQ ID NO: 4342). Figure 1(D) provides a flow chart of a kan experiment in which a chimeric double-stranded hairpin oligonucleotide (SEQ ID NO: 4341) is used. In Figure 1(D), the Kan mutant sequence corresponds to SEQ ID NO: 4343 and SEQ ID NO: 4344; the Kan converted sequence corresponds to SEQ ID NO: 4345 and SEQ ID NO: 4346; the mutant sequence in the sequence trace corresponds to SEQ ID NO: 4347 and the converted sequences in the sequence trace correspond to SEQ ID NO: 4348.

Figure 2. Genetic readout system for correction of a point mutation in plasmid pK^{s}m4021. A mutant kanamycin gene harbored in plasmid pK^{s}m4021 is the target for correction by oligonucleotides. The mutant G is converted to a C by the action of the oligo. Corrected plasmids confer resistance to kanamycin in E. coli (DH10B) after electroporation leading to the genetic readout and colony counts. The wild type sequence corresponds to SEQ ID NO: 4349.

Figure 3: Target plasmid and sequence correction of a frameshift mutation by chimeric and single-stranded oligonucleotides. (A) Plasmid pT^{s}Δ208 contains a single base deletion mutation at position 208 rendering it unable to confer tet resistance. The target sequence presented below indicates the insertion of a T directed by the oligonucleotides to re-establish the resistant phenotype. (B) DNA sequence confirming base insertion directed by Tet 3S/25G; the yellow highlight indicates the position of frameshift repair. The

wild type sequence corresponds to SEQ ID NO: 4350, the mutant sequence corresponds to SEQ ID NO: 4351 and the converted sequence corresponds to SEQ ID NO: 4352. The control sequence in the sequence trace corresponds to SEQ ID NO: 4353 and the 3S/25A sequence in the sequence trace corresponds to SEQ ID NO: 4354.

Figure 4. DNA sequences of representative kan^r colonies. Confirmation of sequence alteration directed by the indicated molecule is presented along with a table outlining codon distribution. Note that 10S/25G and 12S/25G elicit both mixed and unfaithful gene repair. The number of clones sequenced is listed in parentheses next to the designation for the single-stranded oligonucleotide. A plus (+) symbol indicates the codon identified while a figure after the (+) symbol indicates the number of colonies with a particular sequence. TAC/TAG indicates a mixed peak. Representative DNA sequences are presented below the table with yellow highlighting altered residues. The sequences in the sequence traces have been assigned numbers as follows: 3S/25G, 6S/25G and 8S/25G correspond to SEQ ID NO: 4355, 10S/25G corresponds to SEQ ID NO: 4356, 25S/25G on the lower left corresponds to SEQ ID NO: 4357 and 25S/25G on the lower right corresponds to SEQ ID NO: 4358.

Figure 5. *Gene correction in HeLa cells.* Representative oligonucleotides of the invention are co-transfected with the pCMVneo(-)FlAsH plasmid (shown in Figure 9) into HeLa cells. Ligand is diffused into cells after co-transfection of plasmid and oligonucleotides. Green fluorescence indicates gene correction of the mutation in the antibiotic resistance gene. Correction of the mutation results in the expression of a fusion protein that carries a marker ligand binding site and when the fusion protein binds the ligand, a green fluorescence is emitted. The ligand is produced by Aurora Biosciences and can readily diffuse into cells enabling a measurement of corrected protein function; the protein must bind the ligand directly to induce fluorescence. Hence cells bearing the corrected plasmid gene appear green while "uncorrected" cells remain colorless.

Figure 6. *Z-series imaging of corrected cells.* Serial cross-sections of the HeLa cell represented in Figure 5 are produced by Zeiss 510 LSM confocal microscope revealing that the fusion protein is contained within the cell.

Figure 7. *Hygromycin-eGFP target plasmids.* (A) Plasmid pAURHYG(ins)GFP contains a single base insertion mutation between nucleotides 136 and 137, at codon 46, of the Hygromycin B coding sequence (cds) which is transcribed from the constitutive ADH1 promoter. The target sequence presented below indicates the deletion of an A and the substitution of a C for a T directed by the oligonucleotides to re-establish the resistant phenotype. In Figure 7A, the sequence of the normal allele corresponds to SEQ ID NO: 4359, the sequence of the target/existing mutation corresponds to SEQ ID NO: 4360 and the sequence

of the desired alteration corresponds to SEQ ID NO: 4361. (B) Plasmid pAURHYG(rep)GFP contains a base substitution mutation introducing a G at nucleotide 137, at codon 46, of the Hygromycin B coding sequence (cds). The target sequence presented below the diagram indicates the amino acid conservative replacement of G with C, restoring gene function. In Figure 7B, the sequence of the normal allele corresponds to SEQ ID NO: 4359, the sequence of the target/existing mutation corresponds to SEQ ID NO: 4362 and the sequence of the desired alteration corresponds to SEQ ID NO: 4361.

Figure 8. *Oligonucleotides for correction of hygromycin resistance gene.* The sequence of the oligonucleotides used in experiments to assay correction of a hygromycin resistance gene are shown. DNA residues are shown in capital letters, RNA residues are shown in lowercase and nucleotides with a phosphorothioate backbone are capitalized and underlined. In Figure 8, the sequence of HygE3T/25 corresponds to SEQ ID NO: 4363, the sequence of HygE3T/74 corresponds to SEQ ID NO: 4364, the sequence of HygE3T/74a corresponds to SEQ ID NO: 4365, the sequence of HygGG/Rev corresponds to SEQ ID NO: 4366 and the sequence of Kan70T corresponds to SEQ ID NO: 4367.

Figure 9. *pAURNeo(-)FlAsH plasmid.* This figure describes the plasmid structure, target sequence, oligonucleotides, and the basis for detection of the gene alteration event by fluorescence. In Figure 9, the sequence of the Neo/kan target mutant corresponds to SEQ ID NO: 4343 and SEQ ID NO: 4344, the converted sequence corresponds to SEQ ID NO: 4345 and SEQ ID NO: 4346 and the FlAsH peptide sequence corresponds to SEQ ID NO: 4368.

Figure 10. pYESHyg(x)eGFP plasmid. This plasmid is a construct similar to the pAURHyg(x)eGFP construct shown in Figure 7, except the promoter is the inducible GAL1 promoter. This promoter is inducible with galactose, leaky in the presence of raffinose, and repressed in the presence of dextrose.

The following examples are provided by way of illustration only, and are not intended to limit the scope of the invention disclosed herein.

EXAMPLE 1

Assay Method For Base Alteration And Preferred Oligonucleotide Selection

In this example, single-stranded and double-hairpin oligonucleotides with chimeric backbones (see Figure 1 for structures (A and B) and sequences (C and D) of assay oligonucleotides) are used to correct a point mutation in the kanamycin gene of pK^{s}m4021 (Figure 2) or the tetracycline gene of pT^{s}Δ208 (Figure 3). All kan oligonucleotides share the same 25 base sequence surrounding the target base identified for

change, just as all tet oligonucleotides do. The sequence is given in Figures 1C and Figure 1D. Each plasmid contains a functional ampicillin gene. Kanamycin gene function is restored when a G at position 4021 is converted to a C (via a substitution mutation); tetracycline gene function is restored when a deletion at position 208 is replaced by a C (via frameshift mutation). A separate plasmid, pAURNeo(-)FlAsH (Figure 9), bearing the kan^s gene is used in the cell culture experiments. This plasmid was constructed by inserting a synthetic expression cassette containing a neomycin phosphotransferase (kanamycin resistance) gene and an extended reading frame that encodes a receptor for the FlAsH ligand into the pAUR123 shuttle vector (Panvera Corp., Madison, WI). The resulting construct replicates in *S. cerevisiae* at low copy number, confers resistance to aureobasidinA and constitutively expresses either the Neo+/FlAsH fusion product (after alteration) or the truncated Neo-/FlAsH product (before alteration) from the ADH1 promoter. By extending the reading frame of this gene to code for a unique peptide sequence capable of binding a small ligand to form a fluorescent complex, restoration of expression by correction of the stop codon can be detected in real time using confocal microscopy. Additional constructs can be made to test additional gene alteration events.

We also construct three mammalian expression vectors, pHyg(rep)eGFP, pHyg(Δ)eGFP, pHyg(ins)eGFP, that contain a substitution mutation at nucleotide 137 of the hygromycin-B coding sequence. (rep) indicates a T137→G replacement, (Δ) represents a deletion of the G137 and (ins) represents an A insertion between nucleotides 136 and 137. All point mutations create a nonsense termination codon at residue 46. We use pHygEGFP plasmid (Invitrogen, CA) DNA as a template to introduce the mutations into the hygromycin-eGFP fusion gene by a two step site-directed mutagenesis PCR protocol. First, we generate overlapping 5' and a 3' amplicons surrounding the mutation site by PCR for each of the point mutation sites. A 215 bp 5' amplicon for the (rep), (Δ) or (ins) was generated by polymerization from oligonucleotide primer HygEGFPf (5'-AATACGACTCACTATAGG-3'; SEQ ID NO: 4369) to primer Hygrepr (5'GACCTATCCACGCCCTCC-3'; SEQ ID NO: 4370), HygΔr (5'-GACTATCCACGCCCTCC-3'; SEQ ID NO: 4371), or Hyginsr (5'-GACATTATCCACGCCCTCC-3'; SEQ ID NO: 4372), respectively. We generate a 300bp 3' amplicon for the (rep), (Δ) or (ins) by polymerization from oligonucleotide primers Hygrepf (5'-CTGGGATAGGTCCTGCGG-3'; SEQ ID NO: 4373), HygΔf (5'-CGTGGATAGTCCTGCGG-3'; SEQ ID NO: 4374), Hyginsf (5'-CGTGGATAATGTCCTGCGG-3'; SEQ ID NO: 4375), respectively to primer HygEGFPr (5'-AAATCACGCCATGTAGTG-3'; SEQ ID NO: 4376). We mix 20 ng of each of the resultant 5' and 3' overlapping amplicon mutation sets and use the mixture as a template to amplify a 523 bp fragment of the Hygromycin gene spanning the KpnI and RsrII restriction endonuclease sites. We use the Expand PCR system (Roche) to generate all amplicons with 25 cycles of denaturing at 94°C for 10 seconds, annealing at 55 C for 20 seconds and elongation at 68°C for 1 minute. We digest 10 μg of vector pHygEGFP and 5 μg

of the resulting fragments for each mutation with KpnI and RsrII (NEB) and gel purify the fragment for enzymatic ligation. We ligate each mutated insert into pHygEGFP vector at 3:1 molar ratio using T4 DNA ligase (Roche). We screen clones by restriction digest, confirm the mutation by Sanger dideoxy chain termination sequencing and purify the plasmid using a Qiagen maxiprep kit.

Oligonucleotide synthesis and cells. Chimeric oligonucleotides and single-stranded oligonucleotides (including those with the indicated modifications) are synthesized using available phosphoramidites on controlled pore glass supports. After deprotection and detachment from the solid support, each oligonucleotide is gel-purified using, for example, procedures such as those described in Gamper *et al.*, *Biochem.* 39, 5808-5816 (2000) and the concentrations determined spectrophotometrically (33 or 40 μg/ml per A_{260} unit of single-stranded or hairpin oligomer). HUH7 cells are grown in DMEM, 10% FBS, 2mM glutamine, 0.5% pen/strep. The *E.coli* strain, DH10B, is obtained from Life Technologies (Gaithersburg, MD); DH10B cells contain a mutation in the RECA gene (*recA*).

Cell-free extracts. We prepare cell-free extracts from HUH7 cells or other mammalian cells, as follows. We employ this protocol with essentially any mammalian cell including, for example, H1299 cells (human epithelial carcinoma, non-small cell lung cancer), C127I (immortal murine mammary epithelial cells), MEF (mouse embryonic fibroblasts), HEC-1-A (human uterine carcinoma), HCT15 (human colon cancer), HCT116 (human colon carcinoma), LoVo (human colon adenocarcinoma), and HeLa (human cervical carcinoma). We harvest approximately $2x10^8$ cells. We then wash the cells immediately in cold hypotonic buffer (20 mM HEPES, pH7.5; 5 mM KCl; 1.5 mM $MgCl_2$; 1 mM DTT) with 250 mM sucrose. We then resuspend the cells in cold hypotonic buffer without sucrose and after 15 minutes we lyse the cells with 25 strokes of a Dounce homogenizer using a tight fitting pestle. We incubate the lysed cells for 60 minutes on ice and centrifuge the sample for 15 minutes at 12000x*g*. The cytoplasmic fraction is enriched with nuclear proteins due to the extended co-incubation of the fractions following cell breakage. We then immediately aliquote and freeze the supernatant at -80ºC. We determine the protein concentration in the extract by the Bradford assay.

We also perform these experiments with cell-free extracts obtained from fungal cells, including, for example, *S. cerevisiae* (yeast), *Ustilago maydis*, and *Candida albicans*. For example, we grow yeast cells into log phase in 2L YPD medium for 3 days at 30ºC. We then centrifuge the cultures at 5000x*g*, resuspend the pellets in a 10% sucrose, 50 mM Tris, 1mM EDTA lysis solution and freeze them on dry ice. After thawing, we add KCl, spermidine and lyticase to final concentrations of 0.25 mM, 5 mM and 0.1 mg/ml, respectively. We incubate the suspension on ice for 60 minutes, add PMSF and Triton X100 to final concentrations of 0.1 mM and 0.1% and continue to incubate on ice for 20 minutes. We centrifuge the lysate at 3000x*g* for 10 minutes to remove larger debris. We then remove the supernatant and clarify it by

centrifuging at 30000x*g* for 15 minutes. We then add glycerol to the clarified extract to a concentration of 10% (v/v) and freeze aliquots at -80ΘC. We determine the protein concentration of the extract by the Bradford assay.

Reaction mixtures of 50 µl are used, consisting of 10-30 µg protein of cell-free extract, which can be optionally substituted with purified proteins or enriched fractions, about 1.5 µg chimeric double-hairpin oligonucleotide or 0.55 µg single-stranded molecule (3S/25G or 6S/25G, see Figure 1), and 1 µg of plasmid DNA (see Figures 2 and 3) in a reaction buffer of 20 mM Tris, pH 7.4, 15 mM $MgCl_2$, 0.4 mM DTT, and 1.0 mM ATP. Reactions are initiated with extract and incubated at 30ΘC for 45 min. The reaction is stopped by placing the tubes on ice and then immediately deproteinized by two phenol/chloroform (1:1) extractions. Samples are then ethanol precipitated. The nucleic acid is pelleted at 15,000 r.p.m. at 4ΘC for 30 min., is washed with 70% ethanol, resuspended in 50 µl H_20, and is stored at -20ΘC. 5 µl of plasmid from the resuspension (~100 ng) was transfected in 20 µl of DH10B cells by electroporation (400 V, 300 µF, 4 kΩ) in a Cell-Porator apparatus (Life Technologies). After electroporation, cells are transferred to a 14 ml Falcon snap-cap tube with 2 ml SOC and shaken at 37ΘC for 1 h. Enhancement of final kan colony counts is achieved by then adding 3 ml SOC with 10 µg/ml kanamycin and the cell suspension is shaken for a further 2 h at 37ΘC. Cells are then spun down at 3750 x g and the pellet is resuspended in 500 µl SOC. 200 µl is added undiluted to each of two kanamycin (50 µg/ml) agar plates and 200 µl of a 10^5 dilution is added to an ampicillin (100 µg/ml) plate. After overnight 37ΘC incubation, bacterial colonies are counted using an Accucount 1000 (Biologics). Gene conversion effectiveness is measured as the ratio of the average of the kan colonies on both plates per amp colonies multiplied by 10^{-5} to correct for the amp dilution.

The following procedure can also be used. 5 µl of resuspended reaction mixtures (total volume 50 µl) are used to transform 20 µl aliquots of electro-competent ΔH10B bacteria using a Cell-Porator apparatus (Life Technologies). The mixtures are allowed to recover in 1 ml SOC at 37ΘC for 1 hour at which time 50 µg/ml kanamycin or 12 µg/ml tetracycline is added for an additional 3 hours. Prior to plating, the bacteria are pelleted and resuspended in 200 µ1 of SOC. 100 µl aliquots are plated onto kan or tet agar plates and 100 µl of a 10^{-4} dilution of the cultures are concurrently plated on agar plates containing 100 µg/ml of ampicillin. Plating is performed in triplicate using sterile Pyrex beads. Colony counts are determined by an Accu-count 1000 plate reader (Biologics). Each plate contains 200-500 ampicillin resistant colonies or 0-500 tetracycline or kanamycin resistant colonies. Resistant colonies are selected for plasmid extraction and DNA sequencing using an ABI Prism kit on an ABI 310 capillary sequencer (PE Biosystems).

Chimeric single-stranded oligonucleotides. In Figure 1 the upper strands of chimeric oligonucleotides I and II are separated into pathways resulting in the generation of single-stranded oligonucleotides that contain (Figure 1A) 2'-O-methyl RNA nucleotides or (Figure 1B) phosphorothioate linkages. Fold changes in repair activity for correction of kan^s in the HUH7 cell-free extract are presented in parenthesis. Each single-stranded oligonucleotide is 25 bases in length and contains a G residue mismatched to the complementary sequence of the kan^s gene.

Molecules bearing 3, 6, 8, 10 and 12 phosphorothioate linkages in the terminal regions at each end of a backbone with a total of 24 linkages (25 bases) are tested in the kan^s system. Alternatively, molecules bearing 2, 4, 5, 7, 9 and 11 in the terminal regions at each end are tested. The results of one such experiment, presented in Table 1 and Figure 1B, illustrate an enhancement of correction activity directed by some of these modified structures. In this illustrative example, the most efficient molecules contained 3 or 6 phosphorothioate linkages at each end of the 25-mer; the activities are approximately equal (molecules IX and X with results of 3.09 and 3.7 respectively). A reduction in alteration activity may be observed as the number of modified linkages in the molecule is further increased. Interestingly, a single-strand molecule containing 24 phosphorothioate linkages is minimally active suggesting that this backbone modification when used throughout the molecule supports only a low level of targeted gene repair or alteration. Such a non-altering, completely modified molecule can provide a baseline control for determining efficiency of correction for a specific oligonucleotide molecule of known sequence in defining the optimum oligonucleotide for a particular alteration event.

The efficiency of gene repair directed by phosphorothioate-modified, single-stranded molecules, in a length dependent fashion, led us to examine the length of the RNA modification used in the original chimera as it relates to correction. Construct III represents the "RNA-containing" strand of chimera I and, as shown in Table 1 and Figure 2A, it promotes inefficient gene repair. But, as shown in the same figure, reducing the RNA residues on each end from 10 to 3 increases the frequency of repair. At equal levels of modification, however, 25-mers with 2'-O-methyl ribonucleotides were less effective gene repair agents than the same oligomers with phosphorothioate linkages. These results reinforce the fact that an RNA containing oligonucleotide is not as effective in promoting gene repair or alteration as a modified DNA oligonucleotide.

Repair of the kanamycin mutation requires a Go C exchange. To confirm that the specific desired correction alteration was obtained, colonies selected at random from multiple experiments are processed and the isolated plasmid DNA is sequenced. As seen in Figure 4, colonies generated through the action of the single-stranded molecules 3S/25G (IX), 6S/25G (X) and 8S/25G (XI) respectively contained plasmid molecules harboring the targeted base correction. While a few colonies appeared on plates derived from

reaction mixtures containing 25-mers with 10 or 12 thioate linkages on both ends, the sequences of the plasmid molecules from these colonies contain nonspecific base changes. In these illustrative examples, the second base of the codon is changed (see Figure 3). These results show that modified single-strands can direct gene repair, but that efficiency and specificity are reduced when the 25-mers contain 10 or more phosphorothioate linkages at each end.

In Figure 1, the numbers 3, 6, 8, 10, 12 and 12.5 respectively indicate how many phosphorothioate linkages (S) or 2'-O-methyl RNA nucleotides (R) are at each end of the examplified molecule although other molecules with 2, 4, 5, 7, 9 and 11 modifications at each end can also be tested. Hence oligo 12S/25G represents a 25-mer oligonucleotide which contains 12 phosphorothioate linkages on each side of the central G target mismatch base producing a fully phosphorothioate linked backbone, displayed as a dotted line. The dots are merely representative of a linkage in the figure and do not depict the actual number of linkages of the oligonucleotide. Smooth lines indicate DNA residues, wavy lines indicate 2'-O-methyl RNA residues and the carat indicates the mismatched base site (G).

Correction of a mutant kanamycin gene in cultured mammalian cells. The experiments are performed using different mammalian cells, including, for example, 293 cells (transformed human primary kidney cells), HeLa cells (human cervical carcinoma), and H1299 (human epithelial carcinoma, non-small cell lung cancer). HeLa cells are grown at 37°C and 5% CO_2 in a humidified incubator to a density of 2 x 10^5 cells/ml in an 8 chamber slide (Lab-Tek). After replacing the regular DMEM with Optimem, the cells are co-transfected with 10 µg of plasmid pAURNeo(-)FlAsH and 5 µg of modified single-stranded oligonucleotide (3S/25G) that is previously complexed with 10 µg lipofectamine, according to the manufacturer's directions (Life Technologies). The cells are treated with the liposome-DNA-oligo mix for 6 hrs at 37°C. Treated cells are washed with PBS and fresh DMEM is added. After a 16-18 hr recovery period, the culture is assayed for gene repair. The same oligonucleotide used in the cell-free extract experiments is used to target transfected plasmid bearing the kan^s gene. Correction of the point mutation in this gene eliminates a stop codon and restores full expression. This expression can be detected by adding a small non-fluorescent ligand that bound to a C-C-R-E-C-C (SEQ ID NO: 4385) sequence in the genetically modified carboxy terminus of the kan protein, to produce a highly fluorescent complex (FlAsH system, Aurora Biosciences Corporation). Following a 60 min incubation at room temperature with the ligand (FlAsH-EDT2), cells expressing full length kan product acquire an intense green fluorescence detectable by fluorescence microscopy using a fluorescein filter set. Similar experiments are performed using the HygeGFP target as described in Example 2 with a variety of mammalian cells, including, for example, COS-1 and COS-7 cells (African green monkey),

and CHO-K1 cells (Chinese hamster ovary). The experiments are also performed with PG12 cells (rat pheochromocytoma) and ES cells (human embryonic stem cells).

Summary of experimental results. Tables 1, 2 and 3 respectively provide data on the efficiency of gene repair directed by single-stranded oligonucleotides. Table 1 presents data using a cell-free extract from human liver cells (HUH7) to catalyze repair of the point mutation in plasmid pkan^{s}m4021 (see Figure 1). Table 2 illustrates that the oligomers are not dependent on MSH2 or MSH3 for optimal gene repair activity. Table 3 illustrates data from the repair of a frameshift mutation (Figure 3) in the tet gene contained in plasmid pTetΔ208. Table 4 illustrates data from repair of the pkan^{s}m4021 point mutation catalyzed by plant cell extracts prepared from canola and musa (banana). Colony numbers are presented as kan^r or tet^r and fold increases (single strand versus double hairpin) are presented for kan^r in Table 1.

Figure 5A is a confocal picture of HeLa cells expressing the corrected fusion protein from an episomal target. Gene repair is accomplished by the action of a modified single-stranded oligonucleotide containing 3 phosphorothioate linkages at each end (3S/25G). Figure 5B represents a "Z-series" of HeLa cells bearing the corrected fusion gene. This series sections the cells from bottom to top and illustrates that the fluorescent signal is "inside the cells".

Results. In summary, we have designed a novel class of single-stranded oligonucleotides with backbone modifications at the termini and demonstrate gene repair/conversion activity in mammalian and plant cell-free extracts. We confirm that the all DNA strand of the RNA-DNA double-stranded double hairpin chimera is the active component in the process of gene repair. In some cases, the relative frequency of repair by the novel oligonucleotides of the invention is elevated approximately 3-4-fold when compared to frequencies directed by chimeric RNA-DNA double hairpin oligonucleotides.

This strategy centers around the use of extracts from various sources to correct a mutation in a plasmid using a modified single-stranded or a chimeric RNA-DNA double hairpin oligonucleotide. A mutation is placed inside the coding region of a gene conferring antibiotic resistance in bacteria, here kanamycin or tetracycline. The appearance of resistance is measured by genetic readout in *E.coli* grown in the presence of the specified antibiotic. The importance of this system is that both phenotypic alteration and genetic inheritance can be measured. Plasmid pK^{s}m4021 contains a mutation (To G) at residue 4021 rendering it unable to confer antibiotic resistance in *E.coli.* This point mutation is targeted for repair by oligonucleotides designed to restore kanamycin resistance. To avoid concerns of plasmid contamination skewing the colony counts, the directed correction is from Go C rather than Go T (wild-type). After isolation, the plasmid is electroporated into the DH10B strain of *E.coli*, which contains inactive RecA protein. The number of kanamycin colonies is counted and normalized by ascertaining the number of ampicillin colonies, a process

that controls for the influence of electroporation. The number of colonies generated from three to five independent reactions was averaged and is presented for each experiment. A fold increase number is recorded to aid in comparison.

The original RNA-DNA double hairpin chimera design, e.g., as disclosed in U.S. Patent 5,565,350, consists of two hybridized regions of a single-stranded oligonucleotide folded into a double hairpin configuration. The double-stranded targeting region is made up of a 5 base pair DNA/DNA segment bracketed by 10 base pair RNA/DNA segments. The central base pair is mismatched to the corresponding base pair in the target gene. When a molecule of this design is used to correct the kan^s mutation, gene repair is observed (I in Figure 1A). Chimera II (Figure 1B) differs partly from chimera I in that only the DNA strand of the double hairpin is mismatched to the target sequence. When this chimera was used to correct the kan^s mutation, it was twice as active. In the same study, repair function could be further increased by making the targeting region of the chimera a continuous RNA/DNA hybrid.

Frame shift mutations are repaired. By using plasmid $pT^s\Delta208$, described in Figure 1(C) and Figure 3, the capacity of the modified single-stranded molecules that showed activity in correcting a point mutation, can be tested for repair of a frameshift. To determine efficiency of correction of the mutation, a chimeric oligonucleotide (Tet I), which is designed to insert a T residue at position 208, is used. A modified single-stranded oligonucleotide (Tet IX) directs the insertion of a T residue at this same site. Figure 3 illustrates the plasmid and target bases designated for change in the experiments. When all reaction components are present (extract, plasmid, oligomer), tetracycline resistant colonies appear. The colony count increases with the amount of oligonucleotide used up to a point beyond which the count falls off (Table 3). No colonies above background are observed in the absence of either extract or oligonucleotide, nor when a modified single-stranded molecule bearing perfect complementarity is used. Figure 3 represents the sequence surrounding the target site and shows that a T residue is inserted at the correct site. We have isolated plasmids from fifteen colonies obtained in three independent experiments and each analyzed sequence revealed the same precise nucleotide insertion. These data suggest that the single-stranded molecules used initially for point mutation correction can also repair nucleotide deletions.

Comparison of phosphorothioate oligonucleotides to 2'-O-methyl substituted oligonucleotides. From a comparison of molecules VII and XI, it is apparent that gene repair is more subject to inhibition by RNA residues than by phosphorothioate linkages. Thus, even though both of these oligonucleotides contain an equal number of modifications to impart nuclease resistance, XI (with 16 phosphorothioate linkages) has good gene repair activity while VII (with 16 2'-O-methyl RNA residues) is inactive. Hence, the original

chimeric double hairpin oligonucleotide enabled correction directed, in large part, by the strand containing a large region of contiguous DNA residues.

Oligonucleotides can target multiple nucleotide alterations within the same template. The ability of individual single-stranded oligonucleotides to correct multiple mutations in a single target template is tested using the plasmid pK^{s}m4021 and the following single-stranded oligonucleotides modified with 3 phosphorothioate linkages at each end (indicated as underlined nucleotides): Oligo1 is a 25-mer with the sequence TTCGATAAGCCTATGCTGACCCGTG (SEQ ID NO: 4377) corrects the original mutation present in the kanamycin resistance gene of pK^{s}m4021 as well as directing another alteration 2 basepairs away in the target sequence (both indicated in boldface); Oligo2 is a 70-mer with the 5'-end sequence TTCGGCTACGACTGGGCACAACAGACAATTGGC (SEQ ID NO: 4378) with the remaining nucleotides being completely complementary to the kanamycin resistance gene and also ending in 3 phosphorothioate linkages at the 3' end. Oligo2 directs correction of the mutation in pK^{s}m4021 as well as directing another alteration 21 basepairs away in the target sequence (both indicated in boldface).

We also use additional oligonucleotides to assay the ability of individual oligonucleotides to correct multiple mutations in the pK^{s}M4021 plasmid. These include, for example, a second 25-mer that alters two nucleotides that are three nucleotides apart with the sequence 5'-TTGTGCCCAGTCGTATCCGAATAGC-3' (SEQ ID NO: 4379); a 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-CATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAATAGCCTCTCCACCCAAGCGGCCGGAGA -3' (SEQ ID NO: 4380); and another 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-GCTGACAGCCGGAACACGGCGGCATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAATAGCCT-3' (SEQ ID NO: 4381). The nucleotides in the oligonucleotides that direct alteration of the target sequence are underlined and in boldface. These oligonucleotides are modified in the same way as the other oligonucleotides of the invention.

We assay correction of the original mutation in pK^{s}m4021 by monitoring kanamycin resistance (the second alterations which are directed by Oligo2 and Oligo3 are silent with respect to the kanamycin resistance phenotype). In addition, in experiments with Oligo2, we also monitor cleavage of the resulting plasmids using the restriction enzyme Tsp509I which cuts at a specific site present only when the second alteration has occurred (at ATT in Oligo2). We then sequence these clones to determine whether the additional, silent alteration has also been introduced. The results of an analysis are presented below:

	Oligo1 (25-mer)	Oligo2 (70-mer)
Clones with both sites changed	9	7
Clones with a single site changed	0	2
Clones that were not changed	4	1

Nuclease sensitivity of unmodified DNA oligonucleotide. Electrophoretic analysis of nucleic acid recovered from the cell-free extract reactions conducted here confirm that the unmodified single-stranded 25-mer did not survive incubation whereas greater than 90% of the terminally modified oligos did survive (as judged by photo-image analyses of agarose gels).

Plant extracts direct repair. The modified single-stranded constructs can be tested in plant cell extracts. We have observed gene alteration using extracts from multiple plant sources, including, for example, Arabidopsis, tobacco, banana, maize, soybean, canola, wheat, spinach as well as spinach chloroplast extract. We prepare the extracts by grinding plant tissue or cultured cells under liquid nitrogen with a mortar and pestle. We extract 3 ml of the ground plant tissue with 1.5 ml of extraction buffer (20 mM HEPES, pH7.5; 5 mM Kcl; 1.5 mM $MgCl_2$; 10 mM DTT; 10% [v/v] glycerol; and 1 % [w/v] PVP). We then homogenize the samples with 15 strokes of a Dounce homogenizer. Following homogenization, we incubate the samples on ice for 1 hour and centrifuge at 3000x*g* for 5 minutes to remove plant cell debris. We then determine the protein concentration in the supernatants (extracts) by Bradford assay. We dispense 100 µg (protein) aliquots of the extracts which we freeze in a dry ice-ethanol bath and store at -80ºC.

We describe experiments using two sources here: a dicot (canola) and a monocot (banana, *Musa acuminata* cv. Rasthali). Each vector directs gene repair of the kanamycin mutation (Table 4); however, the level of correction is elevated 2-3 fold relative to the frequency observed with the chimeric oligonucleotide. These results are similar to those observed in the mammalian system wherein a significant improvement in gene repair occurred when modified single-stranded molecules were used.

Tables are attached hereto.

TABLE 1

Gene repair activity is directed by single-stranded oligonucleotides

Oligonucleotide	Plasmid	Extract (ug)	kan^r colonies	Fold Increase
I	pK^{S}m4021	10	300	
I		20	418	1.0x
II		10	537	
II		20	748	1.78x
III		10	3	
III		20	5	0.01x
IV		10	112	
IV		20	96	0.22x
V		10	217	
V		20	342	0.81x
VI		10	6	
VI		20	39	0.093x
VII		10	0	
VII		20	0	0x
VIII		10	3	
VIII		20	5	0.01x
IX		10	936	
IX		20	1295	3.09x
X		10	1140	
X		20	1588	3.7x
XI		10	480	
XI		20	681	1.6x
XII		10	18	
XII		20	25	0.059x
XIII		10	0	
XIII		20	4	0.009x
-		20	0	
I		-	0	

Plasmid pK^{S}m4021 (1µg), the indicated oligonucleotide (1.5 µg chimeric oligonucleotide or 0.55 µg single-stranded oligonucleotide; molar ratio of oligo to plasmid of 360 to 1) and either 10 or 20 µg of HUH7 cell-free extract were incubated 45 min at 37ΘC. Isolated plasmid DNA was electroporated into *E. coli* (strain DH10B) and the number of kan^r colonies counted. The data represent the number of kanamycin resistant colonies per 10^3 ampicillin resistant colonies generated from the same reaction and is the average of three experiments (standard deviation usually less than +/-15%). Fold increase is defined relative to 418 kan^r colonies (second reaction) and in all reactions was calculated using the 20µg sample.

TABLE 2

Modified single-stranded oligomers are not dependent on msh2 or msh3 for optimal gene repair activity

A.

Oligonucleotide	Plasmid	Extract	kan^r colonies
IX (3S/25G)		HUH7	637
X (6S/25G)		HUH7	836
IX		MEF2$^{-/-}$	781
X		MEF2$^{-/-}$	676
IX		MEF3$^{-/-}$	582
X		MEF3$^{-/-}$	530
IX		MEF$^{+/+}$	332
X		MEF$^{+/+}$	497
-		MEF2$^{-/-}$	10
-		MEF3$^{-/-}$	5
-		MEF$^{+/+}$	14

Chimeric oligonucleotide (1.5 µg) or modified single-stranded oligonucleotide (0.55 µg) was incubated with 1 µg of plasmid pK^{S}m4021 and 20µg of the indicated extracts. MEF represents mouse embryonic fibroblasts with either MSH2 (2$^{-/-}$) or MSH3 (3$^{-/-}$) deleted. MEF$^{+/+}$ indicates wild-type mouse embryonic fibroblasts. The other reaction components were then added and processed through the bacterial readout system. The data represent the number of kanamycin resistant colonies per 10^6 ampicillin resistant colonies.

TABLE 3

Frameshift mutation repair is directed by single-stranded oligonucleotides

Oligonucleotide	Plasmid	Extract	tet^r colonies
Tet IX (3S/25A; 0.5µg)	pT^{S}Δ208 (1µg)		0
-		20µg	0
Tet IX (0.5µg)			48
Tet IX (1.5µg)			130
Tet IX (2.0µg)			68
Tet I (chimera; 1.5µg)			48

Each reaction mixture contained the indicated amounts of plasmid and oligonucleotide. The extract used for these experiments came from HUH7 cells. The data represent the number of tetracycline resistant colonies per 10^6 ampicillin resistant colonies generated from the same reaction and is the average of 3 independent experiments. Tet I is a chimeric oligonucleotide and Tet IX is a modified single-stranded

oligonucleotide that are designed to insert a T residue at position 208 of pT^{s}Δ208. The oligonucleotides are equivalent to structures I and IX in Figure 2.

TABLE 4

Plant cell-free extracts support gene repair by single-stranded oligonucleotides

Oligonucleotide	Plasmid	Extract		kan^{r} colonies
II (chimera)	pK^{s}m4021	30µg	Canola	337
IX (3S/25G)			Canola	763
X (6S/25G)			Canola	882
II			*Musa*	203
IX			*Musa*	343
X			*Musa*	746
-			Canola	0
-			*Musa*	0
IX		-	Canola	0
X		-	*Musa*	0

Canola or Musa cell-free extracts were tested for gene repair activity on the kanamycin-sensitive gene as previously described in (18). Chimeric oligonucleotide II (1.5µg) and modified single-stranded oligonucleotides IX and X (0.55µg) were used to correct pK^{s}m4021. Total number of kan^{r} colonies are present per 10^{7} ampicillin resistant colonies and represent an average of four independent experiments.

TABLE 5

Gene repair activity in cell-free extracts prepared from yeast (saccharomyces cerevisae)

Cell-type	Plasmid	Chimeric Oligo	SS Oligo	kan^{r} /amp^{r} x 10^{6}
Wild type	pKan^{s}m4021	1µg		0.36
Wild type			1µg	0.81
ΔRAD52		1µg		10.72
ΔRAD52			1µg	17.41
ΔPMS1		1µg		2.02
ΔPMS1			1µg	3.23

In this experiment, the kan^{s} gene in pKan^{s}4021 is corrected by either a chimeric double-hairpin oligonucleotide or a single-stranded oligonucleotide containing three thioate linkages at each end (3S/25G).

EXAMPLE 2

Yeast Cell Targeting Assay Method for Base Alteration and Preferred Oligonucleotide Selection

In this example, single-stranded oligonucleotides with modified backbones and double-hairpin oligonucleotides with chimeric, RNA-DNA backbones are used to measure gene repair using two episomal targets with a fusion between a hygromycin resistance gene and eGFP as a target for gene repair. These plasmids are pAURHYG(rep)GFP, which contains a point mutation in the hygromycin resistance gene (Figure 7), pAURHYG(ins)GFP, which contains a single-base insertion in the hygromycin resistance gene (Figure 7) and pAURHYG(Δ)GFP which has a single base deletion. We also use the plasmid containing a wild-type copy of the hygromycin-eGFP fusion gene, designated pAURHYG(wt)GFP, as a control. These plasmids also contain an aureobasidinA resistance gene. In pAURHYG(rep)GFP, hygromycin resistance gene function and green fluorescence from the eGFP protein are restored when a G at position 137, at codon 46 of the hygromycin B coding sequence, is converted to a C thus removing a premature stop codon in the hygromycin resistance gene coding region. In pAURHYG(ins)GFP, hygromycin resistance gene function and green fluorescence from the eGFP protein are restored when an A inserted between nucleotide positions 136 and 137, at codon 46 of the hygromycin B coding sequence, is deleted and a C is substituted for the T at position 137, thus correcting a frameshift mutation and restoring the reading frame of the hygromycin-eGFP fusion gene.

We synthesize the set of three yeast expression constructs pAURHYG(rep)eGFP, pAURHYG(Δ)eGFP, pAURHYG(ins)eGFP, that contain a point mutation at nucleotide 137 of the hygromycin-B coding sequence as follows. (rep) indicates a T137→G replacement, (Δ) represents a deletion of the G137 and (ins) represents an A insertion between nucleotides 136 and 137. We construct this set of plasmids by excising the respective expression cassettes by restriction digest from pHyg(x)EGFP and ligation into pAUR123 (Panvera, CA). We digest 10 μg pAUR123 vector DNA, as well as, 10 μg of each pHyg(x)EGFP construct with KpnI and SalI (NEB). We gel purify each of the DNA fragments and prepare them for enzymatic ligation. We ligate each mutated insert into pHygEGFP vector at 3:1 molar ration using T4 DNA ligase (Roche). We screen clones by restriction digest, confirm by Sanger dideoxy chain termination sequencing and purify using a Qiagen maxiprep kit.

We use this system to assay the ability of five oligonucleotides (shown in Figure 8) to support correction under a variety of conditions. The oligonucleotides which direct correction of the mutation in pAURHYG(rep)GFP can also direct correction of the mutation in pAURHYG(ins)GFP. Three of the four

oligonucleotides (HygE3T/25, HygE3T/74 and HygGG/Rev) share the same 25-base sequence surrounding the base targeted for alteration. HygGG/Rev is an RNA-DNA chimeric double hairpin oligonucleotide of the type described in the prior art. One of these oligonucleotides, HygE3T/74, is a 74-base oligonucleotide with the 25-base sequence centrally positioned. The fourth oligonucleotide, designated HygE3T/74α, is the reverse complement of HygE3T/74. The fifth oligonucleotide, designated Kan70T, is a non-specific, control oligonucleotide which is not complementary to the target sequence. Alternatively, an oligonucleotide of identical sequence but lacking a mismatch to the target or a completely thioate modified oligonucleotide or a completely 2-0-methylated modified oligonucleotide may be used as a control.

Oligonucleotide synthesis and cells. We synthesized and purified the chimeric, double-hairpin oligonucleotides and single-stranded oligonucleotides (including those with the indicated modifications) as described in Example 1. Plasmids used for assay were maintained stably in yeast (*Saccharomyces cerevisiae*) strain LSY678 *MATα* at low copy number under aureobasidin selection. Plasmids and oligonucleotides are introduced into yeast cells by electroporation as follows: to prepare electrocompetent yeast cells, we inoculate 10 ml of YPD media from a single colony and grow the cultures overnight with shaking at 300 rpm at 30ϴC. We then add 30 ml of fresh YPD media to the overnight cultures and continue shaking at 30ϴC until the OD_{600} was between 0.5 and 1.0 (3-5 hours). We then wash the cells by centrifuging at 4ϴC at 3000 rpm for 5 minutes and twice resuspending the cells in 25 ml ice-cold distilled water. We then centrifuge at 4ϴC at 3000 rpm for 5 minutes and resuspend in 1 ml ice-cold 1M sorbitol and then finally centrifuge the cells at 4ϴC at 5000 rpm for 5 minutes and resuspend the cells in 120 µl 1M sorbitol. To transform electrocompetent cells with plasmids or oligonucleotides, we mix 40 µl of cells with 5 µg of nucleic acid, unless otherwise stated, and incubate on ice for 5 minutes. We then transfer the mixture to a 0.2 cm electroporation cuvette and electroporate with a BIO-RAD Gene Pulser apparatus at 1.5 kV, 25 µF, 200 Ω for one five-second pulse. We then immediately resuspend the cells in 1 ml YPD supplemented with 1M sorbitol and incubate the cultures at 30ϴC with shaking at 300 rpm for 6 hours. We then spread 200 µl of this culture on selective plates containing 300 µg/ml hygromycin and spread 200 µl of a 10^5 dilution of this culture on selective plates containing 500 ng/ml aureobasidinA and/or and incubate at 30ϴC for 3 days to allow individual yeast colonies to grow. We then count the colonies on the plates and calculate the gene conversion efficiency by determining the number of hygromycin resistance colonies per 10^5 aureobasidinA resistant colonies.

Frameshift mutations are repaired in yeast cells. We test the ability of the oligonucleotides shown in Figure 8 to correct a frameshift mutation *in vivo* using LSY678 yeast cells containing the plasmid pAURHYG(ins)GFP. These experiments, presented in Table 6, indicate that these oligonucleotides can

support gene correction in yeast cells. These data reinforce the results described in Example 1 indicating that oligonucleotides comprising phosphorothioate linkages facilitate gene correction much more efficiently than control duplex, chimeric RNA-DNA oligonucleotides. This gene correction activity is also specific as transformation of cells with the control oligonucleotide Kan70T produced no hygromycin resistant colonies above background and thus Kan70T did not support gene correction in this system. In addition, we observe that the 74-base oligonucleotide (HygE3T/74) corrects the mutation in pAURHYG(ins)GFP approximately five-fold more efficiently than the 25-base oligonucleotide (HygE3T/25). We also perform control experiments with LSY678 yeast cells containing the plasmid pAURHYG(wt)GFP. With this strain we observed that even without added oligonucleotides, there are too many hygromycin resistant colonies to count.

We also use additional oligonucleotides to assay the ability of individual oligonucleotides to correct multiple mutations in the pK^{s}M4021 plasmid. These include, for example, a second 25-mer that alters two nucleotides that are three nucleotides apart with the sequence 5'-TTGTGCCCAGTCGTATCCGAATAGC-3' (SEQ ID NO: 4379); a 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-CATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAATAGCCTCTCCACCCAAGCGGCCGGAGA -3' (SEQ ID NO: 4380); and another 70-mer that alters two nucleotides that are 21 nucleotides apart with the sequence 5'-GCTGACAGCCGGAACACGGCGGCATCAGAGCAGCCAATTGTCTGTTGTGCCCAGTCGTAGCCGAATAGCCT-3' (SEQ ID NO: 4381). The nucleotides in the oligonucleotides that direct alteration of the target sequence are underlined and in boldface. These oligonucleotides are modified in the same way as the other oligonucleotides of the invention.

Oligonucleotides targeting the sense strand direct gene correction more efficiently. We compare the ability of single-stranded oligonucleotides to target each of the two strands of the target sequence of both pAURHYG(ins)GFP and pAURHYG(rep)GFP. These experiments, presented in Tables 7 and 8, indicate that an oligonucleotide, HygE3T/74α, with sequence complementary to the sense strand (i.e. the strand of the target sequence that is identical to the mRNA) of the target sequence facilitates gene correction approximately ten-fold more efficiently than an oligonucleotide, HygE3T/74, with sequence complementary to the non-transcribed strand which serves as the template for the synthesis of RNA. As indicated in Table 7, this effect was observed over a range of oligonucleotide concentrations from 0-3.6 μg, although we did observe some variability in the difference between the two oligonucleotides (indicated in Table 7 as a fold difference between HygE3T/74α and HygE3T/74). Furthermore, as shown in Table 8, we observe increased efficiency of correction by HygE3T/74α relative to HygE3T/74 regardless of whether the oligonucleotides were used to correct the base substitution mutation in pAURHYG(rep)GFP or the insertion mutation in

pAURHYG(ins)GFP. The data presented in Table 8 further indicate that the single-stranded oligonucleotides correct a base substitution mutation more efficiently than an insertion mutation. However, this last effect was much less pronounced and the oligonucleotides of the invention are clearly able efficiently to correct both types of mutations in yeast cells. In addition, the role of transcription is investigated using plasmids with inducible promoters such as that described in Figure 10.

Optimization of oligonucleotide concentration. To determine the optimal concentration of oligonucleotide for the purpose of gene alteration, we test the ability of increasing concentrations of Hyg3T/74α to correct the mutation in pAURHYG(rep)GFP contained in yeast LSY678. We chose this assay system because our previous experiments indicated that it supports the highest level of correction. However, this same approach could be used to determine the optimal concentration of any given oligonucleotide. We test the ability of Hyg3T/74α to correct the mutation in pAURHYG(rep)GFP contained in yeast LSY678 over a range of oligonucleotide concentrations from 0-10.0 μg. As shown in Table 9, we observe that the correction efficiency initially increases with increasing oligonucleotide concentration, but then declines at the highest concentration tested.

Tables are attached hereto.

Table 6

Correction of an insertion mutation in pAURHYG(ins)GFP by HygGG/Rev, HygE3T/25 and HygE3T/74

Oligonucleotide Tested	Colonies on Hygromycin	Colonies on Aureobasidin (/10^5)	Correction Efficiency
HygGG/Rev	3	157	0.02
HygE3T/25	64	147	0.44
HygE3T/74	280	174	1.61
Kan70T	0	—	—

Table 7

An oligonucleotide targeting the sense strand of the target sequence corrects more efficiently

Amount of Oligonucleotide (μg)	Colonies per hygromycin plate	
	HygE3T/74	HygE3T/74α
0	0	0
0.6	24	128 (8.4x)*
1.2	69	140 (7.5x)*
2.4	62	167 (3.8x)*
3.6	29	367 (15x)*

* The numbers in parentheses represent the fold increase in efficiency for targeting the non-transcribed strand as compared to the other strand of a DNA duplex that encodes a protein.

Table 8

Correction of a base substitution mutation is more efficient than correction of a frame shift mutation

Oligonucleotide Tested (5 μg)	Plasmid tested (contained in LSY678)	
	pAURHYG(ins)GFP	pAURHYG(rep)GFP
HygE3T/74	72	277
HygE3T/74α	1464	2248
Kan70T	0	0

Table 9

Optimization of oligonucleotide concentration in electroporated yeast cells.

Amount (μg)	Colonies on hygromycin	Colonies on aureobasidin (/10^5)	Correction efficiency
0	0	67	0
1.0	5	64	0.08
2.5	47	30	1.57
5.0	199	33	6.08
7.5	383	39	9.79
10.0	191	33	5.79

EXAMPLE 3

Cultured Cell Manipulation

Mononuclear cells are isolated from human umbilical cord blood of normal donors using Ficoll Hypaque (Pharmacia Biotech, Uppsala, Sweden) density centrifugation. CD34+ cells are immunomagnetically purified from mononuclear cells using either the progenitor or Multisort Kits (Miltenyi Biotec, Auburn, CA). Lin-CD38- cells are purified from the mononuclear cells using negative selection with StemSep system according to the manufacturer's protocol (Stem Cell Technologies, Vancouver, CA). Cells used for microinjection are either freshly isolated or cryopreserved and cultured in Stem Medium (S Medium) for 2 to 5 days prior to microinjection. S Medium contains Iscoves' Modified Dulbecco's Medium without phenol red (IMDM) with 100 μg/ml glutamine/penicillin/streptomycin, 50 mg/ml bovine serum albumin, 50 μg/ml bovine pancreatic insulin, 1 mg/ml human transferrin, and IMDM; Stem Cell Technologies), 40 μg/ml low-density lipoprotein (LDL; Sigma, St. Louis, MO), 50 mM HEPEs buffer and 50 μM 2-mercaptoethanol, 20 ng/ml each of thrombopoietin, flt-3 ligand, stem cell factor and human IL-6 (Pepro Tech Inc., Rocky Hill, NJ). After microinjection, cells are detached and transferred in bulk into wells of 48 well plates for culturing. 35 mm dishes are coated overnight at 4θ C with 50 μg/ml Fibronectin (FN) fragment CH-296 (Retronectin; TaKaRa Biomedicals, Panvera, Madison, WI) in phosphate buffered saline and washed with IMDM containing

glutamine/penicillin/streptomycin. 300 to 2000 cells are added to cloning rings and attached to the plates for 45 minutes at 37θC prior to microinjection. After incubation, cloning rings are removed and 2 ml of S Medium are added to each dish for microinjection. Pulled injection needles with a range of 0.22 μ to 0.3 μ outer tip diameter are used. Cells are visualized with a microscope equipped with a temperature controlled stage set at 37θ C and injected using an electronically interfaced Eppendorf Micromanipulator and Transjector. Successfully injected cells are intact, alive and remain attached to the plate post injection. Molecules that are flourescently labeled allow determination of the amount of oligonucleotide delivered to the cells.

For in vitro erythropoiesis from Lin-CD38- cells, the procedure of Malik, 1998 can be used. Cells are cultured in ME Medium for 4 days and then cultured in E Medium for 3 weeks. Erythropoiesis is evident by glycophorin A expression as well as the presence of red color representing the presence of hemoglobin in the cultured cells. The injected cells are able to retain their proliferative capacity and the ability to generate myeloid and erythoid progeny. CD34+ cells can convert a normal A (β^A) to sickle T (β^S) mutation in the β-globin gene or can be altered using any of the oligonucleotides of the invention herein for correction or alteration of a normal gene to a mutant gene. Alternatively, stem cells can be isolated from blood of humans having genetic disease mutations and the oligonucleotides of the invention can be used to correct a defect or to modify genomes within those cells.

Alternatively, non-stem cell populations of cultured cells can be manipulated using any method known to those of skill in the art including, for example, the use of polycations, cationic lipids, liposomes, polyethylenimine (PEI), electroporation, biolistics, calcium phophate precipitation, or any other method known in the art.

Notes on the tables presented below:

Each of the following tables presents, for the specified human gene, a plurality of mutations that are known to confer a clinically-relevant phenotype and, for each mutation, the oligonucleotides that can be used to correct the respective mutation site-specifically in the human genome according to the present invention.

The left-most column identifies each mutation and the clinical phenotype that the mutation confers.

For most entries, the mutation is identified at both the nucleic acid and protein level. At the amino acid level, mutations are presented according to the following standard nomenclature. The centered number identifies the position of the mutated codon in the protein sequence; to the left of the number is the wild type residue and to the right of the number is the mutant codon. Codon numbering is according to the Human Gene Mutation Database, Cardiff, Wales, UK

(http://archive.uwcm.ac.uk/search/mg/allgenes).

Terminator codons are shown as "TERM". At the nucleic acid level, the entire triplet of the wild type and mutated codons is shown.

The middle column presents, for each mutation, four oligonucleotides capable of repairing the mutation site-specifically in the human genome or in cloned human DNA including human DNA in artificial chromosomes, episomes, plasmids, or other types of vectors. The oligonucleotides of the invention, however, may include any of the oligonucleotides sharing portions of the sequence of the 121 base sequence. Thus, oligonucleotides of the invention for each of the depicted targets may be 18, 19, 20 up to about 121 nucleotides in length. Sequence may be added non-symmetrically.

All oligonucleotides are presented, per convention, in the 5' to 3' orientation. The nucleotide that effects the change in the genome is underlined and presented in bold.

The first of the four oligonucleotides for each mutation is a 121 nt oligonucleotide centered about the repair nucleotide. The second oligonucleotide, its reverse complement, targets the opposite strand of the DNA duplex for repair. The third oligonucleotide is the minimal 17 nt domain of the first oligonucleotide, also centered about the repair nucleotide. The fourth oligonucleotide is the reverse complement of the third, and thus represents the minimal 17 nt domain of the second.

The third column of each table presents the SEQ ID NO: of the respective repair oligonucleotide.

EXAMPLE 4

Adenosine Deaminase (ADA)

Adenosine deaminase (ADA, EC 3.5.4.4) catalyses the deamination of adenosine and 2'-deoxyadenosine to inosine or 2'-deoxyinosine respectively. ADA deficiency has been identified as the metabolic basis for 20-30% of cases with recessively inherited severe combined immunodeficiency (SCID). Affected infants are subject to recurrent chronic viral, fungal, protozoal, and bacterial infections and frequently present with persistent diarrhea, failure to thrive and candidiasis. In patients homozygous for ADA deficiency, 2'-deoxyadenosine accumulating during the rapid turnover of cells rich in DNA is converted back to dATP, either by adenosine kinase or deoxycytidine kinase. Many hypotheses have been advanced to explain the specific toxicity to the immune system in ADA deficiency. The apparently selective accumulation of dATP in thymocytes and peripheral blood B cells, with resultant inhibition of ribonucleotide reductase and DNA synthesis is probably the principal mechanism.

The structural gene for ADA is encoded as a single 32 kb locus containing 12 exons. Studies of the molecular defect in ADA-deficient patients have shown that mRNA is usually detectable in normal or

supranormal amounts. Specific base substitution mutations have been detected in the majority of cases with the complete deficiency. A C-to-T base substitution mutation in exon 11 accounts for a high proportion of these, whilst a few patients are homozygous for large deletions encompassing exon I. A common point mutation resulting in a heat-labile ADA has been characterised in some patients with partial ADA deficiency, a disorder with an apparently increased prevalence in the Caribbean.

As yet no totally effective therapy for ADA deficiency has been reported, except in those few cases where bone marrow from an HLA/MLR compatible sibling donor was available.

Two therapeutic approaches have provided long-term benefit in specific instances. First, reconstitution using T cell depleted mismatched sibling marrow has been encouraging, particularly in early presenters completely deficient in ADA. Secondly, therapy with polyethylene glycol-modified adenosine deaminase (PEG-ADA) for more than 5 years has produced a sustained increase in lymphocyte numbers and mitogen responses together with evidence of in vivo B cell function. Success has generally been achieved in late presenters with residual ADA activity in mononuclear cells.

ADA deficiency has been chosen as the candidate disease for gene replacement therapy and the first human experiment commenced in 1990. The clinical consequences of overexpression of ADA activity - one of the potential hazards of gene implant - are known and take the form of an hereditary haemolytic anaemia associated with a tissue-specific increase in ADA activity. The genetic basis for the latter autosomal dominant disorder seemingly relates to markedly increased levels of structurally normal ADA mRNA.

Table 10

ADA Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency GLN3TERM CAG to TAG	AGAGACCCACCGAGCGGCGGCGGAGGGAGCAGCGCCGGGG CGCACGAGGGCACCATGGCCCAGACGCCCGCCTTCGACAAG CCCAAAGTGAGCGCGCGCGGGGGCTCCGGGGACGGGGGTC	1
	GACCCCCGTCCCCGGAGCCCCCGCGCGCGCTCACTTTGGGC TTGTCGAAGGCGGGCGTCTGGGCCATGGTGCCCTCGTGCGC CCCGGCGCTGCTCCCTCCGCCGCCGCTCGGTGGGTCTCT	2
	CCATGGCCCAGACGCCC	3
	GGGCGTCTGGGCCATGG	4

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency HIS15ASP CAT to GAT	TATTTGTTCTCTCTCTCCCTTTCTCTCTCTCTTCCCCCTGCCCC CTTGCAGGTAGAACTG**C**ATGTCCACCTAGACGGATCCATCAA GCCTGAAACCATCTTATACTATGGCAGGTAAGTCC	5
	GGACTTACCTGCCATAGTATAAGATGGTTTCAGGCTTGATGG ATCCGTCTAGGTGGACAT**G**CAGTTCTACCTGCAAGGGGGCAG GGGGAAGAGAGAGAGAAAGGGAGAGAGAGAACAAATA	6
	TAGAACTG**C**ATGTCCAC	7
	GTGGACAT**G**CAGTTCTA	8
Adenosine deaminase deficiency GLY20ARG GGA to AGA	TCCCTTTCTCTCTCTCTTCCCCCTGCCCCCTTGCAGGTAGAAC TGCATGTCCACCTAGAC**G**GATCCATCAAGCCTGAAACCATCTT ATACTATGGCAGGTAAGTCCATACAGAAGAGCCCT	9
	AGGGCTCTTCTGTATGGACTTACCTGCCATAGTATAAGATGGT TTCAGGCTTGATGGATC**C**GTCTAGGTGGACATGCAGTTCTAC CTGCAAGGGGGCAGGGGGAAGAGAGAGAGAAAGGGA	10
	ACCTAGAC**G**GATCCATC	11
	GATGGATC**C**GTCTAGGT	12
Adenosine deaminase deficiency GLY74CYS GGC to TGC	CCTGGAGCTCCCAAGGGACTTGGGGAAGGTTGTTCCCAACC CCTTTCTTCCCTTCCCAGG**G**GCTGCCGGGAGGCTATCAAAAG GATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGG	13
	CCTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCCTTTT GATAGCCTCCCGGCAGC**C**CCTGGGAAGGGAAGAAAGGGGTT GGGAACAACCTTCCCCAAGTCCCTTGGGAGCTCCAGG	14
	CTATCGCG**G**GCTGCCGG	15
	CCGGCAGC**C**CGCGATAG	16
Adenosine Deaminase Deficiency ARG76TRP CGG to TGG	GCTCCCAAGGGACTTGGGGAAGGTTGTTCCCAACCCCTTTCT TCCCTTCCCAGGGGCTGC**C**GGGAGGCTATCAAAAGGATCGC CTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCGTGG	17
	CCACGCCCTCTTTGGCCTTCATCTCTACAAACTCATAGGCGAT CCTTTTGATAGCCTCCC**G**GCAGCCCCTGGGAAGGGAAGAAA GGGGTTGGGAACAACCTTCCCCAAGTCCCTTGGGAGC	18
	GGGGCTGC**C**GGGAGGCT	19
	AGCCTCCC**G**GCAGCCCC	20

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine Deaminase Deficiency LYS80ARG AAA to AGA	TTGGGGAAGGTTGTTCCCAACCCCTTTCTTCCCTTCCCAGGG GCTGCCGGGAGGCTATCAAAAGGATCGCCTATGAGTTTGTAG AGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAGGT	21
	ACCTCCACATACACCACGCCCTCTTTGGCCTTCATCTCTACAA ACTCATAGGCGATCCTTTTGATAGCCTCCCGGCAGCCCCTGG GAAGGGAAGAAAGGGGTTGGGAACAACCTTCCCCAA	22
	GGCTATCAAAAGGATCG	23
	CGATCCTTTTGATAGCC	24
Adenosine deaminase deficiency ALA83ASP GCC to GAC	GTTGTTCCCAACCCCTTTCTTCCCTTCCCAGGGGCTGCCGGG AGGCTATCAAAAGGATCGCCTATGAGTTTGTAGAGATGAAGG CCAAAGAGGGCGTGGTGTATGTGGAGGTGCGGTACAG	25
	CTGTACCGCACCTCCACATACACCACGCCCTCTTTGGCCTTC ATCTCTACAAACTCATAGGCGATCCTTTTGATAGCCTCCCGGC AGCCCCTGGGAAGGGAAGAAAGGGGTTGGGAACAAC	26
	AAGGATCGCCTATGAGT	27
	ACTCATAGGCGATCCTT	28
Adenosine deaminase deficiency TYR97CYS TAT to TGT	AGGCTATCAAAAGGATCGCCTATGAGTTTGTAGAGATGAAGG CCAAAGAGGGCGTGGTGTATGTGGAGGTGCGGTACAGTCCG CACCTGCTGGCCAACTCCAAAGTGGAGCCAATCCCCTG	29
	CAGGGGATTGGCTCCACTTTGGAGTTGGCCAGCAGGTGCGG ACTGTACCGCACCTCCACATACACCACGCCCTCTTTGGCCTT CATCTCTACAAACTCATAGGCGATCCTTTTGATAGCCT	30
	CGTGGTGTATGTGGAGG	31
	CCTCCACATACACCACG	32
Adenosine deaminase deficiency ARG101GLN CGG to CAG	GGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGC GTGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGC CAACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTGA	33
	TCAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCC AGCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCC CTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCC	34
	GGAGGTGCGGTACAGTC	35
	GACTGTACCGCACCTCC	36

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ARG101LEU CGG to CTG	GGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGC GTGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGC CAACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTGA	37
	TCAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCC AGCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCC CTCTTTGGCCTTCATCTCTACAAACTCATAGGCGATCC	38
	GGAGGTGCGGTACAGTC	39
	GACTGTACCGCACCTCC	40
Adenosine deaminase deficiency ARG101TRP CGG to TGG	AGGATCGCCTATGAGTTTGTAGAGATGAAGGCCAAAGAGGGC GTGGTGTATGTGGAGGTGCGGTACAGTCCGCACCTGCTGGC CAACTCCAAAGTGGAGCCAATCCCCTGGAACCAGGCTG	41
	CAGCCTGGTTCCAGGGGATTGGCTCCACTTTGGAGTTGGCCA GCAGGTGCGGACTGTACCGCACCTCCACATACACCACGCCCT CTTTGGCCTTCATCTCTACAAACTCATAGGCGATCCT	42
	TGGAGGTGCGGTACAGT	43
	ACTGTACCGCACCTCCA	44
Adenosine deaminase deficiency PRO104LEU CCG to CTG	ATGAGTTTGTAGAGATGAAGGCCAAAGAGGGCGTGGTGTATG TGGAGGTGCGGTACAGTCCGCACCTGCTGGCCAACTCCAAA GTGGAGCCAATCCCCTGGAACCAGGCTGAGTGAGTGAT	45
	ATCACTCACTCAGCCTGGTTCCAGGGGATTGGCTCCACTTTG GAGTTGGCCAGCAGGTGCGGACTGTACCGCACCTCCACATA CACCACGCCCTCTTTGGCCTTCATCTCTACAAACTCAT	46
	GTACAGTCCGCACCTGC	47
	GCAGGTGCGGACTGTAC	48
Adenosine deaminase deficiency LEU106VAL CTG to GTG	TTTGTAGAGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAG GTGCGGTACAGTCCGCACCTGCTGGCCAACTCCAAAGTGGA GCCAATCCCCTGGAACCAGGCTGAGTGAGTGATGGGCC	49
	GGCCCATCACTCACTCAGCCTGGTTCCAGGGGATTGGCTCCA CTTTGGAGTTGGCCAGCAGGTGCGGACTGTACCGCACCTCCA CATACACCACGCCCTCTTTGGCCTTCATCTCTACAAA	50
	GTCCGCACCTGCTGGCC	51
	GGCCAGCAGGTGCGGAC	52

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency LEU107PRO CTG to CCG	TAGAGATGAAGGCCAAAGAGGGCGTGGTGTATGTGGAGGTG CGGTACAGTCCGCACCTGCTGGCCAACTCCAAAGTGGAGCC AATCCCCTGGAACCAGGCTGAGTGAGTGATGGGCCTGGA	53
	TCCAGGCCCATCACTCACTCAGCCTGGTTCCAGGGGATTGGC TCCACTTTGGAGTTGGCCAGCAGGTGCGGACTGTACCGCAC CTCCACATACACCACGCCCTCTTTGGCCTTCATCTCTA	54
	GCACCTGCTGGCCAACT	55
	AGTTGGCCAGCAGGTGC	56
Adenosine deaminase deficiency PRO126GLN CCA to CAA	GCCTTCCTTTTGCCTCAGGCCCATCCCTACTCCTCTCCTCACA CAGAGGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTGG GCCAGGGCCTGCAGGAGGGGGAGCGAGACTTCGGGGT	57
	ACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCCCTGGCCCAC TAGGGCCACCACCTCGTCTGGGGTGAGGTCCCCTCTGTGTG AGGAGAGGAGTAGGGATGGGCCTGAGGCAAAAGGAAGGC	58
	CCTCACCCCAGACGAGG	59
	CCTCGTCTGGGGTGAGG	60
Adenosine deaminase deficiency VAL129MET GTG to ATG	TTTGCCTCAGGCCCATCCCTACTCCTCTCCTCACACAGAGGG GACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCAGGG CCTGCAGGAGGGGGAGCGAGACTTCGGGGTCAAGGCCC	61
	GGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCCC TGGCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCC TCTGTGTGAGGAGAGGAGTAGGGATGGGCCTGAGGCAAA	62
	CAGACGAGGTGGTGGCC	63
	GGCCACCACCTCGTCTG	64
Adenosine deaminase deficiency GLY140GLU GGG to GAG	ACAGAGGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTG GGCCAGGGCCTGCAGGAGGGGGAGCGAGACTTCGGGGTCA AGGCCCGGTCCATCCTGTGCTGCATGCGCCACCAGCCCAG	65
	CTGGGCTGGTGGCGCATGCAGCACAGGATGGACCGGGCCTT GACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCCCTGGCCCA CTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCCTCTGT	66
	GCAGGAGGGGGAGCGAG	67
	CTCGCTCCCCCTCCTGC	68

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ARG142GLN CGA to CAA	GGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCAG GGCCTGCAGGAGGGGGAGC**G**AGACTTCGGGGTCAAGGCCC GGTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGTA	69
	TACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGACCG GGCCTTGACCCCGAAGTCT**C**GCTCCCCCTCCTGCAGGCCCT GGCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCC	70
	GGGGGAGC**G**AGACTTCG	71
	CGAAGTCT**C**GCTCCCCC	72
Adenosine deaminase deficiency ARG142TERM CGA to TGA	GGGGACCTCACCCCAGACGAGGTGGTGGCCCTAGTGGGCCA GGGCCTGCAGGAGGGGGAG**C**GAGACTTCGGGGTCAAGGCC CGGTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGT	73
	ACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGACCGG GCCTTGACCCCGAAGTCTC**G**CTCCCCCTCCTGCAGGCCCTG GCCCACTAGGGCCACCACCTCGTCTGGGGTGAGGTCCCC	74
	AGGGGGAG**C**GAGACTTC	75
	GAAGTCTC**G**CTCCCCCT	76
Adenosine deaminase deficiency ARG149GLN CGG to CAG	TGGTGGCCCTAGTGGGCCAGGGCCTGCAGGAGGGGGAGCG AGACTTCGGGGTCAAGGCCC**G**GTCCATCCTGTGCTGCATGC GCCACCAGCCCAGTGAGTAGGATCACCGCCCTGCCCAGGG	77
	CCCTGGGCAGGGCGGTGATCCTACTCACTGGGCTGGTGGCG CATGCAGCACAGGATGGAC**C**GGGCCTTGACCCCGAAGTCTC GCTCCCCCTCCTGCAGGCCCTGGCCCACTAGGGCCACCA	78
	CAAGGCCC**G**GTCCATCC	79
	GGATGGAC**C**GGGCCTTG	80
Adenosine deaminase deficiency ARG149TRP CGG to TGG	GTGGTGGCCCTAGTGGGCCAGGGCCTGCAGGAGGGGGAGC GAGACTTCGGGGTCAAGGCC**C**GGTCCATCCTGTGCTGCATG CGCCACCAGCCCAGTGAGTAGGATCACCGCCCTGCCCAGG	81
	CCTGGGCAGGGCGGTGATCCTACTCACTGGGCTGGTGGCGC ATGCAGCACAGGATGGACC**G**GGCCTTGACCCCGAAGTCTCG CTCCCCCTCCTGCAGGCCCTGGCCCACTAGGGCCACCAC	82
	TCAAGGCC**C**GGTCCATC	83
	GATGGACC**G**GGCCTTGA	84

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency LEU152MET CTG to ATG	CTAGTGGGCCAGGGCCTGCAGGAGGGGGAGCGAGACTTCG GGGTCAAGGCCCGGTCCATCCTGTGCTGCATGCGCCACCAG CCCAGTGAGTAGGATCACCGCCCTGCCCAGGGCCGCCCGT	85
	ACGGGCGGCCCTGGGCAGGGCGGTGATCCTACTCACTGGGC TGGTGGCGCATGCAGCACAGGATGGACCGGGCCTTGACCCC GAAGTCTCGCTCCCCCTCCTGCAGGCCCTGGCCCACTAG	86
	GGTCCATCCTGTGCTGC	87
	GCAGCACAGGATGGACC	88
Adenosine deaminase deficiency ARG156CYS CGC to TGC	GGCCTGCAGGAGGGGGAGCGAGACTTCGGGGGTCAAGGCCC GGTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGTAG GATCACCGCCCTGCCCAGGGCCGCCCGTCTCACCCTGGCC	89
	GGCCAGGGTGAGACGGGCGGCCCTGGGCAGGGCGGTGATC CTACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGACC GGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGCC	90
	GCTGCATGCGCCACCAG	91
	CTGGTGGCGCATGCAGC	92
Adenosine deaminase deficiency ARG156HIS CGC to CAC	GCCTGCAGGAGGGGGAGCGAGACTTCGGGGGTCAAGGCCCG GTCCATCCTGTGCTGCATGCGCCACCAGCCCAGTGAGTAGGA TCACCGCCCTGCCCAGGGCCGCCCGTCTCACCCTGGCCC	93
	GGGCCAGGGTGAGACGGGCGGCCCTGGGCAGGGCGGTGAT CCTACTCACTGGGCTGGTGGCGCATGCAGCACAGGATGGAC CGGGCCTTGACCCCGAAGTCTCGCTCCCCCTCCTGCAGGC	94
	CTGCATGCGCCACCAGC	95
	GCTGGTGGCGCATGCAG	96
Adenosine deaminase deficiency VAL177MET GTG to ATG	CTGCCCACAGACTGGTCCCCCAAGGTGGTGGAGCTGTGTAA GAAGTACCAGCAGCAGACCGTGGTAGCCATTGACCTGGCTG GAGATGAGACCATCCCAGGAAGCAGCCTCTTGCCTGGAC	97
	GTCCAGGCAAGAGGCTGCTTCCTGGGATGGTCTCATCTCCAG CCAGGTCAATGGCTACCACGGTCTGCTGCTGGTACTTCTTAC ACAGCTCCACCACCTTGGGGGACCAGTCTGTGGGCAG	98
	AGCAGACCGTGGTAGCC	99
	GGCTACCACGGTCTGCT	100

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ALA179ASP GCC to GAC	CAGACTGGTCCCCCAAGGTGGTGGAGCTGTGTAAGAAGTAC CAGCAGCAGACCGTGGTAGCCATTGACCTGGCTGGAGATGA GACCATCCCAGGAAGCAGCCTCTTGCCTGGACATGTCCA	101
	TGGACATGTCCAGGCAAGAGGCTGCTTCCTGGGATGGTCTCA TCTCCAGCCAGGTCAATGGCTACCACGGTCTGCTGCTGGTAC TTCTTACACAGCTCCACCACCTTGGGGGACCAGTCTG	102
	CGTGGTAGCCATTGACC	103
	GGTCAATGGCTACCACG	104
Adenosine deaminase deficiency GLN199PRO CAG to CCG	CCATTGACCTGGCTGGAGATGAGACCATCCCAGGAAGCAGC CTCTTGCCTGGACATGTCCAGGCCTACCAGGTGGGTCCTGTG AGAAGGAATGGAGAGGCTGGCCCTGGGTGAGCTTGTCT	105
	AGACAAGCTCACCCAGGGCCAGCCTCTCCATTCCTTCTCACA GGACCCACCTGGTAGGCCTGGACATGTCCAGGCAAGAGGCT GCTTCCTGGGATGGTCTCATCTCCAGCCAGGTCAATGG	106
	ACATGTCCAGGCCTACC	107
	GGTAGGCCTGGACATGT	108
Adenosine deaminase deficiency ARG211CYS CGT to TGT	GCTAGGGCACCCATGACCTGGCTCTCCCCCTTCCAGGAGGC TGTGAAGAGCGGCATTCACCGTACTGTCCACGCCGGGGAGG TGGGCTCGGCCGAAGTAGTAAAAGAGGTGAGGGCCTGGG	109
	CCCAGGCCCTCACCTCTTTTACTACTTCGGCCGAGCCCACCT CCCCGGCGTGGACAGTACGGTGAATGCCGCTCTTCACAGCC TCCTGGAAGGGGGAGAGCCAGGTCATGGGTGCCCTAGC	110
	GCATTCACCGTACTGTC	111
	GACAGTACGGTGAATGC	112
Adenosine deaminase deficiency ARG211HIS CGT to CAT	CTAGGGCACCCATGACCTGGCTCTCCCCCTTCCAGGAGGCT GTGAAGAGCGGCATTCACCGTACTGTCCACGCCGGGGAGGT GGGCTCGGCCGAAGTAGTAAAAGAGGTGAGGGCCTGGGC	113
	GCCCAGGCCCTCACCTCTTTTACTACTTCGGCCGAGCCCACC TCCCCGGCGTGGACAGTACGGTGAATGCCGCTCTTCACAGC CTCCTGGAAGGGGGAGAGCCAGGTCATGGGTGCCCTAG	114
	CATTCACCGTACTGTCC	115
	GGACAGTACGGTGAATG	116

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ALA215THR GCC to ACC	ATGACCTGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGG CATTCACCGTACTGTCCACGCCGGGGAGGTGGGCTCGGCCG AAGTAGTAAAAGAGGTGAGGGCCTGGGCTGGCCATGGGG	117
	CCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTACTTCGG CCGAGCCCACCTCCCCGGCGTGGACAGTACGGTGAATGCCG CTCTTCACAGCCTCCTGGAAGGGGGAGAGCCAGGTCAT	118
	CTGTCCACGCCGGGGAG	119
	CTCCCCGGCGTGGACAG	120
Adenosine deaminase deficiency GLY216ARG GGG to AGG	ACCTGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGGCATT CACCGTACTGTCCACGCCGGGGAGGTGGGCTCGGCCGAAGT AGTAAAAGAGGTGAGGGCCTGGGCTGGCCATGGGGTCC	121
	GGACCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTACTT CGGCCGAGCCCACCTCCCCGGCGTGGACAGTACGGTGAATG CCGCTCTTCACAGCCTCCTGGAAGGGGGAGAGCCAGGT	122
	TCCACGCCGGGGAGGTG	123
	CACCTCCCCGGCGTGGA	124
Adenosine deaminase deficiency GLU217LYS GAG to AAG	TGGCTCTCCCCCTTCCAGGAGGCTGTGAAGAGCGGCATTCAC CGTACTGTCCACGCCGGGGAGGTGGGCTCGGCCGAAGTAGT AAAAGAGGTGAGGGCCTGGGCTGGCCATGGGGTCCCTC	125
	GAGGGACCCCATGGCCAGCCCAGGCCCTCACCTCTTTTACTA CTTCGGCCGAGCCCACCTCCCCGGCGTGGACAGTACGGTGA ATGCCGCTCTTCACAGCCTCCTGGAAGGGGGAGAGCCA	126
	ACGCCGGGGAGGTGGGC	127
	GCCCACCTCCCCGGCGT	128
Adenosine deaminase deficiency THR233ILE ACA to ATA	CTGCCTCCTCCCATACTTGGCTCTATTCTGCTTCTCTACAGGC TGTGGACATACTCAAGACAGAGCGGCTGGGACACGGCTACC ACACCCTGGAAGACCAGGCCCTTTATAACAGGCTGCG	129
	CGCAGCCTGTTATAAAGGGCCTGGTCTTCCAGGGTGTGGTAG CCGTGTCCCAGCCGCTCTGTCTTGAGTATGTCCACAGCCTGT AGAGAAGCAGAATAGAGCCAAGTATGGGAGGAGGCAG	130
	ACTCAAGACAGAGCGGC	131
	GCCGCTCTGTCTTGAGT	132

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency ARG253PRO CGG to CCG	CAGAGCGGCTGGGACACGGCTACCACACCCTGGAAGACCAG GCCCTTTATAACAGGCTGC**G**GCAGGAAAACATGCACTTCGAG GTAAGCGGGCCAGGGAGTGGGGAGGAACCATCCCCGGC	133
	GCCGGGGATGGTTCCTCCCCACTCCCTGGCCCGCTTACCTC GAAGTGCATGTTTTCCTGC**C**GCAGCCTGTTATAAAGGGCCTG GTCTTCCAGGGTGTGGTAGCCGTGTCCCAGCCGCTCTG	134
	CAGGCTGC**G**GCAGGAAA	135
	TTTCCTGC**C**GCAGCCTG	136
Adenosine deaminase deficiency GLN254TERM CAG to TAG	GAGCGGCTGGGACACGGCTACCACACCCTGGAAGACCAGGC CCTTTATAACAGGCTGCGG**C**AGGAAAACATGCACTTCGAGGT AAGCGGGCCAGGGAGTGGGGAGGAACCATCCCCGGCTG	137
	CAGCCGGGGATGGTTCCTCCCCACTCCCTGGCCCGCTTACCT CGAAGTGCATGTTTTCCT**G**CCGCAGCCTGTTATAAAGGGCCT GGTCTTCCAGGGTGTGGTAGCCGTGTCCCAGCCGCTC	138
	GGCTGCGG**C**AGGAAAAC	139
	GTTTTCCT**G**CCGCAGCC	140
Adenosine deaminase deficiency PRO274LEU CCG to CTG	CCACACACCTGCTCTTCCAGATCTGCCCCTGGTCCAGCTACC TCACTGGTGCCTGGAAGC**C**GGACACGGAGCATGCAGTCATT CGGTGAGCTCTGTTCCCCTGGGCCTGTTCAATTTTGTT	141
	AACAAAATTGAACAGGCCCAGGGGAACAGAGCTCACCGAATG ACTGCATGCTCCGTGTCC**G**GCTTCCAGGCACCAGTGAGGTAG CTGGACCAGGGGCAGATCTGGAAGAGCAGGTGTGTGG	142
	CTGGAAGC**C**GGACACGG	143
	CCGTGTCC**G**GCTTCCAG	144
Adenosine deaminase deficiency SER291LEU TCG to TTG	GGAGGCTGATTCTCTCCTCCTCCCTCTTCTGCAGGCTCAAAA ATGACCAGGCTAACTACT**C**GCTCAACACAGATGACCCGCTCA TCTTCAAGTCCACCCTGGACACTGATTACCAGATGAC	145
	GTCATCTGGTAATCAGTGTCCAGGGTGGACTTGAAGATGAGC GGGTCATCTGTGTTGAGC**G**AGTAGTTAGCCTGGTCATTTTTG AGCCTGCAGAAGAGGGAGGAGGAGAGAATCAGCCTCC	146
	TAACTACT**C**GCTCAACA	147
	TGTTGAGC**G**AGTAGTTA	148

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenosine deaminase deficiency PRO297GLN CCG to CAG	CCTCCCTCTTCTGCAGGCTCAAAAATGACCAGGCTAACTACTC GCTCAACACAGATGACCCGCTCATCTTCAAGTCCACCCTGGA CACTGATTACCAGATGACCAAACGGGACATGGGCTT	149
	AAGCCCATGTCCCGTTTGGTCATCTGGTAATCAGTGTCCAGG GTGGACTTGAAGATGAGCGGGTCATCTGTGTTGAGCGAGTAG TTAGCCTGGTCATTTTTGAGCCTGCAGAAGAGGGAGG	150
	AGATGACCCGCTCATCT	151
	AGATGAGCGGGTCATCT	152
Adenosine deaminase deficiency LEU304ARG CTG to CGG	AAAATGACCAGGCTAACTACTCGCTCAACACAGATGACCCGC TCATCTTCAAGTCCACCCTGGACACTGATTACCAGATGACCAA ACGGGACATGGGCTTTACTGAAGAGGAGTTTAAAAG	153
	CTTTTAAACTCCTCTTCAGTAAAGCCCATGTCCCGTTTGGTCA TCTGGTAATCAGTGTCCAGGGTGGACTTGAAGATGAGCGGGT CATCTGTGTTGAGCGAGTAGTTAGCCTGGTCATTTT	154
	GTCCACCCTGGACACTG	155
	CAGTGTCCAGGGTGGAC	156
Adenosine deaminase deficiency ALA329VAL C-to-T at base 1081	GCCTTCTTTGTTCTCTGGTTCCATGTTGTCTGCCATTCTGGCC TTTCCAGAACATCAATGCGGCCAAATCTAGTTTCCTCCCAGAA GATGAAAAGAGGGAGCTTCTCGACCTGCTCTATAA	157
	TTATAGAGCAGGTCGAGAAGCTCCCTCTTTTCATCTTCTGGGA GGAAACTAGATTTGGCCGCATTGATGTTCTGGAAAGGCCAGA ATGGCAGACAACATGGAACCAGAGAACAAAGAAGGC	158
	CATCAATGCGGCCAAAT	159
	ATTTGGCCGCATTGATG	160

EXAMPLE 5

P53 Mutations

The p53 gene codes for a protein that acts as a transcription factor and serves as a key regulator of the cell cycle. Mutation in this gene is probably the most significant genetic change characterizing the transformation of cells from normalcy to malignancy.

Inactivation of p53 by mutation disrupts the cell cycle which, in turn, sets the stage for tumor formation.

Mutations in the p53 gene are among the most commonly diagnosed genetic disorders, occuring in as many as 50% of cancer patients. For some types of cancer, most notably of the breast, lung and colon, p53 mutations are the predominant genetic alternations found thus far. These mutations are associated with genomic instability and thus an increased susceptibility to cancer. Some p53 lesions result in malignancies that are resistant to the most widely used therapeutic regimens and therefore demand more aggressive treatment.

That p53 is associated with different malignant tumors is illustrated in the Li-Fraumeni autosomal dominant hereditary disorder characterized by familial multiple tumors due to mutation in the p53 gene. Affected individuals can develop one or more tumors, including: brain (12%); soft-tissue sarcoma (12%); breast cancer (25%); adrenal tumors (1%); bone cancer (osteosarcoma) (6%); cancer of the lung, prostate, pancreas, and colon as well as lymphoma and melanoma can also occur.

Certain of the most frequently mutated codons are codons 175, 248 and 273, however a variety of oligonucleotides are described below in the atttached table.

Table 11

p53 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In 2 families with Li-Fraumeni syndrome, there was a C-to-T mutation at the first nucleotide of codon 248 which changed arginine to tryptophan.	GACTGTACCACCATCCACTACAACTACATGTGTAACAGTTCCT GCATGGGCGGCATGAACCGGAGGCCCATCCTCACCATCATC ACACTGGAAGACTCCAGGTCAGGAGCCACTTGCCACC	161
	GGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTGTGATGA TGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAGGAA CTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGTC	162
	GCATGAACCGGAGGCCC	163
	GGGCCTCCGGTTCATGC	164
In a family with the Li-Fraumeni syndrome, a G-to-A mutation at the first nucleotide of codon 258 resulting in the substitution of lysine for glutamic acid.	TGTAACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCCAT CCTCACCATCATCACACTGGAAGACTCCAGGTCAGGAGCCAC TTGCCACCCTGCACACTGGCCTGCTGTGCCCCAGCCTC	165
	GAGGCTGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGT GGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGAT GGGCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACA	166
	TCACACTGGAAGACTCC	167
	GGAGTCTTCCAGTGTGA	168
In a family with the Li-Fraumeni syndrome, a G-to-T mutation at the first nucleotide of codon 245 resulting in the substitution of cysteine for glycine.	GTTGGCTCTGACTGTACCACCATCCACTACAACTACATGTGTA ACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCCATCCTC ACCATCATCACACTGGAAGACTCCAGGTCAGGAGCCA	169
A gly245-to-ser, GGC-to-AGC, mutation was found in a patient in whom osteosarcoma was diagnosed at the age of 18 years.	TGGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGGA TGGGCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACACA TGTAGTTGTAGTGGATGGTGGTACAGTCAGAGCCAAC	170
	GCATGGGCGGCATGAAC	171
	GTTCATGCCGCCCATGC	172

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In a family with the Li-Fraumeni syndrome, a germline mutation at codon 252: a T-to-C change at the second position resulted in substitution of proline for leucine.	TCCACTACAACTACATGTGTAACAGTTCCTGCATGGGCGGCA TGAACCGGAGGCCCATCCTCACCATCATCACACTGGAAGACT CCAGGTCAGGAGCCACTTGCCACCCTGCACACTGGCC	173
	GGCCAGTGTGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTC TTCCAGTGTGATGATGGTGAGGATGGGCCTCCGGTTCATGCC GCCCATGCAGGAACTGTTACACATGTAGTTGTAGTGGA	174
	GCCCATCCTCACCATCA	175
	TGATGGTGAGGATGGGC	176
Researchers analyzed for mutations in p53 hepatocellular carcinomas from patients in Qidong, an area of high incidence in China, in which both hepatitis B virus and aflatoxin B1 are risk factors. Eight of 16 tumors had a point mutation at the third base position of codon 249. The G-to-T mutation at codon 249 led to a change from arginine to serine (AGG to AGT).	TACCACCATCCACTACAACTACATGTGTAACAGTTCCTGCATG GGCGGCATGAACCGGAGGCCCATCCTCACCATCATCACACT GGAAGACTCCAGGTCAGGAGCCACTTGCCACCCTGCA	177
	TGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTGT GATGATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGC AGGAACTGTTACACATGTAGTTGTAGTGGATGGTGGTA	178
	AACCGGAGGCCCATCCT	179
	AGGATGGGCCTCCGGTT	180
In cases of hepatocellular carcinoma in southern Africa, a G-to-T substitution in codon 157 resulting in a change from valine to phenylalanine.	CTGGCCAAGACCTGCCCTGTGCAGCTGTGGGTTGATTCCACA CCCCCGCCCGGCACCCGCGTCCGCGCCATGGCCATCTACAA GCAGTCACAGCACATGACGGAGGTTGTGAGGCGCTGCC	181
	GGCAGCGCCTCACAACCTCCGTCATGTGCTGTGACTGCTTGT AGATGGCCATGGCGCGGACGCGGGTGCCGGGCGGGGGTGT GGAATCAACCCACAGCTGCACAGGGCAGGTCTTGGCCAG	182
	GCACCCGCGTCCGCGCC	183
	GGCGCGGACGCGGGTGC	184

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In a family with Li-Fraumeni in which noncancerous skin fibroblasts from affected individuals showed an unusual radiation-resistant phenotype, a point mutation in codon 245 of the P53 gene. A change from GGC to GAC predicted substitution of aspartic acid for glycine.	TTGGCTCTGACTGTACCACCATCCACTACAACTACATGTGTAA CAGTTCCTGCATGGGCGGCATGAACCGGAGGCCCATCCTCA CCATCATCACACTGGAAGACTCCAGGTCAGGAGCCAC	185
	GTGGCTCCTGACCTGGAGTCTTCCAGTGTGATGATGGTGAGG ATGGGCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACAC ATGTAGTTGTAGTGGATGGTGGTACAGTCAGAGCCAA	186
	CATGGGCGGCATGAACC	187
	GGTTCATGCCGCCCATG	188
In 2 of 8 families with Li-Fraumeni syndrome, a mutation in codon 248: a CGG-to-CAG change resulting in substitution of glutamine for arginine.	ACTGTACCACCATCCACTACAACTACATGTGTAACAGTTCCTG CATGGGCGGCATGAACCGGAGGCCCATCCTCACCATCATCA CACTGGAAGACTCCAGGTCAGGAGCCACTTGCCACCC	189
	GGGTGGCAAGTGGCTCCTGACCTGGAGTCTTCCAGTGTGAT GATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAGG AACTGTTACACATGTAGTTGTAGTGGATGGTGGTACAGT	190
	CATGAACCGGAGGCCCA	191
	TGGGCCTCCGGTTCATG	192
In 9 members of an extended family with Li-Fraumeni syndrome, a germline mutation at codon 133 (ATG-to-ACG), resulted in the substitution of threonine for methionine (M133T), and completely cosegregated with the cancer syndrome.	CCCTGACTTTCAACTCTGTCTCCTTCCTCTTCCTACAGTACTC CCCTGCCCTCAACAAGATGTTTTGCCAACTGGCCAAGACCTG CCCTGTGCAGCTGTGGGTTGATTCCACACCCCCGCC	193
	GGCGGGGGTGTGGAATCAACCCACAGCTGCACAGGGCAGGT CTTGGCCAGTTGGCAAAACATCTTGTTGAGGGCAGGGGAGTA CTGTAGGAAGAGGAAGGAGACAGAGTTGAAAGTCAGGG	194
	CAACAAGATGTTTTGCC	195
	GGCAAAACATCTTGTTG	196

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
In 1 pedigree consistent with the Li-Fraumeni syndrome, a germline G-to-T transversion at codon 272 (valine to leucine) was found.	TCTTGCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGG GACGGAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGA GAGACCGGCGCACAGAGGAAGAGAATCTCCGCAAGA	197
	TCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTCCCAG GACAGGCACAAACACGCACCTCAAAGCTGTTCCGTCCCAGTA GATTACCACTACTCAGGATAGGAAAAGAGAAGCAAGA	198
	GCTTTGAGGTGCGTGTT	199
	AACACGCACCTCAAAGC	200
A ser241-to-phe mutation due to a TCC-to-TTC change was found in a patient with hepatoblastoma and multiple foci of osteosarcoma.	TTATCTCCTAGGTTGGCTCTGACTGTACCACCATCCACTACAA CTACATGTGTAACAGTTCCTGCATGGGCGGCATGAACCGGAG GCCCATCCTCACCATCATCACACTGGAAGACTCCAG	201
	CTGGAGTCTTCCAGTGTGATGATGGTGAGGATGGGCCTCCG GTTCATGCCGCCCATGCAGGAACTGTTACACATGTAGTTGTA GTGGATGGTGGTACAGTCAGAGCCAACCTAGGAGATAA	202
	TAACAGTTCCTGCATGG	203
	CCATGCAGGAACTGTTA	204
An AAG-to-TAG change of codon 120, resulting in conversion from lysine to a stop codon, was found in a patient with osteosarcoma and adenocarcinoma of the lung at age 18 and brain tumor (glioma) at the age of 27.	CAGAAAACCTACCAGGGCAGCTACGGTTTCCGTCTGGGCTTC TTGCATTCTGGGACAGCCAAGTCTGTGACTTGCACGGTCAGT TGCCCTGAGGGGCTGGCTTCCATGAGACTTCAATGCC	205
	GGCATTGAAGTCTCATGGAAGCCAGCCCCTCAGGGCAACTG ACCGTGCAAGTCACAGACTTGGCTGTCCCAGAATGCAAGAAG CCCAGACGGAAACCGTAGCTGCCCTGGTAGGTTTTCTG	206
	GGACAGCCAAGTCTGTG	207
	CACAGACTTGGCTGTCC	208
A CGG-to-TGG change at codon 282, resulting in the substitution of tryptophan for arginine, was found in a patient who developed osteosarcoma at the age of 10 years.	GGTAATCTACTGGGACGGAACAGCTTTGAGGTGCGTGTTTGT GCCTGTCCTGGGAGAGACCGGCGCACAGAGGAAGAGAATCT CCGCAAGAAAGGGGAGCCTCACCACGAGCTGCCCCCAG	209
	CTGGGGGCAGCTCGTGGTGAGGCTCCCCTTTCTTGCGGAGA TTCTCTTCCTCTGTGCGCCGGTCTCTCCCAGGACAGGCACAA ACACGCACCTCAAAGCTGTTCCGTCCCAGTAGATTACC	210
	GGAGAGACCGGCGCACA	211

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGTGCGCCGGTCTCTCC	212
In 5 of 6 anaplastic carcinomas of the thyroid and in an anaplastic carcinoma thyroid cell line ARO, a CGT-to-CAT mutation converted arginine-273 to histidine.	GCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGACG GAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGAGA CCGGCGCACAGAGGAAGAGAATCTCCGCAAGAAAGG	213
	CCTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTCC CAGGACAGGCACAAACACGCACCTCAAAGCTGTTCCGTCCCA GTAGATTACCACTACTCAGGATAGGAAAAGAGAAGC	214
	TGAGGTGCGTGTTTGTG	215
	CACAAACACGCACCTCA	216
A germline GGA-to-GTA mutation resulting in a change of glycine-325 to valine was found in a patient who had non-Hodgkin lymphoma diagnosed at age 17 and colon carcinoma at age 26.	TCCTAGCACTGCCCAACAACACCAGCTCCTCTCCCCAGCCAA AGAAGAAACCACTGGATGGAGAATATTTCACCCTTCAGGTAC TAAGTCTTGGGACCTCTTATCAAGTGGAAAGTTTCCA	217
	TGGAAACTTTCCACTTGATAAGAGGTCCCAAGACTTAGTACCT GAAGGGTGAAATATTCTCCATCCAGTGGTTTCTTCTTTGGCTG GGGAGAGGAGCTGGTGTTGTTGGGCAGTGCTAGGA	218
	ACTGGATGGAGAATATT	219
	AATATTCTCCATCCAGT	220
CGC-CCC Arg-72 to Pro association with Lung cancer	AATGGTTCACTGAAGACCCAGGTCCAGATGAAGCTCCCAGAA TGCCAGAGGCTGCTCCCCGCGTGGCCCCTGCACCAGCAGCT CCTACACCGGCGGCCCCTGCACCAGCCCCCTCCTGGCC	221
	GGCCAGGAGGGGGCTGGTGCAGGGGCCGCCGGTGTAGGAG CTGCTGGTGCAGGGGCCACGCGGGGAGCAGCCTCTGGCATT CTGGGAGCTTCATCTGGACCTGGGTCTTCAGTGAACCATT	222
	TGCTCCCCGCGTGGCCC	223
	GGGCCACGCGGGGAGCA	224
CCG-CTG Pro-82 to Leu Breast cancer	AAGCTCCCAGAATGCCAGAGGCTGCTCCCCGCGTGGCCCCT GCACCAGCAGCTCCTACACCGGCGGCCCCTGCACCAGCCCC CTCCTGGCCCCTGTCATCTTCTGTCCCTTCCCAGAAAAC	225
	GTTTTCTGGGAAGGGACAGAAGATGACAGGGGCCAGGAGGG GGCTGGTGCAGGGGCCGCCGGTGTAGGAGCTGCTGGTGCA GGGGCCACGCGGGGAGCAGCCTCTGGCATTCTGGGAGCTT	226
	TCCTACACCGGCGGCCC	227
	GGGCCGCCGGTGTAGGA	228

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
cCAA-TAA Gln-136 to Term Li-Fraumeni syndrome	TTCAACTCTGTCTCCTTCCTCTTCCTACAGTACTCCCCTGCCC TCAACAAGATGTTTTGC**C**AACTGGCCAAGACCTGCCCTGTGC AGCTGTGGGTTGATTC**C**ACACCCCCGCCCGGCACCC	229
	GGGTGCCGGGCGGGGGTGTGGAATCAACCCACAGCTGCACA GGGCAGGTCTTGGCCAGTT**G**GCAAAACATCTTGTTGAGGGCA GGGGAGTACTGTAGGAAGAGGAAGGAGACAGAGTTGAA	230
	TGTTTTGC**C**AACTGGCC	231
	GGCCAGTT**G**GCAAAACA	232
TGC-TAC Cys-141 to Tyr Li-Fraumeni syndrome	TCCTCTTCCTACAGTACTCCCCTGCCCTCAACAAGATGTTTTG CCAACTGGCCAAGACCT**G**CCCTGTGCAGCTGTGGGTTGATTC **C**ACACCCCCGCCCGGCACCCGCGTCCGCGCCATGGC	233
	GCCATGGCGCGGACGCGGGTGCCGGGCGGGGGTGTGGAAT CAACCCACAGCTGCACAGGG**C**AGGTCTTGGCCAGTTGGCAA AACATCTTGTTGAGGGCAGGGGAGTACTGTAGGAAGAGGA	234
	CAAGACCT**G**CCCTGTGC	235
	GCACAGGG**C**AGGTCTTG	236
aCCC-TCC Pro-151 to Ser Li-Fraumeni syndrome	AACAAGATGTTTTGCCAACTGGCCAAGACCTGCCCTGTGCAG CTGTGGGTTGATTC**C**ACA**C**CCCCGCCCGGCACCCGCGTCCG CGCCATGGCCATCTACAAGCAGTCACAGCACATGACGG	237
	CCGTCATGTGCTGTGACTGCTTGTAGATGGCCATGGCGCGGA CGCGGGTGCCGGGCGGGGG**G**TGTGGAATCAACCCACAGCTG CACAGGGCAGGTCTTGGCCAGTTGGCAAAACATCTTGTT	238
	ATTCCACA**C**CCCCGCCC	239
	GGGCGGGGG**G**TGTGGAAT	240
CCG-CTG Pro-152 to Leu Adrenocortical carcinoma	AGATGTTTTGCCAACTGGCCAAGACCTGCCCTGTGCAGCTGT GGGTTGATTC**C**ACACCCC**C**GCCCGGCACCCGCGTCCGCGCC ATGGCCATCTACAAGCAGTCACAGCACATGACGGAGGT	241
	ACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCCATGGCG CGGACGCGGGTGCCGGGC**G**GGGGGTGTGGAATCAACCCACA GCTGCACAGGGCAGGTCTTGGCCAGTTGGCAAAACATCT	242
	CACACCCC**C**GCCCGGCA	243
	TGCCGGGC**G**GGGGTGTG	244

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
GGC-GTC Gly-154 to Val Glioblastoma	TTTGCCAACTGGCCAAGACCTGCCCTGTGCAGCTGTGGGTTG ATTCCACACCCCCGCCCGGCACCCGCGTCCGCGCCATGGCC ATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAG	245
	CTCACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCC ATGGCGCGGACGCGGGTGCCGGGCGGGGGTGTGGAATCAA CCCACAGCTGCACAGGGCAGGTCTTGGCCAGTTGGCAAA	246
	CCCGCCCGGCACCCGCG	247
	CGCGGGTGCCGGGCGGG	248
CGC-CAC Arg-175 to His Li-Fraumeni syndrome	GCCGCGTCCGCGCCATGGCCATCTACAAGCAGTCACAGCAC ATGACGGAGGTTGTGAGGCGCTGCCCCCACCATGAGCGCTG CTCAGATAGCGATGGTGAGCAGCTGGGGCTGGAGAGACG	249
	CGTCTCTCCAGCCCCAGCTGCTCACCATCGCTATCTGAGCAG CGCTCATGGTGGGGGCAGCGCCTCACAACCTCCGTCATGTG CTGTGACTGCTTGTAGATGGCCATGGCGCGGACGCGGG	250
	TGTGAGGCGCTGCCCCC	251
	GGGGGCAGCGCCTCACA	252
tGAG-AAG Glu-180 to Lys Li-Fraumeni syndrome	ATGGCCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTG AGGCGCTGCCCCCACCATGAGCGCTGCTCAGATAGCGATGG TGAGCAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGC	253
	GCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTCACCAT CGCTATCTGAGCAGCGCTCATGGTGGGGGCAGCGCCTCACA ACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGCCAT	254
	CCCACCATGAGCGCTGC	255
	GCAGCGCTCATGGTGGG	256
gCGC-TGC Arg-181 to Cys Breast cancer	GCCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAG GCGCTGCCCCCACCATGAGCGCTGCTCAGATAGCGATGGTG AGCAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGCCCA	257
	TGGGCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTCAC CATCGCTATCTGAGCAGCGCTCATGGTGGGGGCAGCGCCTC ACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGGC	258
	ACCATGAGCGCTGCTCA	259
	TGAGCAGCGCTCATGGT	260

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
CGC-CAC Arg-81 to His Breast cancer	CCATCTACAAGCAGTCACAGCACATGACGGAGGTTGTGAGGC GCTGCCCCCACCATGAGC**G**CTGCTCAGATAGCGATGGTGAG CAGCTGGGGCTGGAGAGACGACAGGGCTGGTTGCCCAG	261
	CTGGGCAACCAGCCCTGTCGTCTCTCCAGCCCCAGCTGCTCA CCATCGCTATCTGAGCAG**C**GCTCATGGTGGGGGCAGCGCCT CACAACCTCCGTCATGTGCTGTGACTGCTTGTAGATGG	262
	CCATGAGC**G**CTGCTCAG	263
	CTGAGCAG**C**GCTCATGG	264
CAT-CGT His-193 to Arg Li-Fraumeni syndrome	CCAGGGTCCCCAGGCCTCTGATTCCTCACTGATTGCTCTTAG GTCTGGCCCCTCCTCAGC**A**TCTTATCCGAGTGGAAGGAAATT TGCGTGTGGAGTATTTGGATGACAGAAACACTTTTCG	265
	CGAAAAGTGTTTCTGTCATCCAAATACTCCACACGCAAATTTC CTTCCACTCGGATAAGA**T**GCTGAGGAGGGGCCAGACCTAAG AGCAATCAGTGAGGAATCAGAGGCCTGGGGACCCTGG	266
	TCCTCAGC**A**TCTTATCC	267
	GGATAAGA**T**GCTGAGGA	268
cCGA-TGA Arg-196 to Term Adrenocortical carcinoma	CCCAGGCCTCTGATTCCTCACTGATTGCTCTTAGGTCTGGCC CCTCCTCAGCATCTTATC**C**GAGTGGAAGGAAATTTGCGTGTG GAGTATTTGGATGACAGAAACACTTTTCGACATAGTG	269
	CACTATGTCGAAAAGTGTTTCTGTCATCCAAATACTCCACACG CAAATTTCCTTCCACTC**G**GATAAGATGCTGAGGAGGGGCCAG ACCTAAGAGCAATCAGTGAGGAATCAGAGGCCTGGG	270
	ATCTTATC**C**GAGTGGAA	271
	TTCCACTC**G**GATAAGAT	272
cAGA-TGA Arg-209 to Term Li-Fraumeni syndrome	GCCCCTCCTCAGCATCTTATCCGAGTGGAAGGAAATTTGCGT GTGGAGTATTTGGATGAC**A**GAAACACTTTTCGACATAGTGTG GTGGTGCCCTATGAGCCGCCTGAGGTCTGGTTTGCAA	273
	TTGCAAACCAGACCTCAGGCGGCTCATAGGGCACCACCACAC TATGTCGAAAAGTGTTTC**T**GTCATCCAAATACTCCACACGCAA ATTTCCTTCCACTCGGATAAGATGCTGAGGAGGGGC	274
	TGGATGAC**A**GAAACACT	275
	AGTGTTTC**T**GTCATCCA	276

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
tCGA-TGA Arg-213 to Term Li-Fraumeni syndrome	CATCTTATCCGAGTGGAAGGAAATTTGCGTGTGGAGTATTTG GATGACAGAAACACTTTTCGACATAGTGTGGTGGTGCCCTAT GAGCCGCCTGAGGTCTGGTTTGCAACTGGGGTCTCTG	277
	CAGAGACCCCAGTTGCAAACCAGACCTCAGGCGGCTCATAG GGCACCACCACACTATGTCGAAAAGTGTTTCTGTCATCCAAAT ACTCCACACGCAAATTTCCTTCCACTCGGATAAGATG	278
	ACACTTTTCGACATAGT	279
	ACTATGTCGAAAAGTGT	280
gCCC-TCC Pro-219 to Ser Adrenocortical carcinoma	GGAAATTTGCGTGTGGAGTATTTGGATGACAGAAACACTTTTC GACATAGTGTGGTGGTGCCCTATGAGCCGCCTGAGGTCTGG TTTGCAACTGGGGTCTCTGGGAGGAGGGGTTAAGGGT	281
	ACCCTTAACCCCTCCTCCCAGAGACCCCAGTTGCAAACCAGA CCTCAGGCGGCTCATAGGGCACCACCACACTATGTCGAAAAG TGTTTCTGTCATCCAAATACTCCACACGCAAATTTCC	282
	TGGTGGTGCCCTATGAG	283
	CTCATAGGGCACCACCA	284
TAT-TGT Tyr-220 to Cys Li-Fraumeni syndrome	ATTTGCGTGTGGAGTATTTGGATGACAGAAACACTTTTCGACA TAGTGTGGTGGTGCCCTATGAGCCGCCTGAGGTCTGGTTTGC AACTGGGGTCTCTGGGAGGAGGGGTTAAGGGTGGTT	285
	AACCACCCTTAACCCCTCCTCCCAGAGACCCCAGTTGCAAAC CAGACCTCAGGCGGCTCATAGGGCACCACCACACTATGTCGA AAAGTGTTTCTGTCATCCAAATACTCCACACGCAAAT	286
	GGTGCCCTATGAGCCGC	287
	GCGGCTCATAGGGCACC	288
cTCT-ACT Ser-227 to Thr Rhabdomyosarcoma	CACAGGTCTCCCCAAGGCGCACTGGCCTCATCTTGGGCCTGT GTTATCTCCTAGGTTGGCTCTGACTGTACCACCATCCACTACA ACTACATGTGTAACAGTTCCTGCATGGGCGGCATGA	289
	TCATGCCGCCCATGCAGGAACTGTTACACATGTAGTTGTAGT GGATGGTGGTACAGTCAGAGCCAACCTAGGAGATAACACAG GCCCAAGATGAGGCCAGTGCGCCTTGGGGAGACCTGTG	290
	AGGTTGGCTCTGACTGT	291
	ACAGTCAGAGCCAACCT	292

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
cCAC-AAC His-233 to Asn Glioma	GCACTGGCCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGC TCTGACTGTACCACCATC**C**ACTACAACTACATGTGTAACAGTT CCTGCATGGGCGGCATGAACCGGAGGCCCATCCTCA	293
	TGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAGGAACTG TTACACATGTAGTTGTAGT**G**GATGGTGGTACAGTCAGAGCCA ACCTAGGAGATAACACAGGCCCAAGATGAGGCCAGTGC	294
	CCACCATC**C**ACTACAAC	295
	GTTGTAGT**G**GATGGTGG	296
cAAC-GAC Asn-235 to Asp Adrenocortical carcinoma	GCCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGCTCTGAC TGTACCACCATCCACTAC**A**ACTACATGTGTAACAGTTCCTGCA TGGGCGGCATGAACCGGAGGCCCATCCTCACCATCA	297
	TGATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCAG GAACTGTTACACATGTAGT**T**GTAGTGGATGGTGGTACAGTCA GAGCCAACCTAGGAGATAACACAGGCCCAAGATGAGGC	298
	TCCACTAC**A**ACTACATG	299
	CATGTAGT**T**GTAGTGGA	300
AAC-AGC Asn-235 to Ser Rhabdomyosarcoma	CCTCATCTTGGGCCTGTGTTATCTCCTAGGTTGGCTCTGACT GTACCACCATCCACTACA**A**CTACATGTGTAACAGTTCCTGCAT GGGCGGCATGAACCGGAGGCCCATCCTCACCATCAT	301
	ATGATGGTGAGGATGGGCCTCCGGTTCATGCCGCCCATGCA GGAACTGTTACACATGTAG**T**TGTAGTGGATGGTGGTACAGTC AGAGCCAACCTAGGAGATAACACAGGCCCAAGATGAGG	302
	CCACTACA**A**CTACATGT	303
	ACATGTAG**T**TGTAGTGG	304
ATCc-ATG Ile-251 to Met Glioma	CATCCACTACAACTACATGTGTAACAGTTCCTGCATGGGCGG CATGAACCGGAGGCCCAT**C**CTCACCATCATCACACTGGAAGA CTCCAGGTCAGGAGCCACTTGCCACCCTGCACACTGG	305
	CCAGTGTGCAGGGTGGCAAGTGGCTCCTGACCTGGAGTCTT CCAGTGTGATGATGGTGAG**G**ATGGGCCTCCGGTTCATGCCG CCCATGCAGGAACTGTTACACATGTAGTTGTAGTGGATG	306
	AGGCCCAT**C**CTCACCAT	307
	ATGGTGAG**G**ATGGGCCT	308

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
ACA-ATA Thr-256 to Ile Glioblastoma	ACATGTGTAACAGTTCCTGCATGGGCGGCATGAACCGGAGG CCCATCCTCACCATCATCA**C**ACTGGAAGACTCCAGGTCAGGA GCCACTTGCCACCCTGCACACTGGCCTGCTGTGCCCCA	309
	TGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGTGGCTCC TGACCTGGAGTCTTCCAGT**G**TGATGATGGTGAGGATGGGCCT CCGGTTCATGCCGCCCATGCAGGAACTGTTACACATGT	310
	CATCATCA**C**ACTGGAAG	311
	C**T**TCCAGT**G**TGATGATG	312
CTG-CAG Leu-257 to Gln Li-Fraumeni syndrome	TGT**G**TAACAGTTCCTGCATGGGCGGCATGAACCGGAGGCCC ATCCTCACCATCATCACAC**T**GGAAGACTCCAGGTCAGGAGCC ACTTGCCACCCTGCACACTGGCCTGCTGTGCCCCAGCC	313
	GGCTGGGGCACAGCAGGCCAGTGTGCAGGGTGGCAAGTGG CTCCTGACCTGGAGTCTTCC**A**GTGTGATGATGGTGAGGATGG GCCTCCGGTTCATGCCGCCCATGCAGGAACTGTTACACA	314
	CATCACAC**T**GGAAGACT	315
	AGTCTTCC**A**GTGTGATG	316
CTG-CCG Leu-265 to Pro Li-Fraumeni syndrome	GACCTGATTTCCTTACTGCCTCTTGCTTCTCTTTTCCTATCCTG AGTAGTGGTAATCTAC**T**GGGACGGAACAGCTTTGAGGTGCGT GTTTGTGCCTGTCCTGGGAGAGACCGGCGCACAGA	317
	TCTGTGCGCCGGTCTCTCCCAGGACAGGCACAAACACGCAC CTCAAAGCTGTTCCGTCCC**A**GTAGATTACCACTACTCAGGATA GGAAAAGAGAAGCAAGAGGCAGTAAGGAAATCAGGTC	318
	TAATCTAC**T**GGGACGGA	319
	TCCGTCCC**A**GTAGATTA	320
gCGT-TGT Arg-273 to Cys Li-Fraumeni syndrome	TGCTTCTCTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGAC GGAACAGCTTTGAGGTG**C**GTGTTTGTGCCTGTCCTGGGAGA GACCGGCGCACAGAGGAAGAGAATCTCCGCAAGAAAG	321
	CTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGGTCTCTCCC AGGACAGGCACAAACAC**G**CACCTCAAAGCTGTTCCGTCCCAG TAGATTACCACTACTCAGGATAGGAAAAGAGAAGCA	322
	TTGAGGTG**C**GTGTTTGT	323
	ACAAACAC**G**CACCTCAA	324

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
TGT-TAT Cys-275 to Tyr Li-Fraumeni syndrome	CTTTTCCTATCCTGAGTAGTGGTAATCTACTGGGACGGAACA GCTTTGAGGTGCGTGTTT**G**TGCCTGTCCTGGGAGAGACCGG CGCACAGAGGAAGAGAATCTCCGCAAGAAAGGGGAGCC	325
	GGCTCCCCTTTCTTGCGGAGATTCTCTTCCTCTGTGCGCCGG TCTCTCCCAGGACAGGCA**C**AAACACGCACCTCAAAGCTGTTC CGTCCCAGTAGATTACCACTACTCAGGATAGGAAAAG	326
	GCGTGTTT**G**TGCCTGTC	327
	GACAGGCA**C**AAACACGC	328
CCT-CTT Pro-278 to Leu Breast cancer	TCCTGAGTAGTGGTAATCTACTGGGACGGAACAGCTTTGAGG TGCGTGTTTGTGCCTGTC**C**TGGGAGAGACCGGCGCACAGAG GAAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGA	329
	TCGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTTCCTCT GTGCGCCGGTCTCTCCCA**G**GACAGGCACAAACACGCACCTC AAAGCTGTTCCGTCCCAGTAGATTACCACTACTCAGGA	330
	TGCCTGTC**C**TGGGAGAG	331
	CTCTCCCA**G**GACAGGCA	332
AGA-AAA Arg-280 to Lys Glioma	GTAGTGGTAATCTACTGGGACGGAACAGCTTTGAGGTGCGTG TTTGTGCCTGTCCTGGGA**G**AGACCGGCGCACAGAGGAAGAG AATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCTGCC	333
	GGCAGCTCGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCT TCCTCTGTGCGCCGGTCT**C**TCCCAGGACAGGCACAAACACG CACCTCAAAGCTGTTCCGTCCCAGTAGATTACCACTAC	334
	TCCTGGGA**G**AGACCGGC	335
	GCCGGTCT**C**TCCCAGGA	336
GAA-GCA Glu-286 to Ala Adrenocortical carcinoma	GGAACAGCTTTGAGGTGCGTGTTTGTGCCTGTCCTGGGAGA GACCGGCGCACAGAGGAAG**A**GAATCTCCGCAAGAAAGGGGA GCCTCACCACGAGCTGCCCCCAGGGAGCACTAAGCGAGG	337
	CCTCGCTTAGTGCTCCCTGGGGGCAGCTCGTGGTGAGGCTC CCCTTTCTTGCGGAGATTC**T**CTTCCTCTGTGCGCCGGTCTCTC CCAGGACAGGCACAAACACGCACCTCAAAGCTGTTCC	338
	AGAGGAAG**A**GAATCTCC	339
	GGAGATTC**T**CTTCCTCT	340

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
CGA-CCA Arg-306 to Pro Rhabdomyosarcoma	AAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCTG CCCCCAGGGAGCACTAAGCGAGGTAAGCAAGCAGGACAAGA AGCGGTGGAGGAGACCAAGGGTGCAGTTATGCCTCAGAT	341
	ATCTGAGGCATAACTGCACCCTTGGTCTCCTCCACCGCTTCTT GTCCTGCTTGCTTACCTCGCTTAGTGCTCCCTGGGGGCAGCT CGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTT	342
	CACTAAGCGAGGTAAGC	343
	GCTTACCTCGCTTAGTG	344
gCGA-TGA Arg-306 to Term Li-Fraumeni syndrome	GAAGAGAATCTCCGCAAGAAAGGGGAGCCTCACCACGAGCT GCCCCCAGGGAGCACTAAGCGAGGTAAGCAAGCAGGACAAG AAGCGGTGGAGGAGACCAAGGGTGCAGTTATGCCTCAGA	345
	TCTGAGGCATAACTGCACCCTTGGTCTCCTCCACCGCTTCTT GTCCTGCTTGCTTACCTCGCTTAGTGCTCCCTGGGGGCAGCT CGTGGTGAGGCTCCCCTTTCTTGCGGAGATTCTCTTC	346
	GCACTAAGCGAGGTAAG	347
	CTTACCTCGCTTAGTGC	348
gCGC-TGC Arg-337 to Cys Osteosarcoma	GGTACTGTGAATATACTTACTTCTCCCCCTCCTCTGTTGCTGC AGATCCGTGGGCGTGAGCGCTTCGAGATGTTCCGAGAGCTG AATGAGGCCTTGGAACTCAAGGATGCCCAGGCTGGGA	349
	TCCCAGCCTGGGCATCCTTGAGTTCCAAGGCCTCATTCAGCT CTCGGAACATCTCGAAGCGCTCACGCCCACGGATCTGCAGC AACAGAGGAGGGGGAGAAGTAAGTATATTCACAGTACC	350
	GGCGTGAGCGCTTCGAG	351
	CTCGAAGCGCTCACGCC	352
CTG-CCG Leu-344 to Pro Li-Fraumeni syndrome	CTCCCCCTCCTCTGTTGCTGCAGATCCGTGGGCGTGAGCGCT TCGAGATGTTCCGAGAGCTGAATGAGGCCTTGGAACTCAAGG ATGCCCAGGCTGGGAAGGAGCCAGGGGGGAGCAGGGC	353
	GCCCTGCTCCCCCCTGGCTCCTTCCCAGCCTGGGCATCCTTG AGTTCCAAGGCCTCATTCAGCTCTCGGAACATCTCGAAGCGC TCACGCCCACGGATCTGCAGCAACAGAGGAGGGGGAG	354
	CCGAGAGCTGAATGAGG	355
	CCTCATTCAGCTCTCGG	356

EXAMPLE 6

beta globin

Hemoglobin, the major protein in the red blood cell, binds oxygen reversibly and is responsible for the cells' capacity to transport oxygen to the tissues. In adults, the major hemoglobin is hemoglobin A, a tetrameric protein consisting of two identical alpha globin chains and two beta globin chains. Disorders involving hemoglobin are among the most common genetic disorders worldwide, with approximately 5% of the world's population being carriers for clinically important hemoglobin mutations. Approximately 300,000 severely affected homozygotes or compound heterozygotes are born each year.

Mutation of the glutamic acid at position 7 in beta globin to valine causes sickle cell anemia, the clinical manifestations of which are well known. Mutations that cause absence of beta chain cause beta-zero-thalassemia. Reduced amounts of detectable beta globin causes beta-plus-thalassemia. For clinical purposes, beta-thalassemia is divided into thalassemia major (transfusion dependent), thalassemia intermedia (of intermediate severity), and thalassemia minor (asymptomatic). Patients with thalassemia major present in the first year of life with severe anemia; they are unable to maintain a hemoglobin level about 5 gm/dl.

The beta-thalassemias were among the first human genetic diseases to be examined by means of recombinant DNA analysis. Baysal et al., *Hemoglobin* 19(3-4):213-36 (1995) and others provide a compendium of mutations that result in beta-thalassemia.

Hemoglobin disorders were among the first to be considered for gene therapy. Transcriptional silencing of genes transferred into hematopoietic stem cells, however, poses one of the most significant challenges to its success. If the transferred gene is not completely silenced, a progressive decline in gene expression is often observed. Position effect variegation (PEV) and silencing mechanisms may act on a transferred globin gene residing in chromatin outside of the normal globin locus during the important terminal phases of erythroblast development when globin transcripts normally accumulate rapidly despite heterochromatization and shutdown of the rest of the genome. The attached table discloses the correcting oligonucleotide base sequences for the beta globin oligonucleotides of the invention.

Table 12

Beta Globin Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Sickle Cell Anemia GLU-7-VAL GAG to GTG	TCTGACACAACTGTGTTCACTAGCAACCTCAAACAGACACCA TGGTGCACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCC CTGTGGGGCAAGGTGAACGTGGATGAAGTTGGTGGTGA	357
	TCACCACCAACTTCATCCACGTTCACCTTGCCCCACAGGGCA GTAACGGCAGACTTCTCCTCAGGAGTCAGGTGCACCATGGT GTCTGTTTGAGGTTGCTAGTGAACACAGTTGTGTCAGA	358
	GACTCCTGAGGAGAAGT	359
	ACTTCTCCTCAGGAGTC	360
Thalassaemia Beta MET-0-ARG ATG to AGG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	361
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCTA GTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	362
	AGACACCATGGTGCACC	363
	GGTGCACCATGGTGTCT	364
Thalassaemia Beta MET-0-ILE ATG to ATA	TATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCAA CCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAA GTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTG	365
	CACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCT CCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCT AGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATA	366
	GACACCATGGTGCACCT	367
	AGGTGCACCATGGTGTC	368
Thalassaemia Beta MET-0-ILE ATG to ATT	TATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCAA CCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAA GTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTG	369
	CACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCT CCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCT AGTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATA	370

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GACACCATGGTGCACCT	371
	AGGTGCACCATGGTGTC	372
Thalassaemia Beta MET-0-LYS ATG to AAG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	373
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCTA GTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	374
	AGACACCATGGTGCACC	375
	GGTGCACCATGGTGTCT	376
Thalassaemia Beta MET-0-THR ATG to ACG	CTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGCA ACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGA AGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGT	377
	ACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTC CTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCTA GTGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAG	378
	AGACACCATGGTGCACC	379
	GGTGCACCATGGTGTCT	380
Thalassaemia Beta MET-0-VAL ATG to GTG	TCTATTGCTTACATTTGCTTCTGACACAACTGTGTTCACTAGC AACCTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAG AAGTCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACG	381
	CGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTTCTCC TCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAGGTTGCTAG TGAACACAGTTGTGTCAGAAGCAAATGTAAGCAATAGA	382
	CAGACACCATGGTGCAC	383
	GTGCACCATGGTGTCTG	384
Thalassaemia Beta TRP-16-Term TGG to TGA	TCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAAGT CTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGAA GTTGGTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTTA	385
	TAACCTTGATACCAACCTGCCCAGGGCCTCACCACCAACTTC ATCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACT TCTCCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGA	386
	GCCCTGTGGGGCAAGGT	387

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACCTTGCCCCACAGGGC	388
Thalassaemia Beta TRP-16-Term TGG to TAG	CTCAAACAGACACCATGGTGCACCTGACTCCTGAGGAGAAG TCTGCCGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGA AGTTGGTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTT	389
	AACCTTGATACCAACCTGCCCAGGGCCTCACCACCAACTTCA TCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCAGACTT CTCCTCAGGAGTCAGGTGCACCATGGTGTCTGTTTGAG	390
	TGCCCTGTGGGGCAAGG	391
	CCTTGCCCCACAGGGCA	392
Thalassaemia Beta LYS-18-Term AAG to TAG	ACAGACACCATGGTGCACCTGACTCCTGAGGAGAAGTCTGC CGTTACTGCCCTGTGGGGCAAGGTGAACGTGGATGAAGTTG GTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTTACAAG	393
	CTTGTAACCTTGATACCAACCTGCCCAGGGCCTCACCACCAA CTTCATCCACGTTCACCTTGCCCCACAGGGCAGTAACGGCA GACTTCTCCTCAGGAGTCAGGTGCACCATGGTGTCTGT	394
	TGTGGGGCAAGGTGAAC	395
	GTTCACCTTGCCCCACA	396
Thalassaemia Beta ASN-20-SER AAC to AGC	CCATGGTGCACCTGACTCCTGAGGAGAAGTCTGCCGTTACT GCCCTGTGGGGCAAGGTGAACGTGGATGAAGTTGGTGGTGA GGCCCTGGGCAGGTTGGTATCAAGGTTACAAGACAGGTT	397
	AACCTGTCTTGTAACCTTGATACCAACCTGCCCAGGGCCTCA CCACCAACTTCATCCACGTTCACCTTGCCCCACAGGGCAGTA ACGGCAGACTTCTCCTCAGGAGTCAGGTGCACCATGG	398
	CAAGGTGAACGTGGATG	399
	CATCCACGTTCACCTTG	400
Thalassaemia Beta GLU-23-ALA GAA to GCA	ACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCCCTGTGG GGCAAGGTGAACGTGGATGAAGTTGGTGGTGAGGCCCTGG GCAGGTTGGTATCAAGGTTACAAGACAGGTTTAAGGAGAC	401
	GTCTCCTTAAACCTGTCTTGTAACCTTGATACCAACCTGCCCA GGGCCTCACCACCAACTTCATCCACGTTCACCTTGCCCCACA GGGCAGTAACGGCAGACTTCTCCTCAGGAGTCAGGT	402
	CGTGGATGAAGTTGGTG	403
	CACCAACTTCATCCACG	404

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta GLU-23-term GAA to TAA	CACCTGACTCCTGAGGAGAAGTCTGCCGTTACTGCCCTGTG GGGCAAGGTGAACGTGGAT**G**AAGTTGGTGGTGAGGCCCTG GGCAGGTTGGTATCAAGGTTACAAGACAGGTTTAAGGAGA	405
	TCTCCTTAAACCTGTCTTGTAACCTTGATACCAACCTGCCCAG GGCCTCACCACCAACTT**C**ATCCACGTTCACCTTGCCCCACAG GGCAGTAACGGCAGACTTCTCCTCAGGAGTCAGGTG	406
	ACGTGGAT**G**AAGTTGGT	407
	ACCAACTT**C**ATCCACGT	408
Thalassaemia Beta GLU-27-LYS GAG to AAG	GAGGAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAA CGTGGATGCAGTTGGTGGT**G**AGGCCCTGGGCAGGTTGGTAT CAAGGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACT	409
	AGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGATAC CAACCTGCCCAGGGCCT**C**ACCACCAACTGCATCCACGTTCA CTTTGCCCCACAGGGCATTGACAGCAGTCTTCTCCTC	410
	TTGGTGGT**G**AGGCCCTG	411
	CAGGGCCT**C**ACCACCAA	412
Thalassaemia Beta GLU-27-Term GAG to TAG	GAGGAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAA CGTGGATGCAGTTGGTGGT**G**AGGCCCTGGGCAGGTTGGTAT CAAGGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACT	413
	AGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGATAC CAACCTGCCCAGGGCCT**C**ACCACCAACTGCATCCACGTTCA CTTTGCCCCACAGGGCATTGACAGCAGTCTTCTCCTC	414
	TTGGTGGT**G**AGGCCCTG	415
	CAGGGCCT**C**ACCACCAA	416
Thalassaemia Beta ALA-28-SER GCC to TCC	GAGAAGACTGCTGTCAATGCCCTGTGGGGCAAAGTGAACGT GGATGCAGTTGGTGGTGAG**G**CCCTGGGCAGGTTGGTATCAA GGTTATAAGAGAGGCTCAAGGAGGCAAATGGAAACTGGG	417
	CCCAGTTTCCATTTGCCTCCTTGAGCCTCTCTTATAACCTTGA TACCAACCTGCCCAGGG**C**CTCACCACCAACTGCATCCACGTT CACTTTGCCCCACAGGGCATTGACAGCAGTCTTCTC	418
	GTGGTGAG**G**CCCTGGGC	419
	GCCCAGGG**C**CTCACCAC	420

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta ARG-31-THR AGG to ACG	CTGTCAATGCCCTGTGGGGCAAAGTGAACGTGGATGCAGTT GGTGGTGAGGCCCTGGGCAGGTTGGTATCAAGGTTATAAGA GAGGCTCAAGGAGGCAAATGGAAACTGGGCATGTGTAGA	421
	TCTACACATGCCCAGTTTCCATTTGCCTCCTTGAGCCTCTCTT ATAACCTTGATACCAACCTGCCCAGGGCCTCACCACCAACTG CATCCACGTTCACTTTGCCCCACAGGGCATTGACAG	422
	CCTGGGCAGGTTGGTAT	423
	ATACCAACCTGCCCAGG	424
Thalassaemia Beta Leu-33-GLN CTG to CAG	TGGGTTTCTGATAGGCACTGACTCTCTGTCCCTTGGGCTGTT TTCCTACCCTCAGATTACTGGTGGTCTACCCTTGGACCCAGA GGTTCTTTGAGTCCTTTGGGGATCTGTCCTCTCCTGA	425
	TCAGGAGAGGACAGATCCCCAAAGGACTCAAAGAACCTCTG GGTCCAAGGGTAGACCACCAGTAATCTGAGGGTAGGAAAAC AGCCCAAGGGACAGAGAGTCAGTGCCTATCAGAAACCCA	426
	CAGATTACTGGTGGTCT	427
	AGACCACCAGTAATCTG	428
Thalassaemia Beta TYR-36-Term TAC to TAA	ATAGGCACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCT CAGATTACTGGTGGTCTACCCTTGGACCCAGAGGTTCTTTGA GTCCTTTGGGGATCTGTCCTCTCCTGATGCTGTTATG	429
	CATAACAGCATCAGGAGAGGACAGATCCCCAAAGGACTCAA AGAACCTCTGGGTCCAAGGGTAGACCACCAGTAATCTGAGG GTAGGAAAACAGCCCAAGGGACAGAGAGTCAGTGCCTAT	430
	GTGGTCTACCCTTGGAC	431
	GTCCAAGGGTAGACCAC	432
Thalassaemia Beta TRP-38-Term TGG to TGA	ACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGATTA CTGGTGGTCTACCCTTGGACCCAGAGGTTCTTTGAGTCCTTT GGGGATCTGTCCTCTCCTGATGCTGTTATGGGCAAC	433
	GTTGCCCATAACAGCATCAGGAGAGGACAGATCCCCAAAGG ACTCAAAGAACCTCTGGGTCCAAGGGTAGACCACCAGTAATC TGAGGGTAGGAAAACAGCCCAAGGGACAGAGAGTCAGT	434
	TACCCTTGGACCCAGAG	435
	CTCTGGGTCCAAGGGTA	436

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta TRP-38-Term TGG to TAG	CACTGACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGATT ACTGGTGGTCTACCCTT**G**GACCCAGAGGTTCTTTGAGTCCTT TGGGGATCTGTCCTCTCCTGATGCTGTTATGGGCAA	437
	TTGCCCATAACAGCATCAGGAGAGGACAGATCCCCAAAGGA CTCAAAGAACCTCTGGGTC**C**AAGGGTAGACCACCAGTAATCT GAGGGTAGGAAAACAGCCCAAGGGACAGAGAGTCAGTG	438
	CTACCCTT**G**GACCCAGA	439
	TCTGGGTC**C**AAGGGTAG	440
Thalassaemia Beta GLN-40-Term CAG-TAG	ACTCTCTGTCCCTTGGGCTGTTTTCCTACCCTCAGATTACTG GTGGTCTACCCTTGGACC**C**AGAGGTTCTTTGAGTCCTTTGGG GATCTGTCCTCTCCTGATGCTGTTATGGGCAACCCTA	441
	TAGGGTTGCCCATAACAGCATCAGGAGAGGACAGATCCCCA AAGGACTCAAAGAACCTCT**G**GGTCCAAGGGTAGACCACCAG TAATCTGAGGGTAGGAAAACAGCCCAAGGGACAGAGAGT	442
	CTTGGACC**C**AGAGGTTC	443
	GAACCTCT**G**GGTCCAAG	444
Thalassaemia Beta GLU-44-Term GAG to TAG	TTGGGCTGTTTTCCTACCCTCAGATTACTGGTGGTCTACCCT TGGACCCAGAGGTTCTTT**G**AGTCCTTTGGGGATCTGTCCTCT CCTGATGCTGTTATGGGCAACCCTAAGGTGAAGGCTC	445
	GAGCCTTCACCTTAGGGTTGCCCATAACAGCATCAGGAGAG GACAGATCCCCAAAGGACT**C**AAAGAACCTCTGGGTCCAAGG GTAGACCACCAGTAATCTGAGGGTAGGAAAACAGCCCAA	446
	GGTTCTTT**G**AGTCCTTT	447
	AAAGGACT**C**AAAGAACC	448
Thalassaemia Beta LYS-62-Term AAG to TAG	TTCTTTGAGTCCTTTGGGGATCTGTCCTCTCCTGATGCTGTTA TGGGCAACCCTAAGGTG**A**AGGCTCATGGCAAGAAGGTGCTA GGTGCCTTTAGTGATGGCCTGGCTCACCTGGACAACC	449
	GGTTGTCCAGGTGAGCCAGGCCATCACTAAAGGCACCTAGC ACCTTCTTGCCATGAGCCT**T**CACCTTAGGGTTGCCCATAACA GCATCAGGAGAGGACAGATCCCCAAAGGACTCAAAGAA	450
	CTAAGGTG**A**AGGCTCAT	451
	ATGAGCCT**T**CACCTTAG	452

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta SER-73-ARG AGT to AGA	TGCTGTTATGGGCAACCCTAAGGTGAAGGCTCATGGCAAGA AGGTGCTAGGTGCCTTTAGTGATGGCCTGGCTCACCTGGAC AACCTCAAGGGCACTTTTTCTCAGCTGAGTGAGCTGCAC	453
	GTGCAGCTCACTCAGCTGAGAAAAAGTGCCCTTGAGGTTGTC CAGGTGAGCCAGGCCATCACTAAAGGCACCTAGCACCTTCT TGCCATGAGCCTTCACCTTAGGGTTGCCCATAACAGCA	454
	GCCTTTAGTGATGGCCT	455
	AGGCCATCACTAAAGGC	456
Haemolytic Anaemia GLY-75-VAL GGC to GTC	TTATGGGCAACCCTAAGGTGAAGGCTCATGGCAAGAAGGTG CTAGGTGCCTTTAGTGATGGCCTGGCTCACCTGGACAACCTC AAGGGCACTTTTTCTCAGCTGAGTGAGCTGCACTGTGA	457
	TCACAGTGCAGCTCACTCAGCTGAGAAAAAGTGCCCTTGAG GTTGTCCAGGTGAGCCAGGCCATCACTAAAGGCACCTAGCA CCTTCTTGCCATGAGCCTTCACCTTAGGGTTGCCCATAA	458
	TAGTGATGGCCTGGCTC	459
	GAGCCAGGCCATCACTA	460
Thalassaemia Beta GLU-91-Term GAG to TAG	GCCTTTAGTGATGGCCTGGCTCACCTGGACAACCTCAAGGG CACCTTTGCCACACTGAGTGAGCTGCACTGTGACAAGCTGCA CGTGGATCCTGAGAACTTCAGGGTGAGTCTATGGGACC	461
	GGTCCCATAGACTCACCCTGAAGTTCTCAGGATCCACGTGCA GCTTGTCACAGTGCAGCTCACTCAGTGTGGCAAAGGTGCCC TTGAGGTTGTCCAGGTGAGCCAGGCCATCACTAAAGGC	462
	CACTGAGTGAGCTGCAC	463
	GTGCAGCTCACTCAGTG	464
Thalassaemia Beta VAL-99-MET GTG to ATG	CTGGACAACCTCAAGGGCACTTTTTCTCAGCTGAGTGAGCTG CACTGTGACAAGCTGCACGTGGATCCTGAGAACTTCAGGGT GAGTCCAGGAGATGCTTCACTTTTCTCTTTTTACTTTC	465
	GAAAGTAAAAAGAGAAAAGTGAAGCATCTCCTGGACTCACCC TGAAGTTCTCAGGATCCACGTGCAGCTTGTCACAGTGCAGCT CACTCAGCTGAGAAAAAGTGCCCTTGAGGTTGTCCAG	466
	AGCTGCACGTGGATCCT	467
	AGGATCCACGTGCAGCT	468

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta LEU-111-PRO CTG-CCG	CCCTTTTGCTAATCATGTTCATACCTCTTATCTTCCTCCCACA GCTCCTGGGCAACGTGCTGGTCTGTGTGCTGGCCCATCACT TTGGCAAAGAATTCACCCCACCAGTGCAGGCTGCCTA	469
	TAGGCAGCCTGCACTGGTGGGGTGAATTCTTTGCCAAAGTG ATGGGCCAGCACACAGACCAGCACGTTGCCCAGGAGCTGTG GGAGGAAGATAAGAGGTATGAACATGATTAGCAAAAGGG	470
	CAACGTGCTGGTCTGTG	471
	CACAGACCAGCACGTTG	472
Thalassaemia Beta CYS-113-Term TGT to TGA	GCTAATCATGTTCATACCTCTTATCTTCCTCCCACAGCTCCTG GGCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAA AGAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAA	473
	TTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTTGCC AAAGTGATGGGCCAGCACACAGACCAGCACGTTGCCCAGGA GCTGTGGGAGGAAGATAAGAGGTATGAACATGATTAGC	474
	CTGGTCTGTGTGCTGGC	475
	GCCAGCACACAGACCAG	476
Thalassaemia Beta LEU-115-PRO CTG to CCG	TCATGTTCATACCTCTTATCTTCCTCCCACAGCTCCTGGGCAA CGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAAGAATT CACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT	477
	ACCACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCT TTGCCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGCC CAGGAGCTGTGGGAGGAAGATAAGAGGTATGAACATGA	478
	CTGTGTGCTGGCCCATC	479
	GATGGGCCAGCACACAG	480
Thalassaemia Beta ALA-116-ASP GCC to GAC	TGTTCATACCTCTTATCTTCCTCCCACAGCTCCTGGGCAACG TGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAAGAATTCA CCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGTGGC	481
	GCCACCACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAA TTCTTTGCCAAAGTGATGGGCCAGCACACAGACCAGCACGTT GCCCAGGAGCTGTGGGAGGAAGATAAGAGGTATGAACA	482
	TGTGCTGGCCCATCACT	483
	AGTGATGGGCCAGCACA	484

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia Beta GLU-122-Term GAA to TAA	TTCCTCCCACAGCTCCTGGGCAACGTGCTGGTCTGTGTGCT GGCCCATCACTTTGGCAAAGAATTCACCCCACCAGTGCAGG CTGCCTATCAGAAAGTGGTGGCTGGTGTGGCTAATGCCC	485
	GGGCATTAGCCACACCAGCCACCACTTTCTGATAGGCAGCC TGCACTGGTGGGGTGAATTCTTTGCCAAAGTGATGGGCCAG CACACAGACCAGCACGTTGCCCAGGAGCTGTGGGAGGAA	486
	TTGGCAAAGAATTCACC	487
	GGTGAATTCTTTGCCAA	488
Thalassaemia Beta GLN-128-PRO CAG to CCG	GCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAAA GAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT GGCTGGTGTGGCTAATGCCCTGGCCCACAAGTATCACTA	489
	TAGTGATACTTGTGGGCCAGGGCATTAGCCACACCAGCCAC CACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTT GCCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGC	490
	ACCAGTGCAGGCTGCCT	491
	AGGCAGCCTGCACTGGT	492
Thalassaemia Beta GLN-128-Term CAG to TAG	GGCAACGTGCTGGTCTGTGTGCTGGCCCATCACTTTGGCAA AGAATTCACCCCACCAGTGCAGGCTGCCTATCAGAAAGTGGT GGCTGGTGTGGCTAATGCCCTGGCCCACAAGTATCACT	493
	AGTGATACTTGTGGGCCAGGGCATTAGCCACACCAGCCACC ACTTTCTGATAGGCAGCCTGCACTGGTGGGGTGAATTCTTTG CCAAAGTGATGGGCCAGCACACAGACCAGCACGTTGCC	494
	CACCAGTGCAGGCTGCC	495
	GGCAGCCTGCACTGGTG	496
Thalassaemia Beta GLN-132-LYS CAG to AAG	GTCTGTGTGCTGGCCCATCACTTTGGCAAAGAATTCACCCCA CCAGTGCAGGCTGCCTATCAGAAAGTGGTGGCTGGTGTGGC TAATGCCCTGGCCCACAAGTATCACTAAGCTCGCTTTC	497
	GAAAGCGAGCTTAGTGATACTTGTGGGCCAGGGCATTAGCC ACACCAGCCACCACTTTCTGATAGGCAGCCTGCACTGGTGG GGTGAATTCTTTGCCAAAGTGATGGGCCAGCACACAGAC	498
	CTGCCTATCAGAAAGTG	499
	CACTTTCTGATAGGCAG	500

EXAMPLE 7

Retinoblastoma

Retinoblastoma (RB) is an embryonic neoplasm of retinal origin. It almost always presents in early childhood and is often bilateral. The risk of osteogenic sarcoma is increased 500-fold in bilateral retinoblastoma patients, the bone malignancy being at sites removed from those exposed to radiation treatment of the eye tumor.

The retinoblastoma susceptibility gene (pRB; pRb) plays a pivotal role in the regulation of the cell cycle. pRB restrains cell cycle progression by maintaining a checkpoint in late G_1 that controls commitment of cells to enter S phase. The critical role that pRB plays in cell cycle regulation explains its status as archetypal tumor suppressor: loss of pRB function results in an inability to maintain control of the G_1 checkpoint; unchecked progression through the cell cycle is, in turn, a hallmark of neoplasia.

Blanquet *et al.*, *Hum. Molec. Genet.* 4: 383-388 (1995) performed a mutation survey of the RB1 gene in 232 patients with hereditary or nonhereditary retinoblastoma. They systematically explored all 27 exons and flanking sequences, as well as the promoter. All types of point mutations were represented and found to be unequally distributed along the RB1 gene sequence. In the population studied, exons 3, 8, 18, and 19 were preferentially altered. The attached table discloses the correcting oligonucleotide base sequences for the retinoblastoma oligonucleotides of the invention.

Table 13

pRB Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Trp99Term TGG-TAG	AATATTTGATCTTTATTTTTTGTTCCCAGGGAGGTTATATTCAA AAGAAAAAGGAACTGT**G**GGGAATCTGTATCTTTATTGCAGCA GTTGACCTAGATGAGATGTCGTTCACTTTTACTGA	501
	TCAGTAAAAGTGAACGACATCTCATCTAGGTCAACTGCTGCA ATAAAGATACAGATTCCC**C**ACAGTTCCTTTTTCTTTTGAATATA ACCTCCCTGGGAACAAAAAATAAAGATCAAATATT	502
	GGAACTGT**G**GGGAATCT	503
	AGATTCCC**C**ACAGTTCC	504

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Glu137Asp GAA-GAT	ATTTACTTTTTTCTATTCTTTCCTTTGTAGTGTCCATAAATTCTT TAACTTACTAAAAGAAATTGATACCAGTACCAAAGTTGATAAT GCTATGTCAAGACTGTTGAAGAAGTATGATGTA	505
	TACATCATACTTCTTCAACAGTCTTGACATAGCATTATCAACT TTGGTACTGGTATCAATTTCTTTTAGTAAGTTAAAGAATTTATG GACACTACAAAGGAAAGAATAGAAAAAAGTAAAT	506
	CTAAAAGAAATTGATAC	507
	GTATCAATTTCTTTTAG	508
Retinoblastoma Glu137Term GAA-TAA	TGATTTACTTTTTTCTATTCTTTCCTTTGTAGTGTCCATAAATT CTTTAACTTACTAAAAGAAATTGATACCAGTACCAAAGTTGAT AATGCTATGTCAAGACTGTTGAAGAAGTATGATG	509
	CATCATACTTCTTCAACAGTCTTGACATAGCATTATCAACTTT GGTACTGGTATCAATTTCTTTTAGTAAGTTAAAGAATTTATGG ACACTACAAAGGAAAGAATAGAAAAAAGTAAATCA	510
	TACTAAAAGAAATTGAT	511
	ATCAATTTCTTTTAGTA	512
Retinoblastoma Gln176Term CAA-TAA	AAAATGTTAAAAAGTCATAATGTTTTTCTTTTCAGGACATGTG AACTTATATATTTGACACAACCCAGCAGTTCGTAAGTAGTTCA CAGAATGTTATTTTTCACTTAAAAAAAAAGATTTT	513
	AAAATCTTTTTTTTTTAAGTGAAAAATAACATTCTGTGAACTACT TACGAACTGCTGGGTTGTGTCAAATATATAAGTTCACATGTCC TGAAAAGAAAAACATTATGACTTTTTAACATTTT	514
	ATTTGACACAACCCAGC	515
	GCTGGGTTGTGTCAAAT	516
Retinoblastoma Ile185Thr ATA-ACA	TGATACATTTTTCCTGTTTTTTTTCTGCTTTCTATTTGTTTAATA GGATATCTACTGAAATAAATTCTGCATTGGTGCTAAAAGTTTC TTGGATCACATTTTTATTAGCTAAAGGTAAGTT	517
	AACTTACCTTTAGCTAATAAAAATGTGATCCAAGAAACTTTTA GCACCAATGCAGAATTTATTTCAGTAGATATCCTATTAAACAA ATAGAAAGCAGAAAAAAAACAGGAAAAATGTATCA	518
	TACTGAAATAAATTCTG	519
	CAGAATTTATTTCAGTA	520

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Gln207Term CAA-TAA	AAAGATCTGAATCTCTAACTTTCTTTAAAAATGTACATTTTTTT TTCAGGGGAAGTATTA**C**AAATGGAAGATGATCTGGTGATTTC ATTTCAGTTAATGCTATGTGTCCTTGACTATTTTA	521
	TAAAATAGTCAAGGACACATAGCATTAACTGAAATGAAATCAC CAGATCATCTTCCATTT**G**TAATACTTCCCCTGAAAAAAAAATG TACATTTTTAAAGAAAGTTAGAGATTCAGATCTTT	522
	AAGTATTA**C**AAATGGAA	523
	TTCCATTT**G**TAATACTT	524
Retinoblastoma Arg251Term CGA to TGA	GTTCTTATCTAATTTACCACTTTTACAGAAACAGCTGTTATAC CCATTAATGGTTCACCT**C**GAACACCCAGGCGAGGTCAGAACA GGAGTGCACGGATAGCAAAACAACTAGAAAATGATA	525
	TATCATTTTCTAGTTGTTTTGCTATCCGTGCACTCCTGTTCTG ACCTCGCCTGGGTGTTC**G**AGGTGAACCATTAATGGGTATAAC AGCTGTTTCTGTAAAAGTGGTAAATTAGATAAGAAC	526
	GTTCACCT**C**GAACACCC	527
	GGGTGTTC**G**AGGTGAAC	528
Retinoblastoma Arg255Term CGA to TGA	TTTACCACTTTTACAGAAACAGCTGTTATACCCATTAATGGTT CACCTCGAACACCCAGG**C**GAGGTCAGAACAGGAGTGCACG GATAGCAAAACAACTAGAAAATGATACAAGAATTATTG	529
	CAATAATTCTTGTATCATTTTCTAGTTGTTTTGCTATCCGTGCA CTCCTGTTCTGACCTC**G**CCTGGGTGTTCGAGGTGAACCATTA ATGGGTATAACAGCTGTTTCTGTAAAAGTGGTAAA	530
	CACCCAGG**C**GAGGTCAG	531
	CTGACCTC**G**CCTGGGTG	532
Retinoblastoma Gln266Term CAA to TAA	ATTAATGGTTCACCTCGAACACCCAGGCGAGGTCAGAACAG GAGTGCACGGATAGCAAAA**C**AACTAGAAAATGATACAAGAAT TATTGAAGTTCTCTGTAAAGAACATGAATGTAATATAG	533
	CTATATTACATTCATGTTCTTTACAGAGAACTTCAATAATTCTT GTATCATTTTCTAGTT**G**TTTTGCTATCCGTGCACTCCTGTTCT GACCTCGCCTGGGTGTTCGAGGTGAACCATTAAT	534
	TAGCAAAA**C**AACTAGAA	535
	TTCTAGTT**G**TTTTGCTA	536

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Arg320Term CGA to TGA	TGACATGTAAAGGATAATTGTCAGTGACTTTTTTCTTTCAAGG TTGAAAATCTTTCTAAA**C**GATACGAAGAAATTTATCTTAAAAAT AAAGATCTAGATGCAAGATTATTTTTGGATCATG	537
	CATGATCCAAAAATAATCTTGCATCTAGATCTTTATTTTTAAGA TAAATTTCTTCGTATC**G**TTTAGAAAGATTTTCAACCTTGAAAG AAAAAAGTCACTGACAATTATCCTTTACATGTCA	538
	TTTCTAAA**C**GATACGAA	539
	TTCGTATC**G**TTT**A**GAAA	540
Retinoblastoma Gln354Term CAG to TAG	ACAAATTGTAAATTT[T]CAGTATGTGAATGACTTCACTTATTGTT ATTTAGTTTTGAAACA**C**AGAGAACACCACGAAAAAGTAACCTT GATGAAGAGGTGAATGTAATTCCTCCACACACTC	541
	GAGTGTGTGGAGGAATTACATTCACCTCTTCATCAAGGTTAC TTTTTCGTGGTGTTCTCT**G**TGTTTCAAAACTAAATAACAATAA GTGAAGTCATTCACATACTGAAAATTTACAATTTGT	542
	TTGAAACA**C**AGAGAACA	543
	TGTTCTCT**G**TGTTTCAA	544
Retinoblastoma Arg358Gly CGA to GGA	TTTTCAGTATGTGAATGACTTCACTTATTGTTATTTAGTTTTGA AACACAGAGAACACCA**C**GAAAAAGTAACCTTGATGAAGAGGT GAATGTAATTCCTCCACACACTCCAGTTAGGTATG	545
	CATACCTAACTGGAGTGTGTGGAGGAATTACATTCACCTCTT CATCAAGGTTACTTTTTC**G**TGGTGTTCTCTGTGTTTCAAAACT AAATAACAATAAGTGAAGTCATTCACATACTGAAAA	546
	GAACACCA**C**GAAAAAGT	547
	ACTTTTTC**G**TGGTGTTC	548
Retinoblastoma Arg358Term CGA to TGA	TTTTCAGTATGTGAATGACTTCACTTATTGTTATTTAGTTTTGA AACACAGAGAACACCA**C**GAAAAAGTAACCTTGATGAAGAGGT GAATGTAATTCCTCCACACACTCCAGTTAGGTATG	549
	CATACCTAACTGGAGTGTGTGGAGGAATTACATTCACCTCTT CATCAAGGTTACTTTTTC**G**TGGTGTTCTCTGTGTTTCAAAACT AAATAACAATAAGTGAAGTCATTCACATACTGAAAA	550
	GAACACCA**C**GAAAAAGT	551
	ACTTTTTC**G**TGGTGTTC	552

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Ser397Term TCA to TAA	CTGTTATGAACACTATCCAACAATTAATGATGATTTTAAATTCA GCAAGTGATCAACCTTCAGAAAATCTGATTTCCTATTTTAACG TAAGCCATATATGAAACATTATTTATTGTAATAT	553
	ATATTACAATAAATAATGTTTCATATATGGCTTACGTTAAAATA GGAAATCAGATTTTCTGAAGGTTGATCACTTGCTGAATTTAAA ATCATCATTAATTGTTGGATAGTGTTCATAACAG	554
	TCAACCTTCAGAAAATC	555
	GATTTTCTGAAGGTTGA	556
Retinoblastoma Arg445Term CGA to TGA	TTTCATAATTGTGATTTTCTAAAATAGCAGGCTCTTATTTTTCT TTTTGTTTGTTTGTAGCGATACAAACTTGGAGTTCGCTTGTAT TACCGAGTAATGGAATCCATGCTTAAATCAGTAA	557
	TTACTGATTTAAGCATGGATTCCATTACTCGGTAATACAAGCG AACTCCAAGTTTGTATCGCTACAAACAAACAAAAAGAAAAATA AGAGCCTGCTATTTTAGAAAATCACAATTATGAAA	558
	GTTTGTAGCGATACAAA	559
	TTTGTATCGCTACAAAC	560
Retinoblastoma Arg455Term CGA to TGA	GCTCTTATTTTTCTTTTTGTTTGTTTGTAGCGATACAAACTTGG AGTTCGCTTGTATTACCGAGTAATGGAATCCATGCTTAAATCA GTAAGTTAAAAACAATATAAAAAAATTTCAGCCG	561
	CGGCTGAAATTTTTTTATATTGTTTTTAACTTACTGATTTAAGC ATGGATTCCATTACTCGGTAATACAAGCGAACTCCAAGTTTGT ATCGCTACAAACAAACAAAAAGAAAAATAAGAGC	562
	TGTATTACCGAGTAATG	563
	CATTACTCGGTAATACA	564
Retinoblastoma Arg552Term CGA to TGA	ATCGAAAGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAA ATGATAAAACATTTAGAACGATGTGAACATCGAATCATGGAAT CCCTTGCATGGCTCTCAGTAAGTAGCTAAATAATTG	565
	CAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGATTCCAT GATTCGATGTTCACATCGTTCTAAATGTTTTATCATTTCTCTTG TCAAGTTGCCTTCTGCTTTGATAAAACTTTCGAT	566
	ATTTAGAACGATGTGAA	567
	TTCACATCGTTCTAAAT	568

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Cys553Term TGT to TGA	AAGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAAATGAT AAAACATTTAGAACGATGTGAACATCGAATCATGGAATCCCTT GCATGGCTCTCAGTAAGTAGCTAAATAATTGAAGAA	569
	TTCTTCAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGAT TCCATGATTCGATGTTCACATCGTTCTAAATGTTTTATCATTTC TCTTGTCAAGTTGCCTTCTGCTTTGATAAAACTT	570
	GAACGATGTGAACATCG	571
	CGATGTTCACATCGTTC	572
Retinoblastoma Glu554Term GAA to TAA	AGTTTTATCAAAGCAGAAGGCAACTTGACAAGAGAAATGATA AAACATTTAGAACGATGTGAACATCGAATCATGGAATCCCTT GCATGGCTCTCAGTAAGTAGCTAAATAATTGAAGAAA	573
	TTTCTTCAATTATTTAGCTACTTACTGAGAGCCATGCAAGGGA TTCCATGATTCGATGTTCACATCGTTCTAAATGTTTTATCATTT CTCTTGTCAAGTTGCCTTCTGCTTTGATAAAACT	574
	AACGATGTGAACATCGA	575
	TCGATGTTCACATCGTT	576
Retinoblastoma Ser567Leu TCA to TTA	TACCTGGGAAAATTATGCTTACTAATGTGGTTTTAATTTCATC ATGTTTCATATAGGATTCACCTTTATTTGATCTTATTAAACAAT CAAAGGACCGAGAAGGACCAACTGATCACCTTGA	577
	TCAAGGTGATCAGTTGGTCCTTCTCGGTCCTTTGATTGTTTAA TAAGATCAAATAAAGGTGAATCCTATATGAAACATGATGAAAT TAAAACCACATTAGTAAGCATAATTTTCCCAGGTA	578
	ATAGGATTCACCTTTAT	579
	ATAAAGGTGAATCCTAT	580
Retinoblastoma Gln575Term CAA to TAA	AATGTGGTTTTAATTTCATCATGTTTCATATAGGATTCACCTTT ATTTGATCTTATTAAACAATCAAAGGACCGAGAAGGACCAAC TGATCACCTTGAATCTGCTTGTCCTCTTAATCTTC	581
	GAAGATTAAGAGGACAAGCAGATTCAAGGTGATCAGTTGGTC CTTCTCGGTCCTTTGATTGTTTAATAAGATCAAATAAAGGTGA ATCCTATATGAAACATGATGAAATTAAAACCACATT	582
	TTATTAAACAATCAAAG	583
	CTTTGATTGTTTAATAA	584

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Arg579Term CGA to TGA	ATTTCATCATGTTTCATATAGGATTCACCTTTATTTGATCTTAT TAAACAATCAAAGGAC**C**GAGAAGGACCAACTGATCACCTTGA ATCTGCTTGTCCTCTTAATCTTCCTCTCCAGAATA	585
	TATTCTGGAGAGGAAGATTAAGAGGACAAGCAGATTCAAGGT GATCAGTTGGTCCTTCTC**G**GTCCTTTGATTGTTTAATAAGATC AAATAAAGGTGAATCCTATATGAAACATGATGAAAT	586
	CAAAGGAC**C**GAGAAGGA	587
	TCCTTCTC**G**GTCCTTTG	588
Retinoblastoma Glu580Term GAA to TAA	TCATCATGTTTCATATAGGATTCACCTTTATTTGATCTTATTAA ACAATCAAAGGACCGA**G**AAGGACCAACTGATCACCTTGAATC TGCTTGTCCTCTTAATCTTCCTCTCCAGAATAATC	589
	GATTATTCTGGAGAGGAAGATTAAGAGGACAAGCAGATTCAA GGTGATCAGTTGGTCCTT**C**TCGGTCCTTTGATTGTTTAATAAG ATCAAATAAAGGTGAATCCTATATGAAACATGATGA	590
	AGGACCGA**G**AAGGACCA	591
	TGGTCCTT**C**TCGGTCCT	592
Retinoblastoma Ser634Term TCA to TGA	AGAAAAAAGGTTCAACTACGCGTGTAAATTCTACTGCAAATG CAGAGACACAAGCAACCT**C**AGCCTTCCAGACCCAGAAGCCA TTGAAATCTACCTCTCTTTCACTGTTTTATAAAAAAGG	593
	CCTTTTTTATAAAACAGTGAAAGAGAGGTAGATTTCAATGGCT TCTGGGTCTGGAAGGCT**G**AGGTTGCTTGTGTCTCTGCATTTG CAGTAGAATTTACACGCGTAGTTGAACCTTTTTTCT	594
	AGCAACCT**C**AGCCTTCC	595
	GGAAGGCT**G**AGGTTGCT	596
Retinoblastoma Ala635Pro GCC to CCC	AAAAAAGGTTCAACTACGCGTGTAAATTCTACTGCAAATGCA GAGACACAAGCAACCTCA**G**CCTTCCAGACCCAGAAGCCATT GAAATCTACCTCTCTTTCACTGTTTTATAAAAAAGGTT	597
	AACCTTTTTTATAAAACAGTGAAAGAGAGGTAGATTTCAATGG CTTCTGGGTCTGGAAGG**C**TGAGGTTGCTTGTGTCTCTGCATT TGCAGTAGAATTTACACGCGTAGTTGAACCTTTTTTT	598
	CAACCTCA**G**CCTTCCAG	599
	CTGGAAGG**C**TGAGGTTG	600

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Gln639Term CAG to TAG	ACTACGCGTGTAAATTCTACTGCAAATGCAGAGACACAAGCA ACCTCAGCCTTCCAGACCCAGAAGCCATTGAAATCTACCTCT CTTTCACTGTTTTATAAAAAAGGTTAGTAGATGATTA	601
	TAATCATCTACTAACCTTTTTTATAAAACAGTGAAAGAGAGGT AGATTTCAATGGCTTCTGGGTCTGGAAGGCTGAGGTTGCTTG TGTCTCTGCATTTGCAGTAGAATTTACACGCGTAGT	602
	TCCAGACCCAGAAGCCA	603
	TGGCTTCTGGGTCTGGA	604
Retinoblastoma Leu657Pro CTA to CCA	TTGTAATTCAAAATGAAGAGTAAAAATGACTAATTTTTCTTATT CCCACAGTGTATCGGCTAGCCTATCTCCGGCTAAATACACTT TGTGAACGCCTTCTGTCTGAGCACCCAGAATTAGA	605
	TCTAATTCTGGGTGCTCAGACAGAAGGCGTTCACAAAGTGTA TTTAGCCGGAGATAGGCTAGCCGATACACTGTGGGAATAAG AAAAATTAGTCATTTTTACTGTTCATTTTGAATTACAA	606
	GTATCGGCTAGCCTATC	607
	GATAGGCTAGCCGATAC	608
Retinoblastoma Arg661Trp CGG to TGG	AATGAACAGTAAAAATGACTAATTTTTCTTATTCCCACAGTGT ATCGGCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGCC TTCTGTCTGAGCACCCAGAATTAGAACATATCATCT	609
	AGATGATATGTTCTAATTCTGGGTGCTCAGACAGAAGGCGTT CACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATACACTG TGGGAATAAGAAAAATTAGTCATTTTTACTGTTCATT	610
	CCTATCTCCGGCTAAAT	611
	ATTTAGCCGGAGATAGG	612
Retinoblastoma Leu662Pro CTA to CCA	AACAGTAAAAATGACTAATTTTTCTTATTCCCACAGTGTATCG GCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGCCTTCT GTCTGAGCACCCAGAATTAGAACATATCATCTGGAC	613
	GTCCAGATGATATGTTCTAATTCTGGGTGCTCAGACAGAAGG CGTTCACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATAC ACTGTGGGAATAAGAAAAATTAGTCATTTTTACTGTT	614
	TCTCCGGCTAAATACAC	615
	GTGTATTTAGCCGGAGA	616

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Glu675Term GAA to TAA	TATCGGCTAGCCTATCTCCGGCTAAATACACTTTGTGAACGC CTTCTGTCTGAGCACCCA**G**AATTAGAACATATCATCTGGACC CTTTTCCAGCACACCCTGCAGAATGAGTATGAACTCA	617
	TGAGTTCATACTCATTCTGCAGGGTGTGCTGGAAAAGGGTCC AGATGATATGTTCTAATT**C**TGGGTGCTCAGACAGAAGGCGTT CACAAAGTGTATTTAGCCGGAGATAGGCTAGCCGATA	618
	AGCACCCA**G**AATTAGAA	619
	TTCTAATT**C**TGGGTGCT	620
Retinoblastoma Gln685Pro CAG to CCG	TTTGTGAACGCCTTCTGTCTGAGCACCCAGAATTAGAACATA TCATCTGGACCCTTTTCC**A**GCACACCCTGCAGAATGAGTATG AACTCATGAGAGACAGGCATTTGGACCAAGTAAGAAA	621
	TTTCTTACTTGGTCCAAATGCCTGTCTCTCATGAGTTCATACT CATTCTGCAGGGTGTGC**T**GGAAAAGGGTCCAGATGATATGTT CTAATTCTGGGTGCTCAGACAGAAGGCGTTCACAAA	622
	CCTTTTCC**A**GCACACCC	623
	GGGTGTGC**T**GGAAAAGG	624
Retinoblastoma Cys706Tyr TGT to TAT	AAAACCATGTAATAAAATTCTGACTACTTTTACATCAATTTATT TACTAGATTATGATGT**G**TTCCATGTATGGCATATGCAAAGTGA AGAATATAGACCTTAAATTCAAAATCATTGTAAC	625
	GTTACAATGATTTTGAATTTAAGGTCTATATTCTTCACTTTGCA TATGCCATACATGGAA**C**ACATCATAATCTAGTAAATAAATTGA TGTAAAAGTAGTCAGAATTTTATTACATGGTTTT	626
	TATGATGT**G**TTCCATGT	627
	ACATGGAA**C**ACATCATA	628
Retinoblastoma Cys712Arg TGC to CGC	TTCTGACTACTTTTACATCAATTTATTTACTAGATTATGATGTG TTCCATGTATGGCATA**T**GCAAAGTGAAGAATATAGACCTTAAA TTCAAAATCATTGTAACAGCATACAAGGATCTTC	629
	GAAGATCCTTGTATGCTGTTACAATGATTTTGAATTTAAGGTC TATATTCTTCACTTTGC**A**TATGCCATACATGGAACACATCATA ATCTAGTAAATAAATTGATGTAAAAGTAGTCAGAA	630
	ATGGCATA**T**GCAAAGTG	631
	CACTTTGC**A**TATGCCAT	632

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Tyr728Term TAC to TAA	GTATGGCATATGCAAAGTGAAGAATATAGACCTTAAATTCAAA ATCATTGTAACAGCATA**C**AAGGATCTTCCTCATGCTGTTCAG GAGGTAGGTAATTTTCCATAGTAAGTTTTTTTGATA	633
	TATCAAAAAAACTTACTATGGAAAATTACCTACCTCCTGAACA GCATGAGGAAGATCCTT**G**TATGCTGTTACAATGATTTTGAATT TAAGGTCTATATTCTTCACTTTGCATATGCCATAC	634
	ACAGCATA**C**AAGGATCT	635
	AGATCCTT**G**TATGCTGT	636
Retinoblastoma Glu748Term GAG to TAG	TTTTTTTTTTTTTTTTACTGT**C**TCTTCCTCAGACATTCAAACGTGT TTTGATCAAAGAAGAG**G**AGTATGATTCTATTATAGTATTCTAT AACTCGGTCTTCATGCAGAGACTGAAAACAAATA	637
	TATTTGTTTTCAGTCTCTGCATGAAGACCGAGTTATAGAATAC TATAATAGAATCATACT**C**CTCTTCTTTGATCAAAACACGTTTG AATGTCTGAGGAAGAACAGTAAAAAAAAAAAAAAA	638
	AAGAAGAG**G**AGTATGAT	639
	ATCATACT**C**CTCTTCTT	640
Retinoblastoma Gln762Term CAG to TAG	GTTTTGATCAAAGAAGAGGAGTATGATTCTATTATAGTATTCT ATAACTCGGTCTTCATG**C**AGAGACTGAAAACAAATATTTTGCA GTATGCTTCCACCAGGGTAGGTCAAAAGTATCCTT	641
	AAGGATACTTTTGACCTACCCTGGTGGAAGCATACTGCAAAA TATTTGTTTTCAGTCTCT**G**CATGAAGACCGAGTTATAGAATAC TATAATAGAATCATACTCCTCTTCTTTGATCAAAAC	642
	TCTTCATG**C**AGAGACTG	643
	CAGTCTCT**G**CATGAAGA	644
Retinoblastoma Arg787Term CGA-TGA	TAATCTACTTTTTTGTTTTTGCTCTAGCCCCCTACCTTGTCAC CAATACCTCACATTCCT**C**GAAGCCCTTACAAGTTTCCTAGTTC ACCCTTACGGATTCCTGGAGGGAACATCTATATTT	645
	AAATATAGATGTTCCCTCCAGGAATCCGTAAGGGTGAACTAG GAAACTTGTAAGGGCTTC**G**AGGAATGTGAGGTATTGGTGACA AGGTAGGGGGCTAGAGCAAAAACAAAAAAGTAGATTA	646
	ACATTCCT**C**GAAGCCCT	647
	AGGGCTTC**G**AGGAATGT	648

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Retinoblastoma Ser816Term TCA to TGA	CCTTACGGATTCCTGGAGGGAACATCTATATTTCACCCCTGA AGAGTCCATATAAAATTTCAGAAGGTCTGCCAACACCAACAA AAATGACTCCAAGATCAAGGTGTGTGTTTTCTCTTTA	649
	TAAAGAGAAAACACACACCTTGATCTTGGAGTCATTTTTGTTG GTGTTGGCAGACCTTCTGAAATTTTATATGGACTCTTCAGGG GTGAAATATAGATGTTCCCTCCAGGAATCCGTAAGG	650
	TAAAATTTCAGAAGGTC	651
	GACCTTCTGAAATTTTA	652

EXAMPLE 8

BRCA1 and BRCA2

Breast cancer is the second major cause of cancer death in American women, with an estimated 44,190 lives lost (290 men and 43,900 women) in the US in 1997. While ovarian cancer accounts for fewer deaths than breast cancer, it still represents 4% of all female cancers. In 1994, two breast cancer susceptibility genes were identified: BRCA1 on chromosome 17 and BRCA2 on chromosome 13. When a woman carries a mutation in either BRCA1 or BRCA2, she is at increased risk of being diagnosed with breast or ovarian cancer at some point in her life.

Ford *et al., Am. J. Hum. Genet.* 62: 676-689 (1998) assessed the contribution of BRCA1 and BRCA2 to inherited breast cancer by linkage and mutation analysis in 237 families, each with at least 4 cases of breast cancer. Families were included without regard to the occurrence of ovarian or other cancers. Overall, disease was linked to BRCA1 in an estimated 52% of families, to BRCA2 in 32% of families, and to neither gene in 16%, suggesting other predisposition genes. The majority (81%) of the breast-ovarian cancer families were due to BRCA1, with most others (14%) due to BRCA2. Conversely, the majority (76%) of families with both male and female breast cancer were due to

BRCA2. The largest proportion (67%) of families due to other genes were families with 4 or 5 cases of female breast cancer only.

More than 75% of the reported mutations in the BRCA1 gene result in truncated proteins. Couch *et al., Hum. Mutat.* 8: 8-18, 1996. (1996) reported a total of 254 BRCA1 mutations, 132 (52%) of which were unique. A total of 221 (87%) of all mutations or 107 (81%) of the unique mutations are small deletions, insertions, nonsense point mutations, splice variants, and regulatory mutations that result in truncation or

absence of the BRCA1 protein. A total of 11 disease-associated missense mutations (5 unique) and 21 variants (19 unique) as yet unclassified as missense mutations or polymorphisms had been detected. Thirty-five independent benign polymorphisms had been described. The most common mutations were 185delAG and 5382insC, which accounted for 30 (11.7%) and 26 (10.1%), respectively, of all the mutations.

Most BRCA2 mutations are predicted to result in a truncated protein product. The smallest known cancer-associated deletion removes from the C terminus only 224 of the 3,418 residues constituting BRCA2, suggesting that these terminal amino acids are critical for BRCA2 function. Studies (Spain *et al.*, Proc. Natl. Acad. Sci. 96:13920-13925 (1999)) suggest that such truncations eliminate or interfere with 2 nuclear localization signals that reside within the final 156 residues of BRCA2, suggesting that the vast majority of BRCA2 mutants are nonfunctional because they are not translocated into the nucleus.

The attached table discloses the correcting oligonucleotide base sequences for the BRACA1 and BRACA2 oligonucleotides of the invention.

Table 14

BRCA1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Met-1-Ile ATG to ATT	CTGCGCTCAGGAGGCCTTCACCCTCTGCTCTGGGTAAAGTT CATTGGAACAGAAAGAAATGGATTTATCTGCTCTTCGCGTTG AAGAAGTACAAAATGTCATTAATGCTATGCAGAAAATC	653
	GATTTTCTGCATAGCATTAATGACATTTTGTACTTCTTCAACG CGAAGAGCAGATAAATCCATTTCTTTCTGTTCCAATGAACTTT ACCCAGAGCAGAGGGTGAAGGCCTCCTGAGCGCAG	654
	AAAGAAATGGATTTATC	655
	GATAAATCCATTTCTTT	656
Breast Cancer Val-11-Ala GTA to GCA	CTGGGTAAAGTTCATTGGAACAGAAAGAAATGGATTTATCTG CTCTTCGCGTTGAAGAAGTACAAAATGTCATTAATGCTATGCA GAAAATCTTAGAGTGTCCCATCTGTCTGGAGTTGAT	657
	ATCAACTCCAGACAGATGGGACACTCTAAGATTTTCTGCATA GCATTAATGACATTTTGTACTTCTTCAACGCGAAGAGCAGATA AATCCATTTCTTTCTGTTCCAATGAACTTTACCCAG	658
	TGAAGAAGTACAAAATG	659
	CATTTTGTACTTCTTCA	660

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Ile-21-Val ATC to GTC	ATGGATTTATCTGCTCTTCGCGTTGAAGAAGTACAAAATGTCA TTAATGCTATGCAGAAA**A**TCTTAGAGTGTCCCATCTGTCTGG AGTTGATCAAGGAACCTGTCTCCACAAAGTGTGACC	661
	GGTCACACTTTGTGGAGACAGGTTCCTTGATCAACTCCAGAC AGATGGGACACTCTAAGA**T**TTTCTGCATAGCATTAATGACATT TTGTACTTCTTCAACGCGAAGAGCAGATAAATCCAT	662
	TGCAGAAA**A**TCTTAGAG	663
	CTCTAAGA**T**TTTCTGCA	664
Breast Cancer Leu-22-Ser TTA to TCA	ATTTATCTGCTCTTCGCGTTGAAGAAGTACAAAATGTCATTAA TGCTATGCAGAAAATCT**T**AGAGTGTCCCATCTGTCTGGAGTT GATCAAGGAACCTGTCTCCACAAAGTGTGACCACAT	665
	ATGTGGTCACACTTTGTGGAGACAGGTTCCTTGATCAACTCC AGACAGATGGGACACTCT**A**AGATTTTCTGCATAGCATTAATG ACATTTTGTACTTCTTCAACGCGAAGAGCAGATAAAT	666
	GAAAATCT**T**AGAGTGTC	667
	GACACTCT**A**AGATTTTC	668
Breast Cancer Cys-39-Tyr TGT to TAT	AGAAAATCTTAGAGTGTCCCATCTGTCTGGAGTTGATCAAGG AACCTGTCTCCACAAAGT**G**TGACCACATATTTTGCAAATTTTG CATGCTGAAACTTCTCAACCAGAAGAAAGGGCCTTC	669
	GAAGGCCCTTTCTTCTGGTTGAGAAGTTTCAGCATGCAAAAT TTGCAAAATATGTGGTCA**C**ACTTTGTGGAGACAGGTTCCTTG ATCAACTCCAGACAGATGGGACACTCTAAGATTTTCT	670
	CACAAAGT**G**TGACCACA	671
	TGTGGTCA**C**ACTTTGTG	672
Breast Cancer Cys-61-Gly TGT to GGT	CACATATTTTGCAAATTTTGCATGCTGAAACTTCTCAACCAGA AGAAAGGGCCTTCACAG**T**GTCCTTTATGTAAGAATGATATAA CCAAAAGGAGCCTACAAGAAAGTACGAGATTTAGTC	673
	GACTAAATCTCGTACTTTCTTGTAGGCTCCTTTTGGTTATATC ATTCTTACATAAAGGAC**A**CTGTGAAGGCCCTTTCTTCTGGTT GAGAAGTTTCAGCATGCAAAATTTGCAAAATATGTG	674
	CTTCACAG**T**GTCCTTTA	675
	TAAAGGAC**A**CTGTGAAG	676

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-63-Stop TTA to TAA	TTTGCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAG GGCCTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAA GGAGCCTACAAGAAAGTACGAGATTTAGTCAACTTGT	677
	ACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTTTGG TTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTTCTT CTGGTTGAGAAGTTTCAGCATGCAAAATTTGCAAA	678
	GTGTCCTTTATGTAAGA	679
	TCTTACATAAAGGACAC	680
Breast Cancer Cys-64-Arg TGT to CGT	TGCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAGGG CCTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAAGGA GCCTACAAGAAAGTACGAGATTTAGTCAACTTGTTG	681
	CAACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTTT GGTTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTTC TTCTGGTTGAGAAGTTTCAGCATGCAAAATTTGCA	682
	GTCCTTTATGTAAGAAT	683
	ATTCTTACATAAAGGAC	684
Breast Cancer Cys-64-Tyr TGT to TAT	GCAAATTTTGCATGCTGAAACTTCTCAACCAGAAGAAAGGGC CTTCACAGTGTCCTTTATGTAAGAATGATATAACCAAAAGGAG CCTACAAGAAAGTACGAGATTTAGTCAACTTGTTGA	685
	TCAACAAGTTGACTAAATCTCGTACTTTCTTGTAGGCTCCTTT TGGTTATATCATTCTTACATAAAGGACACTGTGAAGGCCCTTT CTTCTGGTTGAGAAGTTTCAGCATGCAAAATTTGC	686
	TCCTTTATGTAAGAATG	687
	CATTCTTACATAAAGGA	688
Breast Cancer Gln-74-Stop CAA to TAA	CAGAAGAAAGGGCCTTCACAGTGTCCTTTATGTAAGAATGAT ATAACCAAAAGGAGCCTACAAGAAAGTACGAGATTTAGTCAA CTTGTTGAAGAGCTATTGAAAATCATTTGTGCTTTTC	689
	GAAAAGCACAAATGATTTTCAATAGCTCTTCAACAAGTTGACT AAATCTCGTACTTTCTTGTAGGCTCCTTTTGGTTATATCATTCT TACATAAAGGACACTGTGAAGGCCCTTTCTTCTG	690
	GGAGCCTACAAGAAAGT	691
	ACTTTCTTGTAGGCTCC	692

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Tyr-105-Cys TAT to TGT	AGCTATTGAAAATCATTTGTGCTTTTCAGCTTGACACAGGTTT GGAGTATGCAAACAGCTATAATTTTGCAAAAAAGGAAAATAA CTCTCCTGAACATCTAAAAGATGAAGTTTCTATCAT	693
	ATGATAGAAACTTCATCTTTTAGATGTTCAGGAGAGTTATTTT CCTTTTTTGCAAAATTATAGCTGTTTGCATACTCCAAACCTGT GTCAAGCTGAAAAGCACAAATGATTTTCAATAGCT	694
	AAACAGCTATAATTTTG	695
	CAAAATTATAGCTGTTT	696
Breast Cancer Asn-158-Tyr AAC to TAC	CTACAGAGTGAACCCGAAAATCCTTCCTTGCAGGAAACCAGT CTCAGTGTCCAACTCTCTAACCTTGGAACTGTGAGAACTCTG AGGACAAAGCAGCGGATACAACCTCAAAAGACGTCTG	697
	CAGACGTCTTTTGAGGTTGTATCCGCTGCTTTGTCCTCAGAG TTCTCACAGTTCCAAGGTTAGAGAGTTGGACACTGAGACTGG TTTCCTGCAAGGAAGGATTTTCGGGTTCACTCTGTAG	698
	AACTCTCTAACCTTGGA	699
	TCCAAGGTTAGAGAGTT	700
Breast Cancer Gln-169-Stop CAG to TAG	GAAACCAGTCTCAGTGTCCAACTCTCTAACCTTGGAACTGTG AGAACTCTGAGGACAAAGCAGCGGATACAACCTCAAAAGAC GTCTGTCTACATTGAATTGGGATCTGATTCTTCTGAAG	701
	CTTCAGAAGAATCAGATCCCAATTCAATGTAGACAGACGTCT TTTGAGGTTGTATCCGCTGCTTTGTCCTCAGAGTTCTCACAG TTCCAAGGTTAGAGAGTTGGACACTGAGACTGGTTTC	702
	GGACAAAGCAGCGGATA	703
	TATCCGCTGCTTTGTCC	704
Breast Cancer Trp-353-Stop TGG to TAG	CTCCCAGCACAGAAAAAAAGGTAGATCTGAATGCTGATCCCC TGTGTGAGAGAAAAGAATGGAATAAGCAGAAACTGCCATGCT CAGAGAATCCTAGAGATACTGAAGATGTTCCTTGGAT	705
	ATCCAAGGAACATCTTCAGTATCTCTAGGATTCTCTGAGCAT GGCAGTTTCTGCTTATTCCATTCTTTTCTCTCACACAGGGGAT CAGCATTCAGATCTACCTTTTTTTCTGTGCTGGGAG	706
	AAAAGAATGGAATAAGC	707
	GCTTATTCCATTCTTTT	708

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Ile-379-Met ATT to ATG	ATGCTCAGAGAATCCTAGAGATACTGAAGATGTTCCTTGGAT AACACTAAATAGCAGCATTCAGAAAGTTAATGAGTGGTTTTCC AGAAGTGATGAACTGTTAGGTTCTGATGACTCACAT	709
	ATGTGAGTCATCAGAACCTAACAGTTCATCACTTCTGGAAAA CCACTCATTAACTTTCTGAATGCTGCTATTTAGTGTTATCCAA GGAACATCTTCAGTATCTCTAGGATTCTCTGAGCAT	710
	AGCAGCATTCAGAAAGT	711
	ACTTTCTGAATGCTGCT	712
Breast Cancer Glu-421-Gly GAA to GGA	GGGAGTCTGAATCAAATGCCAAAGTAGCTGATGTATTGGACG TTCTAAATGAGGTAGATGAATATTCTGGTTCTTCAGAGAAAAT AGACTTACTGGCCAGTGATCCTCATGAGGCTTTAAT	713
	ATTAAAGCCTCATGAGGATCACTGGCCAGTAAGTCTATTTTCT CTGAAGAACCAGAATATTCATCTACCTCATTTAGAACGTCCAA TACATCAGCTACTTTGGCATTTGATTCAGACTCCC	714
	GGTAGATGAATATTCTG	715
	CAGAATATTCATCTACC	716
Breast Cancer Phe-461-Leu TTT to CTT	ATATGTAAAAGTGAAAGAGTTCACTCCAAATCAGTAGAGAGT AATATTGAAGACAAAATATTTGGGAAAACCTATCGGAAGAAG GCAAGCCTCCCCAACTTAAGCCATGTAACTGAAAATC	717
	GATTTTCAGTTACATGGCTTAAGTTGGGGAGGCTTGCCTTCT TCCGATAGGTTTTCCCAAATATTTTGTCTTCAATATTACTCTCT ACTGATTTGGAGTGAACTCTTTCACTTTTACATAT	718
	ACAAAATATTTGGGAAA	719
	TTTCCCAAATATTTTGT	720
Breast Cancer Tyr-465-Leu TAT to GAT	GAAAGAGTTCACTCCAAATCAGTAGAGAGTAATATTGAAGAC AAAATATTTGGGAAAACCTATCGGAAGAAGGCAAGCCTCCCC AACTTAAGCCATGTAACTGAAAATCTAATTATAGGAG	721
	CTCCTATAATTAGATTTTCAGTTACATGGCTTAAGTTGGGGAG GCTTGCCTTCTTCCGATAGGTTTTCCCAAATATTTTGTCTTCA ATATTACTCTCTACTGATTTGGAGTGAACTCTTTC	722
	GGAAAACCTATCGGAAG	723
	CTTCCGATAGGTTTTCC	724

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gly-484-Stop GGA to TGA	ACCTATCGGAAGAAGGCAAGCCTCCCCAACTTAAGCCATGTA ACTGAAAATCTAATTATA**G**GAGCATTTGTTACTGAGCCACAGA TAATACAAGAGCGTCCCCTCACAAATAAATTAAAGC	725
	GCTTTAATTTATTTGTGAGGGGACGCTCTTGTATTATCTGTGG CTCAGTAACAAATGCTC**C**TATAATTAGATTTTCAGTTACATGG CTTAAGTTGGGGAGGCTTGCCTTCTTCCGATAGGT	726
	TAATTATA**G**GAGCATTT	727
	AAATGCTC**C**TATAATTA	728
Breast Cancer Arg-507-Ile AGA to ATA	TTACTGAGCCACAGATAATACAAGAGCGTCCCCTCACAAATA AATTAAAGCGTAAAAGGA**G**ACCTACATCAGGCCTTCATCCTG AGGATTTTATCAAGAAAGCAGATTTGGCAGTTCAAAA	729
	TTTTGAACTGCCAAATCTGCTTTCTTGATAAAATCCTCAGGAT GAAGGCCTGATGTAGGT**C**TCCTTTTACGCTTTAATTTATTTGT GAGGGGACGCTCTTGTATTATCTGTGGCTCAGTAA	730
	TAAAAGGA**G**ACCTACAT	731
	ATGTAGGT**C**TCCTTTTA	732
Breast Cancer Ser-510-Stop TCA to TGA	CACAGATAATACAAGAGCGTCCCCTCACAAATAAATTAAAGC GTAAAAGGAGACCTACAT**C**AGGCCTTCATCCTGAGGATTTTA TCAAGAAAGCAGATTTGGCAGTTCAAAAGACTCCTGA	733
	TCAGGAGTCTTTTGAACTGCCAAATCTGCTTTCTTGATAAAAT CCTCAGGATGAAGGCCT**G**ATGTAGGTCTCCTTTTACGCTTTA ATTTATTTGTGAGGGGACGCTCTTGTATTATCTGTG	734
	ACCTACAT**C**AGGCCTTC	735
	GAAGGCCT**G**ATGTAGGT	736
Breast Cancer Gln-526-Stop CAA to TAA	AGGAGACCTACATCAGGCCTTCATCCTGAGGATTTTATCAAG AAAGCAGATTTGGCAGTT**C**AAAAGACTCCTGAAATGATAAAT CAGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA	737
	TCACTTGACCATTCTGCTCCGTTTGGTTAGTTCCCTGATTTAT CATTTCAGGAGTCTTTT**G**AACTGCCAAATCTGCTTTCTTGATA AAATCCTCAGGATGAAGGCCTGATGTAGGTCTCCT	738
	TGGCAGTT**C**AAAAGACT	739
	AGTCTTTT**G**AACTGCCA	740

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-541-Stop CAG to TAG	AGGAGACCTACATCAGGCCTTCATCCTGAGGATTTTATCAAG AAAGCAGATTTGGCAGTTCAAAAGACTCCTGAAATGATAAAT CAGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA	741
	TCACTTGACCATTCTGCTCCGTTTGGTTAGTTCCCTGATTTAT CATTTCAGGAGTCTTTTGAACTGCCAAATCTGCTTTCTTGATA AAATCCTCAGGATGAAGGCCTGATGTAGGTCTCCT	742
	AAACGGAGCAGAATGGT	743
	ACCATTCTGCTCCGTTT	744
Breast Cancer Gly-552-Val GGT to GTT	TAAATCAGGGAACTAACCAAACGGAGCAGAATGGTCAAGTGA TGAATATTACTAATAGTGGTCATGAGAATAAAACAAAAGGTGA TTCTATTCAGAATGAGAAAAATCCTAACCCAATAGA	745
	TCTATTGGGTTAGGATTTTTCTCATTCTGAATAGAATCACCTT TTGTTTTATTCTCATGACCACTATTAGTAATATTCATCACTTGA CCATTCTGCTCCGTTTGGTTAGTTCCCTGATTTA	746
	TAATAGTGGTCATGAGA	747
	TCTCATGACCACTATTA	748
Breast Cancer Gln-563-Stop CAG to TAG	GGTCAAGTGATGAATATTACTAATAGTGGTCATGAGAATAAAA CAAAAGGTGATTCTATTCAGAATGAGAAAAATCCTAACCCAAT AGAATCACTCGAAAAAGAATCTGCTTTCAAAACGA	749
	TCGTTTTGAAAGCAGATTCTTTTTCGAGTGATTCTATTGGGTT AGGATTTTTCTCATTCTGAATAGAATCACCTTTTGTTTTATTCT CATGACCACTATTAGTAATATTCATCACTTGACC	750
	ATTCTATTCAGAATGAG	751
	CTCATTCTGAATAGAAT	752
Ovarian Cancer Lys-607-Stop AAA to TAA	ATAAGCAGCAGTATAAGCAATATGGAACTCGAATTAAATATCC ACAATTCAAAAGCACCTAAAAAAGAATAGGCTGAGGAGGAAGT CTTCTACCAGGCATATTCATGCGCTTGAACTAGTAG	753
	CTACTAGTTCAAGCGCATGAATATGCCTGGTAGAAGACTTCC TCCTCAGCCTATTCTTTTTAGGTGCTTTTGAATTGTGGATATT TAATTCGAGTTCCATATTGCTTATACTGCTGCTTAT	754
	AAGCACCTAAAAAGAAT	755
	ATTCTTTTTAGGTGCTT	756

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-639-Stop TTG to TAG	ATATTCATGCGCTTGAACTAGTAGTCAGTAGAAATCTAAGCC CACCTAATTGTACTGAATTGCAAATTGATAGTTGTTCTAGCAG TGAAGAGATAAAGAAAAAAAAGTACAACCAAATGCC	757
	GGCATTTGGTTGTACTTTTTTTTCTTTATCTCTTCACTGCTAGA ACAACTATCAATTTGCAATTCAGTACAATTAGGTGGGCTTAGA TTTCTACTGACTACTAGTTCAAGCGCATGAATAT	758
	TACTGAATTGCAAATTG	759
	CAATTTGCAATTCAGTA	760
Breast Cancer Asp-693-Asn GAC to AAC	GAACCTGCAACTGGAGCCAAGAAGAGTAACAAGCCAAATGA ACAGACAAGTAAAAGACATGACAGCGATACTTTCCCAGAGCT GAAGTTAACAAATGCACCTGGTTCTTTTACTAAGTGTT	761
	AACACTTAGTAAAAGAACCAGGTGCATTTGTTAACTTCAGCTC TGGGAAAGTATCGCTGTCATGTCTTTTACTTGTCTGTTCATTT GGCTTGTTACTCTTCTTGGCTCCAGTTGCAGGTTC	762
	AAAGACATGACAGCGAT	763
	ATCGCTGTCATGTCTTT	764
Ovarian Cancer Glu-720-Stop GAA to TAA	CTGAAGTTAACAAATGCACCTGGTTCTTTTACTAAGTGTTCAA ATACCAGTGAACTTAAAGAATTTGTCAATCCTAGCCTTCCAAG AGAAGAAAAAGAAGAGAAACTAGAAACAGTTAAAG	765
	CTTTAACTGTTTCTAGTTTCTCTTCTTTTTCTTCTCTTGGAAGG CTAGGATTGACAAATTCTTTAAGTTCACTGGTATTTGAACACT TAGTAAAAGAACCAGGTGCATTTGTTAACTTCAG	766
	AACTTAAAGAATTTGTC	767
	GACAAATTCTTTAAGTT	768
Breast Cancer Glu-755-Stop GAA to TAA	CTAGAAACAGTTAAAGTGTCTAATAATGCTGAAGACCCCAAA GATCTCATGTTAAGTGGAGAAAGGGTTTTGCAAACTGAAAGA TCTGTAGAGAGTAGCAGTATTTCATTGGTACCTGGTA	769
	TACCAGGTACCAATGAAATACTGCTACTCTCTACAGATCTTTC AGTTTGCAAAACCCTTTCTCCACTTAACATGAGATCTTTGGGG TCTTCAGCATTATTAGACACTTTAACTGTTTCTAG	770
	TAAGTGGAGAAAGGGTT	771
	AACCCTTTCTCCACTTA	772

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Ser-770-Stop TCA to TAA	TCATGTTAAGTGGAGAAAGGGTTTTGCAAACTGAAAGATCTG TAGAGAGTAGCAGTATTTCATTGGTACCTGGTACTGATTATG GCACTCAGGAAAGTATCTCGTTACTGGAAGTTAGCAC	773
	GTGCTAACTTCCAGTAACGAGATACTTTCCTGAGTGCCATAA TCAGTACCAGGTACCAATGAAATACTGCTACTCTCTACAGAT CTTTCAGTTTGCAAAACCCTTTCTCCACTTAACATGA	774
	CAGTATTTCATTGGTAC	775
	GTACCAATGAAATACTG	776
Breast Cancer Val-772-Ala GTA to GCA	TAAGTGGAGAAAGGGTTTTGCAAACTGAAAGATCTGTAGAGA GTAGCAGTATTTCATTGGTACCTGGTACTGATTATGGCACTC AGGAAAGTATCTCGTTACTGGAAGTTAGCACTCTAGG	777
	CCTAGAGTGCTAACTTCCAGTAACGAGATACTTTCCTGAGTG CCATAATCAGTACCAGGTACCAATGAAATACTGCTACTCTCTA CAGATCTTTCAGTTTGCAAAACCCTTTCTCCACTTA	778
	TTCATTGGTACCTGGTA	779
	TACCAGGTACCAATGAA	780
Breast Cancer Gln-780-Stop CAG to TAG	ACTGAAAGATCTGTAGAGAGTAGCAGTATTTCATTGGTACCT GGTACTGATTATGGCACTCAGGAAAGTATCTCGTTACTGGAA GTTAGCACTCTAGGGAAGGCAAAAACAGAACCAAATA	781
	TATTTGGTTCTGTTTTTGCCTTCCCTAGAGTGCTAACTTCCAG TAACGAGATACTTTCCTGAGTGCCATAATCAGTACCAGGTAC CAATGAAATACTGCTACTCTCTACAGATCTTTCAGT	782
	ATGGCACTCAGGAAAGT	783
	ACTTTCCTGAGTGCCAT	784
Breast Cancer Glu-797-Stop GAA to TAA	TATGGCACTCAGGAAAGTATCTCGTTACTGGAAGTTAGCACT CTAGGGAAGGCAAAAACAGAACCAAATAAATGTGTGAGTCAG TGTGCAGCATTTGAAAACCCCAAGGGACTAATTCATG	785
	CATGAATTAGTCCCTTGGGGTTTTCAAATGCTGCACACTGAC TCACACATTTATTTGGTTCTGTTTTTGCCTTCCCTAGAGTGCT AACTTCCAGTAACGAGATACTTTCCTGAGTGCCATA	786
	CAAAAACAGAACCAAAT	787
	ATTTGGTTCTGTTTTTG	788

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Lys-820-Glu AAA to GAA	AAATGTGTGAGTCAGTGTGCAGCATTTGAAAACCCCAAGGGA CTAATTCATGGTTGTTCC**A**AAGATAATAGAAATGACACAGAA GGCTTTAAGTATCCATTGGGACATGAAGTTAACCACA	789
	TGTGGTTAACTTCATGTCCCAATGGATACTTAAAGCCTTCTGT GTCATTTCTATTATCTT**T**GGAACAACCATGAATTAGTCCCTTG GGGTTTTCAAATGCTGCACACTGACTCACACATTT	790
	GTTGTTCC**A**AAGATAAT	791
	ATTATCTT**T**GGAACAAC	792
Breast Cancer Thr-826-Lys ACA to AAA	CAGCATTTGAAAACCCCAAGGGACTAATTCATGGTTGTTCCA AAGATAATAGAAATGACA**C**AGAAGGCTTTAAGTATCCATTGG GACATGAAGTTAACCACAGTCGGGAAACAAGCATAGA	793
	TCTATGCTTGTTTCCCGACTGTGGTTAACTTCATGTCCCAATG GATACTTAAAGCCTTCT**G**TGTCATTTCTATTATCTTTGGAACA ACCATGAATTAGTCCCTTGGGGTTTTCAAATGCTG	794
	AAATGACA**C**AGAAGGCT	795
	AGCCTTCT**G**TGTCATTT	796
Breast Cancer Arg-841-Trp CGG to TGG	GATAATAGAAATGACACAGAAGGCTTTAAGTATCCATTGGGA CATGAAGTTAACCACAGT**C**GGGAAACAAGCATAGAAATGGAA GAAAGTGAACTTGATGCTCAGTATTTGCAGAATACAT	797
	ATGTATTCTGCAAATACTGAGCATCAAGTTCACTTTCTTCCAT TTCTATGCTTGTTTCCC**G**ACTGTGGTTAACTTCATGTCCCAAT GGATACTTAAAGCCTTCTGTGTCATTTCTATTATC	798
	ACCACAGT**C**GGGAAACA	799
	TGTTTCCC**G**ACTGTGGT	800
Breast Cancer Pro-871-Leu CCG to CTG	AACTTGATGCTCAGTATTTGCAGAATACATTCAAGGTTTCAAA GCGCCAGTCATTTGCTC**C**GTTTTCAAATCCAGGAAATGCAGA AGAGGAATGTGCAACATTCTCTGCCCACTCTGGGTC	801
	GACCCAGAGTGGGCAGAGAATGTTGCACATTCCTCTTCTGCA TTTCCTGGATTTGAAAAC**G**GAGCAAATGACTGGCGCTTTGAA ACCTTGAATGTATTCTGCAAATACTGAGCATCAAGTT	802
	ATTTGCTC**C**GTTTTCAA	803
	TTGAAAAC**G**GAGCAAAT	804

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-892-Ser TTA to TCA	TTTCAAATCCAGGAAATGCAGAAGAGGAATGTGCAACATTCT CTGCCCACTCTGGGTCCTTAAAGAAACAAAGTCCAAAAGTCA CTTTTGAATGTGAACAAAAGGAAGAAAATCAAGGAAA	805
	TTTCCTTGATTTTCTTCCTTTTGTTCACATTCAAAAGTGACTTT TGGACTTTGTTTCTTTAAGGACCCAGAGTGGGCAGAGAATGT TGCACATTCCTCTTCTGCATTTCCTGGATTTGAAA	806
	TGGGTCCTTAAAGAAAC	807
	GTTTCTTTAAGGACCCA	808
Breast Cancer Glu-908-Stop GAA to TAA	CACTCTGGGTCCTTAAAGAAACAAAGTCCAAAAGTCACTTTT GAATGTGAACAAAAGGAAGAAAATCAAGGAAAGAATGAGTCT AATATCAAGCCTGTACAGACAGTTAATATCACTGCAG	809
	CTGCAGTGATATTAACTGTCTGTACAGGCTTGATATTAGACTC ATTCTTTCCTTGATTTTCTTCCTTTTGTTCACATTCAAAAGTGA CTTTTGGACTTTGTTTCTTTAAGGACCCAGAGTG	810
	AAAAGGAAGAAAATCAA	811
	TTGATTTTCTTCCTTTT	812
Breast Cancer Gly-960-Asp GGC to GAC	ATAATGCCAAATGTAGTATCAAAGGAGGCTCTAGGTTTTGTC TATCATCTCAGTTCAGAGGCAACGAAACTGGACTCATTACTC CAAATAAACATGGACTTTTACAAAACCCATATCGTAT	813
	ATACGATATGGGTTTTGTAAAAGTCCATGTTTATTTGGAGTAA TGAGTCCAGTTTCGTTGCCTCTGAACTGAGATGATAGACAAA ACCTAGAGCCTCCTTTGATACTACATTTGGCATTAT	814
	GTTCAGAGGCAACGAAA	815
	TTTCGTTGCCTCTGAAC	816
Breast Cancer Met-1008-Ile ATG to ATA	ATTTGTTAAAACTAAATGTAAGAAAAATCTGCTAGAGGAAAAC TTTGAGGAACATTCAATGTCACCTGAAAGAGAAATGGGAAAT GAGAACATTCCAAGTACAGTGAGCACAATTAGCCGT	817
	ACGGCTAATTGTGCTCACTGTACTTGGAATGTTCTCATTTCCC ATTTCTCTTTCAGGTGACATTGAATGTTCCTCAAAGTTTTCCT CTAGCAGATTTTTCTTACATTTAGTTTTAACAAAT	818
	CATTCAATGTCACCTGA	819
	TCAGGTGACATTGAATG	820

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Thr-1025-Ile ACA to ATA	ACTTTGAGGAACATTCAATGTCACCTGAAAGAGAAATGGGAA ATGAGAACATTCCAAGTA**C**AGTGAGCACAATTAGCCGTAATA ACATTAGAGAAAATGTTTTTAAAGAAGCCAGCTCAAG	821
	CTTGAGCTGGCTTCTTTAAAAACATTTTCTCTAATGTTATTAC GGCTAATTGTGCTCACT**G**TACTTGGAATGTTCTCATTTCCCAT TTCTCTTTCAGGTGACATTGAATGTTCCTCAAAGT	822
	TCCAAGTA**C**AGTGAGCA	823
	TGCTCACT**G**TACTTGGA	824
Breast Cancer Glu-1038-Gly GAA to GGA	ACATTCCAAGTACAGTGAGCACAATTAGCCGTAATAACATTA GAGAAAATGTTTTTAAAG**A**AGCCAGCTCAAGCAATATTAATGA AGTAGGTTCCAGTACTAATGAAGTGGGCTCCAGTAT	825
	ATACTGGAGCCCACTTCATTAGTACTGGAACCTACTTCATTAA TATTGCTTGAGCTGGCT**T**CTTTAAAAACATTTTCTCTAATGTTA TTACGGCTAATTGTGCTCACTGTACTTGGAATGT	826
	TTTTAAAG**A**AGCCAGCT	827
	AGCTGGCT**T**CTTTAAAA	828
Breast Cancer Ser-1040-Asn AGC to AAC	CAAGTACAGTGAGCACAATTAGCCGTAATAACATTAGAGAAA ATGTTTTTAAAGAAGCCA**G**CTCAAGCAATATTAATGAAGTAGG TTCCAGTACTAATGAAGTGGGCTCCAGTATTAATGA	829
	TCATTAATACTGGAGCCCACTTCATTAGTACTGGAACCTACTT CATTAATATTGCTTGAG**C**TGGCTTCTTTAAAAACATTTTCTCTA ATGTTATTACGGCTAATTGTGCTCACTGTACTTG	830
	AGAAGCCA**G**CTCAAGCA	831
	TGCTTGAG**C**TGGCTTCT	832
Breast Cancer Val-1047-Ala GTA to GCA	GCCGTAATAACATTAGAGAAAATGTTTTTAAAGAAGCCAGCT CAAGCAATATTAATGAAG**T**AGGTTCCAGTACTAATGAAGTGG GCTCCAGTATTAATGAAATAGGTTCCAGTGATGAAAA	833
	TTTTCATCACTGGAACCTATTTCATTAATACTGGAGCCCACTT CATTAGTACTGGAACCT**A**CTTCATTAATATTGCTTGAGCTGGC TTCTTTAAAAACATTTTCTCTAATGTTATTACGGC	834
	TAATGAAG**T**AGGTTCCA	835
	TGGAACCT**A**CTTCATTA	836

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-1080-Stop TTG to TAG	AAATAGGTTCCAGTGATGAAAACATTCAAGCAGAACTAGGTA GAAACAGAGGGCCAAAATTGAATGCTATGCTTAGATTAGGGG TTTTGCAACCTGAGGTCTATAAACAAAGTCTTCCTGG	837
	CCAGGAAGACTTTGTTTATAGACCTCAGGTTGCAAAACCCCT AATCTAAGCATAGCATTCAATTTTGGCCCTCTGTTTCTACCTA GTTCTGCTTGAATGTTTTCATCACTGGAACCTATTT	838
	GCCAAAATTGAATGCTA	839
	TAGCATTCAATTTTGGC	840
Breast Cancer Leu-1086-Stop TTA to TGA	AAAACATTCAAGCAGAACTAGGTAGAAACAGAGGGCCAAAAT TGAATGCTATGCTTAGATTAGGGGTTTTGCAACCTGAGGTCT ATAAACAAAGTCTTCCTGGAAGTAATTGTAAGCATCC	841
	GGATGCTTACAATTACTTCCAGGAAGACTTTGTTTATAGACCT CAGGTTGCAAAACCCCTAATCTAAGCATAGCATTCAATTTTG GCCCTCTGTTTCTACCTAGTTCTGCTTGAATGTTTT	842
	GCTTAGATTAGGGGTTT	843
	AAACCCCTAATCTAAGC	844
Breast Cancer Ser-1130-Stop TCA to TGA	AGCAAGAATATGAAGAAGTAGTTCAGACTGTTAATACAGATTT CTCTCCATATCTGATTTCAGATAACTTAGAACAGCCTATGGGA AGTAGTCATGCATCTCAGGTTTGTTCTGAGACACC	845
	GGTGTCTCAGAACAAACCTGAGATGCATGACTACTTCCCATA GGCTGTTCTAAGTTATCTGAAATCAGATATGGAGAGAAATCT GTATTAACAGTCTGAACTACTTCTTCATATTCTTGCT	846
	TCTGATTTCAGATAACT	847
	AGTTATCTGAAATCAGA	848
Breast Cancer Lys-1183-Arg AAA to AGA	CTAGTTTTGCTGAAAATGACATTAAGGAAAGTTCTGCTGTTTT TAGCAAAAGCGTCCAGAAAGGAGAGCTTAGCAGGAGTCCTA GCCCTTTCACCCATACACATTTGGCTCAGGGTTACCG	849
	CGGTAACCCTGAGCCAAATGTGTATGGGTGAAAGGGCTAGG ACTCCTGCTAAGCTCTCCTTTCTGGACGCTTTTGCTAAAAACA GCAGAACTTTCCTTAATGTCATTTTCAGCAAAACTAG	850
	CGTCCAGAAAGGAGAGC	851
	GCTCTCCTTTCTGGACG	852

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1200-Stop CAG to TAG	AGCGTCCAGAAAGGAGAGCTTAGCAGGAGTCCTAGCCCTTT CACCCATACACATTTGGCT**C**AGGGTTACCGAAGAGGGGCCA AGAAATTAGAGTCCTCAGAAGAGAACTTATCTAGTGAGG	853
	CCTCACTAGATAAGTTCTCTTCTGAGGACTCTAATTTCTTGGC CCCTCTTCGGTAACCCT**G**AGCCAAATGTGTATGGGTGAAAGG GCTAGGACTCCTGCTAAGCTCTCCTTTCTGGACGCT	854
	ATTTGGCT**C**AGGGTTAC	855
	GTAACCCT**G**AGCCAAAT	856
Breast Cancer Arg-1203-Stop CGA to TGA	AAAGGAGAGCTTAGCAGGAGTCCTAGCCCTTTCACCCATACA CATTTGGCTCAGGGTTAC**C**GAAGAGGGGCCAAGAAATTAGA GTCCTCAGAAGAGAACTTATCTAGTGAGGATGAAGAGC	857
	GCTCTTCATCCTCACTAGATAAGTTCTCTTCTGAGGACTCTAA TTTCTTGGCCCCTCTTC**G**GTAACCCTGAGCCAAATGTGTATG GGTGAAAGGGCTAGGACTCCTGCTAAGCTCTCCTTT	858
	AGGGTTAC**C**GAAGAGGG	859
	CCCTCTTC**G**GTAACCCT	860
Breast Cancer Glu-1214-Stop GAG to TAG	ACCCATACACATTTGGCTCAGGGTTACCGAAGAGGGGCCAA GAAATTAGAGTCCTCAGAA**G**AGAACTTATCTAGTGAGGATGA AGAGCTTCCCTGCTTCCAACACTTGTTATTTGGTAAAG	861
	CTTTACCAAATAACAAGTGTTGGAAGCAGGGAAGCTCTTCAT CCTCACTAGATAAGTTCT**C**TTCTGAGGACTCTAATTTCTTGGC CCCTCTTCGGTAACCCTGAGCCAAATGTGTATGGGT	862
	CCTCAGAA**G**AGAACTTA	863
	TAAGTTCT**C**TTCTGAGG	864
Breast Cancer Glu-1219-Asp GAG to GAC	TCAGGGTTACCGAAGAGGGGCCAAGAAATTAGAGTCCTCAG AAGAGAACTTATCTAGTGA**G**GATGAAGAGCTTCCCTGCTTCC AACACTTGTTATTTGGTAAAGTAAACAATATACCTTCT	865
	AGAAGGTATATTGTTTACTTTACCAAATAACAAGTGTTGGAAG CAGGGAAGCTCTTCATC**C**TCACTAGATAAGTTCTCTTCTGAG GACTCTAATTTCTTGGCCCCTCTTCGGTAACCCTGA	866
	TCTAGTGA**G**GATGAAGA	867
	TCTTCATC**C**TCACTAGA	868

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Glu-1221-Stop GAA to TAA	GGTTACCGAAGAGGGGCCAAGAAATTAGAGTCCTCAGAAGA GAACTTATCTAGTGAGGATGAAGAGCTTCCCTGCTTCCAACA CTTGTTATTTGGTAAAGTAAACAATATACCTTCTCAGT	869
	ACTGAGAAGGTATATTGTTTACTTTACCAAATAACAAGTGTTG GAAGCAGGGAAGCTCTTCATCCTCACTAGATAAGTTCTCTTC TGAGGACTCTAATTTCTTGGCCCCTCTTCGGTAACC	870
	GTGAGGATGAAGAGCTT	871
	AAGCTCTTCATCCTCAC	872
Breast Cancer Glu-1250-Stop GAG to TAG	TTATTTGGTAAAGTAAACAATATACCTTCTCAGTCTACTAGGC ATAGCACCGTTGCTACCGAGTGTCTGTCTAAGAACACAGAGG AGAATTTATTATCATTGAAGAATAGCTTAAATGACT	873
	AGTCATTTAAGCTATTCTTCAATGATAATAAATTCTCCTCTGTG TTCTTAGACAGACACTCGGTAGCAACGGTGCTATGCCTAGTA GACTGAGAAGGTATATTGTTTACTTTACCAAATAA	874
	TTGCTACCGAGTGTCTG	875
	CAGACACTCGGTAGCAA	876
Breast Cancer Ser-1262-Stop TCA to TAA	CTAGGCATAGCACCGTTGCTACCGAGTGTCTGTCTAAGAACA CAGAGGAGAATTTATTATCATTGAAGAATAGCTTAAATGACTG CAGTAACCAGGTAATATTGGCAAAGGCATCTCAGGA	877
	TCCTGAGATGCCTTTGCCAATATTACCTGGTTACTGCAGTCAT TTAAGCTATTCTTCAATGATAATAAATTCTCCTCTGTGTTCTTA GACAGACACTCGGTAGCAACGGTGCTATGCCTAG	878
	TTTATTATCATTGAAGA	879
	TCTTCAATGATAATAAA	880
Breast Cancer Gln-1281-Stop CAG to TAG	TTATCATTGAAGAATAGCTTAAATGACTGCAGTAACCAGGTAA TATTGGCAAAGGCATCTCAGGAACATCACCTTAGTGAGGAAA CAAAATGTTCTGCTAGCTTGTTTTCTTCACAGTGCA	881
	TGCACTGTGAAGAAAACAAGCTAGCAGAACATTTTGTTTCCT CACTAAGGTGATGTTCCTGAGATGCCTTTGCCAATATTACCT GGTTACTGCAGTCATTTAAGCTATTCTTCAATGATAA	882
	AGGCATCTCAGGAACAT	883
	ATGTTCCTGAGATGCCT	884

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1313-Stop CAG to TAG	GCTAGCTTGTTTTCTTCACAGTGCAGTGAATTGGAAGACTTG ACTGCAAATACAAACACC**C**AGGATCCTTTCTTGATTGGTTCTT CCAAACAAATGAGGCATCAGTCTGAAAGCCAGGGAG	885
	CTCCCTGGCTTTCAGACTGATGCCTCATTTGTTTGGAAGAAC CAATCAAGAAAGGATCCT**G**GGTGTTTGTATTTGCAGTCAAGT CTTCCAATTCACTGCACTGTGAAGAAAACAAGCTAGC	886
	CAAACACC**C**AGGATCCT	887
	AGGATCCT**G**GGTGTTTG	888
Breast Cancer Ile-1318-Val ATT to GTT	TCACAGTGCAGTGAATTGGAAGACTTGACTGCAAATACAAAC ACCCAGGATCCTTTCTTG**A**TTGGTTCTTCCAAACAAATGAGG CATCAGTCTGAAAGCCAGGGAGTTGGTCTGAGTGACA	889
	TGTCACTCAGACCAACTCCCTGGCTTTCAGACTGATGCCTCA TTTGTTTGGAAGAACCAA**T**CAAGAAAGGATCCTGGGTGTTTG TATTTGCAGTCAAGTCTTCCAATTCACTGCACTGTGA	890
	CTTTCTTG**A**TTGGTTCT	891
	AGAACCAA**T**CAAGAAAG	892
Breast Cancer Gln-1323-Stop CAA to TAA	TTGGAAGACTTGACTGCAAATACAAACACCCAGGATCCTTTC TTGATTGGTTCTTCCAAA**C**AAATGAGGCATCAGTCTGAAAGC CAGGGAGTTGGTCTGAGTGACAAGGAATTGGTTTCAG	893
	CTGAAACCAATTCCTTGTCACTCAGACCAACTCCCTGGCTTT CAGACTGATGCCTCATTT**G**TTTGGAAGAACCAATCAAGAAAG GATCCTGGGTGTTTGTATTTGCAGTCAAGTCTTCCAA	894
	CTTCCAAA**C**AAATGAGG	895
	CCTCATTT**G**TTTGGAAG	896
Breast Cancer Arg-1347-Gly AGA to GGA	CAGTCTGAAAGCCAGGGAGTTGGTCTGAGTGACAAGGAATT GGTTTCAGATGATGAAGAA**A**GAGGAACGGGCTTGGAAGAAA ATAATCAAGAAGAGCAAAGCATGGATTCAAACTTAGGTA	897
	TACCTAAGTTTGAATCCATGCTTTGCTCTTCTTGATTATTTTCT TCCAAGCCCGTTCCTC**T**TTCTTCATCATCTGAAACCAATTCCT. TGTCACTCAGACCAACTCCCTGGCTTTCAGACTG	898
	ATGAAGAA**A**GAGGAACG	899
	CGTTCCTC**T**TTCTTCAT	900

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1395-Stop CAG to TAG	GAAACAAGCGTCTCTGAAGACTGCTCAGGGCTATCCTCTCAG AGTGACATTTTAACCACTCAGGTAAAAAGCGTGTGTGTGTGT GCACATGCGTGTGTGTGGTGTCCTTTGCATTCAGTAG	901
	CTACTGAATGCAAAGGACACCACACACACGCATGTGCACACA CACACACGCTTTTTACCTGAGTGGTTAAAATGTCACTCTGAG AGGATAGCCCTGAGCAGTCTTCAGAGACGCTTGTTTC	902
	TAACCACTCAGGTAAAA	903
	TTTTACCTGAGTGGTTA	904
Breast Cancer Gln-1408-Stop CAG to TAG	TGGTGCCATTTATCGTTTTTGAAGCAGAGGGATACCATGCAA CATAACCTGATAAAGCTCCAGCAGGAAATGGCTGAACTAGAA GCTGTGTTAGAACAGCATGGGAGCCAGCCTTCTAACA	905
	TGTTAGAAGGCTGGCTCCCATGCTGTTCTAACACAGCTTCTA GTTCAGCCATTTCCTGCTGGAGCTTTATCAGGTTATGTTGCAT GGTATCCCTCTGCTTCAAAAACGATAAATGGCACCA	906
	TAAAGCTCCAGCAGGAA	907
	TTCCTGCTGGAGCTTTA	908
Breast Cancer Arg-1443-Gly CGA to GGA Arg-1443-Stop CGA to TGA	AGCCAGCCTTCTAACAGCTACCCTTCCATCATAAGTGACTCT TCTGCCCTTGAGGACCTGCGAAATCCAGAACAAAGCACATCA GAAAAAGGTGTGTATTGTTGGCCAAACACTGATATCT	909
	AGATATCAGTGTTTGGCCAACAATACACACCTTTTTCTGATGT GCTTTGTTCTGGATTTCGCAGGTCCTCAAGGGCAGAAGAGTC ACTTATGATGGAAGGGTAGCTGTTAGAAGGCTGGCT	910
	AGGACCTGCGAAATCCA	911
	TGGATTTCGCAGGTCCT	912
Breast Cancer Ser-1512-Ile AGT to ATT	CAGAATAGAAACTACCCATCTCAAGAGGAGCTCATTAAGGTT GTTGATGTGGAGGAGCAACAGCTGGAAGAGTCTGGGCCACA CGATTTGACGGAAACATCTTACTTGCCAAGGCAAGATC	913
	GATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAAATCGTGTG GCCCAGACTCTTCCAGCTGTTGCTCCTCCACATCAACAACCT TAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCTG	914
	AGGAGCAACAGCTGGAA	915
	TTCCAGCTGTTGCTCCT	916

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1538-Stop CAG to TAG	ATCTTTCTAGGTCATCCCCTTCTAAATGCCCATCATTAGATGA TAGGTGGTACATGCACAGTTGCTCTGGGAGTCTTCAGAATAG AAACTACCCATCTCAAGAGGAGCTCATTAAGGTTGT	917
	ACAACCTTAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCT GAAGACTCCCAGAGCAACTGTGCATGTACCACCTATCATCTA ATGATGGGCATTTAGAAGGGGATGACCTAGAAAGAT	918
	CATGCACAGTTGCTCTG	919
	CAGAGCAACTGTGCATG	920
Breast Cancer Glu-1541-Stop GAG to TAG	CAGAATAGAAACTACCCATCTCAAGAGGAGCTCATTAAGGTT GTTGATGTGGAGGAGCAACAGCTGGAAGAGTCTGGGCCACA CGATTTGACGGAAACATCTTACTTGCCAAGGCAAGATC	921
	GATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAAATCGTGTG GCCCAGACTCTTCCAGCTGTTGCTCCTCCACATCAACAACCT TAATGAGCTCCTCTTGAGATGGGTAGTTTCTATTCTG	922
	AGGAGCAACAGCTGGAA	923
	TTCCAGCTGTTGCTCCT	924
Breast Cancer Thr-1561-Ile ACC to ATC	AACTACCCATCTCAAGAGGAGCTCATTAAGGTTGTTGATGTG GAGGAGCAACAGCTGGAAGAGTCTGGGCCACACGATTTGAC GGAAACATCTTACTTGCCAAGGCAAGATCTAGGTAATA	925
	TATTACCTAGATCTTGCCTTGGCAAGTAAGATGTTTCCGTCAA ATCGTGTGGCCCAGACTCTTCCAGCTGTTGCTCCTCCACATC AACAACCTTAATGAGCTCCTCTTGAGATGGGTAGTT	926
	AGCTGGAAGAGTCTGGG	927
	CCCAGACTCTTCCAGCT	928
Breast Cancer Tyr-1563-Stop TAC to TAG	TTTGTAATTCAACATTCATCGTTGTGTAAATTAAACTTCTCCCA TTCCTTTCAGAGGGAACCCCTTACCTGGAATCTGGAATCAGC CTCTTCTCTGATGACCCTGAATCTGATCCTTCTGA	929
	TCAGAAGGATCAGATTCAGGGTCATCAGAGAAGAGGCTGATT CCAGATTCCAGGTAAGGGGTTCCCTCTGAAAGGAATGGGAG AAGTTTAATTTACACAACGATGAATGTTGAATTACAAA	930
	AGAGGGAACCCCTTACC	931
	GGTAAGGGGTTCCCTCT	932

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Leu-1564-Pro CTG to CCG	CAACATTCATCGTTGTGTAAATTAAACTTCTCCCATTCCTTTC AGAGGGAACCCCTTACCTGGAATCTGGAATCAGCCTCTTCTC TGATGACCCTGAATCTGATCCTTCTGAAGACAGAGC	933
	GCTCTGTCTTCAGAAGGATCAGATTCAGGGTCATCAGAGAAG AGGCTGATTCCAGATTCCAGGTAAGGGGTTCCCTCTGAAAG GAATGGGAGAAGTTTAATTTACACAACGATGAATGTTG	934
	CCCTTACCTGGAATCTG	935
	CAGATTCCAGGTAAGGG	936
Breast Cancer Gln-1604-Stop CAA to TAA	GCCCCAGAGTCAGCTGGTGTTGGCAACATACCATCTTCAACC TCTGCATTGAAAGTTCCCCAATTGAAAGTTGCAGAATCTGCC CAGAGTCCAGCTGCTGCTCATACTACTGATACTGCTG	937
	CAGCAGTATCAGTAGTATGAGCAGCAGCTGGACTCTGGGCA GATTCTGCAACTTTCAATTGGGGAACTTTCAATGCAGAGGTT GAAGATGGTATGTTGCCAACACGAGCTGACTCTGGGGC	938
	AAGTTCCCCAATTGAAA	939
	TTTCAATTGGGGAACTT	940
Breast Cancer Lys-1606-Glu AAA to GAA	GAGTCAGCTCGTGTTGGCAACATACCATCTTCAACCTCTGCA TTGAAAGTTCCCCAATTGAAAGTTGCAGAATCTGCCCAGAGT CCAGCTGCTGCTCATACTACTGATACTGCTGGGTATA	941
	TATACCCAGCAGTATCAGTAGTATGAGCAGCAGCTGGACTCT GGGCAGATTCTGCAACTTTCAATTGGGGAACTTTCAATGCAG AGGTTGAAGATGGTATGTTGCCAACACGAGCTGACTC	942
	CCCAATTGAAAGTTGCA	943
	TGCAACTTTCAATTGGG	944
Breast Cancer Met-1628-Thr ATG to ACG	CAGAATCTGCCCAGAGTCCAGCTGCTGCTCATACTACTGATA CTGCTGGGTATAATGCAATGGAAGAAAGTGTGAGCAGGGAG AAGCCAGAATTGACAGCTTCAACAGAAAGGGTCAACAA	945
	TTGTTGACCCTTTCTGTTGAAGCTGTCAATTCTGGCTTCTCCC TGCTCACACTTTCTTCCATTGCATTATACCCAGCAGTATCAGT AGTATGAGCAGCAGCTGGACTCTGGGCAGATTCTG	946
	TAATGCAATGGAAGAAA	947
	TTTCTTCCATTGCATTA	948

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Met-1628-Val ATG to GTG	GCAGAATCTGCCCAGAGTCCAGCTGCTGCTCATACTACTGAT ACTGCTGGGTATAATGCA**A**TGGAAGAAAGTGTGAGCAGGGA GAAGCCAGAATTGACAGCTTCAACAGAAAGGGTCAACA	949
	TGTTGACCCTTTCTGTTGAAGCTGTCAATTCTGGCTTCTCCCT GCTCACACTTTCTTCCA**T**TGCATTATACCCAGCAGTATCAGTA GTATGAGCAGCAGCTGGACTCTGGGCAGATTCTGC	950
	ATAATGCA**A**TGGAAGAA	951
	TTCTTCCA**T**TGCATTAT	952
Breast Cancer Pro-1637-Leu CCA to CTA	CTCATACTACTGATACTGCTGGGTATAATGCAATGGAAGAAA GTGTGAGCAGGGAGAAGC**C**AGAATTGACAGCTTCAACAGAA AGGGTCAACAAAAGAATGTCCATGGTGGTGTCTGGCCT	953
	AGGCCAGACACCACCATGGACATTCTTTTGTTGACCCTTTCT GTTGAAGCTGTCAATTCT**G**GCTTCTCCCTGCTCACACTTTCTT CCATTGCATTATACCCAGCAGTATCAGTAGTATGAG	954
	GGAGAAGC**C**AGAATTGA	955
	TCAATTCT**G**GCTTCTCC	956
Breast Cancer Met-1652-Ile ATG to ATA	GAGCAGGGAGAAGCCAGAATTGACAGCTTCAACAGAAAGGG TCAACAAAAGAATGTCCAT**G**GTGGTGTCTGGCCTGACCCCAG AAGAATTTGTGAGTGTATCCATATGTATCTCCCTAATG	957
	CATTAGGGAGATACATATGGATACACTCACAAATTCTTCTGG GGTCAGGCCAGACACCAC**C**ATGGACATTCTTTTGTTGACCCT TTCTGTTGAAGCTGTCAATTCTGGCTTCTCCCTGCTC	958
	ATGTCCAT**G**GTGGTGTC	959
	GACACCAC**C**ATGGACAT	960
Breast Cancer Glu-1694-Stop GAG to TAG	CACTTCCTGATTTTGTTTTCAACTTCTAATCCTTTGAGTGTTTT TCATTCTGCAGATGCT**G**AGTTTGTGTGTGAACGGACACTGAA ATATTTTCTAGGAATTGCGGGAGGAAAATGGGTAG	961
	CTACCCATTTTCCTCCCGCAATTCCTAGAAAATATTTCAGTGT CCGTTCACACACAAACT**C**AGCATCTGCAGAATGAAAAACACT CAAAGGATTAGAAGTTGAAAACAAAATCAGGAAGTG	962
	CAGATGCT**G**AGTTTGTG	963
	CACAAACT**C**AGCATCTG	964

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gly-1706-Glu GGA to GAA	GTGTTTTTCATTCTGCAGATGCTGAGTTTGTGTGTGAACGGA CACTGAAATATTTTCTAGGAATTGCGGGAGGAAAATGGGTAG TTAGCTATTTCTGTAAGTATAATACTATTTCTCCCCT	965
	AGGGGAGAAATAGTATTATACTTACAGAAATAGCTAACTACC CATTTTCCTCCCGCAATTCCTAGAAAATATTTCAGTGTCCGTT CACACACAAACTCAGCATCTGCAGAATGAAAAACAC	966
	TTTTCTAGGAATTGCGG	967
	CCGCAATTCCTAGAAAA	968
Breast Cancer Ala-1708-Glu GCG to GAG	TTCATTCTGCAGATGCTGAGTTTGTGTGTGAACGGACACTGA AATATTTTCTAGGAATTGCGGGAGGAAAATGGGTAGTTAGCT ATTTCTGTAAGTATAATACTATTTCTCCCCTCCTCCC	969
	GGGAGGAGGGGAGAAATAGTATTATACTTACAGAAATAGCTA ACTACCCATTTTCCTCCCGCAATTCCTAGAAAATATTTCAGTG TCCGTTCACACACAAACTCAGCATCTGCAGAATGAA	970
	AGGAATTGCGGGAGGAA	971
	TTCCTCCCGCAATTCCT	972
Breast Cancer Val-1713-Ala GTA to GCA	CTGAGTTTGTGTGTGAACGGACACTGAAATATTTTCTAGGAA TTGCGGGAGGAAAATGGGTAGTTAGCTATTTCTGTAAGTATA ATACTATTTCTCCCCTCCTCCCTTTAACACCTCAGAA	973
	TTCTGAGGTGTTAAAGGGAGGAGGGGAGAAATAGTATTATAC TTACAGAAATAGCTAACTACCCATTTTCCTCCCGCAATTCCTA GAAAATATTTCAGTGTCCGTTCACACACAAACTCAG	974
	AAAATGGGTAGTTAGCT	975
	AGCTAACTACCCATTTT	976
Breast Cancer Trp-1718-Stop TGG to TAG	AACGGACACTGAAATATTTTCTAGGAATTGCGGGAGGAAAAT GGGTAGTTAGCTATTTCTGTAAGTATAATACTATTTCTCCCCT CCTCCCTTTAACACCTCAGAATTGCATTTTTACACC	977
	GGTGTAAAAATGCAATTCTGAGGTGTTAAAGGGAGGAGGGG AGAAATAGTATTATACTTACAGAAATAGCTAACTACCCATTTT CCTCCCGCAATTCCTAGAAAATATTTCAGTGTCCGTT	978
	CTATTTCTGTAAGTATA	979
	TATACTTACAGAAATAG	980

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Glu-1725-Stop GAA to TAA	TTCTGCTGTATGTAACCTGTCTTTTCTATGATCTCTTTAGGGG TGACCCAGTCTATTAAAGAAAGAAAAATGCTGAATGAGGTAA GTACTTGATGTTACAAACTAACCAGAGATATTCATT	981
	AATGAATATCTCTGGTTAGTTTGTAACATCAAGTACTTACCTC ATTCAGCATTTTTCTTTCTTTAATAGACTGGGTCACCCCTAAA GAGATCATAGAAAAGACAGGTTACATACAGCAGAA	982
	CTATTAAAGAAAGAAAA	983
	TTTTCTTTCTTTAATAG	984
Breast Cancer Lys-1727-Stop AAA to TAA	TGTATGTAACCTGTCTTTTCTATGATCTCTTTAGGGGTGACCC AGTCTATTAAAGAAAGAAAAATGCTGAATGAGGTAAGTACTT GATGTTACAAACTAACCAGAGATATTCATTCAGTCA	985
	TGACTGAATGAATATCTCTGGTTAGTTTGTAACATCAAGTACT TACCTCATTCAGCATTTTTCTTTCTTTAATAGACTGGGTCACC CCTAAAGAGATCATAGAAAAGACAGGTTACATACA	986
	AAGAAAGAAAAATGCTG	987
	CAGCATTTTTCTTTCTT	988
Breast Cancer Pro-1749-Arg CCA to CGA	TCTTTCAGCATGATTTTGAAGTCAGAGGAGATGTGGTCAATG GAAGAAACCACCAAGGTCCAAAGCGAGCAAGAGAATCCCAG GACAGAAAGGTAAAGCTCCCTCCCTCAAGTTGACAAAA	989
	TTTTGTCAACTTGAGGGAGGGAGCTTTACCTTTCTGTCCTGG GATTCTCTTGCTCGCTTTGGACCTTGGTGGTTTCTTCCATTGA CCACATCTCCTCTGACTTCAAAATCATGCTGAAAGA	990
	CCAAGGTCCAAAGCGAG	991
	CTCGCTTTGGACCTTGG	992
Breast Cancer Arg-1751-Stop CGA to TGA	CAGCATGATTTTGAAGTCAGAGGAGATGTGGTCAATGGAAGA AACCACCAAGGTCCAAAGCGAGCAAGAGAATCCCAGGACAG AAAGGTAAAGCTCCCTCCCTCAAGTTGACAAAAATCTC	993
	GAGATTTTTGTCAACTTGAGGGAGGGAGCTTTACCTTTCTGT CCTGGGATTCTCTTGCTCGCTTTGGACCTTGGTGGTTTCTTC CATTGACCACATCTCCTCTGACTTCAAAATCATGCTG	994
	GTCCAAAGCGAGCAAGA	995
	TCTTGCTCGCTTTGGAC	996

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Gln-1756-Stop CAG to TAG	GTCAGAGGAGATGTGGTCAATGGAAGAAACCACCAAGGTCC AAAGCGAGCAAGAGAATCC**C**AGGACAGAAAGGTAAAGCTCC CTCCCTCAAGTTGACAAAAATCTCACCCCACCACTCTGT	997
	ACAGAGTGGTGGGGTGAGATTTTTGTCAACTTGAGGGAGGG AGCTTTACCTTTCTGTCCT**G**GGATTCTCTTGCTCGCTTTGGAC CTTGGTGGTTTCTTCCATTGACCACATCTCCTCTGAC	998
	GAGAATCC**C**AGGACAGA	999
	TCTGTCCT**G**GGATTCTC	1000
Breast Cancer Met-1775-Arg ATG to AGG	CTCTCTTCTTCCAGATCTTCAGGGG**G**CTAGAAATCTGTTGCT ATGGGCCCTTCACCAACA**T**GCCCACAGGTAAGAGCCTGGGA GAACCCCAGAGTTCCAGCACCAGCCTTTGTCTTACATA	1001
	TATGTAAGACAAAGGCTGGTGCTGGAACTCTGGGGTTCTCCC AGGCTCTTACCTGTGGGC**A**TGTTGGTGAAGGGCCCATAGCA ACAGATTTCTAGCCCCCTGAAGATCTGGAAGAAGAGAG	1002
	CACCAACA**T**GCCCACAG	1003
	CTGTGGGC**A**TGTTGGTG	1004
Breast Cancer Trp-1782-Stop TGG to TGA	AGTATGCAGATTACTGCAGTGATTTTACATCTAAATGTCCATT TTAGATCAACTGGAATG**G**ATGGTACAGCTGTGTGGTGCTTCT GTGGTGAAGGAGCTTTCATCATTCACCCTTGGCACA	1005
	TGTGCCAAGGGTGAATGATGAAAGCTCCTTCACCACAGAAG CACCACACAGCTGTACCAT**C**CATTCCAGTTGATCTAAAATGG ACATTTAGATGTAAAATCACTGCAGTAATCTGCATACT	1006
	CTGGAATG**G**ATGGTACA	1007
	TGTACCAT**C**CATTCCAG	1008
Breast Cancer Gln-1785-His CAG to CAT	ATTACTGCAGTGATTTTACATCTAAATGTCCATTTTAGATCAA CTGGAATGGATGGTACA**G**CTGTGTGGTGCTTCTGTGGTGAA GGAGCTTTCATCATTCACCCTTGGCACAGTAAGTATT	1009
	AATACTTACTGTGCCAAGGGTGAATGATGAAAGCTCCTTCAC CACAGAAGCACCACACAG**C**TGTACCATCCATTCCAGTTGATC TAAAATGGACATTTAGATGTAAAATCACTGCAGTAAT	1010
	ATGGTACA**G**CTGTGTGG	1011
	CCACACAG**C**TGTACCAT	1012

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast Cancer Glu-1794-Asp GAG to GAT	GTCCATTTTAGATCAACTGGAATGGATGGTACAGCTGTGTGG TGCTTCTGTGGTGAAGGAGCTTTCATCATTCACCCTTGGCAC AGTAAGTATTGGGTGCCCTGTCAGAGAGGGAGGACAC	1013
	GTGTCCTCCCTCTCTGACAGGGCACCCAATACTTACTGTGCC AAGGGTGAATGATGAAAGCTCCTTCACCACAGAAGCACCACA CAGCTGTACCATCCATTCCAGTTGATCTAAAATGGAC	1014
	GTGAAGGAGCTTTCATC	1015
	GATGAAAGCTCCTTCAC	1016
Breast Cancer Arg-1835-Stop CGA to TGA	CTCTGCTTGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGT GAGGCACCTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGT AGCACTCTACCAGTGCCAGGAGCTGGACACCTACCTGA	1017
	TCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAGTGCTACA CTGTCCAACACCCACTCTCGGGTCACCACAGGTGCCTCACA CATCTGCCCAATTGCTGGAGACAGAGAACACAAGCAGAG	1018
	TGGTGACCCGAGAGTGG	1019
	CCACTCTCGGGTCACCA	1020
Breast Cancer Trp-1837-Arg TGG to CGG	TTGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGTGAGGCA CCTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGTAGCACT CTACCAGTGCCAGGAGCTGGACACCTACCTGATACCCC	1021
	GGGGTATCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAGT GCTACACTGTCCAACACCCACTCTCGGGTCACCACAGGTGC CTCACACATCTGCCCAATTGCTGGAGACAGAGAACACAA	1022
	CCCGAGAGTGGGTGTTG	1023
	CAACACCCACTCTCGGG	1024
Breast Cancer Trp-1837-Stop TGG to TAG	TGTGTTCTCTGTCTCCAGCAATTGGGCAGATGTGTGAGGCAC CTGTGGTGACCCGAGAGTGGGTGTTGGACAGTGTAGCACTC TACCAGTGCCAGGAGCTGGACACCTACCTGATACCCCA	1025
	TGGGGTATCAGGTAGGTGTCCAGCTCCTGGCACTGGTAGAG TGCTACACTGTCCAACACCCACTCTCGGGTCACCACAGGTG CCTCACACATCTGCCCAATTGCTGGAGACAGAGAACACA	1026
	CCGAGAGTGGGTGTTGG	1027
	CCAACACCCACTCTCGG	1028

Table 15

BRCA2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer PHE32LEU TTT to CTT	GTTAAAACTAAGGTGGGATTTTTTTTTTAAATAGATTTAGGAC CAATAAGTCTTAATTGGTTTGAAGAACTTTCTTCAGAAGCTCC ACCCTATAATTCTGAACCTGCAGAAGAATCTGAAC	1029
	GTTCAGATTCTTCTGCAGGTTCAGAATTATAGGGTGGAGCTT CTGAAGAAAGTTCTTCAAACCAATTAAGACTTATTGGTCCTAA ATCTATTTAAAAAAAAAATCCCACCTTAGTTTTAAC	1030
	TTAATTGGTTTGAAGAA	1031
	TTCTTCAAACCAATTAA	1032
Breast cancer TYR42CYS TAT to TGT	TAGATTTAGGACCAATAAGTCTTAATTGGTTTGAAGAACTTTC TTCAGAAGCTCCACCCTATAATTCTGAACCTGCAGAAGAATC TGAACATAAAAACAACAATTACGAACCAAACCTATT	1033
	AATAGGTTTGGTTCGTAATTGTTGTTTTTATGTTCAGATTCTTC TGCAGGTTCAGAATTATAGGGTGGAGCTTCTGAAGAAAGTTC TTCAAACCAATTAAGACTTATTGGTCCTAAATCTA	1034
	TCCACCCTATAATTCTG	1035
	CAGAATTATAGGGTGGA	1036
Breast cancer LYS53ARG AAA to AGA	AAGAACTTTCTTCAGAAGCTCCACCCTATAATTCTGAACCTGC AGAAGAATCTGAACATAAAAACAACAATTACGAACCAAACCT ATTTAAAACTCCACAAAGGAAACCATCTTATAATCA	1037
	TGATTATAAGATGGTTTCCTTTGTGGAGTTTTAAATAGGTTTG GTTCGTAATTGTTGTTTTATGTTCAGATTCTTCTGCAGGTTC AGAATTATAGGGTGGAGCTTCTGAAGAAAGTTCTT	1038
	TGAACATAAAAACAACA	1039
	TGTTGTTTTTATGTTCA	1040
Breast cancer Phe81Leu TTC to CTC	CTATTTAAAACTCCACAAAGGAAACCATCTTATAATCAGCTGG CTTCAACTCCAATAATATTCAAAGAGCAAGGGCTGACTCTGC CGCTGTACCAATCTCCTGTAAAAGAATTAGATAAAT	1041
	ATTTATCTAATTCTTTTACAGGAGATTGGTACAGCGGCAGAGT CAGCCCTTGCTCTTTGAATATTATTGGAGTTGAAGCCAGCTG ATTATAAGATGGTTTCCTTTGTGGAGTTTTAAATAG	1042

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAATAATATTCAAAGAG	1043
	CTCTTTGAATATTATTG	1044
Breast cancer TRP194TERM TGG to TAG	GTCAGACACCAAAACATATTTCTGAAAGTCTAGGAGCTGAGG TGGATCCTGATATGTCTTGGTCAAGTTCTTTAGCTACACCACC CACCCTTAGTTCTACTGTGCTCATAGGTAATAATAG	1045
	CTATTATTACCTATGAGCACAGTAGAACTAAGGGTGGGTGGT GTAGCTAAAGAACTTGACCAAGACATATCAGGATCCACCTCA GCTCCTAGACTTTCAGAAATATGTTTTGGTGTCTGAC	1046
	TATGTCTTGGTCAAGTT	1047
	AACTTGACCAAGACATA	1048
Breast cancer PRO201ARG CCA to CGA	CTGAAAGTCTAGGAGCTGAGGTGGATCCTGATATGTCTTGGT CAAGTTCTTTAGCTACACCACCCACCCTTAGTTCTACTGTGCT CATAGGTAATAATAGCAAATGTGTATTTACAAGAAA	1049
	TTTCTTGTAAATACACATTTGCTATTATTACCTATGAGCACAGT AGAACTAAGGGTGGGTGGTGTAGCTAAAGAACTTGACCAAG ACATATCAGGATCCACCTCAGCTCCTAGACTTTCAG	1050
	AGCTACACCACCCACCC	1051
	GGGTGGGTGGTGTAGCT	1052
Breast cancer Pro222Ser CCT to TCT	ACAATACACATAAATTTTTATCTTACAGTCAGAAATGAAGAAG CATCTGAAACTGTATTTCCTCATGATACTACTGCTGTAAGTAA ATATGACATTGATTAGACTGTTGAAATTGCTAACA	1053
	TGTTAGCAATTTCAACAGTCTAATCAATGTCATATTTACTTACA GCAGTAGTATCATGAGGAAATACAGTTTCAGATGCTTCTTCAT TTCTGACTGTAAGATAAAAATTTATGTGTATTGT	1054
	CTGTATTTCCTCATGAT	1055
	ATCATGAGGAAATACAG	1056
Breast cancer Leu-414-Term TTG to TAG	AATGGTCTCAACTAACCCTTTCAGGTCTAAATGGAGCCCAGA TGGAGAAAATACCCCTATTGCATATTTCTTCATGTGACCAAAA TATTTCAGAAAAAGACCTATTAGACACAGAGAACAA	1057
	TTGTTCTCTGTGTCTAATAGGTCTTTTTCTGAAATATTTTGGTC ACATGAAGAAATATGCAATAGGGGTATTTTCTCCATCTGGGC TCCATTTAGACCTGAAAGGGTTAGTTGAGACCATT	1058
	ACCCCTATTGCATATTT	1059

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AAATATGCAATAGGGGT	1060
Breast cancer, male Cys554Trp TGT to TGG	AGCCTCTGAAAGTGGACTGGAAATACATACTGTTTGCTCACA GAAGGAGGACTCCTTATGTCCAAATTTAATTGATAATGGAAG CTGGCCAGCCACCACCACACAGAATTCTGTAGCTTTG	1061
	CAAAGCTACAGAATTCTGTGTGGTGGTGGCTGGCCAGCTTC CATTATCAATTAAATTTGGACATAAGGAGTCCTCCTTCTGTGA GCAAACAGTATGTATTTCCAGTCCACTTTCAGAGGCT	1062
	TCCTTATGTCCAAATTT	1063
	AAATTTGGACATAAGGA	1064
Breast cancer Lys944Term AAA to TAA	AACTCTACCATGGTTTTATATGGAGACACAGGTGATAAACAA GCAACCCAAGTGTCAATTAAAAAAGATTTGGTTTATGTTCTTG CAGAGGAGAACAAAAATAGTGTAAAGCAGCATATAA	1065
	TTATATGCTGCTTTACACTATTTTTGTTCTCCTCTGCAAGAAC ATAAACCAAATCTTTTTTAATTGACACTTGGGTTGCTTGTTTAT CACCTGTGTCTCCATATAAAACCATGGTAGAGTT	1066
	TGTCAATTAAAAAAGAT	1067
	ATCTTTTTTAATTGACA	1068
Breast cancer, male Glu1320Term GAA to TAA	ATGACTACTGGCACTTTTGTTGAAGAAATTACTGAAAATTACA AGAGAAATACTGAAAATGAAGATAACAAATATACTGCTGCCA GTAGAAATTCTCATAACTTAGAATTTGATGGCAGTG	1069
	CACTGCCATCAAATTCTAAGTTATGAGAATTTCTACTGGCAGC AGTATATTTGTTATCTTCATTTTCAGTATTTCTCTTGTAATTTTC AGTAATTTCTTCAACAAAAGTGCCAGTAGTCAT	1070
	CTGAAAATGAAGATAAC	1071
	GTTATCTTCATTTTCAG	1072
Breast cancer Glu1876Term GAA to TAA	CATGAAACAATTAAAAAAGTGAAAGACATATTTACAGACAGTT TCAGTAAAGTAATTAAGGAAAACAACGAGAATAAATCAAAAAT TTGCCAAACGAAAATTATGGCAGGTTGTTACGAGG	1073
	CCTCGTAACAACCTGCCATAATTTTCGTTTGGCAAATTTTTGA TTTATTCTCGTTGTTTTCCTTAATTACTTTACTGAAACTGTCTG TAAATATGTCTTTCACTTTTTTAATTGTTTCATG	1074
	TAATTAAGGAAAACAAC	1075
	GTTGTTTTCCTTAATTA	1076

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer Ser1882Term TCA to TAA	TGAAAGACATATTTACAGACAGTTTCAGTAAAGTAATTAAGGA AAACAACGAGAATAAATCAAAAATTTGCCAAACGAAAATTATG GCAGGTTGTTACGAGGCATTGGATGATTCAGAGGA	1077
	TCCTCTGAATCATCCAATGCCTCGTAACAACCTGCCATAATTT TCGTTTGGCAAATTTTTGATTTATTCTCGTTGTTTTCCTTAATT ACTTTACTGAAACTGTCTGTAAATATGTCTTTCA	1078
	GAATAAATCAAAAATTT	1079
	AAATTTTTGATTTATTC	1080
Breast cancer Glu1953Term GAA to TAA	AACCAAAATATGTCTGGATTGGAGAAAGTTTCTAAAATATCAC CTTGTGATGTTAGTTTGGAAACTTCAGATATATGTAAATGTAG TATAGGGAAGCTTCATAAGTCAGTCTCATCTGCAA	1081
	TTGCAGATGAGACTGACTTATGAAGCTTCCCTATACTACATTT ACATATATCTGAAGTTTCCAAACTAACATCACAAGGTGATATT TTAGAAACTTTCTCCAATCCAGACATATTTTGGTT	1082
	TTAGTTTGGAAACTTCA	1083
	TGAAGTTTCCAAACTAA	1084
Breast cancer Ser1970Term TCA to TAA	TTAGTTTGGAAACTTCAGATATATGTAAATGTAGTATAGGGAA GCTTCATAAGTCAGTCTCATCTGCAAATACTTGTGGGATTTTT AGCACAGCAAGTGGAAAATCTGTCCAGGTATCAGA	1085
	TCTGATACCTGGACAGATTTTCCACTTGCTGTGCTAAAAATCC CACAAGTATTTGCAGATGAGACTGACTTATGAAGCTTCCCTAT ACTACATTTACATATATCTGAAGTTTCCAAACTAA	1086
	GTCAGTCTCATCTGCAA	1087
	TTGCAGATGAGACTGAC	1088
Breast cancer Gln1987Term CAG to TAG	AAGTCAGTCTCATCTGCAAATACTTGTGGGATTTTTAGCACAG CAAGTGGAAAATCTGTCCAGGTATCAGATGCTTCATTACAAA ACGCAAGACAAGTGTTTTCTGAAATAGAAGATAGTA	1089
	TACTATCTTCTATTTCAGAAAACACTTGTCTTGCGTTTTGTAAT GAAGCATCTGATACCTGGACAGATTTTCCACTTGCTGTGCTA AAAATCCCACAAGTATTTGCAGATGAGACTGACTT	1090
	AATCTGTCCAGGTATCA	1091
	TGATACCTGGACAGATT	1092

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer Ala2466Val GCA to GTA	AAAATAAGATTAATGACAATGAGATTCATCAGTTTAACAAAAA CAACTCCAATCAAGCAG**C**AGCTGTAACTTTCACAAAGTGTGA AGAAGAACCTTTAGGTATTGTATGACAATTTGTGTG	1093
	CACACAAATTGTCATACAATACCTAAAGGTTCTTCTTCACACT TTGTGAAAGTTACAGCT**G**CTGCTTGATTGGAGTTGTTTTTGTT AAACTGATGAATCTCATTGTCATTAATCTTATTTT	1094
	TCAAGCAG**C**AGCTGTAA	1095
	TTACAGCT**G**CTGCTTGA	1096
Breast cancer Arg2520Term CGA to TGA	AGGCAACGCGTCTTTCCACAGCCAGGCAG**T**CTGTATCTTGCA AAAACATCCACTCTGCCT**C**GAATCTCTCTGAAAGCAGCAGTA GGAGGCCAAGTCCCCTCTGCGTGTCCTCATAAACAGG	1097
	CCTGTTTATGAGGACACGCAGAGGGGACTTGGCCTCCTACT GCTGCTTTCAGAGAGATTC**G**AGGCAGAGTGGATGTTTTTGCA AGATACAGACTGCCTGGCTGTGGAAAGA**C**GCGTTGCCT	1098
	CTCTGCCT**C**GAATCTCT	1099
	AGAGATTC**G**AGGCAGAG	1100
Breast cancer Gln2714Term CAA to TAA	ATTTCATTGAGCGCAAATATATCTGAAACTTCTAGCAATAAAA CTAGTAGTGCAGATACC**C**AAAAAGTGGCCATTATTGAACTTA CAGATGGGTGGTATGCTGTTAAGGCCCAGTTAGATC	1101
	GATCTAACTGGGCCTTAACAGCATACCACCCATCTGTAAGTT CAATAATGGCCACTTTTT**G**GGTATCTGCACTACTAGTTTTATT GCTAGAAGTTTCAGATATATTTGCGCTCAATGAAAT	1102
	CAGATACC**C**AAAAAGTG	1103
	CACTTTTT**G**GGTATCTG	1104
Breast cancer Leu2776Term TTA to TGA	CAGAACTGGTGGGCTCTCCTGATGCCTGTACACCTCTTGAAG CCCCAGAATCTCTTATGT**T**AAAGGTAAATTAATTTGCACTCTT GGTAAAAATCAGTCATTGATTCAGTTAAATTCTAGA	1105
	TCTAGAATTTAACTGAATCAATGACTGATTTTTACCAAGAGTG CAAATTAATTTACCTTT**A**ACATAAGAGATTCTGGGGCTTCAAG AGGTGTACAGGCATCAGGAGAGCCCACCAGTTCTG	1106
	TCTTATGT**T**AAAGATTT	1107
	AAATCTTT**A**ACATAAGA	1108

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer Gln2893Term CAG to TAG	CCTTTTGTTTTCTTAGAAAACACAACAAAACCATATTTACCATC ACGTGCACTAACAAGACAGCAAGTTCGTGCTTTGCAAGATGG TGCAGAGCTTTATGAAGCAGTGAAGAATGCAGCAG	1109
	CTGCTGCATTCTTCACTGCTTCATAAAGCTCTGCACCATCTTG CAAAGCACGAACTTGCTGTCTTGTTAGTGCACGTGATGGTAA ATATGGTTTTGTTGTGTTTTCTAAGAAAACAAAAGG	1110
	TAACAAGACAGCAAGTT	1111
	AACTTGCTGTCTTGTTA	1112
Breast cancer Ala2951Thr GCC to ACC	AATCACAGGCAAATGTTGAATGATAAGAAACAAGCTCAGATC CAGTTGGAAATTAGGAAGGCCATGGAATCTGCTGAACAAAAG GAACAAGGTTTATCAAGGGATGTCACAACCGTGTGGA	1113
	TCCACACGGTTGTGACATCCCTTGATAAACCTTGTTCCTTTTG TTCAGCAGATTCCATGGCCTTCCTAATTTCCAACTGGATCTGA GCTTGTTTCTTATCATTCAACATTTGCCTGTGATT	1114
	TTAGGAAGGCCATGGAA	1115
	TTCCATGGCCTTCCTAA	1116
Breast cancer Met3118Thr ATG to ACG	ACAATTTACTGGCAATAAAGTTTTGGATAGACCTTAATGAGGA CATTATTAAGCCTCATATGTTAATTGCTGCAAGCAACCTCCAG TGGCGACCAGAATCCAAATCAGGCCTTCTTACTTT	1117
	AAAGTAAGAAGGCCTGATTTGGATTCTGGTCGCCACTGGAG GTTGCTTGCAGCAATTAACATATGAGGCTTAATAATGTCCTCA TTAAGGTCTATCCAAAACTTTATTGCCAGTAAATTGT	1118
	GCCTCATATGTTAATTG	1119
	CAATTAACATATGAGGC	1120
Breast cancer Thr3401Met ACG to ATG	GACTGAAACGACGTTGTACTACATCTCTGATCAAAGAACAGG AGAGTTCCCAGGCCAGTACGGAAGAATGTGAGAAAAATAAG CAGGACACAATTACAACTAAAAAATATATCTAAGCATT	1121
	AATGCTTAGATATATTTTTTAGTTGTAATTGTGTCCTGCTTATT TTTCTCACATTCTTCCGTACTGGCCTGGGAACTCTCCTGTTCT TTGATCAGAGATGTAGTACAACGTCGTTTCAGTC	1122
	GGCCAGTACGGAAGAAT	1123
	ATTCTTCCGTACTGGCC	1124

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Breast cancer Ile3412Val ATT to GTT	AAAGAACAGGAGAGTTCCCAGGCCAGTACGGAAGAATGTGA GAAAAATAAGCAGGACACAATTACAACTAAAAAATATATCTAA GCATTTGCAAAGGCGACAATAAATTATTGACGCTTAA	1125
	TTAAGCGTCAATAATTTATTGTCGCCTTTGCAAATGCTTAGAT ATATTTTTTAGTTGTAATTGTGTCCTGCTTATTTTTCTCACATT CTTCCGTACTGGCCTGGGAACTCTCCTGTTCTTT	1126
	AGGACACAATTACAACT	1127
	AGTTGTAATTGTGTCCT	1128

EXAMPLE 9

Cystic Fibrosis – CFTR

Cystic fibrosis is a lethal disease affecting approximately one in 2,500 live Caucasian births and is the most common autosomal recessive disease in Caucasians. Patients with this disease have reduced chloride ion permeability in the secretory and absorptive cells of organs with epithelial cell linings, including the airways, pancreas, intestine, sweat glands and male genital tract. This, in turn, reduces the transport of water across the epithelia. The lungs and the GI tract are the predominant organ systems affected in this disease and the pathology is characterized by blocking of the respiratory and GI tracts with viscous mucus. The chloride impermeability in affected tissues is due to mutations in a specific chloride channel, the cystic fibrosis transmembrane conductance regulator protein (CFTR), which prevents normal passage of chloride ions through the cell membrane (Welsh et al., Neuron, 8:821-829 (1992)). Damage to the lungs due to mucus blockage, frequent bacterial infections and inflammation is the primary cause of morbidity and mortality in CF patients and, although maintenance therapy has improved the quality of patients' lives, the median age at death is still only around 30 years. There is no effective treatment for the disease, and therapeutic research is focused on gene therapy using exogenous transgenes in viral vectors and/or activating the defective or other chloride channels in the cell membrane to normalize chloride permeability (Tizzano et al., J. Pediat., 120:337-349 (1992)). However, the death of a teenage patient treated with an adenovirus vector carrying an exogenous CFTR gene in clinical trials in the late 1990's has impacted this area of research.

The oligonucleotides of the invention for correction of the CFTR gene are attached as a table.

Table 16

CFTR Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Ala46Asp GCT to GAT	AAGGATACAGACAGCGCCTGGAATTGTCAGACATATACCAAA TCCCTTCTGTTGATTCTG**C**TGACAATCTATCTGAAAAATTGGA AAGGTATGTTCATGTACATTGTTTAGTTGAAGAGAG	1129
	CTCTCTTCAACTAAACAATGTACATGAACATACCTTTCCAATT TTTCAGATAGATTGTCA**G**CAGAATCAACAGAAGGGATTTGGT ATATGTCTGACAATTCCAGGCGCTGTCTGTATCCTT	1130
	TGATTCTG**C**TGACAATC	1131
	GATTGTCA**G**CAGAATCA	1132
Cystic fibrosis Ser50Tyr TCT to TAT	AGCGCCTGGAATTGTCAGACATATACCAAATCCCTTCTGTTG ATTCTGCTGACAATCTAT**C**TGAAAAATTGGAAAGGTATGTTCA TGTACATTGTTTAGTTGAAGAGAGAAATTCATATTA	1133
	TAATATGAATTTCTCTCTTCAACTAAACAATGTACATGAACATA CCTTTCCAATTTTTCA**G**ATAGATTGTCAGCAGAATCAACAGAA GGGATTTGGTATATGTCTGACAATTCCAGGCGCT	1134
	CAATCTAT**C**TGAAAAAT	1135
	ATTTTTCA**G**ATAGATTG	1136
Congenital absence of vas deferens Glu56Lys GAA-AAA	AGGACAACTAAAATATTTGCACATGCAACTTATTGGTCCCACT TTTTATTCTTTTGCAGA**G**AATGGGATAGAGAGCTGGCTTCAA AGAAAAATCCTAAACTCATTAATGCCCTTCGGCGAT	1137
	ATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTTGAAG CCAGCTCTCTATCCCATT**C**TCTGCAAAAGAATAAAAAGTGGG ACCAATAAGTTGCATGTGCAAATATTTTAGTTGTCCT	1138
	TTTGCAGA**G**AATGGGAT	1139
	ATCCCATT**C**TCTGCAAA	1140
Cystic fibrosis Trp57Gly TGG to GGG	AGGACAACTAAAATATTTGCACATGCAACTTATTGGTCCCACT TTTTATTCTTTTGCAGA**G**AATGGGATAGAGAGCTGGCTTCAA AGAAAAATCCTAAACTCATTAATGCCCTTCGGCGAT	1141
	ATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTTGAAG CCAGCTCTCTATCCCATT**C**TCTGCAAAAGAATAAAAAGTGGG ACCAATAAGTTGCATGTGCAAATATTTTAGTTGTCCT	1142

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTGCAGAGAATGGGAT	1143
	ATCCCATTCTCTGCAAA	1144
Cystic fibrosis Trp57Term TGG to TGA	AACTAAAATATTTGCACATGCAACTTATTGGTCCCACTTTTTAT TCTTTTGCAGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAA ATCCTAAACTCATTAATGCCCTTCGGCGATGTTTT	1145
	AAAACATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTTT GAAGCCAGCTCTCTATCCCATTCTCTGCAAAAGAATAAAAAG TGGGACCAATAAGTTGCATGTGCAAATATTTTAGTT	1146
	AGAGAATGGGATAGAGA	1147
	TCTCTATCCCATTCTCT	1148
Congenital absence of vas deferens Asp58Asn GAT to AAT	ACTAAAATATTTGCACATGCAACTTATTGGTCCCACTTTTTATT CTTTTGCAGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAA TCCTAAACTCATTAATGCCCTTCGGCGATGTTTTT	1149
	AAAAACATCGCCGAAGGGCATTAATGAGTTTAGGATTTTTCTT TGAAGCCAGCTCTCTATCCCATTCTCTGCAAAAGAATAAAAA GTGGGACCAATAAGTTGCATGTGCAAATATTTTAGT	1150
	GAGAATGGGATAGAGAG	1151
	CTCTCTATCCCATTCTC	1152
Cystic fibrosis Glu60Term GAG to TAG	ATATTTGCACATGCAACTTATTGGTCCCACTTTTTATTCTTTTG CAGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAA ACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGA	1153
	TCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAGGAT TTTTCTTTGAAGCCAGCTCTCTATCCCATTCTCTGCAAAAGAA TAAAAAGTGGGACCAATAAGTTGCATGTGCAAATAT	1154
	GGGATAGAGAGCTGGCT	1155
	AGCCAGCTCTCTATCCC	1156
Cystic fibrosis Pro67Leu CCT to CTT	GGTCCCACTTTTTATTCTTTTGCAGAGAATGGGATAGAGAGC TGGCTTCAAAGAAAAATCCTAAACTCATTAATGCCCTTCGGC GATGTTTTTTCTGGAGATTTATGTTCTATGGAATCTT	1157
	AAGATTCCATAGAACATAAATCTCCAGAAAAAACATCGCCGA AGGGCATTAATGAGTTTAGGATTTTTCTTTGAAGCCAGCTCTC TATCCCATTCTCTGCAAAAGAATAAAAAGTGGGACC	1158
	GAAAAATCCTAAACTCA	1159

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGAGTTTAGGATTTTTC	1160
Cystic fibrosis Arg74Trp CGG to TGG	TGCAGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCT AAACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTA TGTTCTATGGAATCTTTTTATATTTAGGGGTAAGGA	1161
	TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAGGATT TTTCTTTGAAGCCAGCTCTCTATCCCATTCTCTGCA	1162
	ATGCCCTTCGGCGATGT	1163
	ACATCGCCGAAGGGCAT	1164
Congenital absence of vas deferens ARG75GLN CGA to CAA	GAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAAC TCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTATGTT CTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC	1165
	GAGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAA ATCTCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAG GATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTC	1166
	CCTTCGGCGATGTTTTT	1167
	AAAAACATCGCCGAAGG	1168
Cystic fibrosis Arg75Leu CGA to CTA	GAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAAC TCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTATGTT CTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC	1169
	GAGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAA ATCTCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAG GATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTC	1170
	CCTTCGGCGATGTTTTT	1171
	AAAAACATCGCCGAAGG	1172
Cystic fibrosis Arg75Term CGA to TGA	AGAGAATGGGATAGAGAGCTGGCTTCAAAGAAAAATCCTAAA CTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTATGT TCTATGGAATCTTTTTATATTTAGGGGTAAGGATCT	1173
	AGATCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAA TCTCCAGAAAAAACATCGCCGAAGGGCATTAATGAGTTTAGG ATTTTTCTTTGAAGCCAGCTCTCTATCCCATTCTCT	1174
	CCCTTCGGCGATGTTTT	1175
	AAAACATCGCCGAAGGG	1176

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gly85Glu GGA to GAA	AAAATCCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTG GAGATTTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGG ATCTCATTTGTACATTCATTATGTATCACATAACT	1177
	AGTTATGTGATACATAATGAATGTACAAATGAGATCCTTACCC CTAAATATAAAAAGATTCCATAGAACATAAATCTCCAGAAAAA ACATCGCCGAAGGGCATTAATGAGTTTAGGATTTT	1178
	GTTCTATGGAATCTTTT	1179
	AAAAGATTCCATAGAAC	1180
Cystic fibrosis Gly85Val GGA to GTA	AAAATCCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTG GAGATTTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGG ATCTCATTTGTACATTCATTATGTATCACATAACT	1181
	AGTTATGTGATACATAATGAATGTACAAATGAGATCCTTACCC CTAAATATAAAAAGATTCCATAGAACATAAATCTCCAGAAAAA ACATCGCCGAAGGGCATTAATGAGTTTAGGATTTT	1182
	GTTCTATGGAATCTTTT	1183
	AAAAGATTCCATAGAAC	1184
Cystic fibrosis Leu88Ser TTA to TCA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1185
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTT	1186
	AATCTTTTTATATTTAG	1187
	CTAAATATAAAAAGATT	1188
Cystic fibrosis Phe87Leu TTT to CTT	CCTAAACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGAT TTATGTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTC ATTTGTACATTCATTATGTATCACATAACTATATG	1189
	CATATAGTTATGTGATACATAATGAATGTACAAATGAGATCCT TACCCCTAAATATAAAAAGATTCCATAGAACATAAATCTCCAG AAAAAACATCGCCGAAGGGCATTAATGAGTTTAGG	1190
	ATGGAATCTTTTTATAT	1191
	ATATAAAAAGATTCCAT	1192

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Leu88Term TTA to TGA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1193
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTT	1194
	AATCTTTTTATATTTAG	1195
	CTAAATATAAAAAGATT	1196
Cystic fibrosis Leu88Term TTA to TAA	AACTCATTAATGCCCTTCGGCGATGTTTTTTCTGGAGATTTAT GTTCTATGGAATCTTTTTATATTTAGGGGTAAGGATCTCATTT GTACATTCATTATGTATCACATAACTATATGCATT	1197
	AATGCATATAGTTATGTGATACATAATGAATGTACAAATGAGA TCCTTACCCCTAAATATAAAAAGATTCCATAGAACATAAATCT CCAGAAAAAACATCGCCGAAGGGCATTAATGAGTT	1198
	AATCTTTTTATATTTAG	1199
	CTAAATATAAAAAGATT	1200
Cystic fibrosis Gly91Arg GGG to AGG	AATGCCCTTCGGCGATGTTTTTTCTGGAGATTTATGTTCTATG GAATCTTTTTATATTTAGGGGTAAGGATCTCATTTGTACATTC ATTATGTATCACATAACTATATGCATTTTTGTGAT	1201
	ATCACAAAAATGCATATAGTTATGTGATACATAATGAATGTAC AAATGAGATCCTTACCCCTAAATATAAAAAGATTCCATAGAAC ATAAATCTCCAGAAAAAACATCGCCGAAGGGCATT	1202
	TATATTTAGGGGTAAGG	1203
	CCTTACCCCTAAATATA	1204
Cystic fibrosis Gln98Arg CAG to CGG	AATAAATGAAATTTAATTTCTCTGTTTTTCCCCTTTTGTAGGAA GTCACCAAAGCAGTACAGCCTCTCTTACTGGGAAGAATCATA GCTTCCTATGACCCGGATAACAAGGAGGAACGCTC	1205
	GAGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTATGATT CTTCCCAGTAAGAGAGGCTGTACTGCTTTGGTGACTTCCTAC AAAAGGGGAAAAACAGAGAAATTAAATTTCATTTATT	1206
	AGCAGTACAGCCTCTCT	1207
	AGAGAGGCTGTACTGCT	1208

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gln98Term CAG-TAG	AAATAAATGAAATTTAATTTCTCTGTTTTTCCCCTTTTGTAGGA AGTCACCAAAGCAGTA**C**AGCCTCTCTTACTGGGAAGAATCAT AGCTTCCTATGACCCGGATAACAAGGAGGAACGCT	1209
	AGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTATGATTC TTCCCAGTAAGAGAGGCT**G**TACTGCTTTGGTGACTTCCTACA AAAGGGGAAAAACAGAGAAATTAAATTTCATTTATTT	1210
	AAGCAGTA**C**AGCCTCTC	1211
	GAGAGGCT**G**TACTGCTT	1212
Cystic fibrosis Ser108Phe TCC to TTC	CCCTTTTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTAC TGGGAAGAATCATAGCTT**C**CTATGACCCGGATAACAAGGAG GAACGCTCTATCGCGATTTATCTAGGCATAGGCTTATG	1213
	CATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTTCCTCC TTGTTATCCGGGTCATAG**G**AAGCTATGATTCTTCCCAGTAAG AGAGGCTGTACTGCTTTGGTGACTTCCTACAAAAGGG	1214
	CATAGCTT**C**CTATGACC	1215
	GGTCATAG**G**AAGCTATG	1216
Cystic fibrosis Tyr109Cys TAT to TGT	TTTTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGG GAAGAATCATAGCTTCCT**A**TGACCCGGATAACAAGGAGGAAC GCTCTATCGCGATTTATCTAGGCATAGGCTTATGCCT	1217
	AGGCATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTTCC TCCTTGTTATCCGGGTCA**T**AGGAAGCTATGATTCTTCCCAGT AAGAGAGGCTGTACTGCTTTGGTGACTTCCTACAAAA	1218
	AGCTTCCT**A**TGACCCGG	1219
	CCGGGTCA**T**AGGAAGCT	1220
Cystic fibrosis Asp110His GAC to CAC	TTGTAGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGGGA AGAATCATAGCTTCCTAT**G**ACCCGGATAACAAGGAGGAACGC TCTATCGCGATTTATCTAGGCATAGGCTTATGCCTTC	1221
	GAAGGCATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTT CCTCCTTGTTATCCGGGT**C**ATAGGAAGCTATGATTCTTCCCA GTAAGAGAGGCTGTACTGCTTTGGTGACTTCCTACAA	1222
	CTTCCTAT**G**ACCCGGAT	1223
	ATCCGGGT**C**ATAGGAAG	1224

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Congenital absence of vas deferens Pro111Leu CCG to CTG	AGGAAGTCACCAAAGCAGTACAGCCTCTCTTACTGGGAAGAA TCATAGCTTCCTATGACCCGGATAACAAGGAGGAACGCTCTA TCGCGATTTATCTAGGCATAGGCTTATGCCTTCTCTT	1225
	AAGAGAAGGCATAAGCCTATGCCTAGATAAATCGCGATAGAG CGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTATGATTCTT CCCAGTAAGAGAGGCTGTACTGCTTTGGTGACTTCCT	1226
	CTATGACCCGGATAACA	1227
	TGTTATCCGGGTCATAG	1228
Cystic fibrosis Arg117Cys CGC to TGC	GTACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGAC CCGGATAACAAGGAGGAACGCTCTATCGCGATTTATCTAGGC ATAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGC	1229
	GCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCTA GATAAATCGCGATAGAGCGTTCCTCCTTGTTATCCGGGTCAT AGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTAC	1230
	AGGAGGAACGCTCTATC	1231
	GATAGAGCGTTCCTCCT	1232
Cystic fibrosis Arg117His CGC to CAC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAACGCTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1233
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAGCGTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1234
	GGAGGAACGCTCTATCG	1235
	CGATAGAGCGTTCCTCC	1236
Cystic fibrosis Arg117Leu CGC to CTC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAACGCTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1237
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAGCGTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1238
	GGAGGAACGCTCTATCG	1239
	CGATAGAGCGTTCCTCC	1240

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Arg117Pro CGC to CCC	TACAGCCTCTCTTACTGGGAAGAATCATAGCTTCCTATGACC CGGATAACAAGGAGGAACGCTCTATCGCGATTTATCTAGGCA TAGGCTTATGCCTTCTCTTTATTGTGAGGACACTGCT	1241
	AGCAGTGTCCTCACAATAAAGAGAAGGCATAAGCCTATGCCT AGATAAATCGCGATAGAGCGTTCCTCCTTGTTATCCGGGTCA TAGGAAGCTATGATTCTTCCCAGTAAGAGAGGCTGTA	1242
	GGAGGAACGCTCTATCG	1243
	CGATAGAGCGTTCCTCC	1244
Cystic fibrosis Ala120Thr GCG-ACG	CTCTTACTGGGAAGAATCATAGCTTCCTATGACCCGGATAAC AAGGAGGAACGCTCTATCGCGATTTATCTAGGCATAGGCTTA TGCCTTCTCTTTATTGTGAGGACACTGCTCCTACACC	1245
	GGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAGGCATAAG CCTATGCCTAGATAAATCGCGATAGAGCGTTCCTCCTTGTTA TCCGGGTCATAGGAAGCTATGATTCTTCCCAGTAAGAG	1246
	GCTCTATCGCGATTTAT	1247
	ATAAATCGCGATAGAGC	1248
Cystic fibrosis Tyr122Term TAT to TAA	GGGAAGAATCATAGCTTCCTATGACCCGGATAACAAGGAGG AACGCTCTATCGCGATTTATCTAGGCATAGGCTTATGCCTTCT CTTTATTGTGAGGACACTGCTCCTACACCCAGCCATT	1249
	AATGGCTGGGTGTAGGAGCAGTGTCCTCACAATAAAGAGAA GGCATAAGCCTATGCCTAGATAAATCGCGATAGAGCGTTCCT CCTTGTTATCCGGGTCATAGGAAGCTATGATTCTTCCC	1250
	GCGATTTATCTAGGCAT	1251
Cystic fibrosis Gly126Asp GGC-GAC	ATGCCTAGATAAATCGC	1252
	TAGCTTCCTATGACCCGGATAACAAGGAGGAACGCTCTATCG CGATTTATCTAGGCATAGGCTTATGCCTTCTCTTTATTGTGAG GACACTGCTCCTACACCCAGCCATTTTTGGCCTTCA	1253
	TGAAGGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTCAC AATAAAGAGAAGGCATAAGCCTATGCCTAGATAAATCGCGAT AGAGCGTTCCTCCTTGTTATCCGGGTCATAGGAAGCTA	1254
	AGGCATAGGCTTATGCC	1255
	GGCATAAGCCTATGCCT	1256

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis His139Arg CAC to CGC	TCGCGATTTATCTAGGCATAGGCTTATGCCTTCTCTTTATTGT GAGGACACTGCTCCTACACCCAGCCATTTTTGGCCTTCATCA CATTGGAATGCAGATGAGAATAGCTATGTTTAGTTT	1257
	AAACTAAACATAGCTATTCTCATCTGCATTCCAATGTGATGAA GGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTCACAATA AAGAGAAGGCATAAGCCTATGCCTAGATAAATCGCGA	1258
	GCTCCTACACCCAGCCA	1259
	TGGCTGGGTGTAGGAGC	1260
Cystic fibrosis Ala141Asp GCC to GAC	TTTATCTAGGCATAGGCTTATGCCTTCTCTTTATTGTGAGGAC ACTGCTCCTACACCCAGCCATTTTTGGCCTTCATCACATTGG AATGCAGATGAGAATAGCTATGTTTAGTTTGATTTA	1261
	TAAATCAAACTAAACATAGCTATTCTCATCTGCATTCCAATGT GATGAAGGCCAAAAATGGCTGGGTGTAGGAGCAGTGTCCTC ACAATAAAGAGAAGGCATAAGCCTATGCCTAGATAAA	1262
	ACACCCAGCCATTTTTG	1263
	CAAAAATGGCTGGGTGT	1264
Cystic fibrosis Ile148Thr ATT to ACT	GCCTTCTCTTTATTGTGAGGACACTGCTCCTACACCCAGCCA TTTTTGGCCTTCATCACATTGGAATGCAGATGAGAATAGCTAT GTTTAGTTTGATTTATAAGAAGGTAATACTTCCTTG	1265
	CAAGGAAGTATTACCTTCTTATAAATCAAACTAAACATAGCTA TTCTCATCTGCATTCCAATGTGATGAAGGCCAAAAATGGCTG GGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAGGC	1266
	TCATCACATTGGAATGC	1267
	GCATTCCAATGTGATGA	1268
Cystic fibrosis Gly149Arg GGA to AGA	CTTCTCTTTATTGTGAGGACACTGCTCCTACACCCAGCCATTT TTGGCCTTCATCACATTGGAATGCAGATGAGAATAGCTATGT TTAGTTTGATTTATAAGAAGGTAATACTTCCTTGCA	1269
	TGCAAGGAAGTATTACCTTCTTATAAATCAAACTAAACATAGC TATTCTCATCTGCATTCCAATGTGATGAAGGCCAAAAATGGCT GGGTGTAGGAGCAGTGTCCTCACAATAAAGAGAAG	1270
	ATCACATTGGAATGCAG	1271
	CTGCATTCCAATGTGAT	1272

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gln151Term CAG to TAG	TTTATTGTGAGGACACTGCTCCTACACCCAGCCATTTTTGGC CTTCATCACATTGGAATGCAGATGAGAATAGCTATGTTTAGTT TGATTTATAAGAAGGTAATACTTCCTTGCACAGGCC	1273
	GGCCTGTGCAAGGAAGTATTACCTTCTTATAAATCAAACTAAA CATAGCTATTCTCATCTGCATTCCAATGTGATGAAGGCCAAAA ATGGCTGGGTGTAGGAGCAGTGTCCTCACAATAAA	1274
	TTGGAATGCAGATGAGA	1275
	TCTCATCTGCATTCCAA	1276
Cystic fibrosis Lys166Glu AAG-GAG	AATATATTTGTATTTTGTTTGTTGAAATTATCTAACTTTCCATTT TTCTTTTAGACTTTAAAGCTGTCAAGCCGTGTTCTAGATAAAA TAAGTATTGGACAACTTGTTAGTCTCCTTTCCA	1277
	TGGAAAGGAGACTAACAAGTTGTCCAATACTTATTTTATCTAG AACACGGCTTGACAGCTTTAAAGTCTAAAAGAAAAATGGAAA GTTAGATAATTTCAACAAACAAAATACAAATATATT	1278
	AGACTTTAAAGCTGTCA	1279
	TGACAGCTTTAAAGTCT	1280
Cystic fibrosis Ile175Val ATA-GTA	TTATCTAACTTTCCATTTTTCTTTTAGACTTTAAAGCTGTCAAG CCGTGTTCTAGATAAAATAAGTATTGGACAACTTGTTAGTCTC CTTTCCAACAACCTGAACAAATTTGATGAAGTAT	1281
	ATACTTCATCAAATTTGTTCAGGTTGTTGGAAAGGAGACTAAC AAGTTGTCCAATACTTATTTTATCTAGAACACGGCTTGACAGC TTTAAAGTCTAAAAGAAAAATGGAAAGTTAGATAA	1282
	TAGATAAAATAAGTATT	1283
	AATACTTATTTTATCTA	1284
Cystic fibrosis Gly178Arg GGA to AGA	TTTCCATTTTTCTTTTAGACTTTAAAGCTGTCAAGCCGTGTTCT AGATAAAATAAGTATTGGACAACTTGTTAGTCTCCTTTCCAAC AACCTGAACAAATTTGATGAAGTATGTACCTATT	1285
	AATAGGTACATACTTCATCAAATTTGTTCAGGTTGTTGGAAAG GAGACTAACAAGTTGTCCAATACTTATTTTATCTAGAACACGG CTTGACAGCTTTAAAGTCTAAAAGAAAAATGGAAA	1286
	TAAGTATTGGACAACTT	1287
	AAGTTGTCCAATACTTA	1288

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis His199Gln CAT to CAG	AAGATACAATGACACCTGTTTTTGCTGTGCTTTTATTTTCCAG GGACTTGCATTGGCACATTTCGTGTGGATCGCTCCTTTGCAA GTGGCACTCCTCATGGGGCTAATCTGGGAGTTGTTA	1289
	TAACAACTCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAA AGGAGCGATCCACACGAAATGTGCCAATGCAAGTCCCTGGA AAATAAAAGCACAGCAAAAACAGGTGTCATTGTATCTT	1290
	TTGGCACATTTCGTGTG	1291
	CACACGAAATGTGCCAA	1292
Cystic fibrosis His199Tyr CAT to TAT	GGAAGATACAATGACACCTGTTTTTGCTGTGCTTTTATTTTCC AGGGACTTGCATTGGCACATTTCGTGTGGATCGCTCCTTTGC AAGTGGCACTCCTCATGGGGCTAATCTGGGAGTTGT	1293
	ACAACTCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAAAG GAGCGATCCACACGAAATGTGCCAATGCAAGTCCCTGGAAA ATAAAAGCACAGCAAAAACAGGTGTCATTGTATCTTCC	1294
	CATTGGCACATTTCGTG	1295
	CACGAAATGTGCCAATG	1296
Cystic fibrosis Pro205Ser CCT to TCT	TGTTTTTGCTGTGCTTTTATTTTCCAGGGACTTGCATTGGCAC ATTTCGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGG GGCTAATCTGGGAGTTGTTACAGGCGTCTGCCTTCT	1297
	AGAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCCCATG AGGAGTGCCACTTGCAAAGGAGCGATCCACACGAAATGTGC CAATGCAAGTCCCTGGAAAATAAAAGCACAGCAAAAACA	1298
	GGATCGCTCCTTTGCAA	1299
	TTGCAAAGGAGCGATCC	1300
Cystic fibrosis Leu206Trp TTG to TGG	TTTGCTGTGCTTTTATTTTCCAGGGACTTGCATTGGCACATTT CGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGGGGC TAATCTGGGAGTTGTTACAGGCGTCTGCCTTCTGTGG	1301
	CCACAGAAGGCAGACGCCTGTAACAACTCCCAGATTAGCCC CATGAGGAGTGCCACTTGCAAAGGAGCGATCCACACGAAAT GTGCCAATGCAAGTCCCTGGAAAATAAAAGCACAGCAAA	1302
	CGCTCCTTTGCAAGTGG	1303
	CCACTTGCAAAGGAGCG	1304

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Cystic fibrosis Gln220Term CAG to TAG	TTCGTGTGGATCGCTCCTTTGCAAGTGGCACTCCTCATGGGG CTAATCTGGGAGTTGTTA**C**AGGCGTCTGCCTTCTGTGGACTT GGTTTCCTGATAGTCCTTGCCCTTTTTCAGGCTGGGC	1305
	GCCCAGCCTGAAAAAGGGCAAGGACTATCAGGAAACCAAGT CCACAGAAGGCAGACGCCT**G**TAACAACTCCCAGATTAGCCC CATGAGGAGTGCCACTTGCAAAGGAGCGATCCACACGAA	1306
	AGTTGTTA**C**AGGCGTCT	1307
	AGACGCCT**G**TAACAACT	1308
Cystic fibrosis Cys225Arg TGT-CGT	CCTTTGCAAGTGGCACTCCTCATGGGGCTAATCTGGGA**G**TTG TTACAGGCGTCTGCCTTC**T**GTGGACTTGGTTTCCTGATAGTC CTTGCCCTTTTTCAGGCTGGGCTAGGGAGAATGATGA	1309
	TCATCATTCTCCCTAGCCCAGCCTGAAAAAGGGCAAGGACTA TCAGGAAACCAAGTCCA**C**AGAAGGCAGACGCCTGTAACAAC TCCCAGATTAGCCCCATGAGGAGTGCCACTTGCAAAGG	1310
	CTGCCTTC**T**GTGGACTT	1311
	AAGTCCAC**A**GAAGGCAG	1312
Cystic fibrosis Val232Asp GTC to GAC	TGGGGCTAATCTGGGAGTTGTTACAGGCGTCTGCCTTCTGTG GACTTGGTTTCCTGATAG**T**CCTTGCCCTTTTTCAGGCTGGGC TAGGGAGAATGATGATGAAGTACAGGTAGCAACCTAT	1313
	ATAGGTTGCTACCTGTACTTCATCATCATTCTCCCTAGCCCAG CCTGAAAAAGGGCAAGG**A**CTATCAGGAAACCAAGTCCACAG AAGGCAGACGCCTGTAACAACTCCCAGATTAGCCCCA	1314
	CCTGATAG**T**CCTTGCCC	1315
	GGGCAAGG**A**CTATCAGG	1316
Cystic fibrosis Gly239Arg GGG to AGG	GTTACAGGCGTCTGCCTTCTGTGGACTTGGTTTCCTGATAGT CCTTGCCCTTTTTCAGGC**T**GGGCTAGGGAGAATGATGATGAA GTACAGGTAGCAACCTATTTTCATAACTTGAAAGTTT	1317
	AAACTTTCAAGTTATGAAAATAGGTTGCTACCTGTACTTCATC ATCATTCTCCCTAGCCC**A**GCCTGAAAAAGGGCAAGGACTATC AGGAAACCAAGTCCACAGAAGGCAGACGCCTGTAAC	1318
	TTTCAGGC**T**GGGCTAGG	1319
	CCTAGCCC**A**GCCTGAAA	1320

EXAMPLE 10

Cyclin-dependent kinase inhibitor 2A - CDKN2A

The human CDKN2A gene was also designated MTS-1 for multiple tumor suppressor-1 and has been implicated in multiple cancers including, for example, malignant melanoma. Malignant melanoma is a cutaneous neoplasm of melanocytes. Melanomas generally have features of asymmetry, irregular border, variegated color, and diameter greater than 6 mm. The precise cause of melanoma is unknown, but sunlight and heredity are risk factors. Melanoma has been increasing during the past few decades.

The CDKN2A gene has been found to be homozygously deleted at high frequency in cell lines derived from tumors of lung, breast, brain, bone, skin, bladder, kidney, ovary, and lymphocyte. Melanoma cell lines carried at least one copy of CDKN2A in combination with a deleted allele. Melanoma cell lines that carried at least 1 copy of CDKN2A frequently showed nonsense, missense, or frameshift mutations in the gene. Thus, CDKN2A may rival p53 (see Example 5) in the universality of its involvement in tumorigenesis. The attached table discloses the correcting oligonucleotide base sequences for the CDKN2A oligonucleotides of the invention.

Table 17

CDKN2A Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Melanoma Trp15Term TGG-TAG	GGGCGGCGGGGAGCAGCATGGAGCCGGCGGCGGGGAGCAG CATGGAGCCTTCGGCTGACTGGCTGGCCACGGCCGCGGCCC GGGGTCGGGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGG	1321
	CCCGCCTCCAGCAGCGCCCGCACCTCCTCTACCCGACCCCG GGCCGCGGCCGTGGCCAGCCAGTCAGCCGAAGGCTCCATGC TGCTCCCCGCCGCCGGCTCCATGCTGCTCCCCGCCGCCC	1322
	GGCTGACTGGCTGGCCA	1323
	TGGCCAGCCAGTCAGCC	1324
Melanoma Leu16Pro CTG-CCG	CGGCGGGGAGCAGCATGGAGCCGGCGGCGGGGAGCAGCAT GGAGCCTTCGGCTGACTGGCTGGCCACGGCCGCGGCCCGGG GTCGGGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGGC	1325
	GCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTACCCGACC CCGGGCCGCGGCCGTGGCCAGCCAGTCAGCCGAAGGCTCCA TGCTGCTCCCCGCCGCCGGCTCCATGCTGCTCCCCGCCG	1326

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGACTGGCTGGCCACGG	1327
	CCGTGGCCAGCCAGTCA	1328
Melanoma Gly23Asp GGT-GAT	CGGCGGCGGGGAGCAGCATGGAGCCTTCGGCTGACTGGCTG GCCACGGCCGCGGCCCGGGGTCGGGTAGAGGAGGTGCGGG CGCTGCTGGAGGCGGGGGCGCTGCCCAACGCACCGAATAG	1329
	CTATTCGGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGCGC CCGCACCTCCTCTACCCGACCCCGGGCCGCGGCCGTGGCCA GCCAGTCAGCCGAAGGCTCCATGCTGCTCCCCGCCGCCG	1330
	GGCCCGGGGTCGGGTAG	1331
	CTACCCGACCCCGGGCC	1332
Melanoma Arg24Pro CGG-CCG	CGGCGGGGAGCAGCATGGAGCCTTCGGCTGACTGGCTGGCC ACGGCCGCGGCCCGGGGTCGGGTAGAGGAGGTGCGGGCGC TGCTGGAGGCGGGGGCGCTGCCCAACGCACCGAATAGTTA	1333
	TAACTATTCGGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGC GCCCGCACCTCCTCTACCCGACCCCGGGCCGCGGCCGTGGC CAGCCAGTCAGCCGAAGGCTCCATGCTGCTCCCCGCCG	1334
	CCGGGGTCGGGTAGAGG	1335
	CCTCTACCCGACCCCGG	1336
Melanoma Leu32Pro CTG-CCG	CGGCTGACTGGCTGGCCACGGCCGCGGCCCGGGGTCGGGT AGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGGCGCTGCCC AACGCACCGAATAGTTACGGTCGGAGGCCGATCCAGGTGGG	1337
	CCCACCTGGATCGGCCTCCGACCGTAACTATTCGGTGCGTTG GGCAGCGCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTAC CCGACCCCGGGCCGCGGCCGTGGCCAGCCAGTCAGCCG	1338
	GGCGCTGCTGGAGGCGG	1339
	CCGCCTCCAGCAGCGCC	1340
Melanoma Gly35Ala GGG-GCG	GGCTGGCCACGGCCGCGGCCCGGGGTCGGGTAGAGGAGGT GCGGGCGCTGCTGGAGGCGGGGGCGCTGCCCAACGCACCG AATAGTTACGGTCGGAGGCCGATCCAGGTGGGTAGAGGGTC	1341
	GACCCTCTACCCACCTGGATCGGCCTCCGACCGTAACTATTC GGTGCGTTGGGCAGCGCCCCCGCCTCCAGCAGCGCCCGCAC CTCCTCTACCCGACCCCGGGCCGCGGCCGTGGCCAGCC	1342
	GGAGGCGGGGGCGCTGC	1343
	GCAGCGCCCCCGCCTCC	1344
Melanoma Tyr44Term TACg-TAA	GGTAGAGGAGGTGCGGGCGCTGCTGGAGGCGGGGGCGCTG CCCAACGCACCGAATAGTTACGGTCGGAGGCCGATCCAGGTG GGTAGAGGGTCTGCAGCGGGAGCAGGGGATGGCGGGCGA	1345

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCGCCCGCCATCCCCTGCTCCCGCTGCAGACCCTCTACCCAC CTGGATCGGCCTCCGACCGTAACTATTCGGTGCGTTGGGCAG CGCCCCCGCCTCCAGCAGCGCCCGCACCTCCTCTACC	1346
	AATAGTTACGGTCGGAG	1347
	CTCCGACCGTAACTATT	1348
Melanoma Met53Ile ATGa-ATC	TCTCCCATACCTGCCCCCACCCTGGCTCTGACCACTCTGCTCT CTCTGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGGA GCTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCA	1349
	TGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCTCCG CCACGCGGGCGCTGCCCATCATCATGACCTGCCAGAGAGAG CAGAGTGGTCAGAGCCAGGGTGGGGGCAGGTATGGGAGA	1350
	GTCATGATGATGGGCAG	1351
	CTGCCCATCATCATGAC	1352
Melanoma Met54Ile ATGg-ATT	CCCATACCTGCCCCCACCCTGGCTCTGACCACTCTGCTCTCTC TGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGGAGCT GCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCAGAC	1353
	GTCTGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT CCGCCACGCGGGCGCTGCCCATCATCATGACCTGCCAGAGA GAGCAGAGTGGTCAGAGCCAGGGTGGGGGCAGGTATGGG	1354
	ATGATGATGGGCAGCGC	1355
	GCGCTGCCCATCATCAT	1356
Melanoma Ser56Ile AGC-ATC	GCCGGCCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGC AGGTCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTG CTGCTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGC	1357
	GCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCA GCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGACCTGCC AGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGGCCGGC	1358
	GATGGGCAGCGCCCGAG	1359
	CTCGGGCGCTGCCCATC	1360
Melanoma Ala57Val GCC-GTC	GGCCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGG TCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTG CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCAC	1361
	GTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCA GCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGACCT GCCAGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGGCC	1362
	GGGCAGCGCCCGAGTGG	1363
	CCACTCGGGCGCTGCCC	1364

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Melanoma Arg58Term cCGA-TGA	CCCCCACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGGTCA TGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGCTC CACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTC	1365
	GAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG CAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGAC CTGCCAGAGAGAACAGAATGGTCAGAGCCAGGGTGGGGG	1366
	GCAGCGCCCGAGTGGCG	1367
	CGCCACTCGGGCGCTGC	1368
Melanoma Val59Gly GTG-GGG	CACCCTGGCTCTGACCATTCTGTTCTCTCTGGCAGGTCATGATT GATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGCTCCACG GCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCAC	1369
	GTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTG GAGCAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCAT GACCTGCCAGAGAGAACAGAATGGTCAGAGCCAGGGTG	1370
	CGCCCGAGTGGCGGAGC	1371
	GCTCCGCCACTCGGGCG	1372
Melanoma Leu62Pro CTG-CCG	TCTGACCACTCTGCTCTCTCTGGCAGGTCATGATGATGGGCA GCGCCCGCGTGGCGGAGCTGCTGCTGCTCCACGGCGCGGAG CCCAACTGCGCAGACCCTGCCACTCTCACCCGACCGGT	1373
	ACCGGTCGGGTGAGAGTGGCAGGGTCTGCGCAGTTGGGCTC CGCGCCGTGGAGCAGCAGCAGCTCCGCCACGCGGGCGCTGC CCATCATCATGACCTGCCAGAGAGAGCAGAGTGGTCAGA	1374
	GGCGGAGCTGCTGCTGC	1375
	GCAGCAGCAGCTCCGCC	1376
Melanoma Ala68Val GCG-GTG	TCTGGCAGGTCATGATGATGGGCAGCGCCCGCGTGGCGGAG CTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCAGACCC TGCCACTCTCACCCGACCGGTGCATGATGCTGCCCGGGA	1377
	TCCCGGGCAGCATCATGCACCGGTCGGGTGAGAGTGGCAGG GTCTGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT CCGCCACGCGGGCGCTGCCCATCATCATGACCTGCCAGA	1378
	CCACGGCGCGGAGCCCA	1379
	TGGGCTCCGCGCCGTGG	1380
Melanoma Asn71Lys AACt-AAA	CATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTGC TCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTC ACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTG	1381
	CAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGA GAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG CAGCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATG	1382

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGCCCAACTGCGCCGA	1383
	TCGGCGCAGTTGGGCTC	1384
Melanoma Asn71Ser AAC-AGC	TCATGATGATGGGCAGCGCCCGAGTGGCGGAGCTGCTGCTG CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCT CACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCT	1385
	AGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAG AGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCA GCAGCAGCTCCGCCACTCGGGCGCTGCCCATCATCATGA	1386
	GGAGCCCAACTGCGCCG	1387
	CGGCGCAGTTGGGCTCC	1388
Melanoma Pro81Leu CCC-CTC	AGCTGCTGCTGCTCCACGGCGCGGAGCCCAACTGCGCCGAC CCCGCCACTCTCACCCGACCCGTGCACGACGCTGCCCGGGA GGGCTTCCTGGACACGCTGGTGGTGCTGCACCGGGCCGG	1389
	CCGGCCCGGTGCAGCACCACCAGCGTGTCCAGGAAGCCCTC CCGGGCAGCGTCGTGCACGGGTCGGGTGAGAGTGGCGGGG TCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAGCAGCAGCT	1390
	CACCCGACCCGTGCACG	1391
	CGTGCACGGGTCGGGTG	1392
Melanoma Asp84Tyr cGAC-TAC	CTGCTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCAC TCTCACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCT GGACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGC	1393
	GCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGTCCAGG AAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAGAGT GGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAGCAG	1394
	CCGTGCACGACGCTGCC	1395
	GGCAGCGTCGTGCACGG	1396
Melanoma Ala85Thr cGCT-ACT	CTCCACGGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCT CACCCGACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGG ACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGG	1397
	CCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGTCC AGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCGGGTGAG AGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCCGTGGAG	1398
	TGCACGACGCTGCCCGG	1399
	CCGGGCAGCGTCGTGCA	1400
Melanoma Arg87Pro CGG-CCG	GCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCACCCGA CCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGGACACGCT GGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGCG	1401

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAG CGTGTCCAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTC GGGTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGC	1402
	CGCTGCCCGGGAGGGCT	1403
	AGCCCTCCCGGGCAGCG	1404
Melanoma Arg87Trp cCGG-TGG	GGCGCGGAGCCCAACTGCGCCGACCCCGCCACTCTCACCCG ACCCGTGCACGACGCTGCCCGGGAGGGCTTCCTGGACACGC TGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGC	1405
	GCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGC GTGTCCAGGAAGCCCTCCCGGGCAGCGTCGTGCACGGGTCG GGTGAGAGTGGCGGGGTCGGCGCAGTTGGGCTCCGCGCC	1406
	ACGCTGCCCGGGAGGGC	1407
	GCCCTCCCGGGCAGCGT	1408
Melanoma Leu97Arg CTG-CGG	CTCTCACCCGACCGGTGCATGATGCTGCCCGGGAGGGCTTCC TGGACACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCTG GACGTGCGCGATGCCTGGGGTCGTCTGCCCGTGGACTT	1409
	AAGTCCACGGGCAGACGACCCCAGGCATCGCGCACGTCCAG CCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGTCCAGGA AGCCCTCCCGGGCAGCATCATGCACCGGTCGGGTGAGAG	1410
	GGTGGTGCTGCACCGGG	1411
	CCCGGTGCAGCACCACC	1412
Melanoma Arg99Pro CGG-CCG	CCCGACCGGTGCATGATGCTGCCCGGGAGGGCTTCCTGGAC ACGCTGGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGT GCGCGATGCCTGGGGTCGTCTGCCCGTGGACTTGGCCGA	1413
	TCGGCCAAGTCCACGGGCAGACGACCCCAGGCATCGCGCAC GTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAGCGTGT CCAGGAAGCCCTCCCGGGCAGCATCATGCACCGGTCGGG	1414
	GCTGCACCGGGCCGGGG	1415
	CCCCGGCCCGGTGCAGC	1416
Melanoma Gly101Trp cGGG-TGG	CCGGTGCATGATGCTGCCCGGGAGGGCTTCCTGGACACGCT GGTGGTGCTGCACCGGGCCGGGGCGCGGCTGGACGTGCGC GATGCCTGGGGTCGTCTGCCCGTGGACTTGGCCGAGGAGC	1417
	GCTCCTCGGCCAAGTCCACGGGCAGACGACCCCAGGCATCG CGCACGTCCAGCCGCGCCCCGGCCCGGTGCAGCACCACCAG CGTGTCCAGGAAGCCCTCCCGGGCAGCATCATGCACCGG	1418
	ACCGGGCCGGGGCGCGG	1419
	CCGCGCCCCGGCCCGGT	1420

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Melanoma Arg107Cys gCGC-TGC	CGGGAGGGCTTCCTGGACACGCTGGTGGTGCTGCACCGGGC CGGGGCGCGGCTGGACGTGCGCGATGCCTGGGGTCGTCTGC CCGTGGACTTGGCCGAGGAGCGGGGCCACCGCGACGTTG	1421
	CAACGTCGCGGTGGCCCCGCTCCTCGGCCAAGTCCACGGGC AGACGACCCCAGGCATCGCGCACGTCCAGCCGCGCCCCGGC CCGGTGCAGCACCACCAGCGTGTCCAGGAAGCCCTCCCG	1422
	TGGACGTGCGCGATGCC	1423
	GGCATCGCGCACGTCCA	1424
Melanoma Ala118Thr gGCT-ACT	CACGGGGCCGGGGCGCGGCTGGACGTGCGCGATGCCTGGG GCCGTCTGCCCGTGGACCTGGCTGAGGAGCTGGGCCATCGC GATGTCGCACGGTACCTGCGCGCGGCTGCGGGGGGCACCA	1425
	TGGTGCCCCCCGCAGCCGCGCGCAGGTACCGTGCGACATCG CGATGGCCCAGCTCCTCAGCCAGGTCCACGGGCAGACGGCC CCAGGCATCGCGCACGTCCAGCCGCGCCCCGGCCCGGTG	1426
	TGGACCTGGCTGAGGAG	1427
	CTCCTCAGCCAGGTCCA	1428
Melanoma Val126Asp GTC-GAC	TGCGCGATGCCTGGGGCCGTCTGCCCGTGGACCTGGCTGAG GAGCTGGGCCATCGCGATGTCGCACGGTACCTGCGCGCGGC TGCGGGGGGCACCAGAGGCAGTAACCATGCCCGCATAGA	1429
	TCTATGCGGGCATGGTTACTGCCTCTGGTGCCCCCCGCAGCC GCGCGCAGGTACCGTGCGACATCGCGATGGCCCAGCTCCTC AGCCAGGTCCACGGGCAGACGGCCCCAGGCATCGCGCA	1430
	TCGCGATGTCGCACGGT	1431
	ACCGTGCGACATCGCGA	1432

EXAMPLE 11

Adenomatous polyposis of the colon – APC

Adenomatous polyposis of the colon is characterized by adenomatous polyps of the colon and rectum; in extreme cases the bowel is carpeted with a myriad of polyps. This is a viciously premalignant disease with one or more polyps progressing through dysplasia to malignancy in untreated gene carriers with a median age at diagnosis of 40 years.

Mutations in the APC gene are an initiating event for both familial and sporadic colorectal tumorigenesis and many alleles of the APC gene have been identified. Carcinoma may arise at any age from late childhood through the seventh decade with presenting features including, for example, weight loss and

inanition, bowel obstruction, or bloody diarrhea. Cases of new mutation still present in these ways but in areas with well organized registers most other gene carriers are detected. The attached table discloses the correcting oligonucleotide base sequences for the APC oligonucleotides of the invention.

5

Table 18

APC Mutations And Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Arg121Term AGA-TGA	GGATCTGTATCAAGCCGTTCTGGAGAGTGCAGTCCTGTTCCT ATGGGTTCATTTCCAAGAAGAGGGTTTGTAAATGGAAGCAGA GAAAGTACTGGATATTTAGAAGAACTTGAGAAAGAGA	1433
	TCTCTTTCTCAAGTTCTTCTAAATATCCAGTACTTTCTCTGCTT CCATTTACAAACCCTCTTCTTGGAAATGAACCCATAGGAACAG GACTGCACTCTCCAGAACGGCTTGATACAGATCC	1434
	TTCCAAGAAGAGGGTTT	1435
	AAACCCTCTTCTTGGAA	1436
Adenomatous polyposis coli Trp157Term TGG-TAG	AAAAAAAAAATAGGTCATTGCTTCTTGCTGATCTTGACAAAGA AGAAAAGGAAAAAGACTGGTATTACGCTCAACTTCAGAATCTC ACTAAAAGAATAGATAGTCTTCCTTTAACTGAAAA	1437
	TTTTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGAGATTCTG AAGTTGAGCGTAATACCAGTCTTTTTCCTTTTCTTCTTTGTCAA GATCAGCAAGAAGCAATGACCTATTTTTTTTTT	1438
	AAAAGACTGGTATTACG	1439
	CGTAATACCAGTCTTTT	1440
Adenomatous polyposis coli Tyr159Term TAC-TAG	AAATAGGTCATTGCTTCTTGCTGATCTTGACAAAGAAGAAAAG GAAAAAGACTGGTATTACGCTCAACTTCAGAATCTCACTAAAA GAATAGATAGTCTTCCTTTAACTGAAAATGTAAGT	1441
	ACTTACATTTTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGA GATTCTGAAGTTGAGCGTAATACCAGTCTTTTTCCTTTTCTTCT TTGTCAAGATCAGCAAGAAGCAATGACCTATTT	1442
	TGGTATTACGCTCAACT	1443
	AGTTGAGCGTAATACCA	1444
Adenomatous polyposis coli Gln163Term CAG-TAG	TTGCTTCTTGCTGATCTTGACAAAGAAGAAAAGGAAAAAGACT GGTATTACGCTCAACTTCAGAATCTCACTAAAAGAATAGATAG TCTTCCTTTAACTGAAAATGTAAGTAACTGGCAGT	1445
	ACTGCCAGTTACTTACATTTTCAGTTAAAGGAAGACTATCTATT CTTTTAGTGAGATTCTGAAGTTGAGCGTAATACCAGTCTTTTT CCTTTTCTTCTTTGTCAAGATCAGCAAGAAGCAA	1446
	CTCAACTTCAGAATCTC	1447
	GAGATTCTGAAGTTGAG	1448

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Lys586Term AAA-TAA	GACAAATTCCAACTCTAATTAGATGACCCATATTCTGTTTCTTA CTAGGAATCAACCCTCAAAAGCGTATTGAGTGCCTTATGGAAT TTGTCAGCACATTGCACTGAGAATAAAGCTGATA	1513
	TATCAGCTTTATTCTCAGTGCAATGTGCTGACAAATTCCATAA GGCACTCAATACGCTTTTGAGGGTTGATTCCTAGTAAGAAACA GAATATGGGTCATCTAATTAGAGTTGGAATTTGTC	1514
	CAACCCTCAAAAGCGTA	1515
	TACGCTTTTGAGGGTTG	1516
Adenomatous polyposis coli Leu592Term TTA-TGA	TAGATGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAA AAGCGTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACT GAGAATAAAGCTGATATATGTGCTGTAGATGGTGC	1517
	GCACCATCTACAGCACATATATCAGCTTTATTCTCAGTGCAAT GTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGAGGGT TGATTCCTAGTAAGAAACAGAATATGGGTCATCTA	1518
	GAGTGCCTTATGGAATT	1519
	AATTCCATAAGGCACTC	1520
Adenomatous polyposis coli Trp593Term TGG-TAG	ATGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAAAAG CGTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACTGAG AATAAAGCTGATATATGTGCTGTAGATGGTGCACT	1521
	AGTGCACCATCTACAGCACATATATCAGCTTTATTCTCAGTGC AATGTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGAG GGTTGATTCCTAGTAAGAAACAGAATATGGGTCAT	1522
	TGCCTTATGGAATTTGT	1523
	ACAAATTCCATAAGGCA	1524
Adenomatous polyposis coli Trp593Term TGG-TGA	TGACCCATATTCTGTTTCTTACTAGGAATCAACCCTCAAAAGC GTATTGAGTGCCTTATGGAATTTGTCAGCACATTGCACTGAGA ATAAAGCTGATATATGTGCTGTAGATGGTGCACTT	1525
	AAGTGCACCATCTACAGCACATATATCAGCTTTATTCTCAGTG CAATGTGCTGACAAATTCCATAAGGCACTCAATACGCTTTTGA GGGTTGATTCCTAGTAAGAAACAGAATATGGGTCA	1526
	GCCTTATGGAATTTGTC	1527
	GACAAATTCCATAAGGC	1528
Adenomatous polyposis coli Tyr622Term TAC-TAA	TAAAGCTGATATATGTGCTGTAGATGGTGCACTTGCATTTTTG GTTGGCACTCTTACTTACCGGAGCCAGACAAACACTTTAGCC ATTATTGAAAGTGGAGGTGGGATATTACGGAATGTG	1529
	CACATTCCGTAATATCCCACCTCCACTTTCAATAATGGCTAAA GTGTTTGTCTGGCTCCGGTAAGTAAGAGTGCCAACCAAAAAT GCAAGTGCACCATCTACAGCACATATATCAGCTTTA	1530
	CTTACTTACCGGAGCCA	1531
	TGGCTCCGGTAAGTAAG	1532
Adenomatous polyposis coli Gln625Term CAG-TAG	GATATATGTGCTGTAGATGGTGCACTTGCATTTTTGGTTGGCA CTCTTACTTACCGGAGCCAGACAAACACTTTAGCCATTATTGA AAGTGGAGGTGGGATATTACGGAATGTGTCCAGCT	1533

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTATTCCTAATAGCTCTTCGCTGTTTTATCACTTAGAAACAAG TAACTTACCTGTGCTCGTTTTTCCATATCCTGGCAGGTACCTA GTTGTTCTTCCATCGCAACTCTGATTTGCCTTGC	1470
	TGGAAAAACGAGCACAG	1471
	CTGTGCTCGTTTTTCCA	1472
Adenomatous polyposis coli Arg232Term CGA-TGA	GTTTTATTTTAGCGAAGAATAGCCAGAATTCAGCAAATCGAAA AGGACATACTTCGTATACGACAGCTTTTACAGTCCCAAGCAAC AGAAGCAGAGGTTAGTAAATTGCCTTTCTTGTTTG	1473
	CAAACAAGAAAGGCAATTTACTAACCTCTGCTTCTGTTGCTTG GGACTGTAAAAGCTGTCGTATACGAAGTATGTCCTTTTCGATT TGCTGAATTCTGGCTATTCTTCGCTAAAATAAAAC	1474
	TTCGTATACGACAGCTT	1475
	AAGCTGTCGTATACGAA	1476
Adenomatous polyposis coli Gln233Term CAG-TAG	TTATTTTAGCGAAGAATAGCCAGAATTCAGCAAATCGAAAAGG ACATACTTCGTATACGACAGCTTTTACAGTCCCAAGCAACAGA AGCAGAGGTTAGTAAATTGCCTTTCTTGTTTGTGG	1477
	CCACAAACAAGAAAGGCAATTTACTAACCTCTGCTTCTGTTGC TTGGGACTGTAAAAGCTGTCGTATACGAAGTATGTCCTTTTCG ATTTGCTGAATTCTGGCTATTCTTCGCTAAAATAA	1478
	GTATACGACAGCTTTTA	1479
	TAAAAGCTGTCGTATAC	1480
Adenomatous polyposis coli Gln247Term CAG-TAG	AGAAAGCCTACACCATTTTTGCATGTACTGATGTTAACTCCAT CTTAACAGAGGTCATCTCAGAACAAGCATGAAACCGGCTCAC ATGATGCTGAGCGGCAGAATGAAGGTCAAGGAGTGG	1481
	CCACTCCTTGACCTTCATTCTGCCGCTCAGCATCATGTGAGCC GGTTTCATGCTTGTTCTGAGATGACCTCTGTTAAGATGGAGTT AACATCAGTACATGCAAAAATGGTGTAGGCTTTCT	1482
	GGTCATCTCAGAACAAG	1483
	CTTGTTCTGAGATGACC	1484
Adenomatous polyposis coli Gly267Term GGA-TGA	CAGAACAAGCATGAAACCGGCTCACATGATGCTGAGCGGCA GAATGAAGGTCAAGGAGTGGGAGAAATCAACATGGCAACTTC TGGTAATGGTCAGGTAAATAAATTATTTTATCATATTT	1485
	AAATATGATAAAATAATTTATTTACCTGACCATTACCAGAAGTT GCCATGTTGATTTCTCCCACTCCTTGACCTTCATTCTGCCGCT CAGCATCATGTGAGCCGGTTTCATGCTTGTTCTG	1486
	AAGGAGTGGGAGAAATC	1487
	GATTTCTCCCACTCCTT	1488
Adenomatous polyposis coli Glu443Term GAA-TAA	CTTCAAATAACAAAGCATTATGGTTTATGTTGATTTTATTTTTCA GTGCCAGCTCCTGTTGAACATCAGATCTGTCCTGCTGTGTGT GTTCTAATGAAACTTTCATTTGATGAAGAGCATA	1489
	TATGCTCTTCATCAAATGAAAGTTTCATTAGAACACACACAGC AGGACAGATCTGATGTTCAACAGGAGCTGGCACTGAAAAATA AAATCAACATAAACCATAATGCTTTGTTATTTGAAG	1490
	CTCCTGTTGAACATCAG	1491

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTGATGTTCAACAGGAG	1492
Adenomatous polyposis coli SER457TER TCA-TAA	CAGTGCCAGCTCCTGTTGAACATCAGATCTGTCCTGCTGTGT GTGTTCTAATGAAACTTTCATTTGATGAAGAGCATAGACATGC AATGAATGAACTAGGTAAGACAAAAATGTTTTTTAA	1493
	TTAAAAAACATTTTTGTCTTACCTAGTTCATTCATTGCATGTCT ATGCTCTTCATCAAATGAAAGTTTCATTAGAACACACACAGCA GGACAGATCTGATGTTCAACAGGAGCTGGCACTG	1494
	GAAACTTTCATTTGATG	1495
	CATCAAATGAAAGTTTC	1496
Adenomatous polyposis coli Gln473Term CAG-TAG	AGTTGTTTTATTTTAGATGATTGTCTTTTTCCTCTTGCCCTTTTT AAATTAGGGGGACTACAGGCCATTGCAGAATTATTGCAAGTG GACTGTGAAATGTACGGGCTTACTAATGACCACT	1497
	AGTGGTCATTAGTAAGCCCGTACATTTCACAGTCCACTTGCAA TAATTCTGCAATGGCCTGTAGTCCCCCTAATTTAAAAAGGGCA AGAGGAAAAAGACAATCATCTAAAATAAAACAACT	1498
	GGGGACTACAGGCCATT	1499
	AATGGCCTGTAGTCCCC	1500
Adenomatous polyposis coli Tyr486Term TAC-TAG	TTTTAAATTAGGGGGACTACAGGCCATTGCAGAATTATTGCAA GTGGACTGTGAAATGTACGGGCTTACTAATGACCACTACAGT ATTACACTAAGACGATATGCTGGAATGGCTTTGACA	1501
	TGTCAAAGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAG TGGTCATTAGTAAGCCCGTACATTTCACAGTCCACTTGCAATA ATTCTGCAATGGCCTGTAGTCCCCCTAATTTAAAA	1502
	GAAATGTACGGGCTTAC	1503
	GTAAGCCCGTACATTTC	1504
Adenomatous polyposis coli Arg499Term CGA-TGA	TTGCAAGTGGACTGTGAAATGTATGGGCTTACTAATGACCACT ACAGTATTACACTAAGACGATATGCTGGAATGGCTTTGACAAA CTTGACTTTTGGAGATGTAGCCAACAAGGTATGTT	1505
	AACATACCTTGTTGGCTACATCTCCAAAAGTCAAGTTTGTCAA AGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAGTGGTCA TTAGTAAGCCCATACATTTCACAGTCCACTTGCAA	1506
	CACTAAGACGATATGCT	1507
	AGCATATCGTCTTAGTG	1508
Adenomatous polyposis coli Tyr500Term TAT-TAG	AGTGGACTGTGAAATGTATGGGCTTACTAATGACCACTACAGT ATTACACTAAGACGATATGCTGGAATGGCTTTGACAAACTTGA CTTTTGGAGATGTAGCCAACAAGGTATGTTTTTAT	1509
	ATAAAAACATACCTTGTTGGCTACATCTCCAAAAGTCAAGTTT GTCAAAGCCATTCCAGCATATCGTCTTAGTGTAATACTGTAGT GGTCATTAGTAAGCCCATACATTTCACAGTCCACT	1510
	AGACGATATGCTGGAAT	1511
	ATTCCAGCATATCGTCT	1512

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGCTGGACACATTCCGTAATATCCCACCTCCACTTTCAATAAT GGCTAAAGTGTTTGTCTGGCTCCGGTAAGTAAGAGTGCCAAC CAAAAATGCAAGTGCACCATCTACAGCACATATATC	1534
	ACCGGAGCCAGACAAAC	1535
	GTTTGTCTGGCTCCGGT	1536
Adenomatous polyposis coli Leu629Term TTA-TAA	TAGATGGTGCACTTGCATTTTTGGTTGGCACTCTTACTTACCG GAGCCAGACAAACACTTTAGCCATTATTGAAAGTGGAGGTGG GATATTACGGAATGTGTCCAGCTTGATAGCTACAAA	1537
	TTTGTAGCTATCAAGCTGGACACATTCCGTAATATCCCACCTC CACTTTCAATAATGGCTAAAGTGTTTGTCTGGCTCCGGTAAGT AAGAGTGCCAACCAAAAATGCAAGTGCACCATCTA	1538
	AAACACTTTAGCCATTA	1539
	TAATGGCTAAAGTGTTT	1540
Adenomatous polyposis coli Glu650Term GAG-TAG	GCCATTATTGAAAGTGGAGGTGGGATATTACGGAATGTGTCC AGCTTGATAGCTACAAATGAGGACCACAGGTATATATAGAGTT TTATATTACTTTTAAAGTACAGAATTCATACTCTCA	1541
	TGAGAGTATGAATTCTGTACTTTAAAAGTAATATAAAACTCTAT ATATACCTGTGGTCCTCATTTGTAGCTATCAAGCTGGACACAT TCCGTAATATCCCACCTCCACTTTCAATAATGGC	1542
	CTACAAATGAGGACCAC	1543
	GTGGTCCTCATTTGTAG	1544
Adenomatous polyposis coli Trp699Term TGG-TGA	TGCATGTGGAACTTTGTGGAATCTCTCAGCAAGAAATCCTAAA GACCAGGAAGCATTATGGGACATGGGGGCAGTTAGCATGCT CAAGAACCTCATTCATTCAAAGCACAAAATGATTGCT	1545
	AGCAATCATTTTGTGCTTTGAATGAATGAGGTTCTTGAGCATG CTAACTGCCCCCATGTCCCATAATGCTTCCTGGTCTTTAGGAT TTCTTGCTGAGAGATTCCACAAAGTTCCACATGCA	1546
	GCATTATGGGACATGGG	1547
	CCCATGTCCCATAATGC	1548
Adenomatous polyposis coli Ser713Term TCA-TGA	AAGACCAGGAAGCATTATGGGACATGGGGGCAGTTAGCATG CTCAAGAACCTCATTCATTCAAAGCACAAAATGATTGCTATGG GAAGTGCTGCAGCTTTAAGGAATCTCATGGCAAATAG	1549
	CTATTTGCCATGAGATTCCTTAAAGCTGCAGCACTTCCCATAG CAATCATTTTGTGCTTTGAATGAATGAGGTTCTTGAGCATGCT AACTGCCCCCATGTCCCATAATGCTTCCTGGTCTT	1550
	CATTCATTCAAAGCACA	1551
	TGTGCTTTGAATGAATG	1552
Adenomatous polyposis coli Ser722Gly AGT-GGT	GGGGCAGTTAGCATGCTCAAGAACCTCATTCATTCAAAGCAC AAAATGATTGCTATGGGAAGTGCTGCAGCTTTAAGGAATCTCA TGGCAAATAGGCCTGCGAAGTACAAGGATGCCAATA	1553
	TATTGGCATCCTTGTACTTCGCAGGCCTATTTGCCATGAGATT CCTTAAAGCTGCAGCACTTCCCATAGCAATCATTTTGTGCTTT GAATGAATGAGGTTCTTGAGCATGCTAACTGCCCC	1554

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTATGGGAAGTGCTGCA	1555
	TGCAGCACTTCCCATAG	1556
Adenomatous polyposis coli Leu764Term TTA-TAA	TCTCCTGGCTCAGCTTGCCATCTCTTCATGTTAGGAAACAAAA AGCCCTAGAAGCAGAATTAGATGCTCAGCACTTATCAGAAACT TTTGACAATATAGACAATTTAAGTCCCAAGGCATC	1557
	GATGCCTTGGGACTTAAATTGTCTATATTGTCAAAAGTTTCTG ATAAGTGCTGAGCATCTAATTCTGCTTCTAGGGCTTTTTGTTT CCTAACATGAAGAGATGGCAAGCTGAGCCAGGAGA	1558
	AGCAGAATTAGATGCTC	1559
	GAGCATCTAATTCTGCT	1560
Adenomatous polyposis coli Ser784Thr TCT-ACT	TTAGATGCTCAGCACTTATCAGAAACTTTTGACAATATAGACA ATTTAAGTCCCAAGGCATCTCATCGTAGTAAGCAGAGACACA GCAAGTCTCTATGGTGATTATGTTTTTGACACCATC	1561
	GATGGTGTCAAAAACATAATCACCATAGAGACTTGCTGTGTCT CTGCTTACTACGATGAGATGCCTTGGGACTTAAATTGTCTATA TTGTCAAAAGTTTCTGATAAGTGCTGAGCATCTAA	1562
	CCAAGGCATCTCATCGT	1563
	ACGATGAGATGCCTTGG	1564
Adenomatous polyposis coli Arg805Term CGA-TGA	CTCATCGTAGTAAGCAGAGACACAGCAAGTCTCTATGGTGATT ATGTTTTTGACACCAATCGACATGATGATAATAGGTCAGACAT TTTAATACTGGCACATGACTGTCCTTTCACCATAT	1565
	ATATGGTGAAAGGACAGTCATGTGCCAGTATTAAAATGTCTGA CCTATTATCATCATGTCGATTGGTGTCAAAAACATAATCACCAT AGAGACTTGCTGTGTCTCTGCTTACTACGATGAG	1566
	ACACCAATCGACATGAT	1567
	ATCATGTCGATTGGTGT	1568
Adenomatous polyposis coli Gln879Term CAG-TAG	GGTCTAGGCAACTACCATCCAGCAACAGAAAATCCAGGAACT TCTTCAAAGCGAGGTTTGCAGATCTCCACCACTGCAGCCCAG ATTGCCAAAGTCATGGAAGAAGTGTCAGCCATTCATA	1569
	TATGAATGGCTGACACTTCTTCCATGACTTTGGCAATCTGGGC TGCAGTGGTGGAGATCTGCAAACCTCGCTTTGAAGAAGTTCC TGGATTTTCTGTTGCTGGATGGTAGTTGCCTAGACC	1570
	GAGGTTTGCAGATCTCC	1571
	GGAGATCTGCAAACCTC	1572
Adenomatous polyposis coli Ser932Term TCA-TAA	TACATTGTGTGACAGATGAGAGAAATGCACTTAGAAGAAGCT CTGCTGCCCATACACATTCAAACACTTACAATTTCACTAAGTC GGAAAATTCAAATAGGACATGTTCTATGCCTTATGC	1573
	GCATAAGGCATAGAACATGTCCTATTTGAATTTTCCGACTTAG TGAAATTGTAAGTGTTTGAATGTGTATGGGCAGCAGAGCTTCT TCTAAGTGCATTTCTCTCATCTGTCACACAATGTA	1574
	TACACATTCAAACACTT	1575
	AAGTGTTTGAATGTGTA	1576

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Arg168Term AGA-TGA	CTTGACAAAGAAGAAAAGGAAAAAGACTGGTATTACGCTCAA CTTCAGAATCTCACTAAAAGAATAGATAGTCTTCCTTTAACTGA AAATGTAAGTAACTGGCAGTACAACTTATTTGAAA	1449
	TTTCAAATAAGTTGTACTGCCAGTTACTTACATTTTCAGTTAAA GGAAGACTATCTATTCTTTTAGTGAGATTCTGAAGTTGAGCGT AATACCAGTCTTTTTCCTTTTCTTCTTTGTCAAG	1450
	TCACTAAAAGAATAGAT	1451
	ATCTATTCTTTTAGTGA	1452
Adenomatous polyposis coli Ser171Ile AGT-ATT	AAGAAAAGGAAAAAGACTGGTATTACGCTCAACTTCAGAATCT CACTAAAAGAATAGATAGTCTTCCTTTAACTGAAAATGTAAGTA ACTGGCAGTACAACTTATTTGAAACTTTAATAAC	1453
	GTTATTAAAGTTTCAAATAAGTTGTACTGCCAGTTACTTACATT TTCAGTTAAAGGAAGACTATCTATTCTTTTAGTGAGATTCTGAA GTTGAGCGTAATACCAGTCTTTTTCCTTTTCTT	1454
	AATAGATAGTCTTCCTT	1455
	AAGGAAGACTATCTATT	1456
Adenomatous polyposis coli Gln181Term CAA-TAA	GATTAACGTAAATACAAGATATTGATACTTTTTTATTATTTGTG GTTTTAGTTTTCCTTACAAACAGATATGACCAGAAGGCAATTG GAATATGAAGCAAGGCAAATCAGAGTTGCGATGG	1457
	CCATCGCAACTCTGATTTGCCTTGCTTCATATTCCAATTGCCTT CTGGTCATATCTGTTTGTAAGGAAAACTAAAACCACAAATAAT AAAAAAGTATCAATATCTTGTATTTACGTTAATC	1458
	TTTCCTTACAAACAGAT ATCTGTTTGTAAGGAAA	1459 1460
Adenomatous polyposis coli Glu190Term GAA-TAA	CTTTTTTATTATTTGTGGTTTTAGTTTTCCTTACAAACAGATATG ACCAGAAGGCAATTGGAATATGAAGCAAGGCAAATCAGAGTT GCGATGGAAGAACAACTAGGTACCTGCCAGGATA	1461
	TATCCTGGCAGGTACCTAGTTGTTCTTCCATCGCAACTCTGAT TTGCCTTGCTTCATATTCCAATTGCCTTCTGGTCATATCTGTTT GTAAGGAAAACTAAAACCACAAATAATAAAAAAG	1462
	GGCAATTGGGAATATGAA	1463
	TTCATATTCCAATTGCC	1464
Adenomatous polyposis coli Gln208Term CAG-TAG	CAATTGGAATATGAAGCAAGGCAAATCAGAGTTGCGATGGAA GAACAACTAGGTACCTGCCAGGATATGGAAAAACGAGCACAG GTAAGTTACTTGTTTCTAAGTGATAAAACAGCGAAGA	1465
	TCTTCGCTGTTTTATCACTTAGAAACAAGTAACTTACCTGTGCT CGTTTTTCCATATCCTGGCAGGTACCTAGTTGTTCTTCCATCG CAACTCTGATTTGCCTTGCTTCATATTCCAATTG	1466
	GTACCTGCCAGGATATG	1467
	CATATCCTGGCAGGTAC	1468
Adenomatous polyposis coli Arg213Term CGA-TGA	GCAAGGCAAATCAGAGTTGCGATGGAAGAACAACTAGGTACC TGCCAGGATATGGAAAAACGAGCACAGGTAAGTTACTTGTTT CTAAGTGATAAAACAGCGAAGAGCTATTAGGAATAAA	1469

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Ser932Term TCA-TGA	TACATTGTGTGACAGATGAGAGAAATGCACTTAGAAGAAGCT CTGCTGCCCATACACATTCAAACACTTACAATTTCACTAAGTC GGAAAATTCAAATAGGACATGTTCTATGCCTTATGC	1577
	GCATAAGGCATAGAACATGTCCTATTTGAATTTTCCGACTTAG TGAAATTGTAAGTGTTTGAATGTGTATGGGCAGCAGAGCTTCT TCTAAGTGCATTTCTCTCATCTGTCACACAATGTA	1578
	TACACATTCAAACACTT	1579
	AAGTGTTTGAATGTGTA	1580
Adenomatous polyposis coli Tyr935Term TAC-TAG	GACAGATGAGAGAAATGCACTTAGAAGAAGCTCTGCTGCCCA TACACATTCAAACACTTACAATTTCACTAAGTCGGAAAATTCAA ATAGGACATGTTCTATGCCTTATGCCAAATTAGAA	1581
	TTCTAATTTGGCATAAGGCATAGAACATGTCCTATTTGAATTTT CCGACTTAGTGAAATTGTAAGTGTTTGAATGTGTATGGGCAGC AGAGCTTCTTCTAAGTGCATTTCTCTCATCTGTC	1582
	AACACTTACAATTTCAC	1583
	GTGAAATTGTAAGTGTT	1584
Adenomatous polyposis coli Tyr935Term TAC-TAA	GACAGATGAGAGAAATGCACTTAGAAGAAGCTCTGCTGCCCA TACACATTCAAACACTTACAATTTCACTAAGTCGGAAAATTCAA ATAGGACATGTTCTATGCCTTATGCCAAATTAGAA	1585
	TTCTAATTTGGCATAAGGCATAGAACATGTCCTATTTGAATTTT CCGACTTAGTGAAATTGTAAGTGTTTGAATGTGTATGGGCAGC AGAGCTTCTTCTAAGTGCATTTCTCTCATCTGTC	1586
	AACACTTACAATTTCAC	1587
	GTGAAATTGTAAGTGTT	1588
Adenomatous polyposis coli Tyr1000Term TAC-TAA	ACCCTCGATTGAATCCTATTCTGAAGATGATGAAAGTAAGTTT TGCAGTTATGGTCAATACCCAGCCGACCTAGCCCATAAAATAC ATAGTGCAAATCATATGGATGATAATGATGGAGAA	1589
	TTCTCCATCATTATCATCCATATGATTTGCACTATGTATTTTAT GGGCTAGGTCGGCTGGGTATTGACCATAACTGCAAAACTTAC TTTCATCATCTTCAGAATAGGATTCAATCGAGGGT	1590
	GGTCAATACCCAGCCGA	1591
	TCGGCTGGGTATTGACC	1592
Adenomatous polyposis coli Glu1020Term GAA-TAA	TACCCAGCCGACCTAGCCCATAAAATACATAGTGCAAATCATA TGGATGATAATGATGGAGAACTAGATACACCAATAAATTATAG TCTTAAATATTCAGATGAGCAGTTGAACTCTGGAA	1593
	TTCCAGAGTTCAACTGCTCATCTGAATATTTAAGACTATAATTT ATTGGTGTATCTAGTTCTCCATCATTATCATCCATATGATTTGC ACTATGTATTTTATGGGCTAGGTCGGCTGGGTA	1594
	ATGATGGAGAACTAGAT	1595
	ATCTAGTTCTCCATCAT	1596
Adenomatous polyposis coli Ser1032Term TCA-TAA	ATGAAACCCTCGATTGAATCCTATTCTGAAGATGATGAAAGTA AGTTTTGCAGTTATGGTCAATACCCAGCCGACCTAGCCCATAA AATACATAGTGCAAATCATATGGATGATAATGATG	1597

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATCATTATCATCCATATGATTTGCACTATGTATTTTATGGGCT AGGTCGGCTGGGTATTGACCATAACTGCAAAACTTACTTTCAT CATCTTCAGAATAGGATTCAATCGAGGGTTTCAT	1598
	GTTATGGTCAATACCCA	1599
	TGGGTATTGACCATAAC	1600
Adenomatous polyposis coli Gln1041Term CAA-TAA	TGAAGATGATGAAAGTAAGTTTTGCAGTTATGGTCAATACCCA GCCGACCTAGCCCATAAAATACATAGTGCAAATCATATGGATG ATAATGATGGAGAACTAGATACACCAATAAATTAT	1601
	ATAATTTATTGGTGTATCTAGTTCTCCATCATTATCATCCATAT GATTTGCACTATGTATTTTATGGGCTAGGTCGGCTGGGTATTG ACCATAACTGCAAAACTTACTTTCATCATCTTCA	1602
	GCCCATAAAATACATAG	1603
	CTATGTATTTTATGGGC	1604
Adenomatous polyposis coli Gln1045Term CAG-TAG	ATAAATTATAGTCTTAAATATTCAGATGAGCAGTTGAACTCTG GAAGGCAAAGTCCTTCACAGAATGAAAGATGGGCAAGACCCA AACACATAATAGAAGATGAAATAAAACAAAGTGAGC	1605
	GCTCACTTTGTTTTATTTCATCTTCTATTATGTGTTTGGGTCTT GCCCATCTTTCATTCTGTGAAGGACTTTGCCTTCCAGAGTTCA ACTGCTCATCTGAATATTTAAGACTATAATTTAT	1606
	GTCCTTCACAGAATGAA	1607
	TTCATTCTGTGAAGGAC	1608
Adenomatous polyposis coli Gln1067Term CAA-TAA	GAAAGATGGGCAAGACCCAAACACATAATAGAAGATGAAATA AAACAAAGTGAGCAAAGACAATCAAGGAATCAAAGTACAACTT ATCCTGTTTATACTGAGAGCACTGATGATAAACACC	1609
	GGTGTTTATCATCAGTGCTCTCAGTATAAACAGGATAAGTTGT ACTTTGATTCCTTGATTGTCTTTGCTCACTTTGTTTTATTTCATC TTCTATTATGTGTTTGGGTCTTGCCCATCTTTC	1610
	AGCAAAGACAATCAAGG	1611
	CCTTGATTGTCTTTGCT	1612
Adenomatous polyposis coli Tyr1075Term TAT-TAG	AATAGAAGATGAAATAAAACAAAGTGAGCAAAGACAATCAAG GAATCAAAGTACAACTTATCCTGTTTATACTGAGAGCACTGAT GATAAACACCTCAAGTTCCAACCACATTTTGGACAG	1613
	CTGTCCAAAATGTGGTTGGAACTTGAGGTGTTTATCATCAGTG CTCTCAGTATAAACAGGATAAGTTGTACTTTGATTCCTTGATT GTCTTTGCTCACTTTGTTTTATTTCATCTTCTATT	1614
	ACAACTTATCCTGTTTA	1615
	TAAACAGGATAAGTTGT	1616
Adenomatous polyposis coli Tyr1102Term TAC-TAG	TGATGATAAACACCTCAAGTTCCAACCACATTTTGGACAGCAG GAATGTGTTTCTCCATACAGGTCACGGGGAGCCAATGGTTCA GAAACAAATCGAGTGGGTTCTAATCATGGAATTAAT	1617
	ATTAATTCCATGATTAGAACCCACTCGATTTGTTTCTGAACCAT TGGCTCCCCGTGACCTGTATGGAGAAACACATTCCTGCTGTC CAAAATGTGGTTGGAACTTGAGGTGTTTATCATCA	1618

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTCCATACAGGTCACG	1619
	CGTGACCTGTATGGAGA	1620
Adenomatous polyposis coli Ser1110Term TCA-TGA	AACCACATTTTGGACAGCAGGAATGTGTTTCTCCATACAGGTC ACGGGGAGCCAATGGTTCAGAAACAAATCGAGTGGGTTCTAA TCATGGAATTAATCAAAATGTAAGCCAGTCTTTGTG	1621
	CACAAAGACTGGCTTACATTTTGATTAATTCCATGATTAGAAC CCACTCGATTTGTTTCTGAACCATTGGCTCCCCGTGACCTGTA TGGAGAAACACATTCCTGCTGTCCAAAATGTGGTT	1622
	CAATGGTTCAGAAACAA	1623
	TTGTTTCTGAACCATTG	1624
Adenomatous polyposis coli Arg1114Term CGA-TGA	GGACAGCAGGAATGTGTTTCTCCATACAGGTCACGGGGAGCC AATGGTTCAGAAACAAATCGAGTGGGTTCTAATCATGGAATTA ATCAAAATGTAAGCCAGTCTTTGTGTCAAGAAGATG	1625
	CATCTTCTTGACACAAAGACTGGCTTACATTTTGATTAATTCCA TGATTAGAACCCACTCGATTTGTTTCTGAACCATTGGCTCCCC GTGACCTGTATGGAGAAACACATTCCTGCTGTCC	1626
	AAACAAATCGAGTGGGT	1627
	ACCCACTCGATTTGTTT	1628
Adenomatous polyposis coli Tyr1135Term TAT-TAG	GGGTTCTAATCATGGAATTAATCAAAATGTAAGCCAGTCTTTG TGTCAAGAAGATGACTATGAAGATGATAAGCCTACCAATTATA GTGAACGTTACTCTGAAGAAGAACAGCATGAAGAA	1629
	TTCTTCATGCTGTTCTTCTTCAGAGTAACGTTCACTATAATTGG TAGGCTTATCATCTTCATAGTCATCTTCTTGACACAAAGACTG GCTTACATTTTGATTAATTCCATGATTAGAACCC	1630
	GATGACTATGAAGATGA	1631
	TCATCTTCATAGTCATC	1632
Adenomatous polyposis coli Gln1152Term CAG-TAG	GAAGATGACTATGAAGATGATAAGCCTACCAATTATAGTGAAC GTTACTCTGAAGAAGAACAGCATGAAGAAGAAGAGAGACCAA CAAATTATAGCATAAAATATAATGAAGAGAAACGTC	1633
	GACGTTTCTCTTCATTATATTTTATGCTATAATTTGTTGGTCTC TCTTCTTCTTCATGCTGTTCTTCTTCAGAGTAACGTTCACTATA ATTGGTAGGCTTATCATCTTCATAGTCATCTTC	1634
	AAGAAGAACAGCATGAA	1635
	TTCATGCTGTTCTTCTT	1636
Adenomatous polyposis coli Gln1175Term CAG-TAG	GAAGAAGAGAGACCAACAAATTATAGCATAAAATATAATGAAG AGAAACGTCATGTGGATCAGCCTATTGATTATAGTTTAAAATA TGCCACAGATATTCCTTCATCACAGAAACAGTCAT	1637
	ATGACTGTTTCTGTGATGAAGGAATATCTGTGGCATATTTTAA ACTATAATCAATAGGCTGATCCACATGACGTTTCTCTTCATTAT ATTTTATGCTATAATTTGTTGGTCTCTCTTCTTC	1638
	ATGTGGATCAGCCTATT	1639
	AATAGGCTGATCCACAT	1640

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Pro1176Leu CCT-CTT	AAGAGAGACCAACAAATTATAGCATAAAATATAATGAAGAGAA ACGTCATGTGGATCAGCCTATTGATTATAGTTTAAAATATGCC ACAGATATTCCTTCATCACAGAAACAGTCATTTTC	1641
	GAAAATGACTGTTTCTGTGATGAAGGAATATCTGTGGCATATT TTAAACTATAATCAATAGGCTGATCCACATGACGTTTCTCTTCA TTATATTTTATGCTATAATTTGTTGGTCTCTCTT	1642
	GGATCAGCCTATTGATT	1643
	AATCAATAGGCTGATCC	1644
Adenomatous polyposis coli Ala1184Pro GCC-CCC	ATAAAATATAATGAAGAGAAACGTCATGTGGATCAGCCTATTG ATTATAGTTTAAAATATGCCACAGATATTCCTTCATCACAGAAA CAGTCATTTTCATTCTCAAAGAGTTCATCTGGAC	1645
	GTCCAGATGAACTCTTTGAGAATGAAAATGACTGTTTCTGTGA TGAAGGAATATCTGTGGCATATTTTAAACTATAATCAATAGGC TGATCCACATGACGTTTCTCTTCATTATATTTTAT	1646
	TAAAATATGCCACAGAT	1647
	ATCTGTGGCATATTTTA	1648
Adenomatous polyposis coli Ser1194Term TCA-TGA	ATCAGCCTATTGATTATAGTTTAAAATATGCCACAGATATTCCT TCATCACAGAAACAGTCATTTTCATTCTCAAAGAGTTCATCTG GACAAAGCAGTAAAACCGAACATATGTCTTCAAG	1649
	CTTGAAGACATATGTTCGGTTTTACTGCTTTGTCCAGATGAAC TCTTTGAGAATGAAAATGACTGTTTCTGTGATGAAGGAATATC TGTGGCATATTTTAAACTATAATCAATAGGCTGAT	1650
	GAAACAGTCATTTTCAT	1651
	ATGAAAATGACTGTTTC	1652
Adenomatous polyposis coli Ser1198Term TCA-TGA	ATTATAGTTTAAAATATGCCACAGATATTCCTTCATCACAGAAA CAGTCATTTTCATTCTCAAAGAGTTCATCTGGACAAAGCAGTA AAACCGAACATATGTCTTCAAGCAGTGAGAATAC	1653
	GTATTCTCACTGCTTGAAGACATATGTTCGGTTTTACTGCTTT GTCCAGATGAACTCTTTGAGAATGAAAATGACTGTTTCTGTGA TGAAGGAATATCTGTGGCATATTTTAAACTATAAT	1654
	TTCATTCTCAAAGAGTT	1655
	AACTCTTTGAGAATGAA	1656
Adenomatous polyposis coli Gln1228Term CAG-TAG	ACCGAACATATGTCTTCAAGCAGTGAGAATACGTCCACACCTT CATCTAATGCCAAGAGGCAGAATCAGCTCCATCCAGTTCTGC ACAGAGTAGAAGTGGTCAGCCTCAAAGGCTGCCACT	1657
	AGTGGCAGCCTTTGAGGCTGACCACTTCTACTCTGTGCAGAA CTGGATGGAGCTGATTCTGCCTCTTGGCATTAGATGAAGGTG TGGACGTATTCTCACTGCTTGAAGACATATGTTCGGT	1658
	CCAAGAGGCAGAATCAG	1659
	CTGATTCTGCCTCTTGG	1660
Adenomatous polyposis coli Gln1230Term CAG-TAG	CATATGTCTTCAAGCAGTGAGAATACGTCCACACCTTCATCTA ATGCCAAGAGGCAGAATCAGCTCCATCCAGTTCTGCACAGAG TAGAAGTGGTCAGCCTCAAAGGCTGCCACTTGCAAG	1661

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTGCAAGTGGCAGCCTTTGAGGCTGACCACTTCTACTCTGT GCAGAACTGGATGGAGCTGATTCTGCCTCTTGGCATTAGATG AAGGTGTGGACGTATTCTCACTGCTTGAAGACATATG	1662
	GGCAGAATCAGCTCCAT	1663
	ATGGAGCTGATTCTGCC	1664
Adenomatous polyposis coli Cys1249Term TGC-TGA	TCAGCTCCATCCAAGTTCTGCACAGAGTAGAAGTGGTCAGCC TCAAAAGGCTGCCACTTGCAAAGTTTCTTCTATTAACCAAGAA ACAATACAGACTTATTGTGTAGAAGATACTCCAATA	1665
	TATTGGAGTATCTTCTACACAATAAGTCTGTATTGTTTCTTGGT TAATAGAAGAAACTTTGCAAGTGGCAGCCTTTTGAGGCTGAC CACTTCTACTCTGTGCAGAACTTGGATGGAGCTGA	1666
	GCCACTTGCAAAGTTTC	1667
	GAAACTTTGCAAGTGGC	1668
Adenomatous polyposis coli Cys1270Term TGT-TGA	AGTTTCTTCTATTAACCAAGAAACAATACAGACTTATTGTGTAG AAGATACTCCAATATGTTTTTCAAGATGTAGTTCATTATCATCT TTGTCATCAGCTGAAGATGAAATAGGATGTAAT	1669
	ATTACATCCTATTTCATCTTCAGCTGATGACAAAGATGATAATG AACTACATCTTGAAAAACATATTGGAGTATCTTCTACACAATAA GTCTGTATTGTTTCTTGGTTAATAGAAGAAACT	1670
	CCAATATGTTTTTCAAG	1671
	CTTGAAAAACATATTGG	1672
Adenomatous polyposis coli Ser1276Term TCA-TGA	AAGAAACAATACAGACTTATTGTGTAGAAGATACTCCAATATG TTTTTCAAGATGTAGTTCATTATCATCTTTGTCATCAGCTGAAG ATGAAATAGGATGTAATCAGACGACACAGGAAGC	1673
	GCTTCCTGTGTCGTCTGATTACATCCTATTTCATCTTCAGCTG ATGACAAAGATGATAATGAACTACATCTTGAAAAACATATTGG AGTATCTTCTACACAATAAGTCTGTATTGTTTCTT	1674
	ATGTAGTTCATTATCAT	1675
	ATGATAATGAACTACAT	1676
Adenomatous polyposis coli Glu1286Term GAA-TAA	GATACTCCAATATGTTTTTCAAGATGTAGTTCATTATCATCTTT GTCATCAGCTGAAGATGAAATAGGATGTAATCAGACGACACA GGAAGCAGATTCTGCTAATACCCTGCAAATAGCAG	1677
	CTGCTATTTGCAGGGTATTAGCAGAATCTGCTTCCTGTGTCGT CTGATTACATCCTATTTCATCTTCAGCTGATGACAAAGATGATA ATGAACTACATCTTGAAAAACATATTGGAGTATC	1678
	CTGAAGATGAAATAGGA	1679
	TCCTATTTCATCTTCAG	1680
Adenomatous polyposis coli Gln1294Term CAG-TAG	TGTAGTTCATTATCATCTTTGTCATCAGCTGAAGATGAAATAG GATGTAATCAGACGACACAGGAAGCAGATTCTGCTAATACCC TGCAAATAGCAGAAATAAAAGAAAAGATTGGAACTA	1681
	TAGTTCCAATCTTTTCTTTTATTTCTGCTATTTGCAGGGTATTA GCAGAATCTGCTTCCTGTGTCGTCTGATTACATCCTATTTCAT CTTCAGCTGATGACAAAGATGATAATGAACTACA	1682

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGACGACACAGGAAGCA	1683
	TGCTTCCTGTGTCGTCT	1684
Predisposition to, association with, colorectal cancer Ile1307Lys ATA-AAA	TAGGATGTAATCAGACGACACAGGAAGCAGATTCTGCTAATA CCCTGCAAATAGCAGAAATAAAAGAAAAGATTGGAACTAGGT CAGCTGAAGATCCTGTGAGCGAAGTTCCAGCAGTGTC	1685
	GACACTGCTGGAACTTCGCTCACAGGATCTTCAGCTGACCTA GTTCCAATCTTTTCTTTTATTTCTGCTATTTGCAGGGTATTAGC AGAATCTGCTTCCTGTGTCGTCTGATTACATCCTA	1686
	AGCAGAAATAAAAGAAA	1687
	TTTCTTTTATTTCTGCT	1688
Adenomatous polyposis coli Glu1309Term GAA-TAA	CCAAGAAACAATACAGACTTATTGTGTAGAAGATACTCCAATA TGTTTTTCAAGATGTAGTTCATTATCATCTTTGTCATCAGCTGA AGATGAAATAGGATGTAATCAGACGACACAGGAA	1689
	TTCCTGTGTCGTCTGATTACATCCTATTTCATCTTCAGCTGATG ACAAAGATGATAATGAACTACATCTTGAAAAACATATTGGAGT ATCTTCTACACAATAAGTCTGTATTGTTTCTTGG	1690
	AGATGTAGTTCATTATC	1691
	GATAATGAACTACATCT	1692
Predisposition to Colorectal Cancer Glu1317Gln GAA-CAA	GATTCTGCTAATACCCTGCAAATAGCAGAAATAAAAGAAAAGA TTGGAACTAGGTCAGCTGAAGATCCTGTGAGCGAAGTTCCAG CAGTGTCACAGCACCCTAGAACCAAATCCAGCAGAC	1693
	GTCTGCTGGATTTGGTTCTAGGGTGCTGTGACACTGCTGGAA CTTCGCTCACAGGATCTTCAGCTGACCTAGTTCCAATCTTTTC TTTTATTTCTGCTATTTGCAGGGTATTAGCAGAATC	1694
	GGTCAGCTGAAGATCCT	1695
	AGGATCTTCAGCTGACC	1696
Adenomatous polyposis coli Gln1328Term CAG-TAG	AAAGAAAAGATTGGAACTAGGTCAGCTGAAGATCCTGTGAGC GAAGTTCCAGCAGTGTCACAGCACCCTAGAACCAAATCCAGC AGACTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCA	1697
	TGGCTGATTCTGAAGATAAACTAGAACCCTGCAGTCTGCTGG ATTTGGTTCTAGGGTGCTGTGACACTGCTGGAACTTCGCTCA CAGGATCTTCAGCTGACCTAGTTCCAATCTTTTCTTT	1698
	CAGTGTCACAGCACCCT	1699
	AGGGTGCTGTGACACTG	1700
Adenomatous polyposis coli Gln1338Term CAG-TAG	GATCCTGTGAGCGAAGTTCCAGCAGTGTCACAGCACCCTAGA ACCAAATCCAGCAGACTGCAGGGTTCTAGTTTATCTTCAGAAT CAGCCAGGCACAAAGCTGTTGAATTTTCTTCAGGAG	1701
	CTCCTGAAGAAAATTCAACAGCTTTGTGCCTGGCTGATTCTGA AGATAAACTAGAACCCTGCAGTCTGCTGGATTTGGTTCTAGG GTGCTGTGACACTGCTGGAACTTCGCTCACAGGATC	1702
	GCAGACTGCAGGGTTCT	1703
	AGAACCCTGCAGTCTGC	1704

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Adenomatous polyposis coli Leu1342Term TTA-TAA	AAGTTCCAGCAGTGTCACAGCACCCTAGAACCAAATCCAGCA GACTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCAGGCACA AAGCTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTC	1705
	GAGGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTTGTGC CTGGCTGATTCTGAAGATAAACTAGAACCCTGCAGTCTGCTG GATTTGGTTCTAGGGTGCTGTGACACTGCTGGAACTT	1706
	TTCTAGTTTATCTTCAG	1707
	CTGAAGATAAACTAGAA	1708
Adenomatous polyposis coli Arg1348Trp AGG-TGG	CAGCACCCTAGAACCAAATCCAGCAGACTGCAGGGTTCTAGT TTATCTTCAGAATCAGCCAGGCACAAAGCTGTTGAATTTTCTT CAGGAGCGAAATCTCCCTCCCGAAAGTGGTGCTCAG	1709
	CTGAGCACCACTTTCGGGAGGGAGATTTCGCTCCTGAAGAAA ATTCAACAGCTTTGTGCCTGGCTGATTCTGAAGATAAACTAGA ACCCTGCAGTCTGCTGGATTTGGTTCTAGGGTGCTG	1710
	AATCAGCCAGGCACAAA	1711
	TTTGTGCCTGGCTGATT	1712
Adenomatous polyposis coli Gly1357Term GGA-TGA	CTGCAGGGTTCTAGTTTATCTTCAGAATCAGCCAGGCACAAA GCTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTCCCGAAAGT GGTGCTCAGACACCCCAAAGTCCACCTGAACACTAT	1713
	ATAGTGTTCAGGTGGACTTTGGGGTGTCTGAGCACCACTTTC GGGAGGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTTGT GCCTGGCTGATTCTGAAGATAAACTAGAACCCTGCAG	1714
	TTTCTTCAGGAGCGAAA	1715
	TTTCGCTCCTGAAGAAA	1716
Adenomatous polyposis coli Gln1367Term CAG-TAG	CCAGGCACAAAGCTGTTGAATTTTCTTCAGGAGCGAAATCTCC CTCCCGAAAGTGGTGCTCAGACACCCCAAAGTCCACCTGAAC ACTATGTTCAGGAGACCCCACTCATGTTTAGCAGAT	1717
	ATCTGCTAAACATGAGTGGGGTCTCCTGAACATAGTGTTCAG GTGGACTTTGGGGTGTCTGAGCACCACTTTCGGGAGGGAGAT TTCGCTCCTGAAGAAAATTCAACAGCTTTGTGCCTGG	1718
	GTGGTGCTCAGACACCC	1719
	GGGTGTCTGAGCACCAC	1720
Adenomatous polyposis coli Lys1370Term AAA-TAA	AAAGCTGTTGAATTTTCTTCAGGAGCGAAATCTCCCTCCAAAA GTGGTGCTCAGACACCCAAAAGTCCACCTGAACACTATGTTC AGGAGACCCCACTCATGTTTAGCAGATGTACTTCTG	1721
	CAGAAGTACATCTGCTAAACATGAGTGGGGTCTCCTGAACAT AGTGTTCAGGTGGACTTTBGGGTGTCTGAGCACCACTTTTGG AGGGAGATTTCGCTCCTGAAGAAAATTCAACAGCTTT	1722
	AGACACCCBAAAGTCCA	1723
	TGGACTTTTGGGTGTCT	1724
Adenomatous polyposis coli Ser1392Term TCA-TAA	CACCTGAACACTATGTTCAGGAGACCCCACTCATGTTTAGCA GATGTACTTCTGTCAGTTCACTTGATAGTTTTGAGAGTCGTTC GATTGCCAGCTCCGTTCAGAGTGAACCATGCAGTGG	1725

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCACTGCATGGTTCACTCTGAACGGAGCTGGCAATCGAACGA CTCTCAAAACTATCAAGTGAACTGACAGAAGTACATCTGCTAA ACATGAGTGGGGTCTCCTGAACATAGTGTTCAGGTG	1726
	TGTCAGTTCACTTGATA	1727
	TATCAAGTGAACTGACA	1728
Adenomatous polyposis coli Ser1392Term TCA-TGA	CACCTGAACACTATGTTCAGGAGACCCCACTCATGTTTAGCA GATGTACTTCTGTCAGTTCACTTGATAGTTTTGAGAGTCGTTC GATTGCCAGCTCCGTTCAGAGTGAACCATGCAGTGG	1729
	CCACTGCATGGTTCACTCTGAACGGAGCTGGCAATCGAACGA CTCTCAAAACTATCAAGTGAACTGACAGAAGTACATCTGCTAA ACATGAGTGGGGTCTCCTGAACATAGTGTTCAGGTG	1730
	TGTCAGTTCACTTGATA	1731
	TATCAAGTGAACTGACA	1732
Adenomatous polyposis coli Glu1397Term GAG-TAG	GTTCAGGAGACCCCACTCATGTTTAGCAGATGTACTTCTGTCA GTTCACTTGATAGTTTTGAGAGTCGTTCGATTGCCAGCTCCGT TCAGAGTGAACCATGCAGTGGAATGGTAGGTGGCA	1733
	TGCCACCTACCATTCCACTGCATGGTTCACTCTGAACGGAGC TGGCAATCGAACGACTCTCAAAACTATCAAGTGAACTGACAG AAGTACATCTGCTAAACATGAGTGGGGTCTCCTGAAC	1734
	ATAGTTTTGAGAGTCGT	1735
	ACGACTCTCAAAACTAT	1736
Adenomatous polyposis coli Lys1449Term AAG-TAG	CAAACCATGCCACCAAGCAGAAGTAAAACACCTCCACCACCT CCTCAAACAGCTCAAACCAAGCGAGAAGTACCTAAAAATAAA GCACCTACTGCTGAAAAGAGAGAGAGTGGACCTAAGC	1737
	GCTTAGGTCCACTCTCTCTCTTTTCAGCAGTAGGTGCTTTATT TTTAGGTACTTCTCGCTTGGTTTGAGCTGTTTGAGGAGGTGGT GGAGGTGTTTTACTTCTGCTTGGTGGCATGGTTTG	1738
	CTCAAACCAAGCGAGAA	1739
	TTCTCGCTTGGTTTGAG	1740
Adenomatous polyposis coli Arg1450Term CGA-TGA	ACCATGCCACCAAGCAGAAGTAAAACACCTCCACCACCTCCT CAAACAGCTCAAACCAAGCGAGAAGTACCTAAAAATAAAGCA CCTACTGCTGAAAAGAGAGAGAGTGGACCTAAGCAAG	1741
	CTTGCTTAGGTCCACTCTCTCTCTTTTCAGCAGTAGGTGCTTT ATTTTTAGGTACTTCTCGCTTGGTTTGAGCTGTTTGAGGAGGT GGTGGAGGTGTTTTACTTCTGCTTGGTGGCATGGT	1742
	AAACCAAGCGAGAAGTA	1743
	TACTTCTCGCTTGGTTT	1744
Adenomatous polyposis coli Ser1503Term TCA-TAA	CAGATGCTGATACTTTATTACATTTTGCCACGGAAAGTACTCC AGATGGATTTTCTTGTTCATCCAGCCTGAGTGCTCTGAGCCTC GATGAGCCATTTATACAGAAAGATGTGGAATTAAG	1745
	CTTAATTCCACATCTTTCTGTATAAATGGCTCATCGAGGCTCA GAGCACTCAGGCTGGATGAACAAGAAAATCCATCTGGAGTAC TTTCCGTGGCAAAATGTAATAAAGTATCAGCATCTG	1746
	TTCTTGTTCATCCAGCC	1747

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGCTGGATGAACAAGAA	1748
Adenomatous polyposis coli Gln1529Term CAG-TAG	CTGAGCCTCGATGAGCCATTTATACAGAAAGATGTGGAATTAA GAATAATGCCTCCAGTTCAGGAAAATGACAATGGGAATGAAA CAGAATCAGAGCAGCCTAAAGAATCAAATGAAAACC	1749
	GGTTTTCATTTGATTCTTTAGGCTGCTCTGATTCTGTTTCATTC CCATTGTCATTTTCCTGAACTGGAGGCATTATTCTTAATTCCAC ATCTTTCTGTATAAATGGCTCATCGAGGCTCAG	1750
	CTCCAGTTCAGGAAAAT	1751
	ATTTTCCTGAACTGGAG	1752
Adenomatous polyposis coli Ser1539Term TCA-TAA	ATGTGGAATTAAGAATAATGCCTCCAGTTCAGGAAAATGACAA TGGGAATGAAACAGAATCAGAGCAGCCTAAAGAATCAAATGA AAACCAAGAGAAAGAGGCAGAAAAAACTATTGATTC	1753
	GAATCAATAGTTTTTTCTGCCTCTTTCTCTTGGTTTTCATTTGA TTCTTTAGGCTGCTCTGATTCTGTTTCATTCCCATTGTCATTTT CCTGAACTGGAGGCATTATTCTTAATTCCACAT	1754
	AACAGAATCAGAGCAGC	1755
	GCTGCTCTGATTCTGTT	1756
Adenomatous polyposis coli Ser1567Term TCA-TGA	AAAACCAAGAGAAAGAGGCAGAAAAAACTATTGATTCTGAAAA GGACCTATTAGATGATTCAGATGATGATGATATTGAAATACTA GAAGAATGTATTATTTCTGCCATGCCAACAAAGTC	1757
	GACTTTGTTGGCATGGCAGAAATAATACATTCTTCTAGTATTTC AATATCATCATCATCTGAATCATCTAATAGGTCCTTTTCAGAAT CAATAGTTTTTTCTGCCTCTTTCTCTTGGTTTT	1758
	AGATGATTCAGATGATG	1759
	CATCATCTGAATCATCT	1760
Adenomatous polyposis coli Asp1822Val GAC-GTC	AGAGAGTTTTCTCAGACAACAAAGATTCAAAGAAACAGAATTT GAAAAATAATTCCAAGGACTTCAATGATAAGCTCCCAAATAAT GAAGATAGAGTCAGAGGAAGTTTTGCTTTTGATTC	1761
	GAATCAAAAGCAAAACTTCCTCTGACTCTATCTTCATTATTTGG GAGCTTATCATTGAAGTCCTTGGAATTATTTTTCAAATTCTGTT TCTTTGAATCTTTGTTGTCTGAGAAAACTCTCT	1762
	TTCCAAGGACTTCAATG	1763
	CATTGAAGTCCTTGGAA	1764
Adenomatous polyposis coli Leu2839Phe CTT-TTT	AAAACTGACAGCACAGAATCCAGTGGAACCCAAAGTCCTAAG CGCCATTCTGGGTCTTACCTTGTGACATCTGTTTAAAAGAGAG GAAGAATGAAACTAAGAAAATTCTATGTTAATTACA	1765
	TGTAATTAACATAGAATTTTCTTAGTTTCATTCTTCCTCTCTTTT AAACAGATGTCACAAGGTAAGACCCAGAATGGCGCTTAGGAC TTTGGGTTCCACTGGATTCTGTGCTGTCAGTTTT	1766
	GGTCTTACCTTGTGACA	1767
	TGTCACAAGGTAAGACC	1768

EXAMPLE 12

Parahemophilia - Factor V Deficiency

Deficiency in clotting Factor V is associated with a lifelong predisposition to thrombosis. The disease typically manifests itself with usually mild bleeding, although bleeding times and clotting times are consistently prolonged. Individuals that are heterozygous for a mutation in Factor V have lowered levels of factor V but probably never have abnormal bleeding. A large number of alleles with a range of presenting symptoms have been identified. The attached table discloses the correcting oligonucleotide base sequences for the Factor V oligonucleotides of the invention.

Table 19

Factor V Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Factor V deficiency Ala221Val GCC-GTC	TTGACTGAATGCTTATTTTGGCCTGTGTCTCTCCCTCTTTCTCA GATATAACAGTTTGTG**C**CCATGACCACATCAGCTGGCATCTGC TGGGAATGAGCTCGGGGCCAGAATTATTCTCCAT	1768
	ATGGAGAATAATTCTGGCCCCGAGCTCATTCCCAGCAGATGC CAGCTGATGTGGTCATGG**G**CACAAACTGTTATATCTGAGAAA GAGGGAGAGACACAGGCCAAAATAAGCATTCAGTCAA	1769
	AGTTTGTG**C**CCATGACC	1770
	GGTCATGG**G**CACAAACT	1771
Thrombosis Arg306Gly AGG-GGG	TGTCCTAACTCAGCTGGGATGCAGGCTTACATTGACATTAAAA ACTGCCCAAAGAAAACC**A**GGAATCTTAAGAAAATAACTCGTGA GCAGAGGCGGCACATGAAGAGGTGGGAATACTTCA	1772
	TGAAGTATTCCCACCTCTTCATGTGCCGCCTCTGCTCACGAGT TATTTTCTTAAGATTCC**T**GGTTTTCTTTGGGCAGTTTTTAATGT CAATGTAAGCCTGCATCCCAGCTGAGTTAGGACA	1773
	AGAAAACC**A**GGAATCTT	1774
	AAGATTCC**T**GGTTTTCT	1775
Thrombosis Arg306Thr AGG-ACG	GTCCTAACTCAGCTGGGATGCAGGCTTACATTGACATTAAAAA CTGCCCAAAGAAAACCA**G**GAATCTTAAGAAAATAACTCGTGAG CAGAGGCGGCACATGAAGAGGTGGGAATACTTCAT	1776
	ATGAAGTATTCCCACCTCTTCATGTGCCGCCTCTGCTCACGAG TTATTTTCTTAAGATTC**C**TGGTTTTCTTTGGGCAGTTTTTAATG TCAATGTAAGCCTGCATCCCAGCTGAGTTAGGAC	1777
	GAAAACCA**G**GAATCTTA	1778
	TAAGATTC**C**TGGTTTTC	1779

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Increased Risk Thrombosis Arg485Lys AGA-AAA	CCACAGAAAATGATGCCCAGTGCTTAACAAGACCATACTACA GTGACGTGGACATCATGAGAGACATCGCCTCTGGGCTAATAG GACTACTTCTAATCTGTAAGAGCAGATCCCTGGACAG	1780
	CTGTCCAGGGATCTGCTCTTACAGATTAGAAGTAGTCCTATTA GCCCAGAGGCGATGTCTCTCATGATGTCCACGTCACTGTAGT ATGGTCTTGTTAAGCACTGGGCATCATTTTCTGTGG	1781
	CATCATGAGAGACATCG	1782
	CGATGTCTCTCATGATG	1783
Increased Risk Thrombosis Arg506Gln CGA-CAA	ACATCGCCTCTGGGCTAATAGGACTACTTCTAATCTGTAAGAG CAGATCCCTGGACAGGCGAGGAATACAGGTATTTTGTCCTTG AAGTAACCTTTCAGAAATTCTGAGAATTTCTTCTGG	1784
	CCAGAAGAAATTCTCAGAATTTCTGAAAGGTTACTTCAAGGAC AAAATACCTGTATTCCTCGCCTGTCCAGGGATCTGCTCTTACA GATTAGAAGTAGTCCTATTAGCCCAGAGGCGATGT	1785
	GGACAGGCGAGGAATAC	1786
	GTATTCCTCGCCTGTCC	1787
Factor V Deficiency Arg506Term CGA-TGA	GACATCGCCTCTGGGCTAATAGGACTACTTCTAATCTGTAAGA GCAGATCCCTGGACAGGCGAGGAATACAGGTATTTTGTCCTT GAAGTAACCTTTCAGAAATTCTGAGAATTTCTTCTG	1788
	CAGAAGAAATTCTCAGAATTTCTGAAAGGTTACTTCAAGGACA AAATACCTGTATTCCTCGCCTGTCCAGGGATCTGCTCTTACAG ATTAGAAGTAGTCCTATTAGCCCAGAGGCGATGTC	1789
	TGGACAGGCGAGGAATA	1790
	TATTCCTCGCCTGTCCA	1791
Thrombosis Arg712Term CGA-TGA	AGTGATGCTGACTATGATTACCAGAACAGACTGGCTGCAGCA TTAGGAATCAGGTCATTCCGAAACTCATCATTGAATCAGGAAG AAGAAGAGTTCAATCTTACTGCCCTAGCTCTGGAGA	1792
	TCTCCAGAGCTAGGGCAGTAAGATTGAACTCTTCTTCTTCCTG ATTCAATGATGAGTTTCGGAATGACCTGATTCCTAATGCTGCA GCCAGTCTGTTCTGGTAATCATAGTCAGCATCACT	1793
	GGTCATTCCGAAACTCA	1794
	TGAGTTTCGGAATGACC	1795
Thrombosis His1299Arg CAT-CGT	TCAGTCAGACAAACCTTTCCCCAGCCCTCGGTCAGATGCCCA TTTCTCCAGACCTCAGCCATACAACCCTTTCTCTAGACTTCAG CCAGACAAACCTCTCTCCAGAACTCAGTCAAACAAA	1796
	TTTGTTTGACTGAGTTCTGGAGAGAGGTTTGTCTGGCTGAAGT CTAGAGAAAGGGTTGTATGGCTGAGGTCTGGAGAAATGGGCA TCTGACCGAGGGCTGGGGAAAGGTTTGTCTGACTGA	1797
	CCTCAGCCATACAACCC	1798
	GGGTTGTATGGCTGAGG	1799

EXAMPLE 13

Hemophilia - Factor VIII Deficiency

The attached table discloses the correcting oligonucleotide base sequences for the Factor VIII oligonucleotides of the invention.

Table 20

Factor VIII Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A TyrSCys TAC-TGC	AGCTCTCCACCTGCTTCTTTCTGTGCCTTTTGCGATTCTGCTT TAGTGCCACCAGAAGATACTACCTGGGTGCAGTGGAACTGTC ATGGGACTATATGCAAAGTGATCTCGGTGAGCTGCC	1800
	GGCAGCTCACCGAGATCACTTTGCATATAGTCCCATGACAGT TCCACTGCACCCAGGTAGTATCTTCTGGTGGCACTAAAGCAG AATCGCAAAAGGCACAGAAAGAAGCAGGTGGAGAGCT	1801
	CAGAAGATACTACCTGG	1802
	CCAGGTAGTATCTTCTG	1803
Haemophilia A Leu7Arg CTG-CGG	CCACCTGCTTCTTTCTGTGCCTTTTGCGATTCTGCTTTAGTGC CACCAGAAGATACTACCTGGGTGCAGTGGAACTGTCATGGGA CTATATGCAAAGTGATCTCGGTGAGCTGCCTGTGGA	1804
	TCCACAGGCAGCTCACCGAGATCACTTTGCATATAGTCCCAT GACAGTTCCACTGCACCCAGGTAGTATCTTCTGGTGGCACTA AAGCAGAATCGCAAAAGGCACAGAAAGAAGCAGGTGG	1805
	ATACTACCTGGGTGCAG	1806
	CTGCACCCAGGTAGTAT	1807
Haemophilia A Ser(-1)Arg AGTg-AGG	AGTCATGCAAATAGAGCTCTCCACCTGCTTCTTTCTGTGCCTT TTGCGATTCTGCTTTAGTGCCACCAGAAGATACTACCTGGGT GCAGTGGAACTGTCATGGGACTATATGCAAAGTGAT	1808
	ATCACTTTGCATATAGTCCCATGACAGTTCCACTGCACCCAGG TAGTATCTTCTGGTGGCACTAAAGCAGAATCGCAAAAGGCAC AGAAAGAAGCAGGTGGAGAGCTCTATTTGCATGACT	1809
	TGCTTTAGTGCCACCAG	1810
	CTGGTGGCACTAAAGCA	1811
Haemophilia A Arg(-5)Term gCGA-TGA	CATTTGTAGCAATAAGTCATGCAAATAGAGCTCTCCACCTGCT TCTTTCTGTGCCTTTTGCGATTCTGCTTTAGTGCCACCAGAAG ATACTACCTGGGTGCAGTGGAACTGTCATGGGACT	1812
	AGTCCCATGACAGTTCCACTGCACCCAGGTAGTATCTTCTGG TGGCACTAAAGCAGAATCGCAAAAGGCACAGAAAGAAGCAG GTGGAGAGCTCTATTTGCATGACTTATTGCTACAAATG	1813

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCCTTTTGCGATTCTGC	1814
	GCAGAATCGCAAAAGGC	1815
Haemophilia A Glu11Val GAA-GTA	TTCTGTGCCTTTTGCGATTCTGCTTTAGTGCCACCAGAAGATA CTACCTGGGTGCAGTGGAACTGTCATGGGACTATATGCAAAG TGATCTCGGTGAGCTGCCTGTGGACGCAAGGTAAAG	1816
	CTTTACCTTGCGTCCACAGGCAGCTCACCGAGATCACTTTGC ATATAGTCCCATGACAGTTCCACTGCACCCAGGTAGTATCTTC TGGTGGCACTAAAGCAGAATCGCAAAAGGCACAGAA	1817
	TGCAGTGGAACTGTCAT	1818
	ATGACAGTTCCACTGCA	1819
Haemophilia A Trp14Gly aTGG-GGG	CTTTTGCGATTCTGCTTTAGTGCCACCAGAAGATACTACCTGG GTGCAGTGGAACTGTCATGGGACTATATGCAAAGTGATCTCG GTGAGCTGCCTGTGGACGCAAGGTAAAGGCATGTCC	1820
	GGACATGCCTTTACCTTGCGTCCACAGGCAGCTCACCGAGAT CACTTTGCATATAGTCCCATGACAGTTCCACTGCACCCAGGT AGTATCTTCTGGTGGCACTAAAGCAGAATCGCAAAAG	1821
	AACTGTCATGGGACTAT	1822
	ATAGTCCCATGACAGTT	1823
Haemophilia A Tyr46Term TACa-TAA	TTCACGCAGATTTCCTCCTAGAGTGCCAAAATCTTTTCCATTC AACACCTCAGTCGTGTACAAAAAGACTCTGTTTGTAGAATTCA CGGATCACCTTTTCAACATCGCTAAGCCAAGGCCA	1824
	TGGCCTTGGCTTAGCGATGTTGAAAAGGTGATCCGTGAATTC TACAAACAGAGTCTTTTTGTACACGACTGAGGTGTTGAATGGA AAAGATTTTGGCACTCTAGGAGGAAATCTGCGTGAA	1825
	GTCGTGTACAAAAAGAC	1826
	GTCTTTTTGTACACGAC	1827
Haemophilia A Asp56Glu GATc-GAA	ATCTTTTCCATTCAACACCTCAGTCGTGTACAAAAAGACTCTG TTTGTAGAATTCACGGATCACCTTTTCAACATCGCTAAGCCAA GGCCACCCTGGATGGGTAATGAAAACAATGTTGAA	1828
	TTCAACATTGTTTTCATTACCCATCCAGGGTGGCCTTGGCTTA GCGATGTTGAAAAGGTGATCCGTGAATTCTACAAACAGAGTC TTTTTGTACACGACTGAGGTGTTGAATGGAAAAGAT	1829
	TTCACGGATCACCTTTT	1830
	AAAAGGTGATCCGTGAA	1831
Haemophilia A Gly73Val GGT-GTT	TTCTGGAGTACTATCCCCAAGTAACCTTTGGCGGACATCTCAT TCTTACAGGTCTGCTAGGTCCTACCATCCAGGCTGAGGTTTAT GATACAGTGGTCATTACACTTAAGAACATGGCTTC	1832
	GAAGCCATGTTCTTAAGTGTAATGACCACTGTATCATAAACCT CAGCCTGGATGGTAGGACCTAGCAGACCTGTAAGAATGAGAT GTCCGCCAAAGGTTACTTGGGGATAGTACTCCAGAA	1833
	TCTGCTAGGTCCTACCA	1834
	TGGTAGGACCTAGCAGA	1835

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Glu79Lys tGAG-AAG	CAAGTAACCTTTGGCGGACATCTCATTCTTACAGGTCTGCTAG GTCCTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTAC ACTTAAGAACATGGCTTCCCATCCTGTCAGTCTTC	1836
	GAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAATGA CCACTGTATCATAAACCTCAGCCTGGATGGTAGGACCTAGCA GACCTGTAAGAATGAGATGTCCGCCAAAGGTTACTTG	1837
	TCCAGGCTGAGGTTTAT	1838
	ATAAACCTCAGCCTGGA	1839
Haemophilia A ValSOAsp GTT-GAT	TAACCTTTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCC TACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACACTT AAGAACATGGCTTCCCATCCTGTCAGTCTTCATGC	1840
	GCATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTA ATGACCACTGTATCATAAACCTCAGCCTGGATGGTAGGACCT AGCAGACCTGTAAGAATGAGATGTCCGCCAAAGGTTA	1841
	GGCTGAGGTTTATGATA	1842
	TATCATAAACCTCAGCC	1843
Haemophilia A Asp82Val GAT-GTT	TTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCCTACCAT CCAGGCTGAGGTTTATGATACAGTGGTCATTACACTTAAGAA CATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG	1844
	CCAACAGCATGAAGACTGACAGGATGGGAAGCCATGTTCTTA AGTGTAATGACCACTGTATCATAAACCTCAGCCTGGATGGTA GGACCTAGCAGACCTGTAAGAATGAGATGTCCGCCAA	1845
	GGTTTATGATACAGTGG	1846
	CCACTGTATCATAAACC	1847
Haemophilia A Asp82Gly GAT-GGT	TTGGCGGACATCTCATTCTTACAGGTCTGCTAGGTCCTACCAT CCAGGCTGAGGTTTATGATACAGTGGTCATTACACTTAAGAA CATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG	1848
	CCAACAGCATGAAGACTGACAGGATGGGAAGCCATGTTCTTA AGTGTAATGACCACTGTATCATAAACCTCAGCCTGGATGGTA GGACCTAGCAGACCTGTAAGAATGAGATGTCCGCCAA	1849
	GGTTTATGATACAGTGG	1850
	CCACTGTATCATAAACC	1851
Haemophilia A Val85Asp GTC-GAC	ATCTCATTCTTACAGGTCTGCTAGGTCCTACCATCCAGGCTGA GGTTTATGATACAGTGGTCATTACACTTAAGAACATGGCTTCC CATCCTGTCAGTCTTCATGCTGTTGGTGTATCCTA	1852
	TAGGATACACCAACAGCATGAAGACTGACAGGATGGGAAGCC ATGTTCTTAAGTGTAATGACCACTGTATCATAAACCTCAGCCT GGATGGTAGGACCTAGCAGACCTGTAAGAATGAGAT	1853
	TACAGTGGTCATTACAC	1854
	GTGTAATGACCACTGTA	1855
Haemophilia A Lys89Thr AAG-ACG	CAGGTCTGCTAGGTCCTACCATCCAGGCTGAGGTTTATGATA CAGTGGTCATTACACTTAAGAACATGGCTTCCCATCCTGTCAG TCTTCATGCTGTTGGTGTATCCTACTGGAAAGCTTC	1856

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAAGCTTTCCAGTAGGATACACCAACAGCATGAAGACTGACA GGATGGGAAGCCATGTTCTTAAGTGTAATGACCACTGTATCAT AAACCTCAGCCTGGATGGTAGGACCTAGCAGACCTG	1857
	TACACTTAAGAACATGG	1858
	CCATGTTCTTAAGTGTA	1859
Haemophilia A Met91Val cATG-GTG	CTGCTAGGTCCTACCATCCAGGCTGAGGTTTATGATACAGTG GTCATTACACTTAAGAACATGGCTTCCCATCCTGTCAGTCTTC ATGCTGTTGGTGTATCCTACTGGAAAGCTTCTGAGG	1860
	CCTCAGAAGCTTTCCAGTAGGATACACCAACAGCATGAAGAC TGACAGGATGGGAAGCCATGTTCTTAAGTGTAATGACCACTG TATCATAAACCTCAGCCTGGATGGTAGGACCTAGCAG	1861
	TTAAGAACATGGCTTCC	1862
	GGAAGCCATGTTCTTAA	1863
Haemophilia A His94Arg CAT-CGT	CTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACACT TAAGAACATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGGT GTATCCTACTGGAAAGCTTCTGAGGGTGAGTAAAA	1864
	TTTTACTCACCCTCAGAAGCTTTCCAGTAGGATACACCAACAG CATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAA TGACCACTGTATCATAAACCTCAGCCTGGATGGTAG	1865
	GGCTTCCCATCCTGTCA	1866
	TGACAGGATGGGAAGCC	1867
Haemophilia A His94Tyr cCAT-TAT	CCTACCATCCAGGCTGAGGTTTATGATACAGTGGTCATTACAC TTAAGAACATGGCTTCCCATCCTGTCAGTCTTCATGCTGTTGG TGTATCCTACTGGAAAGCTTCTGAGGGTGAGTAAA	1868
	TTTACTCACCCTCAGAAGCTTTCCAGTAGGATACACCAACAGC ATGAAGACTGACAGGATGGGAAGCCATGTTCTTAAGTGTAAT GACCACTGTATCATAAACCTCAGCCTGGATGGTAGG	1869
	TGGCTTCCCATCCTGTC	1870
	GACAGGATGGGAAGCCA	1871
Haemophilia A Leu98Arg CTT-CGT	CTGAGGTTTATGATACAGTGGTCATTACACTTAAGAACATGGC TTCCCATCCTGTCAGTCTTCATGCTGTTGGTGTATCCTACTGG AAAGCTTCTGAGGGTGAGTAAAATACCCTCCTATT	1872
	AATAGGAGGGTATTTTACTCACCCTCAGAAGCTTTCCAGTAG GATACACCAACAGCATGAAGACTGACAGGATGGGAAGCCAT GTTCTTAAGTGTAATGACCACTGTATCATAAACCTCAG	1873
	TGTCAGTCTTCATGCTG	1874
	CAGCATGAAGACTGACA	1875
Haemophilia A Gly102Ser tGGT-AGT	GATACAGTGGTCATTACACTTAAGAACATGGCTTCCCATCCTG TCAGTCTTCATGCTGTTGGTGTATCCTACTGGAAAGCTTCTGA GGGTGAGTAAAATACCCTCCTATTGTCCTGTCATT	1876
	AATGACAGGACAATAGGAGGGTATTTTACTCACCCTCAGAAG CTTTCCAGTAGGATACACCAACAGCATGAAGACTGACAGGAT GGGAAGCCATGTTCTTAAGTGTAATGACCACTGTATC	1877

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGCTGTTGGTGTATCC	1878
	GGATACACCAACAGCAT	1879
Haemophilia A Glu113Asp GAAt-GAC	CTTTGAGTGTACAGTGGATATAGAAAGGACAATTTTATTTCTT CCTGCTATAGGAGCTGAATATGATGATCAGACCAGTCAAAGG GAGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGC	1880
	GCTTCCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGA CTGGTCTGATCATCATATTCAGCTCCTATAGCAGGAAGAAATA AAATTGTCCTTTCTATATCCACTGTACACTCAAAG	1881
	GGAGCTGAATATGATGA	1882
	TCATCATATTCAGCTCC	1883
Haemophilia A Tyr114Cys TAT-TGT	TTGAGTGTACAGTGGATATAGAAAGGACAATTTTATTTCTTCC TGCTATAGGAGCTGAATATGATGATCAGACCAGTCAAAGGGA GAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCCA	1884
	TGGCTTCCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTT GACTGGTCTGATCATCATATTCAGCTCCTATAGCAGGAAGAAA TAAAATTGTCCTTTCTATATCCACTGTACACTCAA	1885
	AGCTGAATATGATGATC	1886
	GATCATCATATTCAGCT	1887
Haemophilia A Asp116Gly GAT-GGT	GTACAGTGGATATAGAAAGGACAATTTTATTTCTTCCTGCTAT AGGAGCTGAATATGATGATCAGACCAGTCAAAGGGAGAAAGA AGATGATAAAGTCTTCCCTGGTGGAAGCCATACATA	1888
	TATGTATGGCTTCCACCAGGGAAGACTTTATCATCTTCTTTCT CCCTTTGACTGGTCTGATCATCATATTCAGCTCCTATAGCAGG AAGAAATAAAATTGTCCTTTCTATATCCACTGTAC	1889
	ATATGATGATCAGACCA	1890
	TGGTCTGATCATCATAT	1891
Haemophilia A Gin 117Term tCAG-TAG	ACAGTGGATATAGAAAGGACAATTTTATTTCTTCCTGCTATAG GAGCTGAATATGATGATCAGACCAGTCAAAGGGAGAAAGAAG ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATG	1892
	CATATGTATGGCTTCCACCAGGGAAGACTTTATCATCTTCTTT CTCCCTTTGACTGGTCTGATCATCATATTCAGCTCCTATAGCA GGAAGAAATAAAATTGTCCTTTCTATATCCACTGT	1893
	ATGATGATCAGACCAGT	1894
	ACTGGTCTGATCATCAT	1895
Haemophilia A Thr118Ile ACC-ATC	TGGATATAGAAAGGACAATTTTATTTCTTCCTGCTATAGGAGC TGAATATGATGATCAGACCAGTCAAAGGGAGAAAGAAGATGA TAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTG	1896
	CAGACATATGTATGGCTTCCACCAGGGAAGACTTTATCATCTT CTTTCTCCCTTTGACTGGTCTGATCATCATATTCAGCTCCTATA GCAGGAAGAAATAAAATTGTCCTTTCTATATCCA	1897
	TGATCAGACCAGTCAAA	1898
	TTTGACTGGTCTGATCA	1899

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Glu122Term gGAG-TAG	AGGACAATTTTATTTCTTCCTGCTATAGGAGCTGAATATGATG ATCAGACCAGTCAAAGGGAGAAAGAAGATGATAAAGTCTTCC CTGGTGGAAGCCATACATATGTCTGGCAGGTCCTGA	1900
	TCAGGACCTGCCAGACATATGTATGGCTTCCACCAGGGAAGA CTTTATCATCTTCTTTCTCCCTTTGACTGGTCTGATCATCATAT TCAGCTCCTATAGCAGGAAGAAATAAAATTGTCCT	1901
	GTCAAAGGGAGAAAGAA	1902
	TTCTTTCTCCCTTTGAC	1903
Haemophilia A Asp126His tGAT-CAT	TTTCTTCCTGCTATAGGAGCTGAATATGATGATCAGACCAGTC AAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCC ATACATATGTCTGGCAGGTCCTGMAGAGAATGGTC	1904
	GACCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTTCC ACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGACTGGTC TGATCATCATATTCAGCTCCTATAGCAGGAAGAAA	1905
	AAGAAGATGATAAAGTC	1906
	GACTTTATCATCTTCTT	1907
Haemophilia A Gln139Term gCAG-TAG	AGTCAAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGA AGCCATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCA ATGGCCTCTGACCCACTGTGCCTTACCTACTCATATC	1908
	GATATGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATTGGA CCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTTCCAC CAGGGAAGACTTTATCATCTTCTTTCTCCCTTTGACT	1909
	ATGTCTGGCAGGTCCTG	1910
	CAGGACCTGCCAGACAT	1911
Haemophilia A Val140Ala GTC-GCC	AAAGGGAGAAAGAAGATGATAAAGTCTTCCCTGGTGGAAGCC ATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGG CCTCTGACCCACTGTGCCTTACCTACTCATATCTTTC	1912
	GAAAGATATGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATT GGACCATTCTCTTTCAGGACCTGCCAGACATATGTATGGCTT CCACCAGGGAAGACTTTATCATCTTCTTTCTCCCTTT	1913
	CTGGCAGGTCCTGAAAG	1914
	CTTTCAGGACCTGCCAG	1915
Haemophilia A Asn144Lys AATg-AAA	AGATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTG GCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACT GTGCCTTACCTACTCATATCTTTCTCATGTGGACCTG	1916
	CAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGTGG GTCAGAGGCCATTGGACCATTCTCTTTCAGGACCTGCCAGAC ATATGTATGGCTTCCACCAGGGAAGACTTTATCATCT	1917
	AAAGAGAATGGTCCAAT	1918
	ATTGGACCATTCTCTTT	1919
Haemophilia AG Gly145Asp GGT-GAT	ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGC AGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACTGT GCCTTACCTACTCATATCTTTCTCATGTGGACCTGGT	1920

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGT GGGTCAGAGGCCATTGGA**C**CATTCTCTTTCAGGACCTGCCAG ACATATGTATGGCTTCCACCAGGGAAGACTTTATCAT	1921
	AGAGAATG**G**TCCAATGG	1922
	CCATTGGA**C**CATTCTCT	1923
Haemophilia A Gly145Val GGT-GTT	ATGATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGC AGGTCCTGAAAGAGAATG**G**TCCAATGGCCTCTGACCCACTGT GCCTTACCTACTCATATCTTTCTCATGTGGACCTGGT	1924
	ACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAGT GGGTCAGAGGCCATTGGA**C**CATTCTCTTTCAGGACCTGCCAG ACATATGTATGGCTTCCACCAGGGAAGACTTTATCAT	1925
	AGAGAATG**G**TCCAATGG	1926
	CCATTGGA**C**CATTCTCT	1927
Haemophilia A Pro146Ser tCCA-TCA	GATAAAGTCTTCCCTGGTGGAAGCCATACATATGTCTGGCAG GTCCTGAAAGAGAATGGT**C**CAATGGCCTCTGACCCACTGTGC CTTACCTACTCATATCTTTCTCATGTGGACCTGGTAA	1928
	TTACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACA GTGGGTCAGAGGCCATTG**G**ACCATTCTCTTTCAGGACCTGCC AGACATATGTATGGCTTCCACCAGGGAAGACTTTATC	1929
	AGAATGGT**C**CAATGGCC	1930
	GGCCATTG**G**ACCATTCT	1931
Haemophilia A Cys153Trp TGCc-TGG	CCATACATATGTCTGGCAGGTCCTGAAAGAGAATGGTCCAAT GGCCTCTGACCCACTGTG**C**CTTACCTACTCATATCTTTCTCAT GTGGACCTGGTAAAAGACTTGAATTCAGGCCTCATT	1932
	AATGAGGCCTGAATTCAAGTCTTTTACCAGGTCCACATGAGAA AGATATGAGTAGGTAAG**G**CACAGTGGGTCAGAGGCCATTGG ACCATTCTCTTTCAGGACCTGCCAGACATATGTATGG	1933
	CCACTGTG**C**CTTACCTA	1934
	TAGGTAAG**G**CACAGTGG	1935
Haemophilia A Tyr156Term TACt-TAA	TGTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGA CCCACTGTGCCTTACCTA**C**TCATATCTTTCTCATGTGGACCTG GTAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTA	1936
	TAGGGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGTC CACATGAGAAAGATATGA**G**TAGGTAAGGCACAGTGGGTCAGA GGCCATTGGACCATTCTCTTTCAGGACCTGCCAGACA	1937
	CTTACCTA**C**TCATATCT	1938
	AGATATGA**G**TAGGTAAG	1939
Haemophilia A Ser157Pro cTCA-CCA	GTCTGGCAGGTCCTGAAAGAGAATGGTCCAATGGCCTCTGAC CCACTGTGCCTTACCTAC**T**CATATCTTTCTCATGTGGACCTGG TAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTAC	1940
	GTAGGGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGT CCACATGAGAAAGATATG**A**GTAGGTAAGGCACAGTGGGTCAG AGGCCATTGGACCATTCTCTTTCAGGACCTGCCAGAC	1941

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTACCTACTCATATCTT	1942
	AAGATATGAGTAGGTAA	1943
Haemophilia A Ser160Pro tTCT-CCT	GTCCTGAAAGAGAATGGTCCAATGGCCTCTGACCCACTGTGC CTTACCTACTCATATCTTTCTCATGTGGACCTGGTAAAAGACT TGAATTCAGGCCTCATTGGAGCCCTACTAGTATGTA	1944
	TACATACTAGTAGGGCTCCAATGAGGCCTGAATTCAAGTCTTT TACCAGGTCCACATGAGAAAGATATGAGTAGGTAAGGCACAG TGGGTCAGAGGCCATTGGACCATTCTCTTTCAGGAC	1945
	CATATCTTTCTCATGTG	1946
	CACATGAGAAAGATATG	1947
Haemophilia A Val162Met tGTG-ATG	AAAGAGAATGGTCCAATGGCCTCTGACCCACTGTGCCTTACC TACTCATATCTTTCTCATGTGGACCTGGTAAAAGACTTGAATT CAGGCCTCATTGGAGCCCTACTAGTATGTAGAGAAG	1948
	CTTCTCTACATACTAGTAGGGCTCCAATGAGGCCTGAATTCAA GTCTTTTACCAGGTCCACATGAGAAAGATATGAGTAGGTAAG GCACAGTGGGTCAGAGGCCATTGGACCATTCTCTTT	1949
	TTTCTCATGTGGACCTG	1950
	CAGGTCCACATGAGAAA	1951
Haemophilia A Lys166Thr AAA-ACA	CAATGGCCTCTGACCCACTGTGCCTTACCTACTCATATCTTTC TCATGTGGACCTGGTAAAAGACTTGAATTCAGGCCTCATTGG AGCCCTACTAGTATGTAGAGAAGGTAAGTGTATGAA	1952
	TTCATACACTTACCTTCTCTACATACTAGTAGGGCTCCAATGA GGCCTGAATTCAAGTCTTTTACCAGGTCCACATGAGAAAGATA TGAGTAGGTAAGGCACAGTGGGTCAGAGGCCATTG	1953
	CCTGGTAAAAGACTTGA	1954
	TCAAGTCTTTTACCAGG	1955
Haemophilia A Ser170Leu TCA-TTA	ACCCACTGTGCCTTACCTACTCATATCTTTCTCATGTGGACCT GGTAAAAGACTTGAATTCAGGCCTCATTGGAGCCCTACTAGT ATGTAGAGAAGGTAAGTGTATGAAAGCGTAGGATTG	1956
	CAATCCTACGCTTTCATACACTTACCTTCTCTACATACTAGTAG GGCTCCAATGAGGCCTGAATTCAAGTCTTTTACCAGGTCCAC ATGAGAAAGATATGAGTAGGTAAGGCACAGTGGGT	1957
	CTTGAATTCAGGCCTCA	1958
	TGAGGCCTGAATTCAAG	1959
Haemophilia A Phe195Val aTTT-GTT	AATGTTCTCACTTCTTTTTCAGGGAGTCTGGCCAAGGAAAAGA CACAGACCTTGCACAAATTTATACTACTTTTTGCTGTATTTGAT GAAGGTTAGTGAGTCTTAATCTGAATTTTGGATT	1960
	AATCCAAAATTCAGATTAAGACTCACTAACCTTCATCAAATACA GCAAAAAGTAGTATAAATTTGTGCAAGGTCTGTGTCTTTTCCT TGGCCAGACTCCCTGAAAAAGAAGTGAGAACATT	1961
	TGCACAAATTTATACTA	1962
	TAGTATAAATTTGTGCA	1963

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Leu198His CTT-CAT	CTTCTTTTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCT TGCACAAATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAG TGAGTCTTAATCTGAATTTTGGATTCCTGAAAGAA	1964
	TTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACTAACCTTC ATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAAGGTCTGT GTCTTTTCCTTGGCCAGACTCCCTGAAAAAGAAG	1965
	TATACTACTTTTTGCTG	1966
	CAGCAAAAAGTAGTATA	1967
Haemophilia A Ala200Asp GCT-GAT	TTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCTTGCACA AATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAGTGAGTC TTAATCTGAATTTTGGATTCCTGAAAGAAATCCTC	1968
	GAGGATTTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACT AACCTTCATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAA GGTCTGTGTCTTTTCCTTGGCCAGACTCCCTGAAA	1969
	ACTTTTTGCTGTATTTG	1970
	CAAATACAGCAAAAAGT	1971
Haemophilia A Ala200Thr tGCT-ACT	TTTTCAGGGAGTCTGGCCAAGGAAAAGACACAGACCTTGCAC AAATTTATACTACTTTTTGCTGTATTTGATGAAGGTTAGTGAGT CTTAATCTGAATTTTGGATTCCTGAAAGAAATCCT	1972
	AGGATTTCTTTCAGGAATCCAAAATTCAGATTAAGACTCACTA ACCTTCATCAAATACAGCAAAAAGTAGTATAAATTTGTGCAAG GTCTGTGTCTTTTCCTTGGCCAGACTCCCTGAAAA	1973
	TACTTTTTGCTGTATTT	1974
	AAATACAGCAAAAAGTA	1975
Haemophilia A Val234Phe aGTC-TTC	AACTCCTTGATGCAGGATAGGGATGCTGCATCTGCTCGGGCC TGGCCTAAAATGCACACAGTCAATGGTTATGTAAACAGGTCTC TGCCAGGTATGTACACACCTGCTCAACAATCCTCAG	1976
	CTGAGGATTGTTGAGCAGGTGTGTACATACCTGGCAGAGACC TGTTTACATAACCATTGACTGTGTGCATTTTAGGCCAGGCCCG AGCAGATGCAGCATCCCTATCCTGCATCAAGGAGTT	1977
	TGCACACAGTCAATGGT	1978
	ACCATTGACTGTGTGCA	1979
Haemophilia A Gly247Glu GGA-GAA	ATTTCAGATTCTCTACTTCATAGCCATAGGTGTCTTATTCCTAC TTTACAGGTCTGATTGGGATGCCACAGGAAATCAGTCTATTGG CATGTGATTGGAATGGGCACCACTCCTGAAGTGCA	1980
	TGCACTTCAGGAGTGGTGCCCATTCCAATCACATGCCAATAG ACTGATTTCCTGTGGCATCCAATCAGACCTGTAAAGTAGGAAT AAGACACCTATGGCTATGAAGTAGAGAATCTGAAAT	1981
	TCTGATTGGATGCCACA	1982
	TGTGGCATCCAATCAGA	1983
Haemophilia A Trp255Cys TGGc-TGT	ATAGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCAC AGGAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACT CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACA	1984

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT GCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGGCAT CCAATCAGACCTGTAAAGTAGGAATAAGACACCTAT	1985
	GTCTATTGGCATGTGAT	1986
	ATCACATGCCAATAGAC	1987
Haemophilia A Trp255Term TGGc-TGA	ATAGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCAC AGGAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACT CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACA	1988
	TGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT GCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGGCAT CCAATCAGACCTGTAAAGTAGGAATAAGACACCTAT	1989
	GTCTATTGGCATGTGAT	1990
	ATCACATGCCAATAGAC	1991
Haemophilia A His256Leu CAT-CTT	AGGTGTCTTATTCCTACTTTACAGGTCTGATTGGATGCCACAG GAAATCAGTCTATTGGCATGTGATTGGAATGGGCACCACTCC TGAAGTGCACTCAATATTCCTCGAAGGTCACACATT	1992
	AATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTG GTGCCCATTCCAATCACATGCCAATAGACTGATTTCCTGTGGC ATCCAATCAGACCTGTAAAGTAGGAATAAGACACCT	1993
	CTATTGGCATGTGATTG	1994
	CAATCACATGCCAATAG	1995
Haemophilia A Gly259Arg tGGA-AGA	TATTCCTACTTTACAGGTCTGATTGGATGCCACAGGAAATCAG TCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAAGTGC ACTCAATATTCCTCGAAGGTCACACATTTCTTGTGA	1996
	TCACAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTC AGGAGTGGTGCCCATTCCAATCACATGCCAATAGACTGATTT CCTGTGGCATCCAATCAGACCTGTAAAGTAGGAATA	1997
	ATGTGATTGGAATGGGC	1998
	GCCCATTCCAATCACAT	1999
Haemophilia A Val266Gly GTG-GGG	TTGGATGCCACAGGAAATCAGTCTATTGGCATGTGATTGGAA TGGGCACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTC ACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTT	2000
	AAGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCT TCGAGGAATATTGAGTGCACTTCAGGAGTGGTGCCCATTCCA ATCACATGCCAATAGACTGATTTCCTGTGGCATCCAA	2001
	TCCTGAAGTGCACTCAA	2002
	TTGAGTGCACTTCAGGA	2003
Haemophilia A Glu272Gly GAA-GGA	CAGTCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAAG TGCACTCAATATTCCTCGAAGGTCACACATTTCTTGTGAGGAA CCATCGCCAGGCGTCCTTGGAAATCTCGCCAATAAC	2004
	GTTATTGGCGAGATTTCCAAGGACGCCTGGCGATGGTTCCTC ACAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCA GGAGTGGTGCCCATTCCAATCACATGCCAATAGACTG	2005
	ATTCCTCGAAGGTCACA	2006

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGTGACCTTCGAGGAAT	2007
Haemophilia A Glu272Lys cGAA-AAA	TCAGTCTATTGGCATGTGATTGGAATGGGCACCACTCCTGAA GTGCACTCAATATTCCTCGAAGGTCACACATTTCTTGTGAGGA ACCATCGCCAGGCGTCCTTGGAAATCTCGCCAATAA	2008
	TTATTGGCGAGATTTCCAAGGACGCCTGGCGATGGTTCCTCA CAAGAAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAG GAGTGGTGCCCATTCCAATCACATGCCAATAGACTGA	2009
	TATTCCTCGAAGGTCAC	2010
	GTGACCTTCGAGGAATA	2011
Haemophilia A Thr275Ile ACA-ATA	GGCATGTGATTGGAATGGGCACCACTCCTGAAGTGCACTCAA TATTCCTCGAAGGTCACACATTTCTTGTGAGGAACCATCGCCA GGCGTCCTTGGAAATCTCGCCAATAACTTTCCTTAC	2012
	GTAAGGAAAGTTATTGGCGAGATTTCCAAGGACGCCTGGCGA TGGTTCCTCACAAGAAATGTGTGACCTTCGAGGAATATTGAGT GCACTTCAGGAGTGGTGCCCATTCCAATCACATGCC	2013
	AGGTCACACATTTCTTG	2014
	CAAGAAATGTGTGACCT	2015
Haemophilia A Val278Ala GTG-GCG	TTGGAATGGGCACCACTCCTGAAGTGCACTCAATATTCCTCG AAGGTCACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCT TGGAAATCTCGCCAATAACTTTCCTTACTGCTCAAAC	2016
	GTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCCAAGGAC GCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCTTCGAGG AATATTGAGTGCACTTCAGGAGTGGTGCCCATTCCAA	2017
	ATTTCTTGTGAGGAACC	2018
	GGTTCCTCACAAGAAAT	2019
Haemophilia A Asn280Ile AAC-ATC	TGGGCACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTC ACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAA TCTCGCCAATAACTTTCCTTACTGCTCAAACACTCTT	2020
	AAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCC AAGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGACCT TCGAGGAATATTGAGTGCACTTCAGGAGTGGTGCCCA	2021
	TGTGAGGAACCATCGCC	2022
	GGCGATGGTTCCTCACA	2023
Haemophilia A Arg282Cys tCGC-TGC	ACCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACAT TTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCTCGC CAATAACTTTCCTTACTGCTCAAACACTCTTGATGG	2024
	CCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGA TTTCCAAGGACGCCTGGCGATGGTTCCTCACAAGAAATGTGT GACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGGT	2025
	GGAACCATCGCCAGGCG	2026
	CGCCTGGCGATGGTTCC	2027

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Arg282His CGC-CAC	CCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATT TCTTGTGAGGAACCATC**G**CCAGGCGTCCTTGGAAATCTCGCC AATAACTTTCCTTACTGCTCAAACACTCTTGATGGA	2028
	TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGG**C**GATGGTTCCTCACAAGAAATGTG TGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGG	2029
	GAACCATC**G**CCAGGCGT	2030
	ACGCCTGG**C**GATGGTTC	2031
Haemophilia A Arg282Leu CGC-CTC	CCACTCCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATT TCTTGTGAGGAACCATC**G**CCAGGCGTCCTTGGAAATCTCGCC AATAACTTTCCTTACTGCTCAAACACTCTTGATGGA	2032
	TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGG**C**GATGGTTCCTCACAAGAAATGTG TGACCTTCGAGGAATATTGAGTGCACTTCAGGAGTGG	2033
	GAACCATC**G**CCAGGCGT	2034
	ACGCCTGG**C**GATGGTTC	2035
Haemophilia A Ala284Glu GCG-GAG	CTGAAGTGCACTCAATATTCCTCGAAGGTCACACATTTCTTGT GAGGAACCATCGCCAGG**C**GTCCTTGGAAATCTCGCCAATAAC TTTCCTTACTGCTCAAACACTCTTGATGGACCTTGG	2036
	CCAAGGTCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATT GGCGAGATTTCCAAGGAC**G**CCTGGCGATGGTTCCTCACAAG AAATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAG	2037
	TCGCCAGG**C**GTCCTTGG	2038
	CCAAGGAC**G**CCTGGCGA	2039
Haemophilia A Ala284Pro gGCG-CCG	CCTGAAGTGCACTCAATATTCCTCGAAGGTCACACATTTCTTG TGAGGAACCATCGCCAG**G**CGTCCTTGGAAATCTCGCCAATAA CTTTCCTTACTGCTCAAACACTCTTGATGGACCTTG	2040
	CAAGGTCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTG GCGAGATTTCCAAGGACG**C**CTGGCGATGGTTCCTCACAAGAA ATGTGTGACCTTCGAGGAATATTGAGTGCACTTCAGG	2041
	ATCGCCAG**G**CGTCCTTG	2042
	CAAGGACG**C**CTGGCGAT	2043
Haemophilia A Ser289Leu TCG-TTG	TATTCCTCGAAGGTCACACATTTCTTGTGAGGAACCATCGCCA GGCGTCCTTGGAAATCT**C**GCCAATAACTTTCCTTACTGCTCAA ACACTCTTGATGGACCTTGGACAGTTTCTACTGTT	2044
	AACAGTAGAAACTGTCCAAGGTCCATCAAGAGTGTTTGAGCA GTAAGGAAAGTTATTGGC**G**AGATTTCCAAGGACGCCTGGCGA TGGTTCCTCACAAGAAATGTGTGACCTTCGAGGAATA	2045
	GGAAATCT**C**GCCAATAA	2046
	TTATTGGC**G**AGATTTCC	2047
Haemophilia A Phe293Ser TTC-TCC	GTCACACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGG AAATCTCGCCAATAACTT**T**CCTTACTGCTCAAACACTCTTGAT GGACCTTGGACAGTTTCTACTGTTTTGTCATATCTC	2048

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCATCAAG AGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTTCCAAG GACGCCTGGCGATGGTTCCTCACAAGAAATGTGTGAC	2049
	AATAACTTTCCTTACTG	2050
	CAGTAAGGAAAGTTATT	2051
Haemophilia A Thr295Ala tACT-GCT	ACATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATC TCGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACC TTGGACAGTTTCTACTGTTTTGTCATATCTCTTCCC	2052
	GGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCA TCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTT CCAAGGACGCCTGGCGATGGTTCCTCACAAGAAATGT	2053
	CTTTCCTTACTGCTCAA	2054
	TTGAGCAGTAAGGAAAG	2055
Haemophilia A Thr295Ile ACT-ATT	CATTTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCT CGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACCT TGGACAGTTTCTACTGTTTTGTCATATCTCTTCCCA	2056
	TGGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCC ATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGATTT CCAAGGACGCCTGGCGATGGTTCCTCACAAGAAATG	2057
	TTTCCTTACTGCTCAAA	2058
	TTTGAGCAGTAAGGAAA	2059
Haemophilia A Ala296Val GCT-GTT	TTCTTGTGAGGAACCATCGCCAGGCGTCCTTGGAAATCTCGC CAATAACTTTCCTTACTGCTCAAACACTCTTGATGGACCTTGG ACAGTTTCTACTGTTTTGTCATATCTCTTCCCACCA	2060
	TGGTGGGAAGAGATATGACAAAACAGTAGAAACTGTCCAAGG TCCATCAAGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAG ATTTCCAAGGACGCCTGGCGATGGTTCCTCACAAGAA	2061
	CCTTACTGCTCAAACAC	2062
	GTGTTTGAGCAGTAAGG	2063
Haemophilia A Leu308Pro CTG-CCG	TCTCGCCAATAACTTTCCTTACTGCTCAAACACTCTTGATGGA CCTTGGACAGTTTCTACTGTTTTGTCATATCTCTTCCCACCAA CATGGTAATATCTTGGATCTTTAAAATGAATATTA	2064
	TAATATTCATTTTAAAGATCCAAGATATTACCATGTTGGTGGG AAGAGATATGACAAAACAGTAGAAACTGTCCAAGGTCCATCA AGAGTGTTTGAGCAGTAAGGAAAGTTATTGGCGAGA	2065
	GTTTCTACTGTTTTGTC	2066
	GACAAAACAGTAGAAAC	2067
Haemophilia A Glu321Lys gGAA-AAA	ACAGCCTAATATAGCAAGACACTCTGACATTGTTTGGTTTGTC TGACTCCAGATGGCATGGAAGCTTATGTCAAAGTAGACAGCT GTCCAGAGGAACCCCAACTACGAATGAAAAATAATG	2068
	CATTATTTTTCATTCGTAGTTGGGGTTCCTCTGGACAGCTGTC TACTTTGACATAAGCTTCCATGCCATCTGGAGTCAGACAAACC AAACAATGTCAGAGTGTCTTGCTATATTAGGCTGT	2069
	ATGGCATGGAAGCTTAT	2070

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATAAGCTTCCATGCCAT	2071
Haemophilia A Tyr323Term TATg-TAA	ATATAGCAAGACACTCTGACATTGTTTGGTTTGTCTGACTCCA GATGGCATGGAAGCTTATGTCAAAGTAGACAGCTGTCCAGAG GAACCCCAACTACGAATGAAAAATAATGAAGAAGCG	2072
	CGCTTCTTCATTATTTTTCATTCGTAGTTGGGGTTCCTCTGGA CAGCTGTCTACTTTGACATAAGCTTCCATGCCATCTGGAGTCA GACAAACCAAACAATGTCAGAGTGTCTTGCTATAT	2073
	GAAGCTTATGTCAAAGT	2074
	ACTTTGACATAAGCTTC	2075
Haemophilia A Val326Leu aGTA-CTA	AAGACACTCTGACATTGTTTGGTTTGTCTGACTCCAGATGGCA TGGAAGCTTATGTCAAAGTAGACAGCTGTCCAGAGGAACCCC AACTACGAATGAAAAATAATGAAGAAGCGGAAGACT	2076
	AGTCTTCCGCTTCTTCATTATTTTTCATTCGTAGTTGGGGTTCC TCTGGACAGCTGTCTACTTTGACATAAGCTTCCATGCCATCTG GAGTCAGACAAACCAAACAATGTCAGAGTGTCTT	2077
	ATGTCAAAGTAGACAGC	2078
	GCTGTCTACTTTGACAT	2079
Haemophilia A Cys329Arg cTGT-CGT	TGACATTGTTTGGTTTGTCTGACTCCAGATGGCATGGAAGCTT ATGTCAAAGTAGACAGCTGTCCAGAGGAACCCCAACTACGAA TGAAAAATAATGAAGAAGCGGAAGACTATGATGATG	2080
	CATCATCATAGTCTTCCGCTTCTTCATTATTTTTCATTCGTAGT TGGGGTTCCTCTGGACAGCTGTCTACTTTGACATAAGCTTCC ATGCCATCTGGAGTCAGACAAACCAAACAATGTCA	2081
	TAGACAGCIGTCCAGAG	2082
	CTCTGGACAGCTGTCTA	2083
Haemophilia A Cys329Tyr TGT-TAT	GACATTGTTTGGTTTGTCTGACTCCAGATGGCATGGAAGCTTA TGTCAAAGTAGACAGCTGTCCAGAGGAACCCCAACTACGAAT GAAAAATAATGAAGAAGCGGAAGACTATGATGATGA	2084
	TCATCATCATAGTCTTCCGCTTCTTCATTATTTTTCATTCGTAG TTGGGGTTCCTCTGGACAGCTGTCTACTTTGACATAAGCTTCC ATGCCATCTGGAGTCAGACAAACCAAACAATGTC	2085
	AGACAGCTGTCCAGAGG	2086
	CCTCTGGACAGCTGTCT	2087
Haemophilia A Arg336Term aCGA-TGA	ACTCCAGATGGCATGGAAGCTTATGTCAAAGTAGACAGCTGT CCAGAGGAACCCCAACTACGAATGAAAAATAATGAAGAAGCG GAAGACTATGATGATGATCTTACTGATTCTGAAATGG	2088
	CCATTTCAGAATCAGTAAGATCATCATCATAGTCTTCCGCTTC TTCATTATTTTTCATTCGTAGTTGGGGTTCCTCTGGACAGCTG TCTACTTTGACATAAGCTTCCATGCCATCTGGAGT	2089
	CCCAACTACGAATGAAA	2090
	TTTCATTCGTAGTTGGG	2091

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Arg372Cys tCGC-TGC	GATTCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTC CTTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAA AACTTGGGTACATTACATTGCTGCTGAAGAGGAGG	2092
	CCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGATG CTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAGAGTT GTCATCATCAAACCTGACCACATCCATTTCAGAATC	2093
	TCCAAATTCGCTCAGTT	2094
	AACTGAGCGAATTTGGA	2095
Haemophilia A Arg372His CGC-CAC	ATTCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCC TTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAAA ACTTGGGTACATTACATTGCTGCTGAAGAGGAGGA	2096
	TCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGAT GCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAGAGT TGTCATCATCAAACCTGACCACATCCATTTCAGAAT	2097
	CCAAATTCGCTCAGTTG	2098
	CAACTGAGCGAATTTGG	2099
Haemophilia A Ser373Leu TCA-TTA	CTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTTC CTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAAAACT TGGGTACATTACATTGCTGCTGAAGAGGAGGACTG	2100
	CAGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAG GATGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAG AGTTGTCATCATCAAACCTGACCACATCCATTTCAG	2101
	AATTCGCTCAGTTGCCA	2102
	TGGCAACTGAGCGAATT	2103
Haemophilia A Ser373Pro cTCA-CCA	TCTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTT CCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAAAAC TTGGGTACATTACATTGCTGCTGAAGAGGAGGACT	2104
	AGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGG ATGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAGA GTTGTCATCATCAAACCTGACCACATCCATTTCAGA	2105
	AAATTCGCTCAGTTGCC	2106
	GGCAACTGAGCGAATTT	2107
Haemophilia A Ser373Term TCA-TAA	CTGAAATGGATGTGGTCAGGTTTGATGATGACAACTCTCCTTC CTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAAAACT TGGGTACATTACATTGCTGCTGAAGAGGAGGACTG	2108
	CAGTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAG GATGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGGAG AGTTGTCATCATCAAACCTGACCACATCCATTTCAG	2109
	AATTCGCTCAGTTGCCA	2110
	TGGCAACTGAGCGAATT	2111
Haemophilia A Ile386Phe cATT-TTT	CCTTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTA AAACTTGGGTACATTACATTGCTGCTGAAGAGGAGGACTGGG ACTATGCTCCCTTAGTCCTCGCCCCCGATGACAGGT	2112

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACCTGTCATCGGGGGCGAGGACTAAGGGAGCATAGTCCCAG TCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGAT GCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAGG	2113
	TACATTACATTGCTGCT	2114
	AGCAGCAATGTAATGTA	2115
Haemophilia A Ile386Ser ATT-AGT	CTTCCTTTATCCAAATTCGCTCAGTTGCCAAGAAGCATCCTAA AACTTGGGTACATTACATTGCTGCTGAAGAGGAGGACTGGGA CTATGCTCCCTTAGTCCTCGCCCCCGATGACAGGTA	2116
	TACCTGTCATCGGGGGCGAGGACTAAGGGAGCATAGTCCCA GTCCTCCTCTTCAGCAGCAATGTAATGTACCCAAGTTTTAGGA TGCTTCTTGGCAACTGAGCGAATTTGGATAAAGGAAG	2117
	ACATTACATTGCTGCTG	2118
	CAGCAGCAATGTAATGT	2119
Haemophilia A Glu390Gly GAG-GGG	AAATTCGCTCAGTTGCCAAGAAGCATCCTAAAACTTGGGTACA TTACATTGCTGCTGAAGAGGAGGACTGGGACTATGCTCCCTT AGTCCTCGCCCCCGATGACAGGTAAGCACTTTTTGA	2120
	TCAAAAAGTGCTTACCTGTCATCGGGGGCGAGGACTAAGGGA GCATAGTCCCAGTCCTCCTCTTCAGCAGCAATGTAATGTACC CAAGTTTTAGGATGCTTCTTGGCAACTGAGCGAATTT	2121
	TGCTGAAGAGGAGGACT	2122
	AGTCCTCCTCTTCAGCA	2123
Haemophilia A Trp393Gly cTGG-GGG	TCAGTTGCCAAGAAGCATCCTAAAACTTGGGTACATTACATTG CTGCTGAAGAGGAGGACTGGGACTATGCTCCCTTAGTCCTCG CCCCCGATGACAGGTAAGCACTTTTTGACTATTGGT	2124
	ACCAATAGTCAAAAAGTGCTTACCTGTCATCGGGGGCGAGGA CTAAGGGAGCATAGTCCCAGTCCTCCTCTTCAGCAGCAATGT AATGTACCCAAGTTTTAGGATGCTTCTTGGCAACTGA	2125
	AGGAGGACTGGGACTAT	2126
	ATAGTCCCAGTCCTCCT	2127
Haemophilia A Lys408Ile AAA-ATA	GCCTACCTAGAATTTTTCTTCCCAACCTCTCATCTTTTTTTCTC TTATACAGAAGTTATAAAAGTCAATATTTGAACAATGGCCCTC AGCGGATTGGTAGGAAGTACAAAAAAGTCCGATT	2128
	AATCGGACTTTTTTGTACTTCCTACCAATCCGCTGAGGGCCAT TGTTCAAATATTGACTTTTATAACTTCTGTATAAGAGAAAAAAA GATGAGAGGTTGGGAAGAAAAATTCTAGGTAGGC	2129
	AAGTTATAAAAGTCAAT	2130
	ATTGACTTTTATAACTT	2131
Haemophilia A Leu412Phe TTGa-TTT	TTTTCTTCCCAACCTCTCATCTTTTTTTCTCTTATACAGAAGTT ATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGATTGGTAG GAAGTACAAAAAAGTCCGATTTATGGCATACACA	2132
	TGTGTATGCCATAAATCGGACTTTTTTGTACTTCCTACCAATC CGCTGAGGGCCATTGTTCAAATATTGACTTTTATAACTTCTGT ATAAGAGAAAAAAAGATGAGAGGTTGGGAAGAAAA	2133

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAATATTTGAACAATGG	2134
	CCATTGTTCAAATATTG	2135
Haemophilia A Arg418Trp gCGG-TGG	TCATCTTTTTTTCTCTTATACAGAAGTTATAAAAGTCAATATTTG AACAATGGCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTC CGATTTATGGCATACACAGATGAAACCTTTAAGA	2136
	TCTTAAAGGTTTCATCTGTGTATGCCATAAATCGGACTTTTTTG TACTTCCTACCAATCCGCTGAGGGCCATTGTTCAAATATTGAC TTTTATAACTTCTGTATAAGAGAAAAAAAGATGA	2137
	GCCCTCAGCGGATTGGT	2138
	ACCAATCCGCTGAGGGC	2139
Haemophilia A Gly420Val GGT-GTT	TTTTTCTCTTATACAGAAGTTATAAAAGTCAATATTTGAACAAT GGCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTCCGATTT ATGGCATACACAGATGAAACCTTTAAGACTCGTGA	2140
	TCACGAGTCTTAAAGGTTTCATCTGTGTATGCCATAAATCGGA CTTTTTTGTACTTCCTACCAATCCGCTGAGGGCCATTGTTCAA ATATTGACTTTTATAACTTCTGTATAAGAGAAAAA	2141
	GCGGATTGGTAGGAAGT	2142
	ACTTCCTACCAATCCGC	2143
Haemophilia A Lys425Arg AAA-AGA	GAAGTTATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGAT TGGTAGGAAGTACAAAAAAGTCCGATTTATGGCATACACAGA TGAAACCTTTAAGACTCGTGAAGCTATTCAGCATGA	2144
	TCATGCTGAATAGCTTCACGAGTCTTAAAGGTTTCATCTGTGT ATGCCATAAATCGGACTTTTTTGTACTTCCTACCAATCCGCTG AGGGCCATTGTTCAAATATTGACTTTTATAACTTC	2145
	GTACAAAAAAGTCCGAT	2146
	ATCGGACTTTTTTGTAC	2147
Haemophilia A Arg427Term cCGA-TGA	TATAAAAGTCAATATTTGAACAATGGCCCTCAGCGGATTGGTA GGAAGTACAAAAAAGTCCGATTTATGGCATACACAGATGAAA CCTTTAAGACTCGTGAAGCTATTCAGCATGAATCAG	2148
	CTGATTCATGCTGAATAGCTTCACGAGTCTTAAAGGTTTCATC TGTGTATGCCATAAATCGGACTTTTTTGTACTTCCTACCAATC CGCTGAGGGCCATTGTTCAAATATTGACTTTTATA	2149
	AAAAAGTCCGATTTATG	2150
	CATAAATCGGACTTTTT	2151
Haemophilia A Tyr431Asn aTAC-AAC	TATTTGAACAATGGCCCTCAGCGGATTGGTAGGAAGTACAAA AAAGTCCGATTTATGGCATACACAGATGAAACCTTTAAGACTC GTGAAGCTATTCAGCATGAATCAGGAATCTTGGGAC	2152
	GTCCCAAGATTCCTGATTCATGCTGAATAGCTTCACGAGTCTT AAAGGTTTCATCTGTGTATGCCATAAATCGGACTTTTTTGTAC TTCCTACCAATCCGCTGAGGGCCATTGTTCAAATA	2153
	TTATGGCATACACAGAT	2154
	ATCTGTGTATGCCATAA	2155

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Thr435Ile ACC-ATC	GCCCTCAGCGGATTGGTAGGAAGTACAAAAAAGTCCGATTTA TGGCATACACAGATGAAA**C**CTTTAAGACTCGTGAAGCTATTCA GCATGAATCAGGAATCTTGGGACCTTTACTTTATGG	2156
	CCATAAAGTAAAGGTCCCAAGATTCCTGATTCATGCTGAATAG CTTCACGAGTCTTAAAG**G**TTTCATCTGTGTATGCCATAAATCG GACTTTTTTGTACTTCCTACCAATCCGCTGAGGGC	2157
	AGATGAAA**C**CTTTAAGA	2158
	TCTTAAAG**G**TTTCATCT	2159
Haemophilia A Pro451Leu CCT-CTT	ACACAGATGAAACCTTTAAGACTCGTGAAGCTATTCAGCATGA ATCAGGAATCTTGGGAC**C**TTTACTTTATGGGGAAGTTGGAGA CACACTGTTGGTAAGTTGAAGAAAAGATTTAAGGTC	2160
	GACCTTAAATCTTTTCTTCAACTTACCAACAGTGTGTCTCCAA CTTCCCCATAAAGTAAA**G**GTCCCAAGATTCCTGATTCATGCTG AATAGCTTCACGAGTCTTAAAGGTTTCATCTGTGT	2161
	CTTGGGAC**C**TTTACTTT	2162
	AAAGTAAA**G**GTCCCAAG	2163
Haemophilia A Pro451Thr aCCT-ACT	TACACAGATGAAACCTTTAAGACTCGTGAAGCTATTCAGCATG AATCAGGAATCTTGGGA**C**CTTTACTTTATGGGGAAGTTGGAG ACACACTGTTGGTAAGTTGAAGAAAAGATTTAAGGT	2164
	ACCTTAAATCTTTTCTTCAACTTACCAACAGTGTGTCTCCAACT TCCCCATAAAGTAAAG**G**TCCCAAGATTCCTGATTCATGCTGAA TAGCTTCACGAGTCTTAAAGGTTTCATCTGTGTA	2165
	TCTTGGGA**C**CTTTACTT	2166
	AAGTAAAG**G**TCCCAAGA	2167
Haemophilia A Gly455Arg tGGG-AGG	ACCTTTAAGACTCGTGAAGCTATTCAGCATGAATCAGGAATCT TGGGACCTTTACTTTAT**G**GGGAAGTTGGAGACACACTGTTGG TAAGTTGAAGAAAAGATTTAAGGTCAGGTAAGAAGA	2168
	TCTTCTTACCTGACCTTAAATCTTTTCTTCAACTTACCAACAGT GTGTCTCCAACTTCCC**C**ATAAAGTAAAGGTCCCAAGATTCCTG ATTCATGCTGAATAGCTTCACGAGTCTTAAAGGT	2169
	TACTTTAT**G**GGGAAGTT	2170
	AACTTCCC**C**ATAAAGTA	2171
Haemophilia A Gly455Glu GGG-GAG	CCTTTAAGACTCGTGAAGCTATTCAGCATGAATCAGGAATCTT GGGACCTTTACTTTATG**G**GGAAGTTGGAGACACACTGTTGGT AAGTTGAAGAAAAGATTTAAGGTCAGGTAAGAAGAA	2172
	TTCTTCTTACCTGACCTTAAATCTTTTCTTCAACTTACCAACAG TGTGTCTCCAACTTCC**C**CATAAAGTAAAGGTCCCAAGATTCCT GATTCATGCTGAATAGCTTCACGAGTCTTAAAGG	2173
	ACTTTATG**G**GGAAGTTG	2174
	CAACTTCC**C**CATAAAGT	2175
Haemophilia A Asp459Asn aGAC-AAC	CGTGAAGCTATTCAGCATGAATCAGGAATCTTGGGACCTTTA CTTTATGGGGAAGTTGGA**G**ACACACTGTTGGTAAGTTGAAGA AAAGATTTAAGGTCAGGTAAGAAGAAAAAGTCTGGAG	2176

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTCCAGACTTTTTCTTCTTACCTGACCTTAAATCTTTTCTTCAA CTTACCAACAGTGTGTCTCCAACTTCCCCATAAAGTAAAGGTC CCAAGATTCCTGATTCATGCTGAATAGCTTCACG	2177
	AAGTTGGAGACACACTG	2178
	CAGTGTGTCTCCAACTT	2179
Haemophilia A Phe465Cys TTT-TGT	TGTTGATCCTAGTCGTTTTAGGATTTGATCTTAGATCTCGCTTA TACTTTCAGATTATATTTTAAGAATCAAGCAAGCAGACCATATAA CATCTACCCTCACGGAATCACTGATGTCCGTCC	2180
	GGACGGACATCAGTGATTCCGTGAGGGTAGATGTTATATGGT CTGCTTGCTTGATTCTTAAATATAATCTGAAAGTATAAGCGAG ATCTAAGATCAAATCCTAAAACGACTAGGATCAACA	2181
	GATTATATTTAAGAATC	2182
	GATTCTTAAATATAATC	2183
Haemophilia A Ala469Gly GCA-GGA	TCGTTTTAGGATTTGATCTTAGATCTCGCTTATACTTTCAGATT ATATTTAAGAATCAAGCAAGCAGACCATATAACATCTACCCTC ACGGAATCACTGATGTCCGTCCTTTGTATTCAAG	2184
	CTTGAATACAAAGGACGGACATCAGTGATTCCGTGAGGGTAG ATGTTATATGGTCTGCTTGCTTGATTCTTAAATATAATCTGAAA GTATAAGCGAGATCTAAGATCAAATCCTAAAACGA	2185
	GAATCAAGCAAGCAGAC	2186
	GTCTGCTTGCTTGATTC	2187
Haemophilia A Arg471Gly cAGA-GGA	TTAGGATTTGATCTTAGATCTCGCTTATACTTTCAGATTATATT TAAGAATCAAGCAAGCAGACCATATAACATCTACCCTCACGG AATCACTGATGTCCGTCCTTTGTATTCAAGGAGAT	2188
	ATCTCCTTGAATACAAAGGACGGACATCAGTGATTCCGTGAG GGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAATATAATC TGAAAGTATAAGCGAGATCTAAGATCAAATCCTAA	2189
	AAGCAAGCAGACCATAT	2190
	ATATGGTCTGCTTGCTT	2191
Haemophilia A Tyr473Cys TAT-TGT	TTGATCTTAGATCTCGCTTATACTTTCAGATTATATTTAAGAAT CAAGCAAGCAGACCATATAACATCTACCCTCACGGAATCACT GATGTCCGTCCTTTGTATTCAAGGAGATTACCAAA	2192
	TTTGGTAATCTCCTTGAATACAAAGGACGGACATCAGTGATTC CGTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAA TATAATCTGAAAGTATAAGCGAGATCTAAGATCAA	2193
	CAGACCATATAACATCT	2194
	AGATGTTATATGGTCTG	2195
Haemophilia A Tyr473His aTAT-CAT	TTTGATCTTAGATCTCGCTTATACTTTCAGATTATATTTAAGAA TCAAGCAAGCAGACCATTATAACATCTACCCTCACGGAATCACT GATGTCCGTCCTTTGTATTCAAGGAGATTACCAA	2196
	TTGGTAATCTCCTTGAATACAAAGGACGGACATCAGTGATTCC GTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATTCTTAAAT ATAATCTGAAAGTATAAGCGAGATCTAAGATCAAA	2197

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCAGACCATATAACATC	2198
	GATGTTATATGGTCTGC	2199
Haemophilia A Ile475Thr ATC-ACC	TTAGATCTCGCTTATACTTTCAGATTATATTTAAGAATCAAGCA AGCAGACCATATAACATCTACCCTCACGGAATCACTGATGTCC GTCCTTTGTATTCAAGGAGATTACCAAAAGGTAA	2200
	TTACCTTTTGGTAATCTCCTTGAATACAAAGGACGGACATCAG TGATTCCGTGAGGGTAGATGTTATATGGTCTGCTTGCTTGATT CTTAAATATAATCTGAAAGTATAAGCGAGATCTAA	2201
	ATATAACATCTACCCTC	2202
	GAGGGTAGATGTTATAT	2203
Haemophilia A Gly479Arg cGGA-AGA	TTATACTTTCAGATTATATTTAAGAATCAAGCAAGCAGACCATA TAACATCTACCCTCACGGAATCACTGATGTCCGTCCTTTGTAT TCAAGGAGATTACCAAAAGGTAAATATTCCCTCG	2204
	CGAGGGAATATTTACCTTTTGGTAATCTCCTTGAATACAAAGG ACGGACATCAGTGATTCCGTGAGGGTAGATGTTATATGGTCT GCTTGCTTGATTCTTAAATATAATCTGAAAGTATAA	2205
	ACCCTCACGGAATCACT	2206
	AGTGATTCCGTGAGGGT	2207
Haemophilia A Thr522Ser aACT-TCT	CCAATTCTGCCAGGAGAAATATTCAAATATAAATGGACAGTGA CTGTAGAAGATGGGCCAACTAAATCAGATCCTCGGTGCCTGA CCCGCTATTACTCTAGTTTCGTTAATATGGAGAGAG	2208
	CTCTCTCCATATTAACGAAACTAGAGTAATAGCGGGTCAGGC ACCGAGGATCTGATTTAGTTGGCCCATCTTCTACAGTCACTGT CCATTTATATTTGAATATTTCTCCTGGCAGAATTGG	2209
	ATGGGCCAACTAAATCA	2210
	TGATTTAGTTGGCCCAT	2211
Haemophilia A Asp525Asn aGAT-AAT	CCAGGAGAAATATTCAAATATAAATGGACAGTGACTGTAGAAG ATGGGCCAACTAAATCAGATCCTCGGTGCCTGACCCGCTATT ACTCTAGTTTCGTTAATATGGAGAGAGATCTAGCTT	2212
	AAGCTAGATCTCTCTCCATATTAACGAAACTAGAGTAATAGCG GGTCAGGCACCGAGGATCTGATTTAGTTGGCCCATCTTCTAC AGTCACTGTCCATTTATATTTGAATATTTCTCCTGG	2213
	CTAAATCAGATCCTCGG	2214
	CCGAGGATCTGATTTAG	2215
Haemophilia A Arg527Trp tCGG-TGG	GAAATATTCAAATATAAATGGACAGTGACTGTAGAAGATGGGC CAACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAG TTTCGTTAATATGGAGAGAGATCTAGCTTCAGGAC	2216
	GTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAGTA ATAGCGGGTCAGGCACCGAGGATCTGATTTAGTTGGCCCATC TTCTACAGTCACTGTCCATTTATATTTGAATATTTC	2217
	CAGATCCTCGGTGCCTG	2218
	CAGGCACCGAGGATCTG	2219

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia A Arg531Cys cCGC-TGC	TATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCA GATCCTCGGTGCCTGACC**C**GCTATTACTCTAGTTTCGTTAATA TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTC	2220
	GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAA CGAAACTAGAGTAATAGC**G**GGTCAGGCACCGAGGATCTGATT TAGTTGGCCCATCTTCTACAGTCACTGTCCATTTATA	2221
	GCCTGACC**C**GCTATTAC	2222
	GTAATAGC**G**GGTCAGGC	2223
Haemophilia A Arg531Gly cCGC-GGC	TATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCA GATCCTCGGTGCCTGACC**C**GCTATTACTCTAGTTTCGTTAATA TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTC	2224
	GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAA CGAAACTAGAGTAATAGC**G**GGTCAGGCACCGAGGATCTGATT TAGTTGGCCCATCTTCTACAGTCACTGTCCATTTATA	2225
	GCCTGACC**C**GCTATTAC	2226
	GTAATAGC**G**GGTCAGGC	2227
Haemophilia A Arg531His CGC-CAC	ATAAATGGACAGTGACTGTAGAAGATGGGCCAACTAAATCAG ATCCTCGGTGCCTGACCC**G**CTATTACTCTAGTTTCGTTAATAT GGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCT	2228
	AGAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATATTA ACGAAACTAGAGTAATAG**C**GGGTCAGGCACCGAGGATCTGAT TTAGTTGGCCCATCTTCTACAGTCACTGTCCATTTAT	2229
	CCTGACCC**G**CTATTACT	2230
	AGTAATAG**C**GGGTCAGG	2231
Haemophilia A Ser534Pro cTCT-CCT	ACAGTGACTGTAGAAGATGGGCCAACTAAATCAGATCCTCGG TGCCTGACCCGCTATTAC**T**CTAGTTTCGTTAATATGGAGAGAG ATCTAGCTTCAGGACTCATTGGCCCTCTCCTCATCT	2232
	AGATGAGGAGAGGGCCAATGAGTCCTGAAGCTAGATCTCTCT CCATATTAACGAAACTAG**A**GTAATAGCGGGTCAGGCACCGAG GATCTGATTTAGTTGGCCCATCTTCTACAGTCACTGT	2233
	GCTATTAC**T**CTAGTTTC	2234
	GAAACTAG**A**GTAATAGC	2235
Haemophilia A Ser535Gly tAGT-GGT	GTGACTGTAGAAGATGGGCCAACTAAATCAGATCCTCGGTGC CTGACCCGCTATTACTCT**A**GTTTCGTTAATATGGAGAGAGATC TAGCTTCAGGACTCATTGGCCCTCTCCTCATCTGCT	2236
	AGCAGATGAGGAGAGGGCCAATGAGTCCTGAAGCTAGATCT CTCTCCATATTAACGAAAC**T**AGAGTAATAGCGGGTCAGGCAC CGAGGATCTGATTTAGTTGGCCCATCTTCTACAGTCAC	2237
	ATTACTCT**A**GTTTCGTT	2238
	AACGAAAC**T**AGAGTAAT	2239
Haemophilia A Val537Asp GTT-GAT	TAGAAGATGGGCCAACTAAATCAGATCCTCGGTGCCTGACCC GCTATTACTCTAGTTTCG**T**TAATATGGAGAGAGATCTAGCTTC AGGACTCATTGGCCCTCTCCTCATCTGCTACAAAGA	2240

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTTTGTAGCAGATGAGGAGAGGGCCAATGAGTCCTGAAGCT AGATCTCTCTCCATATTAACGAAACTAGAGTAATAGCGGGTCA GGCACCGAGGATCTGATTTAGTTGGCCCATCTTCTA	2241
	TAGTTTCGTTAATATGG	2242
	CCATATTAACGAAACTA	2243
Haemophilia A Arg541Thr AGA-ACA	CAACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAG TTTCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGC CCTCTCCTCATCTGCTACAAAGAATCTGTAGATCA	2244
	TGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAATG AGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGAGT AATAGCGGGTCAGGCACCGACGGATCTGATTTAGTTG	2245
	TATGGAGAGAGATCTAG	2246
	CTAGATCTCTCTCCATA	2247
Haemophilia A Asp542Gly GAT-GGT	CTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTTT CGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCC TCTCCTCATCTGCTACAAAGAATCTGTAGATCAAAG	2248
	CTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCA ATGAGTCCTGAAGCTAGATTCTCTCTCCATATTAACGAAACTAG AGTAATAGCGGGTCAGGCACCGAGGATCTGATTTAG	2249
	GGAGAGAGATCTAGCTT	2250
	AAGCTAGATTCTCTCTCC	2251
Haemophilia A Asp542His aGAT-CAT	ACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTT TCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCC CTCTCCTCATCTGCTACAAAGAATCTGTAGATCAAA	2252
	TTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAA TGAGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGA GTAATAGCGGGTCAGGCACCGAGGATCTGATTTAGT	2253
	TGGAGAGAGATCTAGCT	2254
	AGCTAGATCTCTCTCCA	2255
Haemophilia A Asp542Tyr aGAT-TAT	ACTAAATCAGATCCTCGGTGCCTGACCCGCTATTACTCTAGTT TCGTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCC CTCTCCTCATCTGCTACAAAGAATCTGTAGATCAAA	2256
	TTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGCCAA TGAGTCCTGAAGCTAGATCTCTCTCCATATTAACGAAACTAGA GTAATAGCGGGTCAGGCACCGAGGATCTGATTTAGT	2257
	TGGAGAGAGATCTAGCT	2258
	AGCTAGATCTCTCTCCA	2259
Haemophilia A Glu557Term aGAA-TAA	GTTAATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCT CTCCTCATCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACC AGGTGAGTTCTTGCCTTTCCAAGTGCTGGGTTTCAT	2260
	ATGAAACCCAGCACTTGGAAAGGCAAGAACTCACCTGGTTTC CTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAGGGC CAATGAGTCCTGAAGCTAGATCTCTCTCCATATTAAC	2261

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCTACAAAGAATCTGTA	2262
	TACAGATTCTTTGTAGC	2263
Haemophilia A Ser558Phe TCT-TTT	ATATGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCTCC TCATCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACCAGG TGAGTTCTTGCCTTTCCAAGTGCTGGGTTTCATTCTC	2264
	GAGAATGAAACCCAGCACTTGGAAAGGCAAGAACTCACCTGG TTTCCTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGAGAG GGCCAATGAGTCCTGAAGCTAGATCTCTCTCCATAT	2265
	CAAAGAATCTGTAGATC	2266
	GATCTACAGATTCTTTG	2267
Haemophilia A Val559Ala GTA-GCA	TGGAGAGAGATCTAGCTTCAGGACTCATTGGCCCTCTCCTCA TCTGCTACAAAGAATCTGTAGATCAAAGAGGAAACCAGGTGA GTTCTTGCCTTTCCAAGTGCTGGGTTTCATTCTCAGT	2268
	ACTGAGAATGAAACCCAGCACTTGGAAAGGCAAGAACTCACC TGGTTTCCTCTTTGATCTACAGATTCTTTGTAGCAGATGAGGA GAGGGCCAATGAGTCCTGAAGCTAGATCTCTCTCCA	2269
	AGAATCTGTAGATCAAA	2270
	TTTGATCTACAGATTCT	2271

EXAMPLE 14

Hemophilia – Factor Ix Deficiency

The attached table discloses the correcting oligonucleotide base sequences for the Factor IX oligonucleotides of the invention.

Table 21

Factor Ix Mutations And Genome-Correcting Oligs

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Asn2Asp tAAT-GAT	ATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAA TCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAA	2272
	TTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT TCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTT GTTGGCGTTTTCATGATCAAGAAAAACTGAAAT	2273
	AGAGGTATAATTCAGGT	2274
	ACCTGAATTATACCTCT	2275

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Asn2Ile AAT-ATT	TTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAAT CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAA	2276
	TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTT TGTTGGCGTTTTCATGATCAAGAAAAACTGAAA	2277
	GAGGTATAATTCAGGTA	2278
	TACCTGAATTATACCTC	2279
Haemophilia B Asn2Tyr tAAT-TAT	ATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAA TCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAA	2280
	TTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT TCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTT GTTGGCGTTTTCATGATCAAGAAAAACTGAAAT	2281
	AGAGGTATAATTCAGGT	2282
	ACCTGAATTATACCTCT	2283
Haemophilia B Ser3Pro tTCA-CCA	TCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATC GGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTC AAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT	2284
	ACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAAC TCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCAGAAT TTTGTTGGCGTTTTCATGATCAAGAAAAACTGA	2285
	GGTATAATTCAGGTAAA	2286
	TTTACCTGAATTATACC	2287
Haemophilia B Gly4Asp GGT-GAT	TTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCC AAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGG GAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAG	2288
	CTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC AAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCA GAATTTTGTTGGCGTTTTCATGATCAAGAAAAA	2289
	TAATTCAGGTAAATTGG	2290
	CCAATTTACCTGAATTA	2291
Haemophilia B Gly4Ser aGGT-AGT	GTTTTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGC CAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGG GAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTA	2292
	TACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACA AACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATTCA GAATTTTGTTGGCGTTTTCATGATCAAGAAAAAC	2293
	ATAATTCAGGTAAATTG	2294
	CAATTTACCTGAATTAT	2295
Haemophilia B LysSGlu tAAA-GAA	TTTCTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAA AGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGA ACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTT	2296

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGA ACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCGATT CAGAATTTTGTTGGCGTTTTCATGATCAAGAAA	2297
	ATTCAGGTAAATTGGAA	2298
	TTCCAATTTACCTGAAT	2299
Haemophilia B Glu7Ala GAA-GCA	ATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGT ATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGA GAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGA	2300
	TCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTT CCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTG GCCGATTCAGAATTTTGTTGGCGTTTTCATGAT	2301
	TAAATTGGAAGAGTTTG	2302
	CAAACTCTTCCMTTTA	2303
Haemophilia B Glu7Lys gGAA-AAA	GATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGG TATAATTCAGGTAAATTGGGAAGAGTTTGTTCAAGGGAACCTTG AGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAG	2304
	CTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTC CCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGG CCGATTCAGAATTTTGTTGGCGTTTTCATGATC	2305
	GTAAATTGGGAAGAGTTT	2306
	AAACTCTTCCAATTTAC	2307
Haemophilia B Glu7Val GAA-GTA	ATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGT ATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGA GAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGA	2308
	TCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTT CCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTG GCCGATTCAGAATTTTGTTGGCGTTTTCATGAT	2309
	TAAATTGGAAGAGTTTG	2310
	CAAACTCTTCCAATTTA	2311
Haemophilia B Glu8Ala GAG-GCG	ATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATA ATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGA GAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC	2312
	GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCT TTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT	2313
	ATTGGAAGAGTTTGTTC	2314
	GAACAAACTCTTCCAAT	2315
Haemophilia B Glu8Gly GAG-GGG	ATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATA ATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGA GAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC	2316
	GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACCTCT TTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT	2317
	ATTGGAAGAGTTTGTTC	2318

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAACAAACTCTTCCAAT	2319
Haemophilia B Phe9Cys TTT-TGT	AAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAATTC AGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA ATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCACG	2320
	CGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTC AAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATAC CTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTT	2321
	GGAAGAGTTTGTTCAAG	2322
	CTTGAACAAACTCTTCC	2323
Haemophilia B Phe9Ile gTTT-ATT	GAAAACGCCAACAAAATTCTGAATCGGCCAAAGAGGTATAAT TCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGA GAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCAC	2324
	GTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCA AGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAATTATACC TCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTC	2325
	TGGAAGAGTTTGTTCAA	2326
	TTGAACAAACTCTTCCA	2327
Haemophilia B Arg(-1)Ser AGGt-AGC	TTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATTC TGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGT TTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAA	2328
	TTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCC AATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTT GGCGTTTTCATGATCAAGAAAAACTGAAATGTAA	2329
	CCAAAGAGGTATAATTC	2330
	GAATTATACCTCTTTGG	2331
Haemophilia B Arg(-1)Thr AGG-ACG	TTTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAATT CTGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAG TTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA	2332
	TCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCA ATTTACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTTG GCGTTTTCATGATCAAGAAAAACTGAAATGTAAA	2333
	GCCAAAGAGGTATAATT	2334
	AATTATACCTCTTTGGC	2335
Haemophilia B Lys(-2)Asn AAGa-AAT	CTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCCAACAAAA TTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATTGGAAG AGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATG	2336
	CATACATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATT TACCTGAATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGC GTTTTCATGATCAAGAAAAACTGAAATGTAAAAG	2337
	CGGCCAAAGAGGTATAA	2338
	TTATACCTCTTTGGCCG	2339
Haemophilia B Arg(-4)Gln CGG-CAG	AATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCC AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA	2340

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTC ATGATCAAGAAAAACTGAAATGTAAAAGAATAATT	2341
	TCTGAATCGGCCAAAGA	2342
	TCTTTGGCCGATTCAGA	2343
Haemophilia B Arg(-4)Leu CGG-CTG	AATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGCC AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGA	2344
	TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTC ATGATCAAGAAAAACTGAAATGTAAAAGAATAATT	2345
	TCTGAATCGGCCAAAGA	2346
	TCTTTGGCCGATTCAGA	2347
Haemophilia B Arg(-4)Trp tCGG-TGG	GAATTATTCTTTTACATTTCAGTTTTTCTTGATCATGAAAACGC CAACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAA TTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAG	2348
	CTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGA ATTATACCTCTTTGGCCGATTCAGAATTTTGTTGGCGTTTTCAT GATCAAGAAAAACTGAAATGTAAAAGAATAATTC	2349
	TTCTGAATCGGCCAAAG	2350
	CTTTGGCCGATTCAGAA	2351
Haemophilia B GinIITerm tCAA-TAA	GCCAACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGT AAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGT ATGGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAG	2352
	CTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCT CTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTGAAT TATACCTCTTTGGCCGATTCAGAATTTTGTTGGC	2353
	AGTTTGTTCAAGGGAAC	2354
	GTTCCCTTGAACAAACT	2355
Haemophilia B Gly12Ala GGG-GCG	ACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATT GGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA AGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTT	2356
	AAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACA TTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCT GAATTATACCTCTTTGGCCGATTCAGAATTTTGT	2357
	TGTTCAAGGGAACCTTG	2358
	CAAGGTTCCCTTGAACA	2359
Haemophilia B Gly12Arg aGGG-AGG	AACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAAT TGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGG AAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTT	2360
	AAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACAT TCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCTG AATTATACCTCTTTGGCCGATTCAGAATTTTGTT	2361
	TTGTTCAAGGGAACCTT	2362
	AAGGTTCCCTTGAACAA	2363

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Gly12Glu GGG-GAG	ACAAAATTCTGAATCGGCCAAAGAGGTATAATTCAGGTAAATT GGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGA AGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTT	2364
	AAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACA TTCTCTCTCAAGGTTCCCTTGAACAAACTCTTCCAATTTACCT GAATTATACCTCTTTGGCCGATTCAGAATTTTGT	2365
	TGTTCAAGGGAACCTTG	2366
	CAAGGTTCCCTTGAACA	2367
Haemophilia B Glu17Gln aGAA-CAA	CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGT TTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAA	2368
	TTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACAC TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCG	2369
	TTGAGAGAGAATGTATG	2370
	CATACATTCTCTCTCAA	2371
Haemophilia B Glu17Lys aGAA-AAA	CGGCCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTT CAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGT TTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAA	2372
	TTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACAC TTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAACTC TTCCAATTTACCTGAATTATACCTCTTTGGCCG	2373
	TTGAGAGAGAATGTATG	2374
	CATACATTCTCTCTCAA	2375
Haemophilia B Cys18Arg aTGT-CGT	CCAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAG GGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTG AAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA	2376
	TTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTA CACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAAA CTCTTCCAATTTACCTGAATTATACCTCTTTGG	2377
	AGAGAGAATGTATGGAA	2378
	TTCCATACATTCTCTCT	2379
Haemophilia B Cys18Tyr TGT-TAT	CAAAGAGGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGG GAACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGA AGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAAC	2380
	GTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACT ACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTTGAACAA ACTCTTCCAATTTACCTGAATTATACCTCTTTG	2381
	GAGAGAATGTATGGAAG	2382
	CTTCCATACATTCTCTC	2383
Haemophilia B Glu20Val GAA-GTA	GGTATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCT TGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGC ACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAG	2384

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTC AAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCCTT GAACAAACTCTTCCAATTTACCTGAATTATACC	2385
	ATGTATGGAAGAAAAGT	2386
	ACTTTTCTTCCATACAT	2387
Haemophilia B Glu21Lys aGAA-AAA	TATAATTCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTG AGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCAC GAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGTA	2388
	TACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCT TCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAGGTTCCC TTGAACAAACTCTTCCAATTTACCTGAATTATA	2389
	GTATGGAAGAAAAGTGT	2390
	ACACTTTTCTTCCATAC	2391
Haemophilia B Cys23Arg gTGT-CGT	TCAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGA GAATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAA GTTTTTGAAAACACTGAAAGAACAGTGAGTATTTCCA	2392
	TGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGT GCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCAAG GTTCCCTTGAACAAACTCTTCCAATTTACCTGA	2393
	AAGAAAAGTGTAGTTTT	2394
	AAAACTACACTTTTCTT	2395
Haemophilia B Cys23Tyr TGT-TAT	CAGGTAAATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAG AATGTATGGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAG TTTTTGAAAACACTGAAAGAACAGTGAGTATTTCCAC	2396
	GTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAACTTCTC GTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTCTCTCA AGGTTCCCTTGAACAAACTCTTCCAATTTACCTG	2397
	AGAAAAGTGTAGTTTTG	2398
	CAAAACTACACTTTTCT	2399
Haemophilia B Phe25Ser TTT-TCT	AATTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTA TGGAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTG AAAACACTGAAAGAACAGTGAGTATTTCCACATAATA	2400
	TATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAAAC TTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTCTC TCTCAAGGTTCCCTTGAACAAACTCTTCCAATT	2401
	GTGTAGTTTTGAAGAAG	2402
	CTTCTTCAAAACTACAC	2403
Haemophilia B Glu26Gln tGAA-CAA	TTGGAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATG GAAGAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAA ACACTGAAAGAACAGTGAGTATTTCCACATAATACC	2404
	GGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTCAAAA ACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATACATTC TCTCTCAAGGTTCCCTTGAACAAACTCTTCCAA	2405
	GTAGTTTTGAAGAAGCA	2406
	TGCTTCTTCAAAACTAC	2407

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Glu27Ala GAA-GCA	AAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAG AAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACAC TGAAAGAACAGTGAGTATTTCCACATAATACCCTTC	2408
	GAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTT CAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATA CATTCTCTCTCAAGGTTCCCTTGAACAAACTCTT	2409
	TTTTGAAGAAGCACGAG	2410
	CTCGTGCTTCTTCAAAA	2411
Haemophilia B Glu27Asp GAAg-GAC	AGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGA AAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACT GAAAGAACAGTGAGTATTTCCACATAATACCCTTCA	2412
	TGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTT TCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCAT ACATTCTCTCTCAAGGTTCCCTTGAACAAACTCT	2413
	TTTGAAGAAGCACGAGA	2414
	TCTCGTGCTTCTTCAAA	2415
Haemophilia B Glu27Lys aGAA-AAA	GAAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAA GAAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACA CTGAAAGAACAGTGAGTATTTCCACATAATACCCTT	2416
	AAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTTC AAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATAC ATTCTCTCTCAAGGTTCCCTTGAACAAACTCTTC	2417
	GTTTTGAAGAAGCACGA	2418
	TCGTGCTTCTTCAAAAC	2419
Haemophilia B Glu27Val GAA-GTA	AAGAGTTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAG AAAAGTGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACAC TGAAAGAACAGTGAGTATTTCCACATAATACCCTTC	2420
	GAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGTGTTTT CAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTTCCATA CATTCTCTCTCAAGGTTCCCTTGAACAAACTCTT	2421
	TTTTGAAGAAGCACGAG	2422
	CTCGTGCTTCTTCAAAA	2423
Haemophilia B Arg29Gln CGA-CAA	TTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT GTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAG AACAGTGAGTATTTCCACATAATACCCTTCAGATGC	2424
	GCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAG TGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCT TCCATACATTCTCTCTCAAGGTTCCCTTGAACAA	2425
	AGAAGCACGAGAAGTTT	2426
	AAACTTCTCGTGCTTCT	2427
Haemophilia B Arg29Pro CGA-CCA	TTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGT GTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAG AACAGTGAGTATTTCCACATAATACCCTTCAGATGC	2428

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAG TGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCT TCCATACATTCTCTCTCAAGGTTCCCTTGAACAA	2429
	AGAAGCACGAGAAGTTT	2430
	AAACTTCTCGTGCTTCT	2431
Haemophilia B Arg29Term aCGA-TGA	TTTGTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAG TGTAGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAA GAACAGTGAGTATTTCCACATAATACCCTTCAGATG	2432
	CATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTCAGT GTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTTCTT CCATACATTCTCTCTCAAGGTTCCCTTGAACAAA	2433
	AAGAAGCACGAGAAGTT	2434
	AACTTCTCGTGCTTCTT	2435
Haemophilia B Glu30Lys aGAA-AAA	GTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGT AGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA CAGTGAGTATTTCCACATAATACCCTTCAGATGCAG	2436
	CTGCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTC AGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTT CTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC	2437
	AAGCACGAGAAGTTTTT	2438
	AAAAACTTCTCGTGCTT	2439
Haemophilia B Glu30Term aGAA-TAA	GTTCAAGGGAACCTTGAGAGAGAATGTATGGAAGAAAAGTGT AGTTTTGAAGAAGCACGAGAAGTTTTTGAAAACACTGAAAGAA CAGTGAGTATTTCCACATAATACCCTTCAGATGCAG	2440
	CTGCATCTGAAGGGTATTATGTGGAAATACTCACTGTTCTTTC AGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAACTACACTTTT CTTCCATACATTCTCTCTCAAGGTTCCCTTGAAC	2441
	AAGCACGAGAAGTTTTT	2442
	AAAAACTTCTCGTGCTT	2443
Haemophilia B Glu33Asp GAAa-GAC	CCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAAGA AGCACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGTAT TTCCACATAATACCCTTCAGATGCAGAGCATAGAATA	2444
	TATTCTATGCTCTGCATCTGAAGGGTATTATGTGGAAATACTC ACTGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAA ACTACACTTTTCTTCCATACATTCTCTCTCAAGG	2445
	GTTTTTGAAAACACTGA	2446
	TCAGTGTTTTCAAAAAC	2447
Haemophilia B Glu33Term tGAA-TAA	AACCTTGAGAGAGAATGTATGGAAGAAAAGTGTAGTTTTGAA GAAGCACGAGAAGTTTTTGAAAACACTGAAAGAACAGTGAGT ATTTCCACATAATACCCTTCAGATGCAGAGCATAGAA	2448
	TTCTATGCTCTGCATCTGAAGGGTATTATGTGGAAATACTCAC TGTTCTTTCAGTGTTTTCAAAAACTTCTCGTGCTTCTTCAAAAC TACACTTTTCTTCCATACATTCTCTCTCAAGGTT	2449

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AAGTTTTTGAAAACACT	2450
	AGTGTTTTCAAAAACTT	2451
Haemophilia B Trp42Term TGG-TAG	CAAAACACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTT TATAGACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCAT TTTATCCTCTAGCTAATATATGAAACATATGAG	2452
	CTCATATGTTTCATATATTAGCTAGAGGATAAAATGAATTGCTT ACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAATAAAAG AAGACAAATTAACGGTAATATCTAAAGTGTTTTG	2453
	TGAATTTTGGAAGCAGT	2454
	ACTGCTTCCAAAATTCA	2455
Haemophilia B Lys43Glu gAAG-GAG	AAACACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTTTA TAGACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCATTT TATCCTCTAGCTAATATATGAAACATATGAGAA	2456
	TTCTCATATGTTTCATATATTAGCTAGAGGATAAAATGAATTGC TTACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAATAAAA GAAGACAAATTAACGGTAATATCTAAAGTGTTT	2457
	AATTTTGGAAGCAGTAT	2458
	ATACTGCTTCCAAAATT	2459
Haemophilia B Gln44Term gCAG-TAG	CACTTTAGATATTACCGTTAATTTGTCTTCTTTTATTCTTTATAG ACTGAATTTTGGAAGCAGTATGTTGGTAAGCAATTCATTTTAT CCTCTAGCTAATATATGAAACATATGAGAATTA	2460
	TAATTCTCATATGTTTCATATATTAGCTAGAGGATAAAATGAAT TGCTTACCAACATACTGCTTCCAAAATTCAGTCTATAAAGAAT AAAAGAAGACAAATTAACGGTAATATCTAAAGTG	2461
	TTTGGAAGCAGTATGTT	2462
	AACATACTGCTTCCAAA	2463
Haemophilia B Asp49Gly GAT-GGT	CCGGGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAAC CTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTA AATGGCGGCAGTTGCAAGGATGACATTAATTCCTA	2464
	TAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATG GATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAAGAA ATTGAATTGGCACGTAAACTGCTTAGAATGCCCGG	2465
	AGATGGAGATCAGTGTG	2466
	CACACTGATCTCCATCT	2467
Haemophilia B Gln50His CAGt-CAC	GCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTAT CTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATG GCGGCAGTTGCAAGGATGACATTAATTCCTATGAA	2468
	TTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAA CATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTA AGAAATTGAATTGGCACGTAAACTGCTTAGAATGC	2469
	GGAGATCAGTGTGAGTC	2470
	GACTCACACTGATCTCC	2471
Haemophilia B Gln50Pro CAG-CCG	GGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTA TCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAAT GGCGGCAGTTGCAAGGATGACATTAATTCCTATGA	2472

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAAC ATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAA GAAATTGAATTGGCACGTAAACTGCTTAGAATGCC	2473
	TGGAGATCAGTGTGAGT	2474
	ACTCACACTGATCTCCA	2475
Haemophilia B Gln50Term tCAG-TAG	GGGCATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCT ATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAA TGGCGGCAGTTGCAAGGATGACATTAATTCCTATG	2476
	CATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACA TGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTTAAG AAATTGAATTGGCACGTAAACTGCTTAGAATGCCC	2477
	ATGGAGATCAGTGTGAG	2478
	CTCACACTGATCTCCAT	2479
Haemophilia B Cys51Arg gTGT-CGT	CATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCT CAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGG CGGCAGTTGCAAGGATGACATTAATTCCTATGAAT	2480
	ATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAA ACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTT AAGAAATTGAATTGGCACGTAAACTGCTTAGAATG	2481
	GAGATCAGTGTGAGTCC	2482
	GGACTCACACTGATCTC	2483
Haemophilia B Cys51Ser gTGT-AGT	CATTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCT CAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGG CGGCAGTTGCAAGGATGACATTAATTCCTATGAAT	2484
	ATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAA ACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGGTT AAGAAATTGAATTGGCACGTAAACTGCTTAGAATG	2485
	GAGATCAGTGTGAGTCC	2486
	GGACTCACACTGATCTC	2487
Haemophilia B Cys51Trp TGTg-TGG	TTCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCTCA AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGT	2488
	ACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTT AAACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAGG TTAAGAAATTGAATTGGCACGTAAACTGCTTAGAA	2489
	GATCAGTGTGAGTCCAA	2490
	TTGGACTCACACTGATC	2491
Haemophilia B Glu52Term tGAG-TAG	TCTAAGCAGTTTACGTGCCAATTCAATTTCTTAACCTATCTCAA AGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCGG CAGTTGCAAGGATGACATTAATTCCTATGAATGTT	2492
	AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTTTGAGATAG GTTAAGAAATTGAATTGGCACGTAAACTGCTTAGA	2493
	ATCAGTGTGAGTCCAAT	2494
	ATTGGACTCACACTGAT	2495

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Pro55Ala tCCA-GCA	TTTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAG ATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCT	2496
	AGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACT GCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATCT TTGAGATAGGTTAAGAAATTGAATTGGCACGTAAA	2497
	AGTCCAATCCATGTTTA	2498
	TAAACATGGATTGGACT	2499
Haemophilia B Pro55Arg CCA-CGA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2500
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2501
	GTCCAATCCATGTTTAA	2502
	TTAAACATGGATTGGAC	2503
Haemophilia B Pro55Gln CCA-CAA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2504
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2505
	GTCCAATCCATGTTTAA	2506
	TTAAACATGGATTGGAC	2507
Haemophilia B Pro55Leu CCA-CTA	TTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGA TCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAA GGATGACATTAATTCCTATGAATGTTGGTGTCCCTT	2508
	AAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAAC TGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATC TTTGAGATAGGTTAAGAAATTGAATTGGCACGTAA	2509
	GTCCAATCCATGTTTAA	2510
	TTAAACATGGATTGGAC	2511
Haemophilia B Pro55Ser tCCA-TCA	TTTACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAG ATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCT	2512
	AGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACT GCCGCCATTTAAACATGGATTGGACTCACACTGATCTCCATCT TTGAGATAGGTTAAGAAATTGAATTGGCACGTAAA	2513
	AGTCCAATCCATGTTTA	2514
	TAAACATGGATTGGACT	2515
Haemophilia B Cys56Arg aTGT-CGT	ACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATC AGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGG ATGACATTAATTCCTATGAATGTTGGTGTCCCTTTG	2516
	CAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCA ACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACGT	2517

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAATCCATGTTTAAAT	2518
	ATTTAAACATGGATTGG	2519
Haemophilia B Cys56Ser aTGT-AGT	ACGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATC AGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGG ATGACATTAATTCCTATGAATGTTGGTGTCCCTTTG	2520
	CAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGCA ACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACGT	2521
	CCAATCCATGTTTAAAT	2522
	ATTTAAACATGGATTGG	2523
Haemophilia B Cys56Ser TGT-TCT	CGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATCA GTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGA TGACATTAATTCCTATGAATGTTGGTGTCCCTTTGG	2524
	CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACG	2525
	CAATCCATGTTTAAATG	2526
	CATTTAAACATGGATTG	2527
Haemophilia B Cys56Tyr TGT-TAT	CGTGCCAATTCAATTTCTTAACCTATCTCAAAGATGGAGATCA GTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGA TGACATTAATTCCTATGAATGTTGGTGTCCCTTTGG	2528
	CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACACTGATCTCC ATCTTTGAGATAGGTTAAGAAATTGAATTGGCACG	2529
	CAATCCATGTTTAAATG	2530
	CATTTAAACATGGATTG	2531
Haemophilia B Asn58Lys AATg-AAG	ATTCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAG TCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAA	2532
	TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACACT GATCTCCATCTTTGAGATAGGTTAAGAAATTGAAT	2533
	TGTTTAAATGGCGGCAG	2534
	CTGCCGCCATTTAAACA	2535
Haemophilia B Gly59Asp GGC-GAC	TCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTC CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG	2536
	CCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGT CATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACA CTGATCTCCATCTTTGAGATAGGTTAAGAAATTGA	2537
	TTTAAATGGCGGCAGTT	2538
	AACTGCCGCCATTTAAA	2539

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Gly59Val GGC-GTC	TCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTC CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG	2540
	CCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGT CATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACA CTGATCTCCATCTTTGAGATAGGTTAAGAAATTGA	2541
	TTTAAATGGCGGCAGTT	2542
	AACTGCCGCCATTTAAA	2543
Haemophilia B Gly59Ser tGGC-AGC	TTCAATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGT CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAG	2544
	CTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTC ATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCACAC TGATCTCCATCTTTGAGATAGGTTAAGAAATTGAA	2545
	GTTTAAATGGCGGCAGT	2546
	ACTGCCGCCATTTAAAC	2547
Haemophilia B Gly60Ser cGGC-AGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2548
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2549
	TAAATGGCGGCAGTTGC	2550
	GCAACTGCCGCCATTTA	2551
Haemophilia B Gly60Cys cGGC-TGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2552
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2553
	TAAATGGCGGCAGTTGC	2554
	GCAACTGCCGCCATTTA	2555
Haemophilia B Gly60Asp GGC-GAC	ATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAA TCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCC TATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAA	2556
	TTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAA TGTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTC ACACTGATCTCCATCTTTGAGATAGGTTAAGAAAT	2557
	AAATGGCGGCAGTTGCA	2558
	TGCAACTGCCGCCATTT	2559
Haemophilia B Gly60Arg cGGC-CGC	AATTTCTTAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCA ATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTC CTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA	2560

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAAT GTCATCCTTGCAACTGCCGCCATTTAAACATGGATTGGACTCA CACTGATCTCCATCTTTGAGATAGGTTAAGAAATT	2561
	TAAATGGCGGCAGTTGC	2562
	GCAACTGCCGCCATTTA	2563
Haemophilia B Cys62Tyr TGC-TAC	TAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATG TTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAA TGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTG	2564
	CAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGG AATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGATT GGACTCACACTGATCTCCATCTTTGAGATAGGTTA	2565
	CGGCAGTTGCAAGGATG	2566
	CATCCTTGCAACTGCCG	2567
Haemophilia B Cys62Ser TGC-TCC	TAACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATG TTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAA TGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTG	2568
	CAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGG AATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGATT GGACTCACACTGATCTCCATCTTTGAGATAGGTTA	2569
	CGGCAGTTGCAAGGATG	2570
	CATCCTTGCAACTGCCG	2571
Haemophilia B Cys62Term TGCa-TGA	AACCTATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGT TTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAAT GTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGT	2572
	ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACATGGAT TGGACTCACACTGATCTCCATCTTTGAGATAGGTT	2573
	GGCAGTTGCAAGGATGA	2574
	TCATCCTTGCAACTGCC	2575
Haemophilia B Asp64Glu GATg-GAG	TCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAAT GGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGG TGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTA	2576
	TAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACAT TCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAAC ATGGATTGGACTCACACTGATCTCCATCTTTGAGA	2577
	TGCAAGGATGACATTAA	2578
	TTAATGTCATCCTTGCA	2579
Haemophilia B Asp64Gly GAT-GGT	ATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAA TGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTG GTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATT	2580
	AATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATT CATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACA TGGATTGGACTCACACTGATCTCCATCTTTGAGAT	2581
	TTGCAAGGATGACATTA	2582

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAATGTCATCCTTGCAA	2583
Haemophilia B Asp64Asn gGAT-AAT	TATCTCAAAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAA ATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTT GGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAAT	2584
	ATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTC ATAGGAATTAATGTCATCCTTGCAACTGCCGCCATTTAAACAT GGATTGGACTCACACTGATCTCCATCTTTGAGATA	2585
	GTTGCAAGGATGACATT	2586
	AATGTCATCCTTGCAAC	2587
Haemophilia B Ile66Ser ATT-AGT	AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCC CTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAA	2588
	TTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACC AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTT	2589
	GGATGACATTAATTCCT	2590
	AGGAATTAATGTCATCC	2591
Haemophilia B Ile66Thr ATT-ACT	AAGATGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCG GCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCC CTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAA	2592
	TTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACC AACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGCCATT TAAACATGGATTGGACTCACACTGATCTCCATCTT	2593
	GGATGACATTAATTCCT	2594
	AGGAATTAATGTCATCC	2595
Haemophilia B Asn67Lys AATt-AAA	TGGAGATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAG TTGCAAGGATGACATTAATTCCTATGAATGTTGGTGTCCCTTT GGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTAA	2596
	TTACTTACCTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGA CACCAACATTCATAGGAATTAATGTCATCCTTGCAACTGCCGC CATTTAAACATGGATTGGACTCACACTGATCTCCA	2597
	GACATTAATTCCTATGA	2598
	TCATAGGAATTAATGTC	2599
Haemophilia B Tyr69Cys TAT-TGT	ATCAGTGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCA AGGATGACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATT TGAAGGAAAGAACTGTGAATTAGGTAAGTAACTATT	2600
	AATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAATCCAAA GGGACACCAACATTCATAGGAATTAATGTCATCCTTGCAACTG CCGCCATTTAAACATGGATTGGACTCACACTGAT	2601
	TAATTCCTATGAATGTT	2602
	AACATTCATAGGAATTA	2603

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Cys71Term TGTt-TGA	TGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGA CATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGA AAGAACTGTGAATTAGGTAAGTAACTATTTTTTGAA	2604
	TTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAA ATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTT GCAACTGCCGCCATTTAAACATGGATTGGACTCA	2605
	TATGAATGTTGGTGTCC	2606
	GGACACCAACATTCATA	2607
Haemophilia B Cys71Ser TGT-TCT	GTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATG ACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG AAAGAACTGTGAATTAGGTAAGTAACTATTTTTTGA	2608
	TCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAA TCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTG CAACTGCCGCCATTTAAACATGGATTGGACTCAC	2609
	CTATGAATGTTGGTGTC	2610
	GACACCAACATTCATAG	2611
Haemophilia B Cys71Tyr TGT-TAT	GTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATG ACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGG AAAGAACTGTGAATTAGGTAAGTAACTATTTTTTGA	2612
	TCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAA TCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTG CAACTGCCGCCATTTAAACATGGATTGGACTCAC	2613
	CTATGAATGTTGGTGTC	2614
	GACACCAACATTCATAG	2615
Haemophilia B Cys71Ser aTGT-AGT	TGTGAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGAT GACATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAG GAAAGAACTGTGAATTAGGTAAGTAACTATTTTTTG	2616
	CAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCAAAT CCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCTTGC AACTGCCGCCATTTAAACATGGATTGGACTCACA	2617
	CCTATGAATGTTGGTGT	2618
	ACACCAACATTCATAGG	2619
Haemophilia B Trp72Arg tTGG-AGG	GAGTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGAC ATTAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAA AGAACTGTGAATTAGGTAAGTAACTATTTTTTGAAT	2620
	ATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTTCA AATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATCCT TGCAACTGCCGCCATTTAAACATGGATTGGACTC	2621
	ATGAATGTTGGTGTCCC	2622
	GGGACACCAACATTCAT	2623
Haemophilia B Trp72Term TGGt-TGA	GTCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACAT TAATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAG AACTGTGAATTAGGTAAGTAACTATTTTTTGAATAC	2624

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCTT CAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCATC CTTGCAACTGCCGCCATTTAAACATGGATTGGAC	2625
	GAATGTTGGTGTCCCTT	2626
	AAGGGACACCAACATTC	2627
Haemophilia B Cys73Tyr TGT-TAT	CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAA CTGTGAATTAGGTAAGTAACTATTTTTTGAATACTC	2628
	GAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCC TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGG	2629
	ATGTTGGTGTCCCTTTG	2630
	CAAAGGGACACCAACAT	2631
Haemophilia B Cys73Arg gTGT-CGT	TCCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAA CTGTGAATTAGGTAAGTAACTATTTTTTGAATACT	2632
	AGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCCT TCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCAT CCTTGCAACTGCCGCCATTTAAACATGGATTGGA	2633
	AATGTTGGTGTCCCTTT	2634
	AAAGGGACACCAACATT	2635
Haemophilia B Cys73Phe TGT-TTT	CCAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTA ATTCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAA CTGTGAATTAGGTAAGTAACTATTTTTTGAATACTC	2636
	GAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTCC TTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTCA TCCTTGCAACTGCCGCCATTTAAACATGGATTGG	2637
	ATGTTGGTGTCCCTTTG	2638
	CAAAGGGACACCAACAT	2639
Haemophilia B Cys73Term TGTc-TGA	CAATCCATGTTTAAATGGCGGCAGTTGCAAGGATGACATTAAT TCCTATGAATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACT GTGAATTAGGTAAGTAACTATTTTTTGAATACTCA	2640
	TGAGTATTCAAAAAATAGTTACTTACCTAATTCACAGTTCTTTC CTTCAAATCCAAAGGGACACCAACATTCATAGGAATTAATGTC ATCCTTGCAACTGCCGCCATTTAAACATGGATTG	2641
	TGTTGGTGTCCCTTTGG	2642
	CCAAAGGGACACCAACA	2643
Haemophilia B Gly76Val GGA-GTA	GTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGA ATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTA GGTAAGTAACTATTTTTTGAATACTCATGGTTCAA	2644
	TTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTCACA GTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAA TTAATGTCATCCTTGCAACTGCCGCCATTTAAAC	2645
	TCCCTTTGGATTTGAAG	2646

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTCAAATCCAAAGGGA	2647
Haemophilia B Gly76Arg tGGA-AGA	TGTTTAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATG AATGTTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATT AGGTAAGTAACTATTTTTTGAATACTCATGGTTCA	2648
	TGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTCACA GTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAGGAA TTAATGTCATCCTTGCAACTGCCGCCATTTAAACA	2649
	GTCCCTTTGGATTTGAA	2650
	TTCAAATCCAAAGGGAC	2651
Haemophilia B Phe77Cys TTT-TGT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2652
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2653
	CTTTGGATTTGAAGGAA	2654
	TTCCTTCAAATCCAAAG	2655
Haemophilia B Phe77Ser TTT-TCT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2656
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2657
	CTTTGGATTTGAAGGAA	2658
	TTCCTTCAAATCCAAAG	2659
Haemophilia B Phe77Tyr TTT-TAT	TAAATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATG TTGGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGT AAGTAACTATTTTTTGAATACTCATGGTTCAAAGT	2660
	ACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAATTC ACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCATAG GAATTAATGTCATCCTTGCAACTGCCGCCATTTA	2661
	CTTTGGATTTGAAGGAA	2662
	TTCCTTCAAATCCAAAG	2663
Haemophilia B Glu78Lys tGAA-AAA	AATGGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTT GGTGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTA AGTAACTATTTTTTGAATACTCATGGTTCAAAGTTT	2664
	AAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACCTAAT TCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACATTCAT AGGAATTAATGTCATCCTTGCAACTGCCGCCATT	2665
	TTGGATTTGAAGGAAAG	2666
	CTTTCCTTCAAATCCAA	2667
Haemophilia B Gly79Val GGA-GTA	GCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGT GTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTA ACTATTTTTTGAATACTCATGGTTCAAAGTTTCCCT	2668

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTAC CTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACA TTCATAGGAATTAATGTCATCCTTGCAACTGCCGC	2669
	ATTTGAAGGAAAGAACT	2670
	AGTTCTTTCCTTCAAAT	2671
Haemophilia B Gly79Arg aGGA-AGA	GGCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGG TGTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGT AACTATTTTTTGAATACTCATGGTTCAAAGTTTCCC	2672
	GGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTACC TAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACAT TCATAGGAATTAATGTCATCCTTGCAACTGCCGCC	2673
	GATTTGAAGGAAAGAAC	2674
	GTTCTTTCCTTCAAATC	2675
Haemophilia B Gly79Glu GGA-GAA	GCGGCAGTTGCAAGGATGACATTAATTCCTATGAATGTTGGT GTCCCTTTGGATTTGAAGGAAAGAACTGTGAATTAGGTAAGTA ACTATTTTTTGAATACTCATGGTTCAAAGTTTCCCT	2676
	AGGGAAACTTTGAACCATGAGTATTCAAAAAATAGTTACTTAC CTAATTCACAGTTCTTTCCTTCAAATCCAAAGGGACACCAACA TTCATAGGAATTAATGTCATCCTTGCAACTGCCGC	2677
	ATTTGAAGGAAAGAACT	2678
	AGTTCTTTCCTTCAAAT	2679
Haemophilia B Cys88Ser TGT-TCT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2680
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2681
	TGTAACATGTAACATTA	2682
	TAATGTTACATGTTACA	2683
Haemophilia B Cys88Phe TGT-TTT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2684
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2685
	TGTAACATGTAACATTA	2686
	TAATGTTACATGTTACA	2687
Haemophilia B Cys88Arg aTGT-CGT	TTTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCT TCTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAG CAGTTTTGTAAAAATAGTGCTGATAACAAGGTGG	2688
	CCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATCT GCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAAA	2689
	ATGTAACATGTAACATT	2690
	AATGTTACATGTTACAT	2691

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemophilia B Cys88Tyr TGT-TAT	TTAGAAATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTT CTTTTAGATGTAACATGTAACATTAAGAATGGCAGATGCGAGC AGTTTTGTAAAAATAGTGCTGATAACAAGGTGGT	2692
	ACCACCTTGTTATCAGCACTATTTTTACAAAACTGCTCGCATC TGCCATTCTTAATGTTACATGTTACATCTAAAAGAAGCAAAATA GACAGTAACAGCATCATTTAACATGCATTTCTAA	2693
	TGTAACATGTAACATTA	2694
	TAATGTTACATGTTACA	2695
Haemophilia B Ile90Thr ATT-ACT	ATGCATGTTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTA GATGTAACATGTAACATTAAGAATGGCAGATGCGAGCAGTTTT GTAAAAATAGTGCTGATAACAAGGTGGTTTGCTC	2696
	GAGCAAACCACCTTGTTATCAGCACTATTTTTACAAAACTGCT CGCATCTGCCATTCTTAATGTTACATGTTACATCTAAAAGAAG CAAAATAGACAGTAACAGCATCATTTAACATGCAT	2697
	ATGTAACATTAAGAATG	2698
	CATTCTTAATGTTACAT	2699
Haemophilia B Asn92His gAAT-CAT	TGTTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGT AACATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAA AATAGTGCTGATAACAAGGTGGTTTGCTCCTGTA	2700
	TACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACAAAA CTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCTAAA AGAAGCAAAATAGACAGTAACAGCATCATTTAACA	2701
	ACATTAAGAATGGCAGA	2702
	TCTGCCATTCTTAATGT	2703
Haemophilia B Asn92Lys AATg-AAA	TTAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAA CATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAA TAGTGCTGATAACAAGGTGGTTTGCTCCTGTACT	2704
	AGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACAA AACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCTA AAAGAAGCAAAATAGACAGTAACAGCATCATTTAA	2705
	ATTAAGAATGGCAGATG	2706
	CATCTGCCATTCTTAAT	2707
Haemophilia B Gly93Asp GGC-GAC	AAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACA TGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATA GTGCTGATAACAAGGTGGTTTGCTCCTGTACTGA	2708
	TCAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTAC AAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATC TAAAAGAAGCAAAATAGACAGTAACAGCATCATTT	2709
	TAAGAATGGCAGATGCG	2710
	CGCATCTGCCATTCTTA	2711
Haemophilia B Gly93Ser tGGC-AGC	TAAATGATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAAC ATGTAACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAAT AGTGCTGATAACAAGGTGGTTTGCTCCTGTACTG	2712

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTTTTACA AAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTACATCT AAAAGAAGCAAAATAGACAGTAACAGCATCATTTA	2713
	TTAAGAATGGCAGATGC	2714
	GCATCTGCCATTCTTAA	2715
Haemophilia B Arg94Ser AGAt-AGT	GATGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTA ACATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTG CTGATAACAAGGTGGTTTGCTCCTGTACTGAGGGA	2716
	TCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTATTT TTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTTA CATCTAAAAGAAGCAAAATAGACAGTAACAGCATC	2717
	AATGGCAGATGCGAGCA	2718
	TGCTCGCATCTGCCATT	2719
Haemophilia B Cys95Tyr TGC-TAC	TGCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAAC ATTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCT GATAACAAGGTGGTTTGCTCCTGTACTGAGGGATA	2720
	TATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTAT TTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGTT ACATCTAAAAGAAGCAAAATAGACAGTAACAGCA	2721
	TGGCAGATGCGAGCAGT	2722
	ACTGCTCGCATCTGCCA	2723
Haemophilia B Cys95Trp TGCg-TGG	GCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACA TTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTG ATAACAAGGTGGTTTGCTCCTGTACTGAGGGATAT	2724
	ATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTA TTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGT TACATCTAAAAGAAGCAAAATAGACAGTAACAGC	2725
	GGCAGATGCGAGCAGTT	2726
	AACTGCTCGCATCTGCC	2727
Haemophilia B Cys95Term TGCg-TGA	GCTGTTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACA TTAAGAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTG ATAACAAGGTGGTTTGCTCCTGTACTGAGGGATAT	2728
	ATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAGCACTA TTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTACATGT TACATCTAAAAGAAGCAAAATAGACAGTAACAGC	2729
	GGCAGATGCGAGCAGTT	2730
	AACTGCTCGCATCTGCC	2731
Haemophilia B Gln97Pro CAG-CCG	TACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAG AATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAAC AAGGTGGTTTGCTCCTGTACTGAGGGATATCGACT	2732
	AGTCGATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCA GCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTT ACATGTTACATCTAAAAGAAGCAAAATAGACAGTA	2733

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATGCGAGCAGTTTTGTA	2734
	TACAAAACTGCTCGCAT	2735
Haemophilia B Gln97Glu gCAG-GAG	TTACTGTCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAA GAATGGCAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAA CAAGGTGGTTTGCTCCTGTACTGAGGGATATCGAC	2736
	GTCGATATCCCTCAGTACAGGAGCAAACCACCTTGTTATCAG CACTATTTTTACAAAACTGCTCGCATCTGCCATTCTTAATGTTA CATGTTACATCTAAAAGAAGCAAAATAGACAGTAA	2737
	GATGCGAGCAGTTTTGT	2738
	ACAAAACTGCTCGCATC	2739
Haemophilia B Cys99Arg tTGT-CGT	TCTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAGAATGG CAGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAACAAGGT GGTTTGCTCCTGTACTGAGGGATATCGACTTGCAG	2740
	CTGCAAGTCGATATCCCTCAGTACAGGAGCAAACCACCTTGT TATCAGCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTTA ATGTTACATGTTACATCTAAAAGAAGCAAAATAGA	2741
	AGCAGTTTTGTAAAAAT	2742
	ATTTTTACAAAACTGCT	2743
Haemophilia B Cys99Tyr TGT-TAT	CTATTTTGCTTCTTTTAGATGTAACATGTAACATTAAGAATGGC AGATGCGAGCAGTTTTGTAAAAATAGTGCTGATAACAAGGTG GTTTGCTCCTGTACTGAGGGATATCGACTTGCAGA	2744
	TCTGCAAGTCGATATCCCTCAGTACAGGAGCAAACCACCTTG TTATCAGCACTATTTTTACAAAACTGCTCGCATCTGCCATTCTT AATGTTACATGTTACATCTAAAAGAAGCAAAATAG	2745
	GCAGTTTTGTAAAAATA	2746
	TATTTTTACAAAACTGC	2747
Warfarin sensitivity Ala(-10)Thr cGCC-ACC	TTTTTTGCTAAAACTAAAGAATTATTCTTTTACATTTCAGTTTTT CTTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGA GGTATAATTCAGGTAAATTGGAAGAGTTTGTTC	2748
	GAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCCG ATTCAGAATTTTGTTGGCGTTTTCATGATCAAGAAAAACTGAA ATGTAAAAGAATAATTCTTTAGTTTTAGCAAAAAA	2749
	ATGAAAACGCCAACAAA	2750
	TTTGTTGGCGTTTTCAT	2751
Warfarin sensitivity Ala(-10)Val GCC-GTC	TTTTTGCTAAAACTAAAGAATTATTCTTTTACATTTCAGTTTTTC TTGATCATGAAAACGCCAACAAAATTCTGAATCGGCCAAAGA GGTATAATTCAGGTAAATTGGAAGAGTTTGTTCA	2752
	TGAACAAACTCTTCCAATTTACCTGAATTATACCTCTTTGGCC GATTCAGAATTTTGTTGGCGTTTTCATGATCAAGAAAAACTGA AATGTAAAAGAATAATTCTTTAGTTTTAGCAAAAA	2753
	TGAAAACGCCAACAAAA	2754
	TTTTGTTGGCGTTTTCA	2755
Haemophilia B Gly(-26)Val GGA-GTA	TGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCCTCA TCACCATCTGCCTTTTAGGATATCTACTCAGTGCTGAATGTAC AGGTTTGTTTCCTTTTTTAAAATACATTGAGTATGC	2756

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCATACTCAATGTATTTTAAAAAAGGAAACAAACCTGTACATT CAGCACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGC CTGGTGATTCTGCCATGATCATGTTCACGCGCTGCA	2757
	CCTTTTAGGATATCTAC	2758
	GTAGATATCCTAAAAGG	2759
Haemophilia B Leu(-27)Term TTA-TAA	TTATGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCC TCATCACCATCTGCCTTTTAGGATATCTACTCAGTGCTGAATG TACAGGTTTGTTTCCTTTTTTAAAATACATTGAGTA	2760
	TACTCAATGTATTTTAAAAAAGGAAACAAACCTGTACATTCAG CACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGCCTG GTGATTCTGCCATGATCATGTTCACGCGCTGCATAA	2761
	CTGCCTTTTAGGATATC	2762
	GATATCCTAAAAGGCAG	2763
Haemophilia B Ile(-30)Asn ATC-AAC	TAGCAAAGGTTATGCAGCGCGTGAACATGATCATGGCAGAAT CACCAGGCCTCATCACCATCTGCCTTTTAGGATATCTACTCAG TGCTGAATGTACAGGTTTGTTTCCTTTTTTAAAATA	2764
	TATTTTAAAAAAGGAAACAAACCTGTACATTCAGCACTGAGTA GATATCCTAAAAGGCAGATGGTGATGAGGCCTGGTGATTCTG CCATGATCATGTTCACGCGCTGCATAACCTTTGCTA	2765
	CATCACCATCTGCCTTT	2766
	AAAGGCAGATGGTGATG	2767
Haemophilia B Ile(-40)Phe gATC-TTC	ACTAATCGACCTTACCACTTTCACAATCTGCTAGCAAAGGTTA TGCAGCGCGTGAACATGATCATGGCAGAATCACCAGGCCTCA TCACCATCTGCCTTTTAGGATATCTACTCAGTGCTG	2768
	CAGCACTGAGTAGATATCCTAAAAGGCAGATGGTGATGAGGC CTGGTGATTCTGCCATGATCATGTTCACGCGCTGCATAACCTT TGCTAGCAGATTGTGAAAGTGGTAAGGTCGATTAGT	2769
	TGAACATGATCATGGCA	2770
	TGCCATGATCATGTTCA	2771
Haemophilia B Arg(-44)His CGC-CAC	ACTTTGGTACAACTAATCGACCTTACCACTTTCACAATCTGCT AGCAAAGGTTATGCAGCGCGTGAACATGATCATGGCAGAATC ACCAGGCCTCATCACCATCTGCCTTTTAGGATATCT	2772
	AGATATCCTAAAAGGCAGATGGTGATGAGGCCTGGTGATTCT GCCATGATCATGTTCACGCGCTGCATAACCTTTGCTAGCAGA TTGTGAAAGTGGTAAGGTCGATTAGTTGTACCAAAGT	2773
	TATGCAGCGCGTGAACA	2774
	TGTTCACGCGCTGCATA	2775

EXAMPLE 15

Alpha thalassemia - Hemoglobin alpha locus 1

The thalassemia syndromes are a heterogeneous group of inherited anemias characterized by defects in the synthesis of one or more globin chain subunits. For example, beta-thalassemia discussed in Example 6, is caused by a decrease in beta-chain production relative to alpha-chain production; the converse is the case for alpha-thalassemia. The attached table discloses the correcting oligonucleotide base sequences for the hemoglobin alpha locus 1 oligonucleotides of the invention.

Table 22

HBA1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Met(-1)Val cATG-GTG	CCCTGGCGCGCTCGCGGCCCGGCACTCTTCTGGTCCCCACA GACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACAA GACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGC	2776
	GCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTG TCGGCAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGT GGGGACCAGAAGAGTGCCGGGCCGCGAGCGCGCCAGGG	2777
	AACCCACCATGGTGCTG	2778
	CAGCACCATGGTGGGTT	2779
Haemoglobin variant Ala12Asp GCC-GAC	CACAGACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCG ACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCG CACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTG	2780
	CACCTCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGC GCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGG CAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGTG	2781
	CGTCAAGGCCGCCTGGG	2782
	CCCAGGCGGCCTTGACG	2783
Haemoglobin variant Gly15Asp GGT-GAT	AGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAGACCA ACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGG CGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCT	2784
	AGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCC AGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGG TCTTGTCGGCAGGAGACAGCACCATGGTGGGTTCTCTCT	2785
	CGCCTGGGGTAAGGTCG	2786
	CGACCTTACCCCAGGCG	2787

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemoglobin variant Tyr24Cys TAT-TGT	CTGCCGACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTC GGCGCGCACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGA GGTGAGGCTCCCTCCCCTGCTCCGACCCGGGCTCCTCGCC	2788
	GGCGAGGAGCCCGGGTCGGAGCAGGGGAGGGAGCCTCACC TCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGCGCCG ACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGGCAG	2789
	TGGCGAGTATGGTGCGG	2790
	CCGCACCATACTCGCCA	2791
Haemoglobin variant Glu27Asp GAGg-GAT	GACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCAC GCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCT CCCTCCCCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGACC	2792
	GGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGGGGAG GGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCCAG CGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTC	2793
	GGTGCGGAGGCCCTGGA	2794
	TCCAGGGCCTCCGCACC	2795
Haemoglobin variant Asn68Lys AACg-AAG	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2796
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT CGTCCACGTGCGCCACGGCGTTGGTCAGCGCGTCGGCCACC TTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2797
	CTGACCAACGCCGTGGC	2798
	GCCACGGCGTTGGTCAG	2799
Haemoglobin variant Asp74Gly GAC-GGC	AGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGACC AACGCCGTGGCGCACGTGGACGACATGCCCAACGCGCTGTC CGCCCTGAGCGACCTGCACGCGCACAAGCTTCGGGTGGA	2800
	TCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGA CAGCGCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGG TCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCT	2801
	GCACGTGGACGACATGC	2802
	GCATGTCGTCCACGTGC	2803
Haemoglobin variant Asp74His gGAC-CAC	CAGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGAC CAACGCCGTGGCGCACGTGGACGACATGCCCAACGCGCTGT CCGCCCTGAGCGACCTGCACGCGCACAAGCTTCGGGTGG	2804
	CCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGAC AGCGCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGGT CAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTG	2805
	CGCACGTGGACGACATG	2806
	CATGTCGTCCACGTGCG	2807
Haemoglobin variant Asn78His cAAC-CAC	CACGGCAAGAAGGTGGCCGACGCGCTGACCAACGCCGTGG CGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGAGC GACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACT	2808

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCG CTCAGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGC CACGGCGTTGGTCAGCGCGTCGGCCACCTTCTTGCCGTG	2809
	ACATGCCCAACGCGCTG	2810
	CAGCGCGTTGGGCATGT	2811
Haemoglobin variant His87Tyr gCAC-TAC	ACCAACGCCGTGGCGCACGTGGACGACATGCCCAACGCGCT GTCCGCCCTGAGCGACCTGCACGCGCACAAGCTTCGGGTGG ACCCGGTCAACTTCAAGGTGAGCGGCGGGCCGGGAGCGA	2812
	TCGCTCCCGGCCCGCCGCTCACCTTGAAGTTGACCGGGTCC ACCCGAAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGACAG CGCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGGT	2813
	GCGACCTGCACGCGCAC	2814
	GTGCGCGTGCAGGTCGC	2815
Haemoglobin variant Lys90Asn AAGc-AAC	GGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGA GCGACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAAC TTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAG	2816
	CTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGT TGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTC AGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCC	2817
	GCGCACAAGCTTCGGGT	2818
	ACCCGAAGCTTGTGCGC	2819
Haemoglobin variant Lys90Thr AAG-ACG	TGGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTG AGCGACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAA CTTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGA	2820
	TCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGTT GACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTCA GGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCCA	2821
	CGCGCACAAGCTTCGGG	2822
	CCCGAAGCTTGTGCGCG	2823
Haemoglobin variant Arg92Gln CGG-CAG	ACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGAGCGAC CTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACTTCAA GGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCG	2824
	CGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTT GAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGT CGCTCAGGGCGGACAGCGCGTTGGGCATGTCGTCCACGT	2825
	CAAGCTTCGGGTGGACC	2826
	GGTCCACCCGAAGCTTG	2827
Haemoglobin variant Asp94Gly GAC-GGC	ACGACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCAC GCGCACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAG CGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGG	2828
	CCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCT CACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGT GCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGTCGT	2829

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCGGGTGGACCCGGTCA	2830
	TGACCGGGTCCACCCGA	2831
Haemoglobin variant Pro95Arg CCG-CGG	ACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG CACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAGCGG CGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGGCGC	2832
	GCGCCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCC GCTCACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCG CGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT	2833
	GGTGGACCCGGTCAACT	2834
	AGTTGACCGGGTCCACC	2835
Haemoglobin variant Ser102Arg AGCc-AGA	CGGCGGCTGCGGGCCTGGGCCCTCGGCCCCACTGACCCTC TTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTG GCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCAC	2836
	GTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCC AGGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAA GAGGGTCAGTGGGGCCGAGGGCCCAGGCCCGCAGCCGCCG	2837
	CTCCTAAGCCACTGCCT	2838
	AGGCAGTGGCTTAGGAG	2839
Haemoglobin variant Glu116Lys cGAG-AAG	TTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTG GCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGC CTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGC	2840
	GCACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCG TGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCA GGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAA	2841
	TCCCCGCCGAGTTCACC	2842
	GGTGAACTCGGCGGGGA	2843
Haemoglobin variant Ala120Glu GCG-GAG	TCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGCCCACCTC CCCGCCGAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAA GTTCCTGGCTTCTGTGAGCACCGTGCTGACCTCCAAATA	2844
	TATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAGGAACTTG TCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGA GGTGGGCGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGA	2845
	CACCCCTGCGGTGCACG	2846
	CGTGCACCGCAGGGGTG	2847
Thalassaemia alpha Leu129Pro CTG-CCG	TGGCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCAC GCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCTG ACCTCCAAATACCGTTAAGCTGGAGCCTCGGTGGCCAT	2848
	ATGGCCACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGC ACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG CACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCA	2849
	CAAGTTCCTGGCTTCTG	2850
	CAGAAGCCAGGAACTTG	2851

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemoglobin variant Arg141Leu CGT-CTT	TGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCG TGCTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTGGCCA TGCTTCTTGCCCCTTGGGCCTCCCCCCAGCCCCTCCT	2852
	AGGAGGGGCTGGGGGGAGGCCCAAGGGGCAAGAAGCATGG CCACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCACG GTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTGCA	2853
	CAAATACCGTTAAGCTG	2854
	CAGCTTAACGGTATTTG	2855

EXAMPLE 16

Alpha-thalassemia - Hemoglobin alpha locus 2

The attached table discloses the correcting oligonucleotide base sequences for the hemoglobin alpha locus 2 oligonucleotides of the invention.

Table 23

HBA2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Met(-1)Thr ATG-ACG	CCTGGCGCGCTCGCGGGCCGGCACTCTTCTGGTCCCCACAG ACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAG ACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCA	2856
	TGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTT GTCGGCAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCT GTGGGGACCAGAAGAGTGCCGGCCCGCGAGCGCGCCAGG	2857
	ACCCACCATGGTGCTGT	2858
	ACAGCACCATGGTGGGT	2859
Haemoglobin variant Ala12Asp GCC-GAC	CACAGACTCAGAGAGAACCCACCATGGTGCTGTCTCCTGCCG ACAAGACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCG CACGCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTG	2860
	CACCTCTCCAGGGCCTCCGCACCATACTCGCCAGCGTGCGC GCCGACCTTACCCCAGGCGGCCTTGACGTTGGTCTTGTCGG CAGGAGACAGCACCATGGTGGGTTCTCTCTGAGTCTGTG	2861
	CGTCAAGGCCGCCTGGG	2862
	CCCAGGCGGCCTTGACG	2863

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemoglobin variant Lys16Glu tAAG-GAG	AGAGAACCCACCATGGTGCTGTCTCCTGCCGACAAGACCAAC GTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGGCG AGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCTCC	2864
	GGAGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCG CCAGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTT GGTCTTGTCGGCAGGAGACAGCACCATGGTGGGTTCTCT	2865
	CCTGGGGTAAGGTCGGC	2866
	GCCGACCTTACCCCAGG	2867
Haemoglobin variant His20Gln CACg-CAA	GGTGCTGTCTCCTGCCGACAAGACCAACGTCAAGGCCGCCT GGGGTAAGGTCGGCGCGCACGCTGGCGAGTATGGTGCGGA GGCCCTGGAGAGGTGAGGCTCCCTCCCCTGCTCCGACCCG	2868
	CGGGTCGGAGCAGGGGAGGGAGCCTCACCTCTCCAGGGCC TCCGCACCATACTCGCCAGCGTGCGCGCCGACCTTACCCCA GGCGGCCTTGACGTTGGTCTTGTCGGCAGGAGACAGCACC	2869
	GGCGCGCACGCTGGCGA	2870
	TCGCCAGCGTGCGCGCC	2871
Haemoglobin variant Glu27Asp GAGg-GAC	GACCAACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCAC GCTGGCGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCT CCCTCCCCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGACC	2872
	GGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGGGGAG GGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCGCCAG CGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGTTGGTC	2873
	GGTGCGGAGGCCCTGGA	2874
	TCCAGGGCCTCCGCACC	2875
Thalassaemia alpha Leu29Pro CTG-CCG	ACGTCAAGGCCGCCTGGGGTAAGGTCGGCGCGCACGCTGG CGAGTATGGTGCGGAGGCCCTGGAGAGGTGAGGCTCCCTCC CCTGCTCCGACCCGGGCTCCTCGCCCGCCCGGACCCACAG	2876
	CTGTGGGTCCGGGCGGGCGAGGAGCCCGGGTCGGAGCAGG GGAGGGAGCCTCACCTCTCCAGGGCCTCCGCACCATACTCG CCAGCGTGCGCGCCGACCTTACCCCAGGCGGCCTTGACGT	2877
	GGAGGCCCTGGAGAGGT	2878
	ACCTCTCCAGGGCCTCC	2879
Haemoglobin variant Asp47His cGAC-CAC	GCTTCTCCCCGCAGGATGTTCCTGTCCTTCCCCACCACCAAG ACCTACTTCCCGCACTTCGACCTGAGCCACGGCTCTGCCCAG GTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCTGA	2880
	TCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTGG GCAGAGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAGGTCTT GGTGGTGGGGAAGGACAGGAACATCCTGCGGGGAGAAGC	2881
	CGCACTTCGACCTGAGC	2882
	GCTCAGGTCGAAGTGCG	2883

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Haemoglobin variant Leu48Arg CTG-CGG	CTCCCCGCAGGATGTTCCTGTCCTTCCCCACCACCAAGACCT ACTTCCCGCACTTCGACCTGAGCCACGGCTCTGCCCAGGTTA AGGGCCACGGCAAGAAGGTGGCCGACGCGCTGACCAA	2884
	TTGGTCAGCGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACC TGGGCAGAGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAGGT CTTGGTGGTGGGGAAGGACAGGAACATCCTGCGGGGAG	2885
	CTTCGACCTGAGCCACG	2886
	CGTGGCTCAGGTCGAAG	2887
Haemoglobin variant Gln54Glu cCAG-GAG	CTGTCCTTCCCCACCACCAAGACCTACTTCCCGCACTTCGAC CTGAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAA GGTGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGG	2888
	CCACGTGCGCCACGGCGTTGGTCAGCGCGTCGGCCACCTTC TTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTCAGGTC GAAGTGCGGGAAGTAGGTCTTGGTGGTGGGGAAGGACAG	2889
	GCTCTGCCCAGGTTAAG	2890
	CTTAACCTGGGCAGAGC	2891
Haemoglobin variant Gly59Asp GGC-GAC	CCAAGACCTACTTCCCGCACTTCGACCTGAGCCACGGCTCTG CCCAGGTTAAGGGCCACGGCAAGAAGGTGGCCGACGCGCT GACCAACGCCGTGGCGCACGTGGACGACATGCCCAACGC	2892
	GCGTTGGGCATGTCGTCCACGTGCGCCACGGCGTTGGTCAG CGCGTCGGCCACCTTCTTGCCGTGGCCCTTAACCTGGGCAG AGCCGTGGCTCAGGTCGAAGTGCGGGAAGTAGGTCTTGG	2893
	GGGCCACGGCAAGAAGG	2894
	CCTTCTTGCCGTGGCCC	2895
Haemoglobin variant Asn68Lys AACg-AAG	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2896
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT CGTCCACGTGCGCCACGGCGTTGGTCAGCGCGTCGGCCACC TTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2897
	CTGACCAACGCCGTGGC	2898
	GCCACGGCGTTGGTCAG	2899
Haemoglobin variant Asn68Lys AACg-AAA	GAGCCACGGCTCTGCCCAGGTTAAGGGCCACGGCAAGAAGG TGGCCGACGCGCTGACCAACGCCGTGGCGCACGTGGACGA CATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG	2900
	CGCGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT CGTCCACGTGCGCCACGGCGTTGGTCAGCGCGTCGGCCACC TTCTTGCCGTGGCCCTTAACCTGGGCAGAGCCGTGGCTC	2901
	CTGACCAACGCCGTGGC	2902
	GCCACGGCGTTGGTCAG	2903
Haemoglobin variant Asn78Lys AACg-AAA	CGGCAAGAAGGTGGCCGACGCGCTGACCAACGCCGTGGCG CACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGAGCGA CCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAACTTC	2904

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGT CGCTCAGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGC GCCACGGCGTTGGTCAGCGCGTCGGCCACCTTCTTGCCG	2905
	ATGCCCAACGCGCTGTC	2906
	GACAGCGCGTTGGGCAT	2907
Haemoglobin variant Asp85Val GAC-GTC	CGCTGACCAACGCCGTGGCGCACGTGGACGACATGCCCAAC GCGCTGTCCGCCCTGAGCGACCTGCACGCGCACAAGCTTCG GGTGGACCCGGTCAACTTCAAGGTGAGCGGCGGGCCGGG	2908
	CCCGGCCCGCCGCTCACCTTGAAGTTGACCGGGTCCACCCG AAGCTTGTGCGCGTGCAGGTCGCTCAGGGCGGACAGCGCGT TGGGCATGTCGTCCACGTGCGCCACGGCGTTGGTCAGCG	2909
	CCTGAGCGACCTGCACG	2910
	CGTGCAGGTCGCTCAGG	2911
Haemoglobin variant Lys90Asn AAGc-AAT	GGCGCACGTGGACGACATGCCCAACGCGCTGTCCGCCCTGA GCGACCTGCACGCGCACAAGCTTCGGGTGGACCCGGTCAAC TTCAAGGTGAGCGGCGGGCCGGGAGCGATCTGGGTCGAG	2912
	CTCGACCCAGATCGCTCCCGGCCCGCCGCTCACCTTGAAGT TGACCGGGTCCACCCGAAGCTTGTGCGCGTGCAGGTCGCTC AGGGCGGACAGCGCGTTGGGCATGTCGTCCACGTGCGCC	2913
	GCGCACAAGCTTCGGGT	2914
	ACCCGAAGCTTGTGCGC	2915
Haemoglobin variant Asp94His gGAC-CAC	GACGACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCA CGCGCACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGA GCGGCGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATG	2916
	CATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCCGCTC ACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCGCGTG CAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGTCGTC	2917
	TTCGGGTGGACCCGGTC	2918
	GACCGGGTCCACCCGAA	2919
Haemoglobin variant Pro95Leu CCG-CTG	ACATGCCCAACGCGCTGTCCGCCCTGAGCGACCTGCACGCG CACAAGCTTCGGGTGGACCCGGTCAACTTCAAGGTGAGCGG CGGGCCGGGAGCGATCTGGGTCGAGGGGCGAGATGGCGC	2920
	GCGCCATCTCGCCCCTCGACCCAGATCGCTCCCGGCCCGCC GCTCACCTTGAAGTTGACCGGGTCCACCCGAAGCTTGTGCG CGTGCAGGTCGCTCAGGGCGGACAGCGCGTTGGGCATGT	2921
	GGTGGACCCGGTCAACT	2922
	AGTTGACCGGGTCCACC	2923
Haemoglobin variant Ser102Arg aAGC-CGC	TAGCGCAGGCGGCGGCTGCGGGCCTGGGCCGCACTGACCC TCTTCTCTGCACAGCTCCTAAGCCACTGCCTGCTGGTGACCC TGGCCGCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGC	2924
	GCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGCGGCCAG GGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCAGAGAAGA GGGTCAGTGCGGCCCAGGCCCGCAGCCGCCGCCTGCGCTA	2925

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGCTCCTAAGCCACTGC	2926
	GCAGTGGCTTAGGAGCT	2927
Haemoglobin H disease Cys104Tyr TGC-TAC	GGCGGCGGCTGCGGGCCTGGGCCGCACTGACCCTCTTCTCT GCACAGCTCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGC CCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGCCTC	2928
	GAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGG CGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGAGCTGTGCA GAGAAGAGGGTCAGTGCGGCCCAGGCCCGCAGCCGCCGCC	2929
	AAGCCACTGCCTGCTGG	2930
	CCAGCAGGCAGTGGCTT	2931
Haemoglobin variant Ala111Val GCC-GTC	CCGCACTGACCCTCTTCTCTGCACAGCTCCTAAGCCACTGCC TGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGAGTTCACC CCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGGCTTC	2932
	GAAGCCAGGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGT GAACTCGGCGGGGAGGTGGGCGGCCAGGGTCACCAGCAGG CAGTGGCTTAGGAGCTGTGCAGAGAAGAGGGTCAGTGCGG	2933
	CCTGGCCGCCCACCTCC	2934
	GGAGGTGGGCGGCCAGG	2935
Haemoglobin variant Ala120Glu GCG-GAG	TCCTAAGCCACTGCCTGCTGGTGACCCTGGCCGCCCACCTC CCCGCCGAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAA GTTCCTGGCTTCTGTGAGCACCGTGCTGACCTCCAAATA	2936
	TATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAGGAACTTG TCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGGCGGGGA GGTGGGCGGCCAGGGTCACCAGCAGGCAGTGGCTTAGGA	2937
	CACCCCTGCGGTGCACG	2938
	CGTGCACCGCAGGGGTG	2939
Haemoglobin variant His122Gln CACg-CAG	CCACTGCCTGCTGGTGACCCTGGCCGCCCACCTCCCCGCCG AGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTG GCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAA	2940
	TTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAG GAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTCGG CGGGGAGGTGGGCGGCCAGGGTCACCAGCAGGCAGTGG	2941
	GCGGTGCACGCCTCCCT	2942
	AGGGAGGCGTGCACCGC	2943
Haemoglobin variant Ala123Ser cGCC-TCC	CACTGCCTGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGA GTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGG CTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAG	2944
	CTTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCA GGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTCG GCGGGGAGGTGGGCGGCCAGGGTCACCAGCAGGCAGTG	2945
	CGGTGCACGCCTCCCTG	2946
	CAGGGAGGCGTGCACCG	2947

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Thalassaemia alpha Leu125Pro CTG-CCG	TGCTGGTGACCCTGGCCGCCCACCTCCCCGCCGAGTTCACC CCTGCGGTGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGTG AGCACCGTGCTGACCTCCAAATACCGTTAAGCTGGAGC	2948
	GCTCCAGCTTAACGGTATTTGGAGGTCAGCACGGTGCTCACA GAAGCCAGGAACTTGTCCAGGGAGGCGTGCACCGCAGGGG TGAACTCGGCGGGGAGGTGGGCGGCCAGGGTCACCAGCA	2949
	CGCCTCCCTGGACAAGT	2950
	ACTTGTCCAGGGAGGCG	2951
Haemoglobin variant Ser131Pro tTCT-CCT	GCCCACCTCCCCGCCGAGTTCACCCCTGCGGTGCACGCCTC CCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCTGACCTC CAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTTCCTC	2952
	GAGGAACGGCTACCGAGGCTCCAGCTTAACGGTATTTGGAG GTCAGCACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGA GGCGTGCACCGCAGGGGTGAACTCGGCGGGGAGGTGGGC	2953
	TCCTGGCTTCTGTGAGC	2954
	GCTCACAGAAGCCAGGA	2955
Haemoglobin variant Leu136Met gCTG-ATG	GAGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCT GGCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAGC TGGAGCCTCGGTAGCCGTTCCTCCTGCCCGCTGGGCCT	2956
	AGGCCCAGCGGGCAGGAGGAACGGCTACCGAGGCTCCAGC TTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCAG GAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACTC	2957
	GCACCGTGCTGACCTCC	2958
	GGAGGTCAGCACGGTGC	2959
Haemoglobin variant Leu136Pro CTG-CCG	AGTTCACCCCTGCGGTGCACGCCTCCCTGGACAAGTTCCTG GCTTCTGTGAGCACCGTGCTGACCTCCAAATACCGTTAAGCT GGAGCCTCGGTAGCCGTTCCTCCTGCCCGCTGGGCCTC	2960
	GAGGCCCAGCGGGCAGGAGGAACGGCTACCGAGGCTCCAG CTTAACGGTATTTGGAGGTCAGCACGGTGCTCACAGAAGCCA GGAACTTGTCCAGGGAGGCGTGCACCGCAGGGGTGAACT	2961
	CACCGTGCTGACCTCCA	2962
	TGGAGGTCAGCACGGTG	2963
Haemoglobin variant Arg141Cys cCGT-TGT	GTGCACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACC GTGCTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCC GTTCCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCC	2964
	GGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAACGGC TACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCACGGT GCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTGCAC	2965
	CCAAATACCGTTAAGCT	2966
	AGCTTAACGGTATTTGG	2967
Haemoglobin variant Term142Gln tTAA-CAA	CACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTG CTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTT CCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCC	2968

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAAC GGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCA CGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG	2969
	AATACCGTTAAGCTGGA	2970
	TCCAGCTTAACGGTATT	2971
Haemoglobin variant Term142Lys tTAA-AAA	CACGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTG CTGACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTT CCTCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCC	2972
	GGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGGAAC GGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAGCA CGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCGTG	2973
	AATACCGTTAAGCTGGA	2974
	TCCAGCTTAACGGTATT	2975
Haemoglobin variant Term142Tyr TAAg-TAT	CGCCTCCCTGGACAAGTTCCTGGCTTCTGTGAGCACCGTGCT GACCTCCAAATACCGTTAAGCTGGAGCCTCGGTAGCCGTTCC TCCTGCCCGCTGGGCCTCCCAACGGGCCCTCCTCCCC	2976
	GGGGAGGAGGGCCCGTTGGGAGGCCCAGCGGGCAGGAGG AACGGCTACCGAGGCTCCAGCTTAACGGTATTTGGAGGTCAG CACGGTGCTCACAGAAGCCAGGAACTTGTCCAGGGAGGCG	2977
	TACCGTTAAGCTGGAGC	2978
	GCTCCAGCTTAACGGTA	2979

EXAMPLE 17

Human mismatch repair - MLH1

The human MLH1 gene is homologous to the bacterial *mutL* gene, which is involved in mismatch repair. Mutations in the MLH1 gene have been identified in many individuals with hereditary nonpolyposis colorectal cancer (HNPCC). Mutations in the MLH1 gene are also implicated in predisposition to a variety of cancers associated with, for example, Muir-Torre syndrome and Turcot syndrome. The attached table discloses the correcting oligonucleotide base sequences for the MLH1 oligonucleotides of the invention.

Table 24

MLH1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Met1Arg ATG-AGG	TTGGCTGAAGGCACTTCCGTTGAGCATCTAGACGTTTCCTTG GCTCTTCTGGCGCCAAAATGTCGTTCGTGGCAGGGGTTATTC GGCGGCTGGACGAGACAGTGGTGAACCGCATCGCGGC	2980
	GCCGCGATGCGGTTCACCACTGTCTCGTCCAGCCGCCGAAT AACCCCTGCCACGAACGACATTTTGGCGCCAGAAGAGCCAA GGAAACGTCTAGATGCTCAACGGAAGTGCCTTCAGCCAA	2981
	CGCCAAAATGTCGTTCG	2982
	CGAACGACATTTTGGCG	2983
Non-polyposis colorectal cancer Met1Lys ATG-AAG	TTGGCTGAAGGCACTTCCGTTGAGCATCTAGACGTTTCCTTG GCTCTTCTGGCGCCAAAATGTCGTTCGTGGCAGGGGTTATTC GGCGGCTGGACGAGACAGTGGTGAACCGCATCGCGGC	2984
	GCCGCGATGCGGTTCACCACTGTCTCGTCCAGCCGCCGAAT AACCCCTGCCACGAACGACATTTTGGCGCCAGAAGAGCCAA GGAAACGTCTAGATGCTCAACGGAAGTGCCTTCAGCCAA	2985
	CGCCAAAATGTCGTTCG	2986
	CGAACGACATTTTGGCG	2987
Non-polyposis colorectal cancer Met35Arg ATG-AGG	TGGTGAACCGCATCGCGGCGGGGGAAGTTATCCAGCGGCCA GCTAATGCTATCAAAGAGATGATTGAGAACTGGTACGGAGGG AGTCGAGCCGGGCTCACTTAAGGGCTACGACTTAACGG	2988
	CCGTTAAGTCGTAGCCCTTAAGTGAGCCCGGCTCGACTCCCT CCGTACCAGTTCTCAATCATCTCTTTGATAGCATTAGCTGGCC GCTGGATAACTTCCCCCGCCGCGATGCGGTTCACCA	2989
	CAAAGAGATGATTGAGA	2990
	TCTCAATCATCTCTTTG	2991
Non-polyposis colorectal cancer Ser44Phe TCC-TTC	TAGAGTAGTTGCAGACTGATAAATTATTTTCTGTTTGATTTGCC AGTTTAGATGCAAAATCCACAAGTATTCAAGTGATTGTTAAAG AGGGAGGCCTGAAGTTGATTCAGATCCAAGACAA	2992
	TTGTCTTGGATCTGAATCAACTTCAGGCCTCCCTCTTTAACAA TCACTTGAATACTTGTGGATTTTGCATCTAAACTGGCAAATCA AACAGAAAATAATTTATCAGTCTGCAACTACTCTA	2993
	TGCAAAATCCACAAGTA	2994
	TACTTGTGGATTTTGCA	2995
Non-polyposis colorectal cancer Gln62Lys CAA-AAA	GCAAAATCCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGC CTGAAGTTGATTCAGATCCAAGACAATGGCACCGGGATCAGG GTAAGTAAAACCTCAAAGTAGCAGGATGTTTGTGCGC	2996
	GCGCACAAACATCCTGCTACTTTGAGGTTTTACTTACCCTGAT CCCGGTGCCATTGTCTTGGATCTGAATCAACTTCAGGCCTCC CTCTTTAACAATCACTTGAATACTTGTGGATTTTGC	2997
	TTCAGATCCAAGACAAT	2998

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATTGTCTTGGATCTGAA	2999
Non-polyposis colorectal cancer Gln62Term CAA-TAA	GCAAAATCCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGC CTGAAGTTGATTCAGATCCAAGACAATGGCACCGGGATCAGG GTAAGTAAAACCTCAAAGTAGCAGGATGTTTGTGCGC	3000
	GCGCACAAACATCCTGCTACTTTGAGGTTTTACTTACCCTGAT CCCGGTGCCATTGTCTTGGATCTGAATCAACTTCAGGCCTCC CTCTTTAACAATCACTTGAATACTTGTGGATTTTGC	3001
	TTCAGATCCAAGACAAT	3002
	ATTGTCTTGGATCTGAA	3003
Non-polyposis colorectal cancer Asn64Ser AAT-AGT	CCACAAGTATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGT TGATTCAGATCCAAGACAATGGCACCGGGATCAGGGTAAGTA AAACCTCAAAGTAGCAGGATGTTTGTGCGCTTCATGG	3004
	CCATGAAGCGCACAAACATCCTGCTACTTTGAGGTTTTACTTA CCCTGATCCCGGTGCCATTGTCTTGGATCTGAATCAACTTCA GGCCTCCCTCTTTAACAATCACTTGAATACTTGTGG	3005
	CCAAGACAATGGCACCG	3006
	CGGTGCCATTGTCTTGG	3007
Non-polyposis colorectal cancer Gly67Arg GGG-AGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAG ATCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCA AAGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3008
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3009
	ATGGCACCGGGATCAGG	3010
	CCTGATCCCGGTGCCAT	3011
Non-polyposis colorectal cancer Gly67Arg GGG-CGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAG ATCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCA AAGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3012
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3013
	ATGGCACCGGGATCAGG	3014
	CCTGATCCCGGTGCCAT	3015
Non-polyposis colorectal cancer Gly67Trp GGG-TGG	ATTCAAGTGATTGTTAAAGAGGGAGGCCTGAAGTTGATTCAG ATCCAAGACAATGGCACCGGGATCAGGGTAAGTAAAACCTCA AAGTAGCAGGATGTTTGTGCGCTTCATGGAAGAGTCA	3016
	TGACTCTTCCATGAAGCGCACAAACATCCTGCTACTTTGAGGT TTTACTTACCCTGATCCCGGTGCCATTGTCTTGGATCTGAATC AACTTCAGGCCTCCCTCTTTAACAATCACTTGAAT	3017
	ATGGCACCGGGATCAGG	3018
	CCTGATCCCGGTGCCAT	3019

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Cys77Arg TGT-CGT	GTAACATGATTATTTACTCATCTTTTTGGTATCTAACAGAAAGA AGATCTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTG CAGTCCTTTGAGGATTTAGCCAGTATTTCTACCT	3020
	AGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGTTTACT AGTAGTGAACCTTTCACATACAATATCCAGATCTTCTTTCTGTT AGATACCAAAAAGATGAGTAAATAATCATGTTAC	3021
	ATATTGTATGTGAAAGG	3022
	CCTTTCACATACAATAT	3023
Non-polyposis colorectal cancer Cys77Tyr TGT-TAT	TAACATGATTATTTACTCATCTTTTTGGTATCTAACAGAAAGAA GATCTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTGC AGTCCTTTGAGGATTTAGCCAGTATTTCTACCTA	3024
	TAGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGTTTAC TAGTAGTGAACCTTTCACATACAATATCCAGATCTTCTTTCTGT TAGATACCAAAAAGATGAGTAAATAATCATGTTA	3025
	TATTGTATGTGAAAGGT	3026
	ACCTTTCACATACAATA	3027
Non-polyposis colorectal cancer Ser93Gly AGT-GGT	CTGGATATTGTATGTGAAAGGTTCACTACTAGTAAACTGCAGT CCTTTGAGGATTTAGCCAGTATTTCTACCTATGGCTTTCGAGG TGAGGTAAGCTAAAGATTCAAGAAATGTGTAAAAT	3028
	ATTTTACACATTTCTTGAATCTTTAGCTTACCTCACCTCGAAAG CCATAGGTAGAAATACTGGCTAAATCCTCAAAGGACTGCAGT TTACTAGTAGTGAACCTTTCACATACAATATCCAG	3029
	ATTTAGCCAGTATTTCT	3030
	AGAAATACTGGCTAAAT	3031
Non-polyposis colorectal cancer Arg100Term CGA-TGA	TTCACTACTAGTAAACTGCAGTCCTTTGAGGATTTAGCCAGTA TTTCTACCTATGGCTTTCGAGGTGAGGTAAGCTAAAGATTCAA GAAATGTGTAAAATATCCTCCTGTGATGACATTGT	3032
	ACAATGTCATCACAGGAGGATATTTTACACATTTCTTGAATCTT TAGCTTACCTCACCTCGAAAGCCATAGGTAGAAATACTGGCT AAATCCTCAAAGGACTGCAGTTTACTAGTAGTGAA	3033
	ATGGCTTTCGAGGTGAG	3034
	CTCACCTCGAAAGCCAT	3035
Non-polyposis colorectal cancer Ile107Arg ATA-AGA	ACCCAGCAGTGAGTTTTTCTTTCAGTCTATTTTCTTTTCTTCCT TAGGCTTTGGCCAGCATAAGCCATGTGGCTCATGTTACTATTA CAACGAAAACAGCTGATGGAAAGTGTGCATACAG	3036
	CTGTATGCACACTTTCCATCAGCTGTTTTCGTTGTAATAGTAA CATGAGCCACATGGCTTATGCTGGCCAAAGCCTAAGGAAGAA AAGAAAATAGACTGAAAGAAAAACTCACTGCTGGGT	3037
	GGCCAGCATAAGCCATG	3038
	CATGGCTTATGCTGGCC	3039
Non-polyposis colorectal cancer Thr117Arg ACG-AGG	TTTCTTTTCTTCCTTAGGCTTTGGCCAGCATAAGCCATGTGGC TCATGTTACTATTACAACGAAAACAGCTGATGGAAAGTGTGCA TACAGGTATAGTGCTGACTTCTTTTACTCATATAT	3040

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATATATGAGTAAAAGAAGTCAGCACTATACCTGTATGCACACT TTCCATCAGCTGTTTTCGTTGTAATAGTAACATGAGCCACATG GCTTATGCTGGCCAAAGCCTAAGGAAGAAAAGAAA	3041
	TATTACAACGAAAACAG	3042
	CTGTTTTCGTTGTAATA	3043
Non-polyposis colorectal cancer Thr117Met ACG-ATG	TTTCTTTTCTTCCTTAGGCTTTGGCCAGCATAAGCCATGTGGC TCATGTTACTATTACAACGAAAACAGCTGATGGAAAGTGTGCA TACAGGTATAGTGCTGACTTCTTTTACTCATATAT	3044
	ATATATGAGTAAAAGAAGTCAGCACTATACCTGTATGCACACT TTCCATCAGCTGTTTTCGTTGTAATAGTAACATGAGCCACATG GCTTATGCTGGCCAAAGCCTAAGGAAGAAAAGAAA	3045
	TATTACAACGAAAACAG	3046
	CTGTTTTCGTTGTAATA	3047
Non-polyposis colorectal cancer Gly133Term GGA-TGA	TCTATCTCTCTACTGGATATTAATTTGTTATATTTTCTCATTAGA GCAAGTTACTCAGATGGAAAACTGAAAGCCCCTCCTAAACCA TGTGCTGGCAATCAAGGGACCCAGATCACGGTAA	3048
	TTACCGTGATCTGGGTCCCTTGATTGCCAGCACATGGTTTAG GAGGGGCTTTCAGTTTTCCATCTGAGTAACTTGCTCTAATGAG AAAATATAACAAATTAATATCCAGTAGAGAGATAGA	3049
	ACTCAGATGGAAAACTG	3050
	CAGTTTTCCATCTGAGT	3051
Non-polyposis colorectal cancer Val185Gly GTA-GGA	TAGTGTGTGTTTTTGGCAACTCTTTTCTTACTCTTTTGTTTTTC TTTTCCAGGTATTCAGTACACAATGCAGGCATTAGTTTCTCAG TTAAAAAAGTAAGTTCTTGGTTTATGGGGGATGG	3052
	CCATCCCCCATAAACCAAGAACTTACTTTTTTAACTGAGAAAC TAATGCCTGCATTGTGTACTGAATACCTGGAAAAGAAAAACAA AAGAGTAAGAAAAGAGTTGCCAAAAACACACACTA	3053
	GTATTCAGTACACAATG	3054
	CATTGTGTACTGAATAC	3055
Non-polyposis colorectal cancer Ser193Pro TCA-CCA	TTTCTTACTCTTTTGTTTTTCTTTTCCAGGTATTCAGTACACAA TGCAGGCATTAGTTTCTCAGTTAAAAAAGTAAGTTCTTGGTTT ATGGGGGATGGTTTTGTTTTATGAAAAGAAAAAA	3056
	TTTTTTCTTTTCATAAAACAAAACCATCCCCCATAAACCAAGAA CTTACTTTTTTAACTGAGAAACTAATGCCTGCATTGTGTACTG AATACCTGGAAAAGAAAAACAAAAGAGTAAGAAA	3057
	TTAGTTTCTCAGTTAAA	3058
	TTTAACTGAGAAACTAA	3059
Non-polyposis colorectal cancer Val213Met GTG-ATG	TTTGTTTATCAGCAAGGAGAGACAGTAGCTGATGTTAGGACA CTACCCAATGCCTCAACCGTGGACAATATTCGCTCCATCTTTG GAAATGCTGTTAGTCGGTATGTCGATAACCTATATA	3060
	TATATAGGTTATCGACATACCGACTAACAGCATTTCCAAAGAT GGAGCGAATATTGTCCACGGTTGAGGCATTGGGTAGTGTCCT AACATCAGCTACTGTCTCTCCTTGCTGATAAACAAA	3061
	CCTCAACCGTGGACAAT	3062

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATTGTCCACGGTTGAGG	3063
Non-polyposis colorectal cancer Arg217Cys CGC-TGC	CAAGGAGAGACAGTAGCTGATGTTAGGACACTACCCAATGCC TCAACCGTGGACAATATTCGCTCCATCTTTGGAAATGCTGTTA GTCGGTATGTCGATAACCTATATAAAAAAATCTTTT	3064
	AAAAGATTTTTTTATATAGGTTATCGACATACCGACTAACAGC ATTTCCAAAGATGGAGCGAATATTGTCCACGGTTGAGGCATT GGGTAGTGTCCTAACATCAGCTACTGTCTCTCCTTG	3065
	ACAATATTCGCTCCATC	3066
	GATGGAGCGAATATTGT	3067
Non-polyposis colorectal cancer Ile219Val ATC-GTC	GAGACAGTAGCTGATGTTAGGACACTACCCAATGCCTCAACC GTGGACAATATTCGCTCCATCTTTGGAAATGCTGTTAGTCGGT ATGTCGATAACCTATATAAAAAAATCTTTTACATTT	3068
	AAATGTAAAAGATTTTTTTATATAGGTTATCGACATACCGACTA ACAGCATTTCCAAAGATGGAGCGAATATTGTCCACGGTTGAG GCATTGGGTAGTGTCCTAACATCAGCTACTGTCTC	3069
	TTCGCTCCATCTTTGGA	3070
	TCCAAAGATGGAGCGAA	3071
Non-polyposis colorectal cancer Gly244Asp GGT-GAT	CTAATAGAGAACTGATAGAAATTGGATGTGAGGATAAAACCCT AGCCTTCAAAATGAATGGTTACATATCCAATGCAAACTACTCA GTGAAGAAGTGCATCTTCTTACTCTTCATCAACCG	3072
	CGGTTGATGAAGAGTAAGAAGATGCACTTCTTCACTGAGTAG TTTGCATTGGATATGTAACCATTCATTTTGAAGGCTAGGGTTT TATCCTCACATCCAATTTCTATCAGTTCTCTATTAG	3073
	AATGAATGGTTACATAT	3074
	ATATGTAACCATTCATT	3075
Non-polyposis colorectal cancer Ser252Term TCA-TAA	GATGTGAGGATAAAACCCTAGCCTTCAAAATGAATGGTTACAT ATCCAATGCAAACTACTCAGTGAAGAAGTGCATCTTCTTACTC TTCATCAACCGTAAGTTAAAAAGAACCACATGGGA	3076
	TCCCATGTGGTTCTTTTTAACTTACGGTTGATGAAGAGTAAGA AGATGCACTTCTTCACTGAGTAGTTTGCATTGGATATGTAACC ATTCATTTTGAAGGCTAGGGTTTTATCCTCACATC	3077
	AAACTACTCAGTGAAGA	3078
	TCTTCACTGAGTAGTTT	3079
Non-polyposis colorectal cancer Glu268Gly GAA-GGA	CACCCCTCAGGACAGTTTTGAACTGGTTGCTTTCTTTTTATTG TTTAGATCGTCTGGTAGAATCAACTTCCTTGAGAAAAGCCATA GAAACAGTGTATGCAGCCTATTTGCCCAAAAACAC	3080
	GTGTTTTTGGGCAAATAGGCTGCATACACTGTTTCTATGGCTT TTCTCAAGGAAGTTGATTCTACCAGACGATCTAAACAATAAAA AGAAAGCAACCAGTTCAAAACTGTCCTGAGGGGTG	3081
	TCTGGTAGAATCAACTT	3082
	AAGTTGATTCTACCAGA	3083

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Ser269Term TCA-TGA	CCCTCAGGACAGTTTTGAACTGGTTGCTTTCTTTTTATTGTTTA GATCGTCTGGTAGAATCAACTTCCTTGAGAAAAGCCATAGAA ACAGTGTATGCAGCCTATTTGCCCAAAAACACACA	3084
	TGTGTGTTTTTGGGCAAATAGGCTGCATACACTGTTTCTATGG CTTTTCTCAAGGAAGTTGATTCTACCAGACGATCTAAACAATA AAAAGAAAGCAACCAGTTCAAAACTGTCCTGAGGG	3085
	GGTAGAATCAACTTCCT	3086
	AGGAAGTTGATTCTACC	3087
Non-polyposis colorectal cancer Glu297Term GAA-TAA	CTTTTTCTCCCCCTCCCACTATCTAAGGTAATTGTTCTCTCTTA TTTTCCTGACAGTTTAGAAATCAGTCCCCAGAATGTGGATGTT ~~A~~ATGTGCACCCCACAAAGCATGAAGTTCACTTCC	3088
	GGAAGTGAACTTCATGCTTTGTGGGGTGCACATTAACATCCA CATTCTGGGGACTGATTTCTAAACTGTCAGGAAAATAAGAGA GAACAATTACCTTAGATAGTGGGAGGGGGAGAAAAAG	3089
	ACAGTTTAGAAATCAGT	3090
	ACTGATTTCTAAACTGT	3091
Non-polyposis colorectal cancer Gln301Term CAG-TAG	CTCCCACTATCTAAGGTAATTGTTCTCTCTTATTTTCCTGACAG TTTAGAAATCAGTCCCCAGAATGTGGATGTTAATGTGCACCCC ACAAAGCATGAAGTTCACTTCCTGCACGAGGAGA	3092
	TCTCCTCGTGCAGGAAGTGAACTTCATGCTTTGTGGGGTGCA CATTAACATCCACATTCTGGGGACTGATTTCTAAACTGTCAGG AAAATAAGAGAGAACAATTACCTTAGATAGTGGGAG	3093
	TCAGTCCCCAGAATGTG	3094
	CACATTCTGGGGACTGA	3095
Non-polyposis colorectal cancer Val326Ala GTG-GCG	ATGTGCACCCCACAAAGCATGAAGTTCACTTCCTGCACGAGG AGAGCATCCTGGAGCGGGTGCAGCAGCACATCGAGAGCAAG CTCCTGGGCTCCAATTCCTCCAGGATGTACTTCACCCA	3096
	TGGGTGAAGTACATCCTGGAGGAATTGGAGCCCAGGAGCTT GCTCTCGATGTGCTGCTGCACCCGCTCCAGGATGCTCTCCTC GTGCAGGAAGTGAACTTCATGCTTTGTGGGGTGCACAT	3097
	GGAGCGGGTGCAGCAGC	3098
	GCTGCTGCACCCGCTCC	3099
Non-polyposis colorectal cancer His329Pro CAC-CCC	CCACAAAGCATGAAGTTCACTTCCTGCACGAGGAGAGCATCC TGGAGCGGGTGCAGCAGCACATCGAGAGCAAGCTCCTGGGC TCCAATTCCTCCAGGATGTACTTCACCCAGGTCAGGGC	3100
	GCCCTGACCTGGGTGAAGTACATCCTGGAGGAATTGGAGCC CAGGAGCTTGCTCTCGATGTGCTGCTGCACCCGCTCCAGGAT GCTCTCCTCGTGCAGGAAGTGAACTTCATGCTTTGTGG	3101
	GCAGCAGCACATCGAGA	3102
	TCTCGATGTGCTGCTGC	3103
Non-polyposis colorectal cancer Val384Asp GTT-GAT	CAAGTCTGACCTCGTCTTCTACTTCTGGAAGTAGTGATAAGGT CTATGCCCACCAGATGGTTCGTACAGATTCCCGGGAACAGAA GCTTGATGCATTTCTGCAGCCTCTGAGCAAACCCCT	3104

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGGGTTTGCTCAGAGGCTGCAGAAATGCATCAAGCTTCTGT TCCCGGGAATCTGTACGAACCATCTGGTGGGCATAGACCTTA TCACTACTTCCAGAAGTAGAAGACGAGGTCAGACTTG	3105
	CCAGATGGTTCGTACAG	3106
	CTGTACGAACCATCTGG	3107
Non-polyposis colorectal cancer Ala441Thr GCT-ACT	AGTGGCAGGGCTAGGCAGCAAGATGAGGAGATGCTTGAACT CCCAGCCCCTGCTGAAGTGGGCTGCCAAAAATCAGAGCTTGGA GGGGGATACAACAAAGGGGACTTCAGAAATGTCAGAGA	3108
	TCTCTGACATTTCTGAAGTCCCCTTTGTTGTATCCCCCTCCAA GCTCTGATTTTTGGCAGCCACTTCAGCAGGGGCTGGGAGTTC AAGCATCTCCTCATCTTGCTGCCTAGCCCTGCCACT	3109
	CTGAAGTGGGCTGCCAAA	3110
	TTTGGCAGCCACTTCAG	3111
Non-polyposis colorectal cancer Arg487Term CGA-TGA	CTTCATTGCAGAAAGAGACATCGGGAAGATTCTGATGTGGAA ATGGTGGAAGATGATTCCCGAAAGGAAATGACTGCAGCTTGT ACCCCCCGGAGAAGGATCATTAACCTCACTAGTGTTT	3112
	AAACACTAGTGAGGTTAATGATCCTTCTCCGGGGGGTACAAG CTGCAGTCATTTCCTTTCGGGAATCATCTTCCACCATTTCCAC ATCAGAATCTTCCCGATGTCTCTTTCTGCAATGAAG	3113
	ATGATTCCCGAAAGGAA	3114
	TTCCTTTCGGGAATCAT	3115
Non-polyposis colorectal cancer Ala492Thr GCA-ACA	AGACATCGGGAAGATTCTGATGTGGAAATGGTGGAAGATGAT TCCCGAAAGGAAATGACTGCAGCTTGTACCCCCCGGAGAAG GATCATTAACCTCACTAGTGTTTTGAGTCTCCAGGAAG	3116
	CTTCCTGGAGACTCAAAACACTAGTGAGGTTAATGATCCTTCT CCGGGGGGTACAAGCTGCAGTCATTTCCTTTCGGGAATCATC TTCCACCATTTCCACATCAGAATCTTCCCGATGTCT	3117
	AAATGACTGCAGCTTGT	3118
	ACAAGCTGCAGTCATTT	3119
Non-polyposis colorectal cancer Val506Ala GTT-GCT	CCCGAAAGGAAATGACTGCAGCTTGTACCCCCCGGAGAAGG ATCATTAACCTCACTAGTGTTTTGAGTCTCCAGGAAGAAATTA ATGAGCAGGGACATGAGGGTACGTAAACGCTGTGGCC	3120
	GGCCACAGCGTTTACGTACCCTCATGTCCCTGCTCATTAATTT CTTCCTGGAGACTCAAAACACTAGTGAGGTTAATGATCCTTCT CCGGGGGGTACAAGCTGCAGTCATTTCCTTTCGGG	3121
	CACTAGTGTTTTGAGTC	3122
	GACTCAAAACACTAGTG	3123
Non-polyposis colorectal cancer Gln542Leu CAG-CTG	GGGAGATGTTGCATAACCACTCCTTCGTGGGCTGTGTGAATC CTCAGTGGGCCTTGGCACAGCATCAAACCAAGTTATACCTTC TCAACACCACCAAGCTTAGGTAAATCAGCTGAGTGTG	3124
	CACACTCAGCTGATTTACCTAAGCTTGGTGGTGTTGAGAAGG TATAACTTGGTTTGATGCTGTGCCAAGGCCCACTGAGGATTC ACACAGCCCACGAAGGAGTGGTTATGCAACATCTCCC	3125

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTTGGCACAGCATCAAA	3126
	TTTGATGCTGTGCCAAG	3127
Non-polyposis colorectal cancer Leu549Pro CTT-CCT	CCTTCGTGGGCTGTGTGAATCCTCAGTGGGCCTTGGCACAG CATCAAACCAAGTTATACCTTCTCAACACCACCAAGCTTAGGT AAATCAGCTGAGTGTGTGAACAAGCAGAGCTACTACA	3128
	TGTAGTAGCTCTGCTTGTTCACACACTCAGCTGATTTACCTAA GCTTGGTGGTGTTGAGAAGGTATAACTTGGTTTGATGCTGTG CCAAGGCCCACTGAGGATTCACACAGCCCACGAAGG	3129
	GTTATACCTTCTCAACA	3130
	TGTTGAGAAGGTATAAC	3131
Non-polyposis colorectal cancer Asn551Thr AAC-ACC	TGGGCTGTGTGAATCCTCAGTGGGCCTTGGCACAGCATCAAA CCAAGTTATACCTTCTCAACACCACCAAGCTTAGGTAAATCAG CTGAGTGTGTGAACAAGCAGAGCTACTACAACAATG	3132
	CATTGTTGTAGTAGCTCTGCTTGTTCACACACTCAGCTGATTT ACCTAAGCTTGGTGGTGTTGAGAAGGTATAACTTGGTTTGAT GCTGTGCCAAGGCCCACTGAGGATTCACACAGCCCA	3133
	CCTTCTCAACACCACCA	3134
	TGGTGGTGTTGAGAAGG	3135
Non-polyposis colorectal cancer Gln562Term CAG-TAG	ATGAATTCAGCTTTTCCTTAAAGTCACTTCATTTTTATTTTCAG TGAAGAACTGTTCTACCAGATACTCATTTATGATTTTGCCAATT TTGGTGTTCTCAGGTTATCGGTAAGTTTAGATC	3136
	GATCTAAACTTACCGATAACCTGAGAACACCAAAATTGGCAAA ATCATAAATGAGTATCTGGTAGAACAGTTCTTCACTGAAAATA AAAATGAAGTGACTTTAAGGAAAAGCTGAATTCAT	3137
	TGTTCTACCAGATACTC	3138
	GAGTATCTGGTAGAACA	3139
Non-polyposis colorectal cancer Ile565Phe ATT-TTT	GCTTTTCCTTAAAGTCACTTCATTTTTATTTTCAGTGAAGAACT GTTCTACCAGATACTCATTTATGATTTTGCCAATTTTGGTGTTC TCAGGTTATCGGTAAGTTTAGATCCTTTTCACT	3140
	AGTGAAAAGGATCTAAACTTACCGATAACCTGAGAACACCAA AATTGGCAAAATCATAAATGAGTATCTGGTAGAACAGTTCTTC ACTGAAAATAAAAATGAAGTGACTTTAAGGAAAAGC	3141
	AGATACTCATTTATGAT	3142
	ATCATAAATGAGTATCT	3143
Non-polyposis colorectal cancer Leu574Pro CTC-CCC	TTTTCAGTGAAGAACTGTTCTACCAGATACTCATTTATGATTTT GCCAATTTTGGTGTTCTCAGGTTATCGGTAAGTTTAGATCCTT TTCACTTCTGAAATTTCAACTGATCGTTTCTGAA	3144
	TTCAGAAACGATCAGTTGAAATTTCAGAAGTGAAAAGGATCTA AACTTACCGATAACCTGAGAACACCAAAATTGGCAAAATCATA AATGAGTATCTGGTAGAACAGTTCTTCACTGAAAA	3145
	TGGTGTTCTCAGGTTAT	3146
	ATAACCTGAGAACACCA	3147

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Leu582Val CTC-GTC	TGGATGCTCCGTTAAAGCTTGCTCCTTCATGTTCTTGCTTCTT CCTAGGAGCCAGCACCGCTCTTTGACCTTGCCATGCTTGCCT TAGATAGTCCAGAGAGTGGCTGGACAGAGGAAGATG	3148
	CATCTTCCTCTGTCCAGCCACTCTCTGGACTATCTAAGGCAA GCATGGCAAGGTCAAAGAGCGGTGCTGGCTCCTAGGAAGAA GCAAGAACATGAAGGAGCAAGCTTTAACGGAGCATCCA	3149
	CAGCACCGCTCTTTGAC	3150
	GTCAAAGAGCGGTGCTG	3151
Non-polyposis colorectal cancer Leu607His CTT-CAT	TGCTTGCCTTAGATAGTCCAGAGAGTGGCTGGACAGAGGAA GATGGTCCCAAAGAAGGACTTGCTGAATACATTGTTGAGTTT CTGAAGAAGAAGGCTGAGATGCTTGCAGACTATTTCTC	3152
	GAGAAATAGTCTGCAAGCATCTCAGCCTTCTTCTTCAGAAACT CAACAATGTATTCAGCAAGTCCTTCTTTGGGACCATCTTCCTC TGTCCAGCCACTCTCTGGACTATCTAAGGCAAGCA	3153
	AGAAGGACTTGCTGAAT	3154
	ATTCAGCAAGTCCTTCT	3155
Non-polyposis colorectal cancer Lys618Term AAG-TAG	ACAGAGGAAGATGGTCCCAAAGAAGGACTTGCTGAATACATT GTTGAGTTTCTGAAGAAGAAGGCTGAGATGCTTGCAGACTAT TTCTCTTTGGAAATTGATGAGGTGTGACAGCCATTCT	3156
	AGAATGGCTGTCACACCTCATCAATTTCCAAAGAGAAATAGTC TGCAAGCATCTCAGCCTTCTTCTTCAGAAACTCAACAATGTAT TCAGCAAGTCCTTCTTTGGGACCATCTTCCTCTGT	3157
	TGAAGAAGAAGGCTGAG	3158
	CTCAGCCTTCTTCTTCA	3159
Non-polyposis colorectal cancer Lys618Thr AAG-ACG	CAGAGGAAGATGGTCCCAAAGAAGGACTTGCTGAATACATTG TTGAGTTTCTGAAGAAGAAGGCTGAGATGCTTGCAGACTATT TCTCTTTGGAAATTGATGAGGTGTGACAGCCATTCTT	3160
	AAGAATGGCTGTCACACCTCATCAATTTCCAAAGAGAAATAGT CTGCAAGCATCTCAGCCTTCTTCTTCAGAAACTCAACAATGTA TTCAGCAAGTCCTTCTTTGGGACCATCTTCCTCTG	3161
	GAAGAAGAAGGCTGAGA	3162
	TCTCAGCCTTCTTCTTC	3163
Non-polyposis colorectal cancer Arg659Leu CGA-CTA	TACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGAC TGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATCA AGCAGATACTAAGCATTTCGGTACATGCATGTGTGC	3164
	GCACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCAC TGACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCT CCAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTA	3165
	CATTCTTCGACTAGCCA	3166
	TGGCTAGTCGAAGAATG	3167
Non-polyposis colorectal cancer Arg659Pro CGA-CCA	TACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGAC TGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATCA AGCAGATACTAAGCATTTCGGTACATGCATGTGTGC	3168

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCAC TGACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCT CCAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTA	3169
	CATTCTTCGACTAGCCA	3170
	TGGCTAGTCGAAGAATG	3171
Non-polyposis colorectal cancer Arg659Term CGA-TGA	TTACCCCTTCTGATTGACAACTATGTGCCCCCTTTGGAGGGA CTGCCTATCTTCATTCTTCGACTAGCCACTGAGGTCAGTGATC AAGCAGATACTAAGCATTTCGGTACATGCATGTGTG	3172
	CACACATGCATGTACCGAAATGCTTAGTATCTGCTTGATCACT GACCTCAGTGGCTAGTCGAAGAATGAAGATAGGCAGTCCCTC CAAAGGGGGCACATAGTTGTCAATCAGAAGGGGTAA	3173
	TCATTCTTCGACTAGCC	3174
	GGCTAGTCGAAGAATGA	3175
Non-polyposis colorectal cancer Ala681Thr GCT-ACT	TTGGACCAGGTGAATTGGGACGAAGAAAAGGAATGTTTTGAA AGCCTCAGTAAAGAATGCGCTATGTTCTATTCCATCCGGAAG CAGTACATATCTGAGGAGTCGACCCTCTCAGGCCAGC	3176
	GCTGGCCTGAGAGGGTCGACTCCTCAGATATGTACTGCTTCC GGATGGAATAGAACATAGCGCATTCTTTACTGAGGCTTTCAAA ACATTCCTTTTCTTCGTCCCAATTCACCTGGTCCAA	3177
	AAGAATGCGCTATGTTC	3178
	GAACATAGCGCATTCTT	3179
Non-polyposis colorectal cancer Trp712Term TGG-TAG	AGGCTTATGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTG GCTCCATTCCAAACTCCTGGAAGTGGACTGTGGAACACATTG TCTATAAAGCCTTGCGCTCACACATTCTGCCTCCTAA	3180
	TTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATAGACAATG TGTTCCACAGTCCACTTCCAGGAGTTTGGAATGGAGCCAGGC ACTTCACTCTGGAAAACACATTAGATGTCATAAGCCT	3181
	AAACTCCTGGAAGTGGA	3182
	TCCACTTCCAGGAGTTT	3183
Non-polyposis colorectal cancer Trp714Term TGG-TAG	ATGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCAT TCCAAACTCCTGGAAGTGGACTGTGGAACACATTGTCTATAA AGCCTTGCGCTCACACATTCTGCCTCCTAAACATTT	3184
	AAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATAG ACAATGTGTTCCACAGTCCACTTCCAGGAGTTTGGAATGGAG CCAGGCACTTCACTCTGGAAAACACATTAGATGTCAT	3185
	CTGGAAGTGGACTGTGG	3186
	CCACAGTCCACTTCCAG	3187
Non-polyposis colorectal cancer Trp714Term TGG-TGA	TGACATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCATT CCAAACTCCTGGAAGTGGACTGTGGAACACATTGTCTATAAA GCCTTGCGCTCACACATTCTGCCTCCTAAACATTTC	3188
	GAAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTTTATA GACAATGTGTTCCACAGTCCACTTCCAGGAGTTTGGAATGGA GCCAGGCACTTCACTCTGGAAAACACATTAGATGTCA	3189

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGGAAGTGGACTGTGGA	3190
	TCCACAGTCCACTTCCA	3191
Non-polyposis colorectal cancer Val716Met GTG-ATG	ATCTAATGTGTTTTCCAGAGTGAAGTGCCTGGCTCCATTCCAA ACTCCTGGAAGTGGACTGTGGAACACATTGTCTATAAAGCCT TGCGCTCACACATTCTGCCTCCTAAACATTTCACAG	3192
	CTGTGAAATGTTTAGGAGGCAGAATGTGTGAGCGCAAGGCTT TATAGACAATGTGTTCCACAGTCCACTTCCAGGAGTTTGGAAT GGAGCCAGGCACTTCACTCTGGAAAACACATTAGAT	3193
	AGTGGACTGTGGAACAC	3194
	GTGTTCCACAGTCCACT	3195
Non-polyposis colorectal cancer Tyr721Term TAT-TAA	GAGTGAAGTGCCTGGCTCCATTCCAAACTCCTGGAAGTGGAC TGTGGAACACATTGTCTATAAAGCCTTGCGCTCACACATTCTG CCTCCTAAACATTTCACAGAAGATGGAAATATCCTG	3196
	CAGGATATTTCCATCTTCTGTGAAATGTTTAGGAGGCAGAATG TGTGAGCGCAAGGCTTTATAGACAATGTGTTCCACAGTCCAC TTCCAGGAGTTTGGAATGGAGCCAGGCACTTCACTC	3197
	ATTGTCTATAAAGCCTT	3198
	AAGGCTTTATAGACAAT	3199
Non-polyposis colorectal cancer Lys751Arg AAA-AGA	CTAAACATTTCACAGAAGATGGAAATATCCTGCAGCTTGCTAA CCTGCCTGATCTATACAAAGTCTTTGAGAGGTGTTAAATATGG TTATTTATGCACTGTGGGATGTGTTCTTCTTTCTC	3200
	GAGAAAGAAGAACACATCCCACAGTGCATAAATAACCATATTT AACACCTCTCAAAGACTTTGTATAGATCAGGCAGGTTAGCAA GCTGCAGGATATTTCCATCTTCTGTGAAATGTTTAG	3201
	TCTATACAAAGTCTTTG	3202
	CAAAGACTTTGTATAGA	3203
Non-polyposis colorectal cancer Arg755Trp AGG-TGG	ACAGAAGATGGAAATATCCTGCAGCTTGCTAACCTGCCTGAT CTATACAAAGTCTTTGAGAGGTGTTAAATATGGTTATTTATGC ACTGTGGGATGTGTTCTTCTTTCTCTGTATTCCGAT	3204
	ATCGGAATACAGAGAAAGAAGAACACATCCCACAGTGCATAA ATAACCATATTTAACACCTCTCAAAGACTTTGTATAGATCAGG CAGGTTAGCAAGCTGCAGGATATTTCCATCTTCTGT	3205
	TCTTTGAGAGGTGTTAA	3206
	TTAACACCTCTCAAAGA	3207

EXAMPLE 18

Human mismatch repair - MSH2

The human MSH2 gene is homologous to the bacterial *mutS* gene, which is involved in mismatch repair. Mutations in the MSH2 gene have been identified in a variety of cancers, including, for example,

ovarian tumors, colorectal cancer, endometrial cancer, uterine cancer. The attached table discloses the correcting oligonucleotide base sequences for the MSH2 oligonucleotides of the invention.

Table 25

MSH2 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Gln252Term CAG-TAG	TTTTCCACAAAAGACATTTATCAGGACCTCAACCGGTTGTTGA AAGGCAAAAAGGGAGAGCAGATGAATAGTGCTGTATTGCCAG AAATGGAGAATCAGGTACATGGATTATAAATGTGAA	3208
	TTCACATTTATAATCCATGTACCTGATTCTCCATTTCTGGCAAT ACAGCACTATTCATCTGCTCTCCCTTTTTGCCTTTCAACAACC GGTTGAGGTCCTGATAAATGTCTTTTGTGGAAAA	3209
	AGGGAGAGCAGATGAAT	3210
	ATTCATCTGCTCTCCCT	3211
Non polyposis colorectal cancer Gln288Term CAG-TAG	TCATCACTGTCTGCGGTAATCAAGTTTTTAGAACTCTTATCAG ATGATTCCAACTTTGGACAGTTTGAACTGACTACTTTTGACTT CAGCCAGTATATGAAATTGGATATTGCAGCAGTCA	3212
	TGACTGCTGCAATATCCAATTTCATATACTGGCTGAAGTCAAA AGTAGTCAGTTCAAACTGTCCAAAGTTGGAATCATCTGATAAG AGTTCTAAAAACTTGATTACCGCAGACAGTGATGA	3213
	ACTTTGGACAGTTTGAA	3214
	TTCAAACTGTCCAAAGT	3215
Non polyposis colorectal cancer Ala305Thr GCA-ACA	AACTTTGGACAGTTTGAACTGACTACTTTTGACTTCAGCCAGT ATATGAAATTGGATATTGCAGCAGTCAGAGCCCTTAACCTTTT TCAGGTAAAAAAAAAAAAAAAAAAAAAAAAAAAGG	3216
	CCTTTTTTTTTTTTTTTTTTTTTTTTTTTACCTGAAAAAGGTTAA GGGCTCTGACTGCTGCAATATCCAATTTCATATACTGGCTGAA GTCAAAAGTAGTCAGTTCAAACTGTCCAAAGTT	3217
	TGGATATTGCAGCAGTC	3218
	GACTGCTGCAATATCCA	3219
Non polyposis colorectal cancer Gly322Asp GGC-GAC	AGCTTGCCATTCTTTCTATTTTATTTTTTGTTTACTAGGGTTCT GTTGAAGATACCACTGGCTCTCAGTCTCTGGCTGCCTTGCTG AATAAGTGTAAAACCCCTCAAGGACAAAGACTTGT	3220
	ACAAGTCTTTGTCCTTGAGGGGTTTTACACTTATTCAGCAAGG CAGCCAGAGACTGAGAGCCAGTGGTATCTTCAACAGAACCCT AGTAAACAAAAAATAAAATAGAAAGAATGGCAAGCT	3221
	TACCACTGGCTCTCAGT	3222
	ACTGAGAGCCAGTGGTA	3223
Non polyposis colorectal cancer Ser323Cys TCT-TGT	TTGCCATTCTTTCTATTTTATTTTTTGTTTACTAGGGTTCTGTT GAAGATACCACTGGCTCTCAGTCTCTGGCTGCCTTGCTGAAT AAGTGTAAAACCCCTCAAGGACAAAGACTTGTTAA	3224

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTAACAAGTCTTTGTCCTTGAGGGGTTTTACACTTATTCAGCA AGGCAGCCAGAGACTGAGAGCCAGTGGTATCTTCAACAGAA CCCTAGTAAACAAAAAATAAAATAGAAAGAATGGCAA	3225
	CACTGGCTCTCAGTCTC	3226
	GAGACTGAGAGCCAGTG	3227
Non polyposis colorectal cancer Arg383Term CGA-TGA	GTGGAAGCTTTTGTAGAAGATGCAGAATTGAGGCAGACTTTA CAAGAAGATTTACTTCGTCGATTCCCAGATCTTAACCGACTTG CCAAGAAGTTTCAAAGACAAGCAGCAAACTTACAAG	3228
	CTTGTAAGTTTGCTGCTTGTCTTTGAAACTTCTTGGCAAGTCG GTTAAGATCTGGGAATCGACGAAGTAAATCTTCTTGTAAAGTC TGCCTCAATTCTGCATCTTCTACAAAAGCTTCCAC	3229
	TACTTCGTCGATTCCCA	3230
	TGGGAATCGACGAAGTA	3231
Non polyposis colorectal cancer Gln397Term CAA-TAA	CAAGAAGATTTACTTCGTCGATTCCCAGATCTTAACCGACTTG CCAAGAAGTTTCAAAGACAAGCAGCAAACTTACAAGATTGTTA CCGACTCTATCAGGGTATAAATCAACTACCTAATG	3232
	CATTAGGTAGTTGATTTATACCCTGATAGAGTCGGTAACAATC TTGTAAGTTTGCTGCTTGTCTTTGAAACTTCTTGGCAAGTCGG TTAAGATCTGGGAATCGACGAAGTAAATCTTCTTG	3233
	TTCAAAGACAAGCAGCA	3234
	TGCTGCTTGTCTTTGAA	3235
Non polyposis colorectal cancer Arg406Term CGA-TGA	GATCTTAACCGACTTGCCAAGAAGTTTCAAAGACAAGCAGCA AACTTACAAGATTGTTACCGACTCTATCAGGGTATAAATCAAC TACCTAATGTTATACAGGCTCTGGAAAAACATGAAG	3236
	CTTCATGTTTTTCCAGAGCCTGTATAACATTAGGTAGTTGATTT ATACCCTGATAGAGTCGGTAACAATCTTGTAAGTTTGCTGCTT GTCTTTGAAACTTCTTGGCAAGTCGGTTAAGATC	3237
	ATTGTTACCGACTCTAT	3238
	ATAGAGTCGGTAACAAT	3239
Non polyposis colorectal cancer Gln419Term CAG-TAG	GCAAACTTACAAGATTGTTACCGACTCTATCAGGGTATAAATC AACTACCTAATGTTATACAGGCTCTGGAAAAACATGAAGGTAA CAAGTGATTTTGTTTTTTTGTTTTCCTTCAACTCA	3240
	TGAGTTGAAGGAAAACAAAAAAACAAAATCACTTGTTACCTTC ATGTTTTTCCAGAGCCTGTATAACATTAGGTAGTTGATTTATAC CCTGATAGAGTCGGTAACAATCTTGTAAGTTTGC	3241
	ATGTTATACAGGCTCTG	3242
	CAGAGCCTGTATAACAT	3243
Non polyposis colorectal cancer Gln429Term CAG-TAG	TATTCTGTAAAATGAGATCTTTTTATTTGTTTGTTTTACTACTTT CTTTTAGGAAAACACCAGAAATTATTGTTGGCAGTTTTTGTGA CTCCTCTTACTGATCTTCGTTCTGACTTCTCCA	3244
	TGGAGAAGTCAGAACGAAGATCAGTAAGAGGAGTCACAAAAA CTGCCAACAATAATTTCTGGTGTTTTCCTAAAAGAAAGTAGTA AAACAAACAAATAAAAAGATCTCATTTTACAGAATA	3245

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GAAAACACCAGAAATTA	3246
	TAATTTCTGGTGTTTTC	3247
Non polyposis colorectal cancer Leu458Term TTA-TGA	CTCCTCTTACTGATCTTCGTTCTGACTTCTCCAAGTTTCAGGA AATGATAGAAACAACTTTAGATATGGATCAGGTATGCAATATA CTTTTTAATTTAAGCAGTAGTTATTTTTAAAAAGC	3248
	GCTTTTTAAAAATAACTACTGCTTAAATTAAAAAGTATATTGCA TACCTGATCCATATCTAAAGTTGTTTCTATCATTTCCTGAAACT TGGAGAAGTCAGAACGAAGATCAGTAAGAGGAG	3249
	AACAACTTTAGATATGG	3250
	CCATATCTAAAGTTGTT	3251
Non polyposis colorectal cancer Gln518Term CAG-TAG	TTTCTTCTTGATTATCAAGGCTTGGACCCTGGCAAACAGATTA AACTGGATTCCAGTGCACAGTTTGGATATTACTTTCGTGTAAC CTGTAAGGAAGAAAAAGTCCTTCGTAACAATAAAA	3252
	TTTTATTGTTACGAAGGACTTTTTCTTCCTTACAGGTTACACGA AAGTAATATCCAAACTGTGCACTGGAATCCAGTTTAATCTGTT TGCCAGGGTCCAAGCCTTGATAATCAAGAAGAAA	3253
	CCAGTGCACAGTTTGGA	3254
	TCCAAACTGTGCACTGG	3255
Non polyposis colorectal cancer Arg524Pro CGT-CCT	GCTTGGACCCTGGCAAACAGATTAAACTGGATTCCAGTGCAC AGTTTGGATATTACTTTCGTGTAACCTGTAAGGAAGAAAAAGT CCTTCGTAACAATAAAAACTTTAGTACTGTAGATAT	3256
	ATATCTACAGTACTAAAGTTTTTATTGTTACGAAGGACTTTTTC TTCCTTACAGGTTACACGAAAGTAATATCCAAACTGTGCACTG GAATCCAGTTTAATCTGTTTGCCAGGGTCCAAGC	3257
	TTACTTTCGTGTAACCT	3258
	AGGTTACACGAAAGTAA	3259
Non polyposis colorectal cancer Glu562Val GAG-GTG	TTAATATTTTTAATAAAACTGTTATTTCGATTTGCAGCAAATTG ACTTCTTTAAATGAAGAGTATACCAAAAATAAAACAGAATATG AAGAAGCCCAGGATGCCATTGTTAAAGAAATTGT	3260
	ACAATTTCTTTAACAATGGCATCCTGGGCTTCTTCATATTCTGT TTTATTTTTGGTATACTCTTCATTTAAAGAAGTCAATTTGCTGC AAATCGAAATAACAGTTTTATTAAAAATATTAA	3261
	AAATGAAGAGTATACCA	3262
	TGGTATACTCTTCATTT	3263
Glioma Glu580Term GAA-TAA	AATGAAGAGTATACCAAAAATAAAACAGAATATGAAGAAGCCC AGGATGCCATTGTTAAAGAAATTGTCAATATTTCTTCAGGTAA ACTTAATAGAACTAATAATGTTCTGAATGTCACCT	3264
	AGGTGACATTCAGAACATTATTAGTTCTATTAAGTTTACCTGA AGAAATATTGACAATTTCTTTAACAATGGCATCCTGGGCTTCT TCATATTCTGTTTTATTTTTGGTATACTCTTCATT	3265
	TTGTTAAAGAAATTGTC	3266
	GACAATTTCTTTAACAA	3267

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Gln601Term CAG-TAG	TGTTTTTATTTTTATACAGGCTATGTAGAACCAATGCAGACACT CAATGATGTGTTAGCTCAGCTAGATGCTGTTGTCAGCTTTGCT CACGTGTCAAATGGAGCACCTGTTCCATATGTAC	3268
	GTACATATGGAACAGGTGCTCCATTTGACACGTGAGCAAAGC TGACAACAGCATCTAGCTGAGCTAACACATCATTGAGTGTCT GCATTGGTTCTACATAGCCTGTATAAAAATAAAAACA	3269
	TGTTAGCTCAGCTAGAT	3270
	ATCTAGCTGAGCTAACA	3271
Non polyposis colorectal cancer Tyr619Term TAT-TAG	AGCTCAGCTAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAAT GGAGCACCTGTTCCATATGTACGACCAGCCATTTTGGAGAAA GGACAAGGAAGAATTATATTAAAAGCATCCAGGCAT	3272
	ATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTTTCTCCA AAATGGCTGGTCGTACATATGGAACAGGTGCTCCATTTGACA CGTGAGCAAAGCTGACAACAGCATCTAGCTGAGCT	3273
	GTTCCATATGTACGACC	3274
	GGTCGTACATATGGAAC	3275
Non polyposis colorectal cancer Arg621Term CGA-TGA	CAGCTAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAATGGA GCACCTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGA CAAGGAAGAATTATATTAAAAGCATCCAGGCATGCTT	3276
	AAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTTTC TCCAAAATGGCTGGTCGTACATATGGAACAGGTGCTCCATTT GACACGTGAGCAAAGCTGACAACAGCATCTAGCTG	3277
	CATATGTACGACCAGCC	3278
	GGCTGGTCGTACATATG	3279
Non polyposis colorectal cancer Pro622Leu CCA-CTA	TAGATGCTGTTGTCAGCTTTGCTCACGTGTCAAATGGAGCAC CTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGACAAG GAAGAATTATATTAAAAGCATCCAGGCATGCTTGTGT	3280
	ACACAAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTC CTTTCTCCAAAATGGCTGGTCGTACATATGGAACAGGTGCTC CATTTGACACGTGAGCAAAGCTGACAACAGCATCTA	3281
	TGTACGACCAGCCATTT	3282
	AAATGGCTGGTCGTACA	3283
Non polyposis colorectal cancer Ala636Pro GCA-CCA	CCTGTTCCATATGTACGACCAGCCATTTTGGAGAAAGGACAA GGAAGAATTATATTAAAAGCATCCAGGCATGCTTGTGTTGAAG TTCAAGATGAAATTGCATTTATTCCTAATGACGTAT	3284
	ATACGTCATTAGGAATAAATGCAATTTCATCTTGAACTTCAAC ACAAGCATGCCTGGATGCTTTTAATATAATTCTTCCTTGTCCTT TCTCCAAAATGGCTGGTCGTACATATGGAACAGG	3285
	TATTAAAAGCATCCAGG	3286
	CCTGGATGCTTTTAATA	3287
Non polyposis colorectal cancer His639Arg CAT-CGT	ATGTACGACCAGCCATTTTGGAGAAAGGACAAGGAAGAATTA TATTAAAAGCATCCAGGCATGCTTGTGTTGAAGTTCAAGATGA AATTGCATTTATTCCTAATGACGTATACTTTGAAAA	3288

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TTTTCAAAGTATACGTCATTAGGAATAAATGCAATTTCATCTTG AACTTCAACACAAGCATGCCTGGATGCTTTTAATATAATTCTT CCTTGTCCTTTCTCCAAAATGGCTGGTCGTACAT	3289
	ATCCAGGCATGCTTGTG	3290
	CACAAGCATGCCTGGAT	3291
Non polyposis colorectal cancer His639Tyr CAT-TAT	TATGTACGACCAGCCATTTTGGAGAAAGGACAAGGAAGAATT ATATTAAAAGCATCCAGGCATGCTTGTGTTGAAGTTCAAGATG AAATTGCATTTATTCCTAATGACGTATACTTTGAAA	3292
	TTTCAAAGTATACGTCATTAGGAATAAATGCAATTTCATCTTGA ACTTCAACACAAGCATGCCTGGATGCTTTTAATATAATTCTTC CTTGTCCTTTCTCCAAAATGGCTGGTCGTACATA	3293
	CATCCAGGCATGCTTGT	3294
	ACAAGCATGCCTGGATG	3295
Non polyposis colorectal cancer Glu647Lys GAA-AAA	AAAGGACAAGGAAGAATTATATTAAAAGCATCCAGGCATGCTT GTGTTGAAGTTCAAGATGAAATTGCATTTATTCCTAATGACGT ATACTTTGAAAAAGATAAACAGATGTTCCACATCA	3296
	TGATGTGGAACATCTGTTTATCTTTTTCAAAGTATACGTCATTA GGAATAAATGCAATTTCATCTTGAACTTCAACACAAGCATGCC TGGATGCTTTTAATATAATTCTTCCTTGTCCTTT	3297
	TTCAAGATGAAATTGCA	3298
	TGCAATTTCATCTTGAA	3299
Non polyposis colorectal cancer Tyr656Term TAC-TAG	ATCCAGGCATGCTTGTGTTGAAGTTCAAGATGAAATTGCATTT ATTCCTAATGACGTATACTTTGAAAAAGATAAACAGATGTTCC ACATCATTACTGGTAAAAAACCTGGTTTTTGGGCT	3300
	AGCCCAAAAACCAGGTTTTTTACCAGTAATGATGTGGAACATC TGTTTATCTTTTTCAAAGTATACGTCATTAGGAATAAATGCAAT TTCATCTTGAACTTCAACACAAGCATGCCTGGAT	3301
	GACGTATACTTTGAAAA	3302
	TTTTCAAAGTATACGTC	3303
Non polyposis colorectal cancer Gly674Asp GGT-GAT	GAAAGAAGTTTAAAATCTTGCTTTCTGATATAATTTGTTTTGTA GGCCCCAATATGGGAGGTAAATCAACATATATTCGACAAACT GGGGTGATAGTACTCATGGCCCAAATTGGGTGTTT	3304
	AAACACCCAATTTGGGCCATGAGTACTATCACCCCAGTTTGTC GAATATATGTTGATTTACCTCCCATATTGGGGCCTACAAAACA AATTATATCAGAAAGCAAGATTTTAAACTTCTTTC	3305
	TATGGGAGGTAAATCAA	3306
	TTGATTTACCTCCCATA	3307
Non polyposis colorectal cancer Arg680Term CGA-TGA	TTGCTTTCTGATATAATTTGTTTTGTAGGCCCCAATATGGGAG GTAAATCAACATATATTCGACAAACTGGGGTGATAGTACTCAT GGCCCAAATTGGGTGTTTTGTGCCATGTGAGTCAG	3308
	CTGACTCACATGGCACAAAACACCCAATTTGGGCCATGAGTA CTATCACCCCAGTTTGTCGAATATATGTTGATTTACCTCCCAT ATTGGGGCCTACAAAACAAATTATATCAGAAAGCAA	3309

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATATATTCGACAAACT	3310
	AGTTTGTCGAATATATG	3311
Non polyposis colorectal cancer Gly692Arg GGG-CGG	ATGGGAGGTAAATCAACATATATTCGACAAACTGGGGTGATA GTACTCATGGCCCAAATTGGGTGTTTTGTGCCATGTGAGTCA GCAGAAGTGTCCATTGTGGACTGCATCTTAGCCCGAG	3312
	CTCGGGCTAAGATGCAGTCCACAATGGACACTTCTGCTGACT CACATGGCACAAAACACCCAATTTGGGCCATGAGTACTATCA CCCCAGTTTGTCGAATATATGTTGATTTACCTCCCAT	3313
	CCCAAATTGGGTGTTTT	3314
	AAAACACCCAATTTGGG	3315
Non polyposis colorectal cancer Cys697Arg TGT-CGT	ACATATATTCGACAAACTGGGGTGATAGTACTCATGGCCCAA ATTGGGTGTTTTGTGCCATGTGAGTCAGCAGAAGTGTCCATT GTGGACTGCATCTTAGCCCGAGTAGGGGCTGGTGACA	3316
	TGTCACCAGCCCCTACTCGGGCTAAGATGCAGTCCACAATGG ACACTTCTGCTGACTCACATGGCACAAAACACCCAATTTGGG CCATGAGTACTATCACCCCAGTTTGTCGAATATATGT	3317
	TTGTGCCATGTGAGTCA	3318
	TGACTCACATGGCACAA	3319
Non polyposis colorectal cancer Cys697Phe TGT-TTT	CATATATTCGACAAACTGGGGTGATAGTACTCATGGCCCAAA TTGGGTGTTTTGTGCCATGTGAGTCAGCAGAAGTGTCCATTG TGGACTGCATCTTAGCCCGAGTAGGGGCTGGTGACAG	3320
	CTGTCACCAGCCCCTACTCGGGCTAAGATGCAGTCCACAATG GACACTTCTGCTGACTCACATGGCACAAAACACCCAATTTGG GCCATGAGTACTATCACCCCAGTTTGTCGAATATATG	3321
	TGTGCCATGTGAGTCAG	3322
	CTGACTCACATGGCACA	3323
Non polyposis colorectal cancer Gln718Term CAA-TAA	GAGTCAGCAGAAGTGTCCATTGTGGACTGCATCTTAGCCCGA GTAGGGGCTGGTGACAGTCAATTGAAAGGAGTCTCCACGTTC ATGGCTGAAATGTTGGAAACTGCTTCTATCCTCAGGT	3324
	ACCTGAGGATAGAAGCAGTTTCCAACATTTCAGCCATGAACG TGGAGACTCCTTTCAATTGACTGTCACCAGCCCCTACTCGGG CTAAGATGCAGTCCACAATGGACACTTCTGCTGACTC	3325
	GTGACAGTCAATTGAAA	3326
	TTTCAATTGACTGTCAC	3327
Non polyposis colorectal cancer Leu811Term TTA-TGA	CCAATCAGATACCAACTGTTAATAATCTACATGTCACAGCACT CACCACTGAAGAGACCTTAACTATGCTTTATCAGGTGAAGAAA GGTATGTACTATTGGAGTACTCTAAATTCAGAACT	3328
	AGTTCTGAATTTAGAGTACTCCAATAGTACATACCTTTCTTCAC CTGATAAAGCATAGTTAAGGTCTCTTCAGTGGTGAGTGCTGT GACATGTAGATTATTAACAGTTGGTATCTGATTGG	3329
	AGAGACCTTAACTATGC	3330
	GCATAGTTAAGGTCTCT	3331

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non polyposis colorectal cancer Ala834Thr GCT-ACT	TTCCCCAAATTTCTTATAGGTGTCTGTGATCAAAGTTTTGGGA TTCATGTTGCAGAGCTTGCTAATTTCCCTAAGCATGTAATAGA GTGTGCTAAACAGAAAGCCCTGGAACTTGAGGAGT	3332
	ACTCCTCAAGTTCCAGGGCTTTCTGTTTAGCACACTCTATTAC ATGCTTAGGGAAATTAGCAAGCTCTGCAACATGAATCCCAAA ACTTTGATCACAGACACCTATAAGAAATTTGGGGAA	3333
	CAGAGCTTGCTAATTTC	3334
	GAAATTAGCAAGCTCTG	3335
Non polyposis colorectal cancer Gln861Term CAA-TAA	ATAGAGTGTGCTAAACAGAAAGCCCTGGAACTTGAGGAGTTT CAGTATATTGGAGAATCGCAAGGATATGATATCATGGAACCA GCAGCAAAGAAGTGCTATCTGGAAAGAGAGGTTTGTC	3336
	GACAAACCTCTCTTTCCAGATAGCACTTCTTTGCTGCTGGTTC CATGATATCATATCCTTGCGATTCTCCAATATACTGAAACTCC TCAAGTTCCAGGGCTTTCTGTTTAGCACACTCTAT	3337
	GAGAATCGCAAGGATAT	3338
	ATATCCTTGCGATTCTC	3339
Non polyposis colorectal cancer Thr905Arg ACA-AGA	AGGAGTTCCTGTCCAAGGTGAAACAAATGCCCTTTACTGAAA TGTCAGAAGAAAACATCACAATAAAGTTAAAACAGCTAAAAGC TGAAGTAATAGCAAAGAATAATAGCTTTGTAAATGA	3340
	TCATTTACAAAGCTATTATTCTTTGCTATTACTTCAGCTTTTAG CTGTTTTAACTTTATTGTGATGTTTTCTTCTGACATTTCAGTAA AGGGCATTTGTTTCACCTTGGACAGGAACTCCT	3341
	AAACATCACAATAAAGT	3342
	ACTTTATTGTGATGTTT	3343

EXAMPLE 19

Human mismatch repair - MSH6

The human MSH6 gene is homologous to the bacterial *mutS* gene, which is involved in mismatch repair. Mutations in the MSH6 gene have been identified in a variety of cancers, including particularly hereditary nonpolyposis colorectal cancer. The attached table discloses the correcting oligonucleotide base sequences for the MSH6 oligonucleotides of the invention.

Table 26

MSH6 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Non-polyposis colorectal cancer Ser144Ile AGC-ATC	GGAAATCAGTCCGTGTTCATGTACAGTTTTTTGATGACAGCCC AACAAGGGGCTGGGTTAGCAAAAGGCTTTTAAAGCCATATAC AGGTAAGAGTCACTACTGCCATGTGTGTGTGTTTGT	3344
	ACAAACACACACACATGGCAGTAGTGACTCTTACCTGTATATG GCTTTAAAAGCCTTTTGCTAACCCAGCCCCTTGTTGGGCTGT CATCAAAAAACTGTACATGAACACGGACTGATTTCC	3345
	CTGGGTTAGCAAAAGGC	3346
	GCCTTTTGCTAACCCAG	3347
Endometrial cancer Ser156Term TCA-TGA	CGTGAGCCTCTGCACCCGGCCCTTATTGTTTATAAATACATTT CTTTCTAGGTTCAAAATCAAAGGAAGCCCAGAAGGGAGGTCA TTTTTACAGTGCAAAGCCTGAAATACTGAGAGCAAT	3348
	ATTGCTCTCAGTATTTCAGGCTTTGCACTGTAAAAATGACCTC CCTTCTGGGCTTCCTTTGATTTTGAACCTAGAAAGAAATGTAT TTATAAACAATAAGGGCCGGGTGCAGAGGCTCACG	3349
	TTCAAAATCAAAGGAAG	3350
	CTTCCTTTGATTTTGAA	3351
Early onset colorectal cancer Tyr214Term TAC-TAG	TTCCAAATTTTGATTTGTTTTTAAATACTCTTTCCTTGCCTGGC AGGTAGGCACAACTTACGTAACAGATAAGAGTGAAGAAGATA ATGAAATTGAGAGTGAAGAGGAAGTACAGCCTAAG	3352
	CTTAGGCTGTACTTCCTCTTCACTCTCAATTTCATTATCTTCTT CACTCTTATCTGTTACGTAAGTTGTGCCTACCTGCCAGGCAA GGAAAGAGTATTTAAAAACAAATCAAAATTTGGAA	3353
	ACAACTTACGTAACAGA	3354
	TCTGTTACGTAAGTTGT	3355
Endometrial cancer Arg248Term CGA-TGA	GAAGAGGAAGTACAGCCTAAGACACAAGGATCTAGGCGAAG TAGCCGCCAAATAAAAAAACGAAGGGTCATATCAGATTCTGA GAGTGACATTGGTGGCTCTGATGTGGAATTTAAGCCAG	3356
	CTGGCTTAAATTCCACATCAGAGCCACCAATGTCACTCTCAGA ATCTGATATGACCCTTCGTTTTTTTATTTGGCGGCTACTTCGC CTAGATCCTTGTGTCTTAGGCTGTACTTCCTCTTC	3357
	TAAAAAAACGAAGGGTC	3358
	GACCCTTCGTTTTTTTA	3359
Colorectal cancer Ser285Ile AGT-ATT	TTAAGCCAGACACTAAGGAGGAAGGAAGCAGTGATGAAATAA GCAGTGGAGTGGGGGATAGTGAGAGTGAAGGCCTGAACAGC CCTGTCAAAGTTGCTCGAAAGCGGAAGAGAATGGTGAC	3360

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GTCACCATTCTCTTCCGCTTTCGAGCAACTTTGACAGGGCTG TTCAGGCCTTCACTCTCACTATCCCCCACTCCACTGCTTATTT CATCACTGCTTCCTTCCTCCTTAGTGTCTGGCTTAA	3361
	GGGGGATAGTGAGAGTG	3362
	CACTCTCACTATCCCCC	3363
Colorectal cancer Gly566Arg GGA-AGA	GAGGAAGATTCTTCTGGCCATACTCGTGCATATGGTGTGTGC TTTGTTGATACTTCACTGGGAAAGTTTTTCATAGGTCAGTTTTC AGATGATCGCCATTGTTCGAGATTTAGGACTCTAG	3364
	CTAGAGTCCTAAATCTCGAACAATGGCGATCATCTGAAAACT GACCTATGAAAAACTTTCCCAGTGAAGTATCAACAAAGCACAC ACCATATGCACGAGTATGGCCAGAAGAATCTTCCTC	3365
	CTTCACTGGGAAAGTTT	3366
	AAACTTTCCCAGTGAAG	3367
Non-polyposis colorectal cancer Gln698Glu CAG-GAG	GAATTGGCCCTCTCTGCTCTAGGTGGTTGTGTCTTCTACCTCA AAAAATGCCTTATTGATCAGGAGCTTTTATCAATGGCTAATTTT GAAGAATATATTCCCTTGGATTCTGACACAGTCA	3368
	TGACTGTGTCAGAATCCAAGGGAATATATTCTTCAAAATTAGC CATTGATAAAAGCTCCTGATCAATAAGGCATTTTTTGAGGTAG AAGACACAACCACCTAGAGCAGAGAGGGCCAATTC	3369
	TTATTGATCAGGAGCTT	3370
	AAGCTCCTGATCAATAA	3371
Endometrial cancer Gln731Term CAA-TAA	CCCTTGGATTCTGACACAGTCAGCACTACAAGATCTGGTGCT ATCTTCACCAAAGCCTATCAACGAATGGTGCTAGATGCAGTG ACATTAAACAACTTGGAGATTTTTCTGAATGGAACAA	3372
	TTGTTCCATTCAGAAAAATCTCCAAGTTGTTTAATGTCACTGC ATCTAGCACCATTCGTTGATAGGCTTTGGTGAAGATAGCACC AGATCTTGTAGTGCTGACTGTGTCAGAATCCAAGGG	3373
	AAGCCTATCAACGAATG	3374
	CATTCGTTGATAGGCTT	3375
Colorectal cancer Val800Leu GTT-CTT	GCCCCACTCTGTAACCATTATGCTATTAATGATCGTCTAGATG CCATAGAAGACCTCATGGTTGTGCCTGACAAAATCTCCGAAG TTGTAGAGCTTCTAAAGAAGCTTCCAGATCTTGAGA	3376
	TCTCAAGATCTGGAAGCTTCTTTAGAAGCTCTACAACTTCGGA GATTTTGTCAGGCACAACCATGAGGTCTTCTATGGCATCTAGA CGATCATTAATAGCATAATGGTTACAGAGTGGGGC	3377
	ACCTCATGGTTGTGCCT	3378
	AGGCACAACCATGAGGT	3379

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Colorectal cancer Asp803Gly GAC-GGC	GTAACCATTATGCTATTAATGATCGTCTAGATGCCATAGAAGA CCTCATGGTTGTGCCTG**A**CAAAATCTCCGAAGTTGTAGAGCT TCTAAAGAAGCTTCCAGATCTTGAGAGGCTACTCAG	3380
	CTGAGTAGCCTCTCAAGATCTGGAAGCTTCTTTAGAAGCTCTA CAACTTCGGAGATTTTG**T**CAGGCACAACCATGAGGTCTTCTAT GGCATCTAGACGATCATTAATAGCATAATGGTTAC	3381
	TGTGCCTG**A**CAAAATCT	3382
	AGATTTTG**T**CAGGCACA	3383
Non-polyposis colorectal cancer Tyr850Cys TAC-TGC	CTCCCCTGAAGAGTCAGAACCACCCAGACAGCAGGGCTATAA TGTATGAAGAAACTACAT**A**CAGCAAGAAGAAGATTATTGATTT TCTTTCTGCTCTGGAAGGATTCAAAGTAATGTGTAA	3384
	TTACACATTACTTTGAATCCTTCCAGAGCAGAAAGAAAATCAA TAATCTTCTTCTTGCTG**T**ATGTAGTTTCTTCATACATTATAGCC CTGCTGTCTGGGTGGTTCTGACTCTTCAGGGGAG	3385
	AACTACAT**A**CAGCAAGA	3386
	TCTTGCTG**T**ATGTAGTT	3387
Colorectal cancer Pro1087Thr CCC-ACC	TATAGTCGAGGGGGTGATGGTCCTATGTGTCGCCCAGTAATT CTGTTGCCGGAAGATACC**C**CCCCCTTCTTAGAGCTTAAAGGA TCACGCCATCCTTGCATTACGAAGACTTTTTTTGGAG	3388
	CTCCAAAAAAAGTCTTCGTAATGCAAGGATGGCGTGATCCTTT AAGCTCTAAGAAGGGGGG**G**GGTATCTTCCGGCAACAGAATTAC TGGGCGACACATAGGACCATCACCCCCTCGACTATA	3389
	AAGATACC**C**CCCCCTTC	3390
	GAAGGGGGG**G**GGTATCTT	3391
Non-polyposis colorectal cancer Gln1258Term CAA-TAA	ACTATAAAATGTCGTACATTATTTTCAACTCACTACCATTCATT AGTAGAAGATTATTCT**C**AAAATGTTGCTGTGCGCCTAGGACAT ATGGTATGTGCAAATTGTTTTTTTCCACAAATTC	3392
	GAATTTGTGGAAAAAAACAATTTGCACATACCATATGTCCTAG GCGCACAGCAACATTTT**G**AGAATAATCTTCTACTAATGAATGG TAGTGAGTTGAAAATAATGTACGACATTTTATAGT	3393
	ATTATTCT**C**AAAATGTT	3394
	AACATTTT**G**AGAATAAT	3395

EXAMPLE 20

Hyperlipidemia – APOE

Hyperlipidemia is the abnormal elevation of plasma cholesterol and/or triglyceride levels and it is one of the most common diseases. The human apolipoprotein E protein is involved in the transport of

endogenous lipids and appears to be crucial for both the direct removal of cholesterol-rich LDL from plasma and conversion of IDL particles to LDL particles. Individuals who either lack apolipoprotein E or who are homozygous for particular alleles of apoE may have have a condition known as dysbetalipoproteinemia, which is characterized by elevated plasma cholesterol and triglyceride levels and an increased risk for atherosclerosis.

In a comprehensive review of apoE variants, de Knijff et al., *Hum. Mutat.* 4:178-194 (1994) found that 30 variants had been characterized, including the most common variant, apoE3. To that time, 14 apoE variants had been found to be associated with familial dysbetalipoproteinemia. The attached table discloses the correcting oligonucleotide base sequences for the APOE oligonucleotides of the invention.

Table 27

Apoe Mutations And Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Apolipoprotein E Glu13Lys cGAG-AAG	TTGTTCCACACAGGATGCCAGGCCAAGGTGGAGCAAGCGGT GGAGACAGAGCCGGAGCCCGAGCTGCGCCAGCAGACCGAG TGGCAGAGCGGCCAGCGCTGGGAACTGGCACTGGGTCGCT	3396
	AGCGACCCAGTGCCAGTTCCCAGCGCTGGCCGCTCTGCCAC TCGGTCTGCTGGCGCAGCTCGGGCTCCGGCTCTGTCTCCAC CGCTTGCTCCACCTTGGCCTGGCATCCTGTGTGGAACAA	3397
	CGGAGCCCGAGCTGCGC	3398
	GCGCAGCTCGGGCTCCG	3399
Apolipoprotein E Trp20Term TGGc-TGA	CAAGGTGGAGCAAGCGGTGGAGACAGAGCCGGAGCCCGAG CTGCGCCAGCAGACCGAGTGGCAGAGCGGCCAGCGCTGGG AACTGGCACTGGGTCGCTTTTGGGATTACCTGCGCTGGGTG	3400
	CACCCAGCGCAGGTAATCCCAAAAGCGACCCAGTGCCAGTT CCCAGCGCTGGCCGCTCTGCCACTCGGTCTGCTGGCGCAGC TCGGGCTCCGGCTCTGTCTCCACCGCTTGCTCCACCTTG	3401
	ACCGAGTGGCAGAGCGG	3402
	CCGCTCTGCCACTCGGT	3403
Apolipoprotein E Leu28Pro CTG-CCG	CAGAGCCGGAGCCCGAGCTGCGCCAGCAGACCGAGTGGCA GAGCGGCCAGCGCTGGGAACTGGCACTGGGTCGCTTTTGGG ATTACCTGCGCTGGGTGCAGACACTGTCTGAGCAGGTGCA	3404
	TGCACCTGCTCAGACAGTGTCTGCACCCAGCGCAGGTAATCC CAAAAGCGACCCAGTGCCAGTTCCCAGCGCTGGCCGCTCTG CCACTCGGTCTGCTGGCGCAGCTCGGGCTCCGGCTCTG	3405
	CTGGGAACTGGCACTGG	3406
	CCAGTGCCAGTTCCCAG	3407

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Apolipoprotein E Cys112Arg gTGC-CGC	CGGCTGTCCAAGGAGCTGCAGGCGGCGCAGGCCCGGCTGG GCGCGGACATGGAGGACGTGTGCGGCCGCCTGGTGCAGTA CCGCGGCGAGGTGCAGGCCATGCTCGGCCAGAGCACCGAG G	3408
	CCTCGGTGCTCTGGCCGAGCATGGCCTGCACCTCGCCGCGG TACTGCACCAGGCGGCCGCACACGTCCTCCATGTCCGCGCC CAGCCGGGCCTGCGCCGCCTGCAGCTCCTTGGACAGCCG	3409
	AGGACGTGTGCGGCCGC	3410
	GCGGCCGCACACGTCCT	3411
Apolipoprotein E Gly127Asp GGC-GAC	ACATGGAGGACGTGTGCGGCCGCCTGGTGCAGTACCGCGGC GAGGTGCAGGCCATGCTCGGCCAGAGCACCGAGGAGCTGC GGGTGCGCCTCGCCTCCCACCTGCGCAAGCTGCGTAAGCG	3412
	CGCTTACGCAGCTTGCGCAGGTGGGAGGCGAGGCGCACCC GCAGCTCCTCGGTGCTCTGGCCGAGCATGGCCTGCACCTCG CCGCGGTACTGCACCAGGCGGCCGCACACGTCCTCCATGT	3413
	CATGCTCGGCCAGAGCA	3414
	TGCTCTGGCCGAGCATG	3415
Apolipoprotein E Arg136Cys gCGC-TGC	GTGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGA GCACCGAGGAGCTGCGGGTGCGCCTCGCCTCCCACCTGCG CAAGCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGC	3416
	GCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTTG CGCAGGTGGGAGGCGAGGCGCACCCGCAGCTCCTCGGTGC TCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCAC	3417
	TGCGGGTGCGCCTCGCC	3418
	GGCGAGGCGCACCCGCA	3419
Apolipoprotein E Arg136His CGC-CAC	TGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGAGC ACCGAGGAGCTGCGGGTGCGCCTCGCCTCCCACCTGCGCAA GCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGCA	3420
	TGCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTT GCGCAGGTGGGAGGCGAGGCGCACCCGCAGCTCCTCGGTG CTCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCA	3421
	GCGGGTGCGCCTCGCCT	3422
	AGGCGAGGCGCACCCGC	3423
Apolipoprotein E Arg136Ser gCGC-AGC	GTGCAGTACCGCGGCGAGGTGCAGGCCATGCTCGGCCAGA GCACCGAGGAGCTGCGGGTGCGCCTCGCCTCCCACCTGCG CAAGCTGCGTAAGCGGCTCCTCCGCGATGCCGATGACCTGC	3424
	GCAGGTCATCGGCATCGCGGAGGAGCCGCTTACGCAGCTTG CGCAGGTGGGAGGCGAGGCGCACCCGCAGCTCCTCGGTGC TCTGGCCGAGCATGGCCTGCACCTCGCCGCGGTACTGCAC	3425
	TGCGGGTGCGCCTCGCC	3426
	GGCGAGGCGCACCCGCA	3427
Apolipoprotein E Arg142Cys gCGC-TGC	GTGCAGGCCATGCTCGGCCAGAGCACCGAGGAGCTGCGGG TGCGCCTCGCCTCCCACCTGCGCAAGCTGCGTAAGCGGCTC CTCCGCGATGCCGATGACCTGCAGAAGCGCCTGGCAGTGT	3428

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACACTGCCAGGCGCTTCTGCAGGTCATCGGCATCGCGGAGG AGCCGCTTACGCAGCTTGCGCAGGTGGGAGGCGAGGCGCA CCCGCAGCTCCTCGGTGCTCTGGCCGAGCATGGCCTGCAC	3429
	CCCACCTGCGCAAGCTG	3430
	CAGCTTGCGCAGGTGGG	3431
Apolipoprotein E Arg142Leu CGC-CTC	TGCAGGCCATGCTCGGCCAGAGCACCGAGGAGCTGCGGGT GCGCCTCGCCTCCCACCTGCGCAAGCTGCGTAAGCGGCTCC TCCGCGATGCCGATGACCTGCAGAAGCGCCTGGCAGTGTA	3432
	TACACTGCCAGGCGCTTCTGCAGGTCATCGGCATCGCGGAG GAGCCGCTTACGCAGCTTGCGCAGGTGGGAGGCGAGGCGC ACCCGCAGCTCCTCGGTGCTCTGGCCGAGCATGGCCTGCA	3433
	CCACCTGCGCAAGCTGC	3434
	GCAGCTTGCGCAGGTGG	3435
Apolipoprotein E Arg145Cys gCGT-TGT	ATGCTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCG CCTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGAT GCCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCG	3436
	CGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGCA TCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAGG CGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAGCAT	3437
	GCAAGCTGCGTAAGCGG	3438
	CCGCTTACGCAGCTTGC	3439
Apolipoprotein E Arg145Pro CGT-CCT	TGCTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGC CTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATG CCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGG	3440
	CCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGC ATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAG GCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAGCA	3441
	CAAGCTGCGTAAGCGGC	3442
	GCCGCTTACGCAGCTTG	3443
Apolipoprotein E Lys146Gln tAAG-CAG	CTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGCCT CCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATGCC GATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGGGG	3444
	CCCCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCG GCATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGA GGCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAG	3445
	AGCTGCGTAAGCGGCTC	3446
	GAGCCGCTTACGCAGCT	3447
Apolipoprotein E Lys146Glu tAAG-GAG	CTCGGCCAGAGCACCGAGGAGCTGCGGGTGCGCCTCGCCT CCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGATGCC GATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCGGGG	3448
	CCCCGGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCG GCATCGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGA GGCGAGGCGCACCCGCAGCTCCTCGGTGCTCTGGCCGAG	3449

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGCTGCGTAAGCGGCTC	3450
	GAGCCGCTTACGCAGCT	3451
Apolipoprotein E Arg158Cys gCGC-TGC	GCCTCCCACCTGCGCAAGCTGCGTAAGCGGCTCCTCCGCGA TGCCGATGACCTGCAGAAGCGCCTGGCAGTGTACCAGGCCG GGGCCCGCGAGGGCGCCGAGCGCGGCCTCAGCGCCATCC	3452
	GGATGGCGCTGAGGCCGCGCTCGGCGCCCTCGCGGGCCCC GGCCTGGTACACTGCCAGGCGCTTCTGCAGGTCATCGGCAT CGCGGAGGAGCCGCTTACGCAGCTTGCGCAGGTGGGAGGC	3453
	TGCAGAAGCGCCTGGCA	3454
	TGCCAGGCGCTTCTGCA	3455
Apolipoprotein E Gln187Glu aCAG-GAG	CGCGAGGGCGCCGAGCGCGGCCTCAGCGCCATCCGCGAGC GCCTGGGGCCCCTGGTGGAACAGGGCCGCGTGCGGGCCGC CACTGTGGGCTCCCTGGCCGGCCAGCCGCTACAGGAGCGG G	3456
	CCCGCTCCTGTAGCGGCTGGCCGGCCAGGGAGCCCACAGT GGCGGCCCGCACGCGGCCCTGTTCCACCAGGGGCCCCAGG CGCTCGCGGATGGCGCTGAGGCCGCGCTCGGCGCCCTCGC G	3457
	TGGTGGAACAGGGCCGC	3458
	GCGGCCCTGTTCCACCA	3459
Apolipoprotein E Trp210Term TGG-TAG	TGCGGGCCGCCACTGTGGGCTCCCTGGCCGGCCAGCCGCT ACAGGAGCGGGCCCAGGCCTGGGGCGAGCGGCTGCGCGCG CGGATGGAGGAGATGGGCAGCCGGACCCGCGACCGCCTGG A	3460
	TCCAGGCGGTCGCGGGTCCGGCTGCCCATCTCCTCCATCCG CGCGCGCAGCCGCTCGCCCCAGGCCTGGGCCCGCTCCTGT AGCGGCTGGCCGGCCAGGGAGCCCACAGTGGCGGCCCGCA	3461
	CCAGGCCTGGGGCGAGC	3462
	GCTCGCCCCAGGCCTGG	3463
Apolipoprotein E Arg228Cys cCGC-TGC	CAGGCCTGGGGCGAGCGGCTGCGCGCGCGGATGGAGGAGA TGGGCAGCCGGACCCGCGACCGCCTGGACGAGGTGAAGGA GCAGGTGGCGGAGGTGCGCGCCAAGCTGGAGGAGCAGGCC C	3464
	GGGCCTGCTCCTCCAGCTTGGCGCGCACCTCCGCCACCTGC TCCTTCACCTCGTCCAGGCGGTCGCGGGTCCGGCTGCCCAT CTCCTCCATCCGCGCGCGCAGCCGCTCGCCCCAGGCCTG	3465
	CCCGCGACCGCCTGGAC	3466
	GTCCAGGCGGTCGCGGG	3467
Apolipoprotein E Glu244Lys gGAG-AAG	CGGACCCGCGACCGCCTGGACGAGGTGAAGGAGCAGGTGG CGGAGGTGCGCGCCAAGCTGGAGGAGCAGGCCCAGCAGAT ACGCCTGCAGGCCGAGGCCTTCCAGGCCCGCCTCAAGAGCT	3468
	AGCTCTTGAGGCGGGCCTGGAAGGCCTCGGCCTGCAGGCGT ATCTGCTGGGCCTGCTCCTCCAGCTTGGCGCGCACCTCCGC CACCTGCTCCTTCACCTCGTCCAGGCGGTCGCGGGTCCG	3469

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAAGCTGGAGGAGCAG	3470
	CTGCTCCTCCAGCTTGG	3471

EXAMPLE 21

Familial hypercholesterolemia – LDLR

Familial hypercholesterolemia is characterized by elevation of serum cholesterol bound to low density lipoprotein (LDL) and is, hence, one of the conditions producing a hyperlipoproteinemia phenotype. Familial hypercholesterolemia is an autosomal dominant disorder characterized by elevation of serum cholesterol bound to low density lipoprotein (LDL). Mutations in the LDL receptor (LDLR) gene cause this disorder. The attached table discloses the correcting oligonucleotide base sequences for the LDLR oligonucleotides of the invention.

Table 28

LDLR Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Glu10Term CGAG-TAG	GCGTTGAGAGACCCTTTCTCCTTTTCCTCTCTCTCAGTGGGC GACAGATGCGAAAGAAACGAGTTCCAGTGCCAAGACGGGAA ATGCATCTCCTACAAGTGGGTCTGCGATGGCAGCGCTG	3472
	CAGCGCTGCCATCGCAGACCCACTTGTAGGAGATGCATTTCC CGTCTTGGCACTGGAACTCGTTTCTTTCGCATCTGTCGCCCA CTGAGAGAGAGGAAAAGGAGAAAGGGTCTCTCAACGC	3473
	AAAGAAACGAGTTCCAG	3474
	CTGGAACTCGTTTCTTT	3475
Hypercholesterolaemia Gln12Term cCAG-TAG	AGAGACCCTTTCTCCTTTTCCTCTCTCTCAGTGGGCGACAGAT GCGAAAGAAACGAGTTCCAGTGCCAAGACGGGAAATGCATCT CCTACAAGTGGGTCTGCGATGGCAGCGCTGAGTGCC	3476
	GGCACTCAGCGCTGCCATCGCAGACCCACTTGTAGGAGATG CATTTCCCGTCTTGGCACTGGAACTCGTTTCTTTCGCATCTGT CGCCCACTGAGAGAGAGGAAAAGGAGAAAGGGTCTCT	3477
	ACGAGTTCCAGTGCCAA	3478
	TTGGCACTGGAACTCGT	3479
Hypercholesterolaemia Gln14Term cCAA-TAA	CCTTTCTCCTTTTCCTCTCTCTCAGTGGGCGACAGATGCGAAA GAAACGAGTTCCAGTGCCAAGACGGGAAATGCATCTCCTACA AGTGGGTCTGCGATGGCAGCGCTGAGTGCCAGGATG	3480

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CATCCTGGCACTCAGCGCTGCCATCGCAGACCCACTTGTAGG AGATGCATTTCCCGTCTTGGCACTGGAACTCGTTTCTTTCGCA TCTGTCGCCCACTGAGAGAGAGGAAAAGGAGAAAGG	3481
	TCCAGTGCCAAGACGGG	3482
	CCCGTCTTGGCACTGGA	3483
Hypercholesterolaemia Trp23Term TGG-TAG	GCGACAGATGCGAAAGAAACGAGTTCCAGTGCCAAGACGGG AAATGCATCTCCTACAAGTGGGTCTGCGATGGCAGCGCTGAG TGCCAGGATGGCTCTGATGAGTCCCAGGAGACGTGCTG	3484
	CAGCACGTCTCCTGGGACTCATCAGAGCCATCCTGGCACTCA GCGCTGCCATCGCAGACCCACTTGTAGGAGATGCATTTCCCG TCTTGGCACTGGAACTCGTTTCTTTCGCATCTGTCGC	3485
	CTACAAGTGGGTCTGCG	3486
	CGCAGACCCACTTGTAG	3487
Hypercholesterolaemia Ala29Ser cGCT-TCT	AACGAGTTCCAGTGCCAAGACGGGAAATGCATCTCCTACAAG TGGGTCTGCGATGGCAGCGCTGAGTGCCAGGATGGCTCTGA TGAGTCCCAGGAGACGTGCTGTGAGTCCCCTTTGGGCA	3488
	TGCCCAAAGGGGACTCACAGCACGTCTCCTGGGACTCATCA GAGCCATCCTGGCACTCAGCGCTGCCATCGCAGACCCACTT GTAGGAGATGCATTTCCCGTCTTGGCACTGGAACTCGTT	3489
	ATGGCAGCGCTGAGTGC	3490
	GCACTCAGCGCTGCCAT	3491
Hypercholesterolaemia Cys31Tyr TGC-TAC	TCCAGTGCCAAGACGGGAAATGCATCTCCTACAAGTGGGTCT GCGATGGCAGCGCTGAGTGCCAGGATGGCTCTGATGAGTCC CAGGAGACGTGCTGTGAGTCCCCTTTGGGCATGATATG	3492
	CATATCATGCCCAAAGGGGACTCACAGCACGTCTCCTGGGAC TCATCAGAGCCATCCTGGCACTCAGCGCTGCCATCGCAGACC CACTTGTAGGAGATGCATTTCCCGTCTTGGCACTGGA	3493
	CGCTGAGTGCCAGGATG	3494
	CATCCTGGCACTCAGCG	3495
Hypercholesterolaemia Arg57Cys cCGT-TGT	AATCCTGTCTCTTCTGTAGTGTCTGTCACCTGCAAATCCGGG GACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATTCCTCA GTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAACG	3496
	CGTTGTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAG GAATGCAGCGGTTGACACGGGCCCCCACAGCTGAAGTCCCCG GATTTGCAGGTGACAGACACTACAGAAGAGACAGGATT	3497
	GTGGGGGCCGTGTCAAC	3498
	GTTGACACGGCCCCCAC	3499
Hypercholesterolaemia Gln64Term tCAG-TAG	TCTGTCACCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCG TGTCAACCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCA AGTGGACTGCGACAACGGCTCAGACGAGCAAGGCTGTC	3500
	GACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCACTTGGC CATCGCACCTCCAGAACTGAGGAATGCAGCGGTTGACACGG CCCCCACAGCTGAAGTCCCCGGATTTGCAGGTGACAGA	3501
	GCATTCCTCAGTTCTGG	3502

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAGAACTGAGGAATGC	3503
Hypercholesterolaemia Trp66Gly cTGG-GGG	ACCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAA CCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGG ACTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGT	3504
	ACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCA CTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTG ACACGGCCCCCACAGCTGAAGTCCCCGGATTTGCAGGT	3505
	CTCAGTTCTGGAGGTGC	3506
	GCACCTCCAGAACTGAG	3507
Hypercholesterolaemia Trp66Term TGG-TAG	CCTGCAAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAAC CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTG	3508
	CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTG ACACGGCCCCCACAGCTGAAGTCCCCGGATTTGCAGG	3509
	TCAGTTCTGGAGGTGCG	3510
	CGCACCTCCAGAACTGA	3511
Hypercholesterolaemia Cys68Arg gTGC-CGC	AAATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTG CATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGA CAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCC	3512
	GGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGC AGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGC GGTTGACACGGCCCCCACAGCTGAAGTCCCCGGATTT	3513
	TCTGGAGGTGCGATGGC	3514
	GCCATCGCACCTCCAGA	3515
Hypercholesterolaemia Cys68Trp TGCg-TGG	ATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCA TTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCT	3516
	AGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTC GCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCA GCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGAT	3517
	TGGAGGTGCGATGGCCA	3518
	TGGCCATCGCACCTCCA	3519
Hypercholesterolaemia Cys68Tyr TGC-TAC	AATCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGC ATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGAC AACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCC	3520
	GGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCG CAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAG CGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGATT	3521
	CTGGAGGTGCGATGGCC	3522
	GGCCATCGCACCTCCAG	3523

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Asp69Asn cGAT-AAT	TCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCAT TCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTG	3524
	CAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGT CGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGC AGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGA	3525
	GGAGGTGCGATGGCCAA	3526
	TTGGCCATCGCACCTCC	3527
Hypercholesterolaemia Asp69Gly GAT-GGT	CCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATT CCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAA CGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTGC	3528
	GCAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTT GTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAAT GCAGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGG	3529
	GAGGTGCGATGGCCAAG	3530
	CTTGGCCATCGCACCTC	3531
Hypercholesterolaemia Asp69Tyr cGAT-TAT	TCCGGGGACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCAT TCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACA ACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTG	3532
	CAGGGCCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGT CGCAGTCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGC AGCGGTTGACACGGCCCCCACAGCTGAAGTCCCCGGA	3533
	GGAGGTGCGATGGCCAA	3534
	TTGGCCATCGCACCTCC	3535
Hypercholesterolaemia Gln71Glu cCAA-GAA	GACTTCAGCTGTGGGGGCCGTGTCAACCGCTGCATTCCTCA GTTCTGGAGGTGCGATGGCCAAGTGGACTGCGACAACGGCT CAGACGAGCAAGGCTGTCGTAAGTGTGGCCCTGCCTTTG	3536
	CAAAGGCAGGGCCACACTTACGACAGCCTTGCTCGTCTGAG CCGTTGTCGCAGTCCACTTGGCCATCGCACCTCCAGAACTGA GGAATGCAGCGGTTGACACGGCCCCCACAGCTGAAGTC	3537
	GCGATGGCCAAGTGGAC	3538
	GTCCACTTGGCCATCGC	3539
Hypercholesterolaemia Cys74Gly cTGC-GGC	TGTGGGGGCCGTGTCAACCGCTGCATTCCTCAGTTCTGGAG GTGCGATGGCCAAGTGGACTGCGACAACGGCTCAGACGAGC AAGGCTGTCGTAAGTGTGGCCCTGCCTTTGCTATTGAGC	3540
	GCTCAATAGCAAAGGCAGGGCCACACTTACGACAGCCTTGCT CGTCTGAGCCGTTGTCGCAGTCCACTTGGCCATCGCACCTCC AGAACTGAGGAATGCAGCGGTTGACACGGCCCCCACA	3541
	AAGTGGACTGCGACAAC	3542
	GTTGTCGCAGTCCACTT	3543
Hypercholesterolaemia Ser78Term TCA-TGA	TCAACCGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAG TGGACTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAG TGTGGCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCT	3544

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCACACTTA CGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCCACTTGG CCATCGCACCTCCAGAACTGAGGAATGCAGCGGTTGA	3545
	CAACGGCTCAGACGAGC	3546
	GCTCGTCTGAGCCGTTG	3547
Hypercholesterolaemia Glu80Lys cGAG-AAG	CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTG GCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGA	3548
	TCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCA CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCG	3549
	GCTCAGACGAGCAAGGC	3550
	GCCTTGCTCGTCTGAGC	3551
Hypercholesterolaemia Glu80Term cGAG-TAG	CGCTGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGA CTGCGACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTG GCCCTGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGA	3552
	TCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGGCCA CACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAGTCC ACTTGGCCATCGCACCTCCAGAACTGAGGAATGCAGCG	3553
	GCTCAGACGAGCAAGGC	3554
	GCCTTGCTCGTCTGAGC	3555
Hypercholesterolaemia Gln81Term gCAA-TAA	TGCATTCCTCAGTTCTGGAGGTGCGATGGCCAAGTGGACTGC GACAACGGCTCAGACGAGCAAGGCTGTCGTAAGTGTGGCCC TGCCTTTGCTATTGAGCCTATCTGAGTCCTGGGGAGTG	3556
	CACTCCCCAGGACTCAGATAGGCTCAATAGCAAAGGCAGGG CCACACTTACGACAGCCTTGCTCGTCTGAGCCGTTGTCGCAG TCCACTTGGCCATCGCACCTCCAGAACTGAGGAATGCA	3557
	CAGACGAGCAAGGCTGT	3558
	ACAGCCTTGCTCGTCTG	3559
Hypercholesterolaemia Cys88Arg gTGC-CGC	TGGGAGACTTCACACGGTGATGGTGGTCTCGGCCCATCCATC CCTGCAGCCCCCAAGACGTGCTCCCAGGACGAGTTTCGCTG CCACGATGGGAAGTGCATCTCTCGGCAGTTCGTCTGTG	3560
	CACAGACGAACTGCCGAGAGATGCACTTCCCATCGTGGCAG CGAAACTCGTCCTGGGAGCACGTCTTGGGGGCTGCAGGGAT GGATGGGCCGAGACCACCATCACCGTGTGAAGTCTCCCA	3561
	CCAAGACGTGCTCCCAG	3562
	CTGGGAGCACGTCTTGG	3563
Hypercholesterolaemia Glu92Term cGAG-TAG	CACGGTGATGGTGGTCTCGGCCCATCCATCCCTGCAGCCCC CAAGACGTGCTCCCAGGACGAGTTTCGCTGCCACGATGGGA AGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGG	3564
	CCCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTTC CCATCGTGGCAGCGAAACTCGTCCTGGGAGCACGTCTTGGG GGCTGCAGGGATGGATGGGCCGAGACCACCATCACCGTG	3565
	CCCAGGACGAGTTTCGC	3566

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCGAAACTCGTCCTGGG	3567
Hypercholesterolaemia Cys95Arg cTGC-CGC	GGTGGTCTCGGCCCATCCATCCCTGCAGCCCCCAAGACGTG CTCCCAGGACGAGTTTCGCTGCCACGATGGGAAGTGCATCTC TCGGCAGTTCGTCTGTGACTCAGACCGGGACTGCTTGG	3568
	CCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGCCGAGAG ATGCACTTCCCATCGTGGCAGCGAAACTCGTCCTGGGAGCA CGTCTTGGGGGCTGCAGGGATGGATGGGCCGAGACCACC	3569
	AGTTTCGCTGCCACGAT	3570
	ATCGTGGCAGCGAAACT	3571
Hypercholesterolaemia Asp97Tyr cGAT-TAT	CTCGGCCCATCCATCCCTGCAGCCCCCAAGACGTGCTCCCA GGACGAGTTTCGCTGCCACGATGGGAAGTGCATCTCTCGGC AGTTCGTCTGTGACTCAGACCGGGACTGCTTGGACGGCT	3572
	AGCCGTCCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGC CGAGAGATGCACTTCCCATCGTGGCAGCGAAACTCGTCCTG GGAGCACGTCTTGGGGGCTGCAGGGATGGATGGGCCGAG	3573
	GCTGCCACGATGGGAAG	3574
	CTTCCCATCGTGGCAGC	3575
Hypercholesterolaemia Trp(-12)Arg cTGG-AGG	GGGTCGGGACACTGCCTGGCAGAGGCTGCGAGCATGGGGC CCTGGGGCTGGAAATTGCGCTGGACCGTCGCCTTGCTCCTC GCCGCGGCGGGGACTGCAGGTAAGGCTTGCTCCAGGCGCC	3576
	GGCGCCTGGAGCAAGCCTTACCTGCAGTCCCCGCCGCGGCG AGGAGCAAGGCGACGGTCCAGCGCAATTTCCAGCCCCAGGG CCCCATGCTCGCAGCCTCTGCCAGGCAGTGTCCCGACCC	3577
	AATTGCGCTGGACCGTC	3578
	GACGGTCCAGCGCAATT	3579
Hypercholesterolaemia Trp(-18)Term TGGg-TGA	CAGCAGGTCGTGATCCGGGTCGGGACACTGCCTGGCAGAGG CTGCGAGCATGGGGCCCTGGGGCTGGAAATTGCGCTGGACC GTCGCCTTGCTCCTCGCCGCGGCGGGGACTGCAGGTAAG	3580
	CTTACCTGCAGTCCCCGCCGCGGCGAGGAGCAAGGCGACG GTCCAGCGCAATTTCCAGCCCCAGGGCCCCATGCTCGCAGC CTCTGCCAGGCAGTGTCCCGACCCGGATCACGACCTGCTG	3581
	GGGCCCTGGGGGCTGGAA	3582
	TTCCAGCCCCAGGGCCC	3583
Hypercholesterolaemia Met(-21)Leu cATG-TTG	CAGCTAGGACACAGCAGGTCGTGATCCGGGTCGGGACACTG CCTGGCAGAGGCTGCGAGCATGGGGCCCTGGGGCTGGAAA TTGCGCTGGACCGTCGCCTTGCTCCTCGCCGCGGCGGGGA	3584
	TCCCCGCCGCGGCGAGGAGCAAGGCGACGGTCCAGCGCAA TTTCCAGCCCCAGGGCCCCATGCTCGCAGCCTCTGCCAGGC AGTGTCCCGACCCGGATCACGACCTGCTGTGTCCTAGCTG	3585
	CTGCGAGCATGGGGCCC	3586
	GGGCCCCATGCTCGCAG	3587

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Met(-21)Val cATG-GTG	CAGCTAGGACACAGCAGGTCGTGATCCGGGTCGGGACACTG CCTGGCAGAGGCTGCGAGCATGGGGCCCTGGGGCTGGAAA TTGCGCTGGACCGTCGCCTTGCTCCTCGCCGCGGCGGGGA	3588
	TCCCCGCCGCGGCGAGGAGCAAGGCGACGGTCCAGCGCAA TTTCCAGCCCCAGGGCCCCATGCTCGCAGCCTCTGCCAGGC AGTGTCCCGACCCGGATCACGACCTGCTGTGTCCTAGCTG	3589
	CTGCGAGCATGGGGCCC	3590
	GGGCCCCATGCTCGCAG	3591
Hypercholesterolaemia Ile101Phe cATC-TTC	ATCCCTGCAGCCCCCAAGACGTGCTCCCAGGACGAGTTTCG CTGCCACGATGGGAAGTGCATCTCTCGGCAGTTCGTCTGTGA CTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGCCT	3592
	AGGCCTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCA CAGACGAACTGCCGAGAGATGCACTTCCCATCGTGGCAGCG AAACTCGTCCTGGGAGCACGTCTTGGGGGCTGCAGGGAT	3593
	GGAAGTGCATCTCTCGG	3594
	CCGAGAGATGCACTTCC	3595
Hypercholesterolaemia Gln104Term gCAG-TAG	GCCCCCAAGACGTGCTCCCAGGACGAGTTTCGCTGCCACGA TGGGAAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCG GGACTGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGG	3596
	CCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTCCCGG TCTGAGTCACAGACGAACTGCCGAGAGATGCACTTCCCATCG TGGCAGCGAAACTCGTCCTGGGAGCACGTCTTGGGGGC	3597
	TCTCTCGGCAGTTCGTC	3598
	GACGAACTGCCGAGAGA	3599
Hypercholesterolaemia Cys113Arg cTGC-CGC	TTTCGCTGCCACGATGGGAAGTGCATCTCTCGGCAGTTCGTC TGTGACTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGC CTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTCC	3600
	GGAAGCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGC CTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCACAGA CGAACTGCCGAGAGATGCACTTCCCATCGTGGCAGCGAAA	3601
	ACCGGGACTGCTTGGAC	3602
	GTCCAAGCAGTCCCGGT	3603
Hypercholesterolaemia Glu119Lys cGAG-AAG	AAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGGAC TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCT	3604
	AGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGGTG AGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTC CCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTT	3605
	GCTCAGACGAGGCCTCC	3606
	GGAGGCCTCGTCTGAGC	3607
Hypercholesterolaemia Glu119Term cGAG-TAG	AAGTGCATCTCTCGGCAGTTCGTCTGTGACTCAGACCGGGAC TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCT	3608

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGGTG AGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCAGTC CCGGTCTGAGTCACAGACGAACTGCCGAGAGATGCACTT	3609
	GCTCAGACGAGGCCTCC	3610
	GGAGGCCTCGTCTGAGC	3611
Hypercholesterolaemia Cys122Term TGCc-TGA	TCGGCAGTTCGTCTGTGACTCAGACCGGGACTGCTTGGACG GCTCAGACGAGGCCTCCTGCCCGGTGCTCACCTGTGGTCCC GCCAGCTTCCAGTGCAACAGCTCCACCTGCATCCCCCAG	3612
	CTGGGGGATGCAGGTGGAGCTGTTGCACTGGAAGCTGGCGG GACCACAGGTGAGCACCGGGCAGGAGGCCTCGTCTGAGCC GTCCAAGCAGTCCCGGTCTGAGTCACAGACGAACTGCCGA	3613
	GCCTCCTGCCCGGTGCT	3614
	AGCACCGGGCAGGAGGC	3615
Hypercholesterolaemia Cys127Trp TGTg-TGG	TGACTCAGACCGGGACTGCTTGGACGGCTCAGACGAGGCCT CCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTCCAGTGC AACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGAC	3616
	GTCGCAGGCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGC ACTGGAAGCTGGCGGGACCACAGGTGAGCACCGGGCAGGA GGCCTCGTCTGAGCCGTCCAAGCAGTCCCGGTCTGAGTCA	3617
	CTCACCTGTGGTCCCGC	3618
	GCGGGACCACAGGTGAG	3619
Hypercholesterolaemia Gln133Term cCAG-TAG	TGCTTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCAC CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCAT CCCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCG	3620
	CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATG CAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAGGT GAGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAAGCA	3621
	CCAGCTTCCAGTGCAAC	3622
	GTTGCACTGGAAGCTGG	3623
Hypercholesterolaemia Cys134Gly gTGC-GGC	TTGGACGGCTCAGACGAGGCCTCCTGCCCGGTGCTCACCTG TGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATCCC CCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAG	3624
	CTTCGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGG ATGCAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA GGTGAGCACCGGGCAGGAGGCCTCGTCTGAGCCGTCCAA	3625
	GCTTCCAGTGCAACAGC	3626
	GCTGTTGCACTGGAAGC	3627
Hypercholesterolaemia Cys139Gly cTGC-GGC	GAGGCCTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTT CCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCT GCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGT	3628
	ACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAG GCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA GCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGCCTC	3629
	GCTCCACCTGCATCCCC	3630

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGGGATGCAGGTGGAGC	3631
Hypercholesterolaemia Cys139Tyr TGC-TAC	AGGCCTCCTGCCCGGTGCTCACCTGTGGTCCCGCCAGCTTC CAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTG CGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTG	3632
	CACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAG GCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA GCTGGCGGGACCACAGGTGAGCACCGGGCAGGAGGCCT	3633
	CTCCACCTGCATCCCCC	3634
	GGGGGATGCAGGTGGAG	3635
Hypercholesterolaemia Cys146Term TGCg-TGA	CTGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCAT CCCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAG ATGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTT	3636
	AAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTTC GCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATGC AGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACAG	3637
	TGGGCCTGCGACAACGA	3638
	TCGTTGTCGCAGGCCCA	3639
Hypercholesterolaemia Asp147Asn cGAC-AAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3640
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATG CAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3641
	GGGCCTGCGACAACGAC	3642
	GTCGTTGTCGCAGGCCC	3643
Hypercholesterolaemia Asp147His cGAC-CAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3644
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATG CAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3645
	GGGCCTGCGACAACGAC	3646
	GTCGTTGTCGCAGGCCC	3647
Hypercholesterolaemia Asp147Tyr cGAC-TAC	TGTGGTCCCGCCAGCTTCCAGTGCAACAGCTCCACCTGCATC CCCCAGCTGTGGGCCTGCGACAACGACCCCGACTGCGAAGA TGGCTCGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTT	3648
	AAAGACCCCTACAGCGCTGCGGCCACTCATCCGAGCCATCTT CGCAGTCGGGGTCGTTGTCGCAGGCCCACAGCTGGGGGATG CAGGTGGAGCTGTTGCACTGGAAGCTGGCGGGACCACA	3649
	GGGCCTGCGACAACGAC	3650
	GTCGTTGTCGCAGGCCC	3651

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Cys152Arg cTGC-CGC	TTCCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCC TGCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTG GCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGG	3652
	CCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCCAC TCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGC CCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA	3653
	ACCCCGACTGCGAAGAT	3654
	ATCTTCGCAGTCGGGGT	3655
Hypercholesterolaemia Cys152Gly cTGC-GGC	TTCCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCC TGCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTG GCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGG	3656
	CCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCCAC TCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGC CCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGGAA	3657
	ACCCCGACTGCGAAGAT	3658
	ATCTTCGCAGTCGGGGT	3659
Hypercholesterolaemia Cys152Trp TGCg-TGG	CCAGTGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCT GCGACAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGG CCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGAC	3660
	GTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCGGCC ACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCGCAG GCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCACTGG	3661
	CCCGACTGCGAAGATGG	3662
	CCATCTTCGCAGTCGGG	3663
Hypercholesterolaemia Asp154Asn aGAT-AAT	TGCAACAGCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGA CAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGGCCGC AGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGACAGTA	3664
	TACTGTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCG GCCACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTGTCG CAGGCCCACAGCTGGGGGATGCAGGTGGAGCTGTTGCA	3665
	ACTGCGAAGATGGCTCG	3666
	CGAGCCATCTTCGCAGT	3667
Hypercholesterolaemia Ser156Leu TCG-TTG	GCTCCACCTGCATCCCCCAGCTGTGGGCCTGCGACAACGAC CCCGACTGCGAAGATGGCTCGGATGAGTGGCCGCAGCGCTG TAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGCCCCTG	3668
	CAGGGGCTACTGTCCCCTTGGAACACGTAAAGACCCCTACAG CGCTGCGGCCACTCATCCGAGCCATCTTCGCAGTCGGGGTC GTTGTCGCAGGCCCACAGCTGGGGGATGCAGGTGGAGC	3669
	AGATGGCTCGGATGAGT	3670
	ACTCATCCGAGCCATCT	3671
Hypercholesterolaemia Cys163Tyr TGT-TAT	TGTGGGCCTGCGACAACGACCCCGACTGCGAAGATGGCTCG GATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAA GGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG	3672

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CAGTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTG GAACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG AGCCATCTTCGCAGTCGGGGTCGTTGTCGCAGGCCCACA	3673
	GCAGCGCTGTAGGGGTC	3674
	GACCCCTACAGCGCTGC	3675
Hypercholesterolaemia Tyr167Term TACg-TAG	CAACGACCCCGACTGCGAAGATGGCTCGGATGAGTGGCCGC AGCGCTGTAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGC CCCTGCTCGGCCTTCGAGTTCCACTGCCTAAGTGGCGAG	3676
	CTCGCCACTTAGGCAGTGGAACTCGAAGGCCGAGCAGGGGC TACTGTCCCCTTGGAACACGTAAAGACCCCTACAGCGCTGCG GCCACTCATCCGAGCCATCTTCGCAGTCGGGGTCGTTG	3677
	GGTCTTTACGTGTTCCA	3678
	TGGAACACGTAAAGACC	3679
Hypercholesterolaemia Gln170Term cCAA-TAA	CCCGACTGCGAAGATGGCTCGGATGAGTGGCCGCAGCGCTG TAGGGGTCTTTACGTGTTCCAAGGGGACAGTAGCCCCTGCTC GGCCTTCGAGTTCCACTGCCTAAGTGGCGAGTGCATCC	3680
	GGATGCACTCGCCACTTAGGCAGTGGAACTCGAAGGCCGAG CAGGGGCTACTGTCCCCTTGGAACACGTAAAGACCCCTACAG CGCTGCGGCCACTCATCCGAGCCATCTTCGCAGTCGGG	3681
	ACGTGTTCCAAGGGGAC	3682
	GTCCCCTTGGAACACGT	3683
Hypercholesterolaemia Cys176Phe TGC-TTC	CGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTC CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGA	3684
	TCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA GTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTGGA ACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG	3685
	TAGCCCCTGCTCGGCCT	3686
	AGGCCGAGCAGGGGCTA	3687
Hypercholesterolaemia Cys176Tyr TGC-TAC	CGGATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTC CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGA	3688
	TCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA GTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTGGA ACACGTAAAGACCCCTACAGCGCTGCGGCCACTCATCCG	3689
	TAGCCCCTGCTCGGCCT	3690
	AGGCCGAGCAGGGGCTA	3691
Hypercholesterolaemia Ser177Leu TCG-TTG	ATGAGTGGCCGCAGCGCTGTAGGGGTCTTTACGTGTTCCAAG GGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTGCCTAA GTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATGG	3692
	CCATCACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAG GCAGTGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTT GGAACACGTAAAGACCCCTACAGCGCTGCGGCCACTCAT	3693
	CCCCTGCTCGGCCTTCG	3694

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CGAAGGCCGAGCAGGGG	3695
Hypercholesterolaemia Glu187Lys cGAG-AAG	TACGTGTTCCAAGGGGACAGTAGCCCCTGCTCGGCCTTCGA GTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGC GCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACG	3696
	CGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCC AGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTCG AAGGCCGAGCAGGGGCTACTGTCCCCTTGGAACACGTA	3697
	TAAGTGGCGAGTGCATC	3698
	GATGCACTCGCCACTTA	3699
Hypercholesterolaemia His190Tyr cCAC-TAC	CAAGGGGACAGTAGCCCCTGCTCGGCCTTCGAGTTCCACTG CCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATG GTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACT	3700
	AGTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCAT CACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCAG TGGAACTCGAAGGCCGAGCAGGGGCTACTGTCCCCTTG	3701
	AGTGCATCCACTCCAGC	3702
	GCTGGAGTGGATGCACT	3703
Hypercholesterolaemia Gly198Asp GGC-GAC	CCTTCGAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCA GCTGGCGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCT GACGAGGAAAACTGCGGTATGGGCGGGGCCAGGGTGGG	3704
	CCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCGTCAGAT TTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCAGCTGGA GTGGATGCACTCGCCACTTAGGCAGTGGAACTCGAAGG	3705
	TGATGGTGGCCCCGACT	3706
	AGTCGGGGCCACCATCA	3707
Hypercholesterolaemia Asp200Asn cGAC-AAC	GAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGG CGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGA GGAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGG	3708
	CCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCGT CAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCAG CTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTC	3709
	GTGGCCCCGACTGCAAG	3710
	CTTGCAGTCGGGGCCAC	3711
Hypercholesterolaemia Asp200Gly GAC-GGC	AGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGC GCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAG GAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGG	3712
	CCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTC GTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCA GCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACT	3713
	TGGCCCCGACTGCAAGG	3714
	CCTTGCAGTCGGGGCCA	3715

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Asp200Tyr cGAC-TAC	GAGTTCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGG CGCTGTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGA GGAAAACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGG	3716
	CCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTCCTCGT CAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCGCCAG CTGGAGTGGATGCACTCGCCACTTAGGCAGTGGAACTC	3717
	GTGGCCCCGACTGCAAG	3718
	CTTGCAGTCGGGGCCAC	3719
Hypercholesterolaemia Cys201Term TGCa-TGA	CCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCT GTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAA AACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGT	3720
	ACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTT CCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGC GCCAGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGG	3721
	CCCGACTGCAAGGACAA	3722
	TTGTCCTTGCAGTCGGG	3723
Hypercholesterolaemia Cys201Tyr TGC-TAC	TCCACTGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCT GTGATGGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAA AACTGCGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCG	3724
	CGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCAGTTTTC CTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCACAGCG CCAGCTGGAGTGGATGCACTCGCCACTTAGGCAGTGGA	3725
	CCCCGACTGCAAGGACA	3726
	TGTCCTTGCAGTCGGGG	3727
Hypercholesterolaemia Asp203Asn gGAC-AAC	TGCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGAT GGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACTG CGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTA	3728
	TAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCAG TTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATCA CAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGCA	3729
	ACTGCAAGGACAAATCT	3730
	AGATTTGTCCTTGCAGT	3731
Hypercholesterolaemia Asp203Gly GAC-GGC	GCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGAT GGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACTG CGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTAT	3732
	ATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCA GTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATC ACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGC	3733
	CTGCAAGGACAAATCTG	3734
	CAGATTTGTCCTTGCAG	3735
Hypercholesterolaemia Asp203Val GAC-GTC	GCCTAAGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGAT GGTGGCCCCGACTGCAAGGACAAATCTGACGAGGAAAACTG CGGTATGGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTAT	3736

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATACCGCA GTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCACCATC ACAGCGCCAGCTGGAGTGGATGCACTCGCCACTTAGGC	3737
	CTGCAAGGACAAATCTG	3738
	CAGATTTGTCCTTGCAG	3739
Hypercholesterolaemia Ser205Pro aTCT-CCT	AGTGGCGAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGG CCCCGACTGCAAGGACAAATCTGACGAGGAAAACTGCGGTAT GGGCGGGGCCAGGGTGGGGGCGGGGCGTCCTATCACCT	3740
	AGGTGATAGGACGCCCCGCCCCCACCCTGGCCCCGCCCATA CCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTCGGGGCCA CCATCACAGCGCCAGCTGGAGTGGATGCACTCGCCACT	3741
	AGGACAAATCTGACGAG	3742
	CTCGTCAGATTTGTCCT	3743
Hypercholesterolaemia Asp206Glu GACg-GAG	CGAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCG ACTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGC GGGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCC	3744
	GGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCCG CCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTCG GGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTCG	3745
	AAATCTGACGAGGAAAA	3746
	TTTTCCTCGTCAGATTT	3747
Hypercholesterolaemia Glu207Gln cGAG-CAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3748
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3749
	AATCTGACGAGGAAAAC	3750
	GTTTTCCTCGTCAGATT	3751
Hypercholesterolaemia Glu207Lys cGAG-AAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3752
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3753
	AATCTGACGAGGAAAAC	3754
	GTTTTCCTCGTCAGATT	3755
Hypercholesterolaemia Glu207Term cGAG-TAG	GAGTGCATCCACTCCAGCTGGCGCTGTGATGGTGGCCCCGA CTGCAAGGACAAATCTGACGAGGAAAACTGCGGTATGGGCG GGGCCAGGGTGGGGGCGGGGCGTCCTATCACCTGTCCCT	3756

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGGGACAGGTGATAGGACGCCCCGCCCCCACCCTGGCCCC GCCCATACCGCAGTTTTCCTCGTCAGATTTGTCCTTGCAGTC GGGGCCACCATCACAGCGCCAGCTGGAGTGGATGCACTC	3757
	AATCTGACGAGGAAAAC	3758
	GTTTTCCTCGTCAGATT	3759
Hypercholesterolaemia Glu219Lys cGAA-AAA	TCTTGAGAAAATCAACACACTCTGTCCTGTTTTCCAGCTGTGG CCACCTGTCGCCCTGACGAATTCCAGTGCTCTGATGGAAACT GCATCCATGGCAGCCGGCAGTGTGACCGGGAATATG	3760
	CATATTCCCGGTCACACTGCCGGCTGCCATGGATGCAGTTTC CATCAGAGCACTGGAATTCGTCAGGGCGACAGGTGGCCACA GCTGGAAAACAGGACAGAGTGTGTTGATTTTCTCAAGA	3761
	GCCCTGACGAATTCCAG	3762
	CTGGAATTCGTCAGGGC	3763
Hypercholesterolaemia Gln221Term cCAG-TAG	GAAAATCAACACACTCTGTCCTGTTTTCCAGCTGTGGCCACCT GTCGCCCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCC ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCA	3764
	TGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGATGC AGTTTCCATCAGAGCACTGGAATTCGTCAGGGCGACAGGTG GCCACAGCTGGAAAACAGGACAGAGTGTGTTGATTTTC	3765
	ACGAATTCCAGTGCTCT	3766
	AGAGCACTGGAATTCGT	3767
Hypercholesterolaemia Cys227Phe TGC-TTC	CCTGTTTTCCAGCTGTGGCCACCTGTCGCCCTGACGAATTCC AGTGCTCTGATGGAAACTGCATCCATGGCAGCCGGCAGTGT GACCGGGAATATGACTGCAAGGACATGAGCGATGAAGT	3768
	ACTTCATCGCTCATGTCCTTGCAGTCATATTCCCGGTCACACT GCCGGCTGCCATGGATGCAGTTTCCATCAGAGCACTGGAATT CGTCAGGGCGACAGGTGGCCACAGCTGGAAAACAGG	3769
	TGGAAACTGCATCCATG	3770
	CATGGATGCAGTTTCCA	3771
Hypercholesterolaemia Asp235Glu GACc-GAA	TCGCCCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCCA TGGCAGCCGGCAGTGTGACCGGGAATATGACTGCAAGGACA TGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGG	3772
	CCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTC CTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGAT GCAGTTTCCATCAGAGCACTGGAATTCGTCAGGGCGA	3773
	CAGTGTGACCGGGAATA	3774
	TATTCCCGGTCACACTG	3775
Hypercholesterolaemia Asp235Gly GAC-GGC	GTCGCCCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCC ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCAAGGAC ATGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTG	3776
	CAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTCC TTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCATGGATG CAGTTTCCATCAGAGCACTGGAATTCGTCAGGGCGAC	3777

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GCAGTGTGACCGGGAAT	3778
	ATTCCCGGTCACACTGC	3779
Hypercholesterolaemia Glu237Lys gGAA-AAA	CCTGACGAATTCCAGTGCTCTGATGGAAACTGCATCCATGGC AGCCGGCAGTGTGACCGGGAATATGACTGCAAGGACATGAG CGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGGCCAT	3780
	ATGGCCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCA TGTCCTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCAT GGATGCAGTTTCCATCAGAGCACTGGAATTCGTCAGG	3781
	GTGACCGGGAATATGAC	3782
	GTCATATTCCCGGTCAC	3783
Hypercholesterolaemia Cys240Phe TGC-TTC	TCCAGTGCTCTGATGGAAACTGCATCCATGGCAGCCGGCAGT GTGACCGGGAATATGACTGCAAGGACATGAGCGATGAAGTT GGCTGCGTTAATGGTGAGCGCTGGCCATCTGGTTTTCC	3784
	GGAAAACCAGATGGCCAGCGCTCACCATTAACGCAGCCAACT TCATCGCTCATGTCCTTGCAGTCATATTCCCGGTCACACTGCC GGCTGCCATGGATGCAGTTTCCATCAGAGCACTGGA	3785
	ATATGACTGCAAGGACA	3786
	TGTCCTTGCAGTCATAT	3787
Hypercholesterolaemia Asp245Glu GATg-GAA	AAACTGCATCCATGGCAGCCGGCAGTGTGACCGGGAATATG ACTGCAAGGACATGAGCGATGAAGTTGGCTGCGTTAATGGTG AGCGCTGGCCATCTGGTTTTCCATCCCCCATTCTCTGT	3788
	ACAGAGAATGGGGGATGGAAAACCAGATGGCCAGCGCTCAC CATTAACGCAGCCAACTTCATCGCTCATGTCCTTGCAGTCATA TTCCCGGTCACACTGCCGGCTGCCATGGATGCAGTTT	3789
	ATGAGCGATGAAGTTGG	3790
	CCAACTTCATCGCTCAT	3791
Hypercholesterolaemia Cys249Tyr TGC-TAC	ATGGCAGCCGGCAGTGTGACCGGGAATATGACTGCAAGGAC ATGAGCGATGAAGTTGGCTGCGTTAATGGTGAGCGCTGGCC ATCTGGTTTTCCATCCCCCATTCTCTGTGCCTTGCTGCT	3792
	AGCAGCAAGGCACAGAGAATGGGGGATGGAAAACCAGATGG CCAGCGCTCACCATTAACGCAGCCAACTTCATCGCTCATGTC CTTGCAGTCATATTCCCGGTCACACTGCCGGCTGCCAT	3793
	AGTTGGCTGCGTTAATG	3794
	CATTAACGCAGCCAACT	3795
Hypercholesterolaemia Glu256Lys cGAG-AAG	AGGCTCAGACACACCTGACCTTCCTCCTTCCTCTCTCTGGCT CTCACAGTGACACTCTGCGAGGGACCCAACAAGTTCAAGTGT CACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCA	3796
	TGCAGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACACT TGAACTTGTTGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCA GAGAGAGGAAGGAGGAAGGTCAGGTGTGTCTGAGCCT	3797
	CACTCTGCGAGGGACCC	3798
	GGGTCCCTCGCAGAGTG	3799

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Ser265Arg AGCg-AGA	CCTCTCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAA CAAGTTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAA AGTCTGCAACATGGCTAGAGACTGCCGGGACTGGTCA	3800
	TGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTTTGTC CAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGTTGGG TCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGAGAGG	3801
	TGTCACAGCGGCGAATG	3802
	CATTCGCCGCTGTGACA	3803
Hypercholesterolaemia Glu267Lys cGAA-AAA	TCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAACAAG TTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTC TGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATG	3804
	CATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTT TGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGT TGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGA	3805
	ACAGCGGCGAATGCATC	3806
	GATGCATTCGCCGCTGT	3807
Hypercholesterolaemia Glu267Term cGAA-TAA	TCTCTGGCTCTCACAGTGACACTCTGCGAGGGACCCAACAAG TTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTC TGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATG	3808
	CATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTT TGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACTTGT TGGGTCCCTCGCAGAGTGTCACTGTGAGAGCCAGAGA	3809
	ACAGCGGCGAATGCATC	3810
	GATGCATTCGCCGCTGT	3811
Hypercholesterolaemia Lys273Glu cAAA-GAA	ACACTCTGCGAGGGACCCAACAAGTTCAAGTGTCACAGCGG CGAATGCATCACCCTGGACAAAGTCTGCAACATGGCTAGAGA CTGCCGGGACTGGTCAGATGAACCCATCAAAGAGTGCG	3812
	CGCACTCTTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTC TAGCCATGTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGC TGTGACACTTGAACTTGTTGGGTCCCTCGCAGAGTGT	3813
	CCCTGGACAAAGTCTGC	3814
	GCAGACTTTGTCCAGGG	3815
Hypercholesterolaemia Cys275Term TGCa-TGA	CGAGGGACCCAACAAGTTCAAGTGTCACAGCGGCGAATGCA TCACCCTGGACAAAGTCTGCAACATGGCTAGAGACTGCCGG GACTGGTCAGATGAACCCATCAAAGAGTGCGGTGAGTCT	3816
	AGACTCACCGCACTCTTTGATGGGTTCATCTGACCAGTCCCG GCAGTCTCTAGCCATGTTGCAGACTTTGTCCAGGGTGATGCA TTCGCCGCTGTGACACTTGAACTTGTTGGGTCCCTCG	3817
	AAAGTCTGCAACATGGC	3818
	GCCATGTTGCAGACTTT	3819
Hypercholesterolaemia Asp280Gly GAC-GGC	AGTTCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAG TCTGCAACATGGCTAGAGACTGCCGGGACTGGTCAGATGAA CCCATCAAAGAGTGCGGTGAGTCTCGGTGCAGGCGGCT	3820

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AGCCGCCTGCACCGAGACTCACCGCACTCTTTGATGGGTTCA TCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGACTTTG TCCAGGGTGATGCATTCGCCGCTGTGACACTTGAACT	3821
	GGCTAGAGACTGCCGGG	3822
	CCCGGCAGTCTCTAGCC	3823
Hypercholesterolaemia Cys281Tyr TGC-TAC	TCAAGTGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCT GCAACATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCC ATCAAAGAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGC	3824
	GCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGATGGG TTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGCAGAC TTTGTCCAGGGTGATGCATTCGCCGCTGTGACACTTGA	3825
	TAGAGACTGCCGGGACT	3826
	AGTCCCGGCAGTCTCTA	3827
Hypercholesterolaemia Asp283Asn gGAC-AAC	TGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAAC ATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAA GAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGT	3828
	ACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGA TGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGC AGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACA	3829
	ACTGCCGGGACTGGTCA	3830
	TGACCAGTCCCGGCAGT	3831
Hypercholesterolaemia Asp283Glu GACt-GAG	TCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAACAT GGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAAG AGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTT	3832
	AAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTT GATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTT GCAGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGA	3833
	TGCCGGGACTGGTCAGA	3834
	TCTGACCAGTCCCGGCA	3835
Hypercholesterolaemia Asp283Tyr gGAC-TAC	TGTCACAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAAC ATGGCTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAA GAGTGCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGT	3836
	ACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTCTTTGA TGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCATGTTGC AGACTTTGTCCAGGGTGATGCATTCGCCGCTGTGACA	3837
	ACTGCCGGGACTGGTCA	3838
	TGACCAGTCCCGGCAGT	3839
Hypercholesterolaemia Trp284Term TGGt-TGA	CAGCGGCGAATGCATCACCCTGGACAAAGTCTGCAACATGG CTAGAGACTGCCGGGACTGGTCAGATGAACCCATCAAAGAGT GCGGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTTGTG	3840
	CACAAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCACTC TTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGCCAT GTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGCTG	3841
	CGGGACTGGTCAGATGA	3842

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCATCTGACCAGTCCCG	3843
Hypercholesterolaemia Ser285Leu TCA-TTA	GCGGCGAATGCATCACCCTGGACAAAGTCTGCAACATGGCTA GAGACTGCCGGGACTGGTCAGATGAACCCATCAAAGAGTGC GGTGAGTCTCGGTGCAGGCGGCTTGCAGAGTTTGTGGG	3844
	CCCACAAACTCTGCAAGCCGCCTGCACCGAGACTCACCGCA CTCTTTGATGGGTTCATCTGACCAGTCCCGGCAGTCTCTAGC CATGTTGCAGACTTTGTCCAGGGTGATGCATTCGCCGC	3845
	GGACTGGTCAGATGAAC	3846
	GTTCATCTGACCAGTCC	3847
Hypercholesterolaemia Lys290Arg AAA-AGA	CCCTGGACAAAGTCTGCAACATGGCTAGAGACTGCCGGGAC TGGTCAGATGAACCCATCAAAGAGTGCGGTGAGTCTCGGTG CAGGCGGCTTGCAGAGTTTGTGGGGAGCCAGGAAAGGGA	3848
	TCCCTTTCCTGGCTCCCCACAAACTCTGCAAGCCGCCTGCAC CGAGACTCACCGCACTCTTTGATGGGTTCATCTGACCAGTCC CGGCAGTCTCTAGCCATGTTGCAGACTTTGTCCAGGG	3849
	ACCCATCAAAGAGTGCG	3850
	CGCACTCTTTGATGGGT	3851
Hypercholesterolaemia Cys297Phe TGC-TTC	GGGTAGGGGCCCGAGAGTGACCAGTCTGCATCCCCTGGCCC TGCGCAGGGACCAACGAATGCTTGGACAACAACGGCGGCTG TTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTG	3852
	CACTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACAG CCGCCGTTGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGC CAGGGGATGCAGACTGGTCACTCTCGGGCCCCTACCC	3853
	CAACGAATGCTTGGACA	3854
	TGTCCAAGCATTCGTTG	3855
Hypercholesterolaemia Cys297Tyr TGC-TAC	GGGTAGGGGCCCGAGAGTGACCAGTCTGCATCCCCTGGCCC TGCGCAGGGACCAACGAATGCTTGGACAACAACGGCGGCTG TTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTG	3856
	CACTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACAG CCGCCGTTGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGC CAGGGGATGCAGACTGGTCACTCTCGGGCCCCTACCC	3857
	CAACGAATGCTTGGACA	3858
	TGTCCAAGCATTCGTTG	3859
Hypercholesterolaemia His306Tyr cCAC-TAC	TGCATCCCCTGGCCCTGCGCAGGGACCAACGAATGCTTGGA CAACAACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGAT CGGCTACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGG	3860
	CCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCGATC TTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTTGTTGTCC AAGCATTCGTTGGTCCCTGCGCAGGGCCAGGGGATGCA	3861
	GCTGTTCCCACGTCTGC	3862
	GCAGACGTGGGAACAGC	3863

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Cys308Gly cTGC-GGC	CCCTGGCCCTGCGCAGGGACCAACGAATGCTTGGACAACAA CGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTA CGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCC	3864
	GGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGT TGTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGCCAGGG	3865
	CCCACGTCTGCAATGAC	3866
	GTCATTGCAGACGTGGG	3867
Hypercholesterolaemia Cys308Tyr TGC-TAC	CCTGGCCCTGCGCAGGGACCAACGAATGCTTGGACAACAAC GGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTAC GAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCA	3868
	TGGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTT GTTGTCCAAGCATTCGTTGGTCCCTGCGCAGGGCCAGG	3869
	CCACGTCTGCAATGACC	3870
	GGTCATTGCAGACGTGG	3871
Hypercholesterolaemia Gly314Ser cGGC-AGC	ACCAACGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTC TGCAATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGAC GGCTTCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTG	3872
	CACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCCGTCG GGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCAGAC GTGGGAACAGCCGCCGTTGTTGTCCAAGCATTCGTTGGT	3873
	TTAAGATCGGCTACGAG	3874
	CTCGTAGCCGATCTTAA	3875
Hypercholesterolaemia Gly314Val GGC-GTC	CCAACGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTCT GCAATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGAC GGCTTCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGA	3876
	TCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCCGTCG GGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCAGACG TGGGAACAGCCGCCGTTGTTGTCCAAGCATTCGTTGG	3877
	TAAGATCGGCTACGAGT	3878
	ACTCGTAGCCGATCTTA	3879
Hypercholesterolaemia Tyr315Term TACg-TAA	CGAATGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAA TGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTT CCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTC	3880
	GAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCC GTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCATTGCA GACGTGGGAACAGCCGCCGTTGTTGTCCAAGCATTCG	3881
	ATCGGCTACGAGTGCCT	3882
	AGGCACTCGTAGCCGAT	3883
Hypercholesterolaemia Cys317Gly gTGC-GGC	TGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAATGAC CTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTTCCA GCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCCGGG	3884

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCCGGAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGA AGCCGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCA TTGCAGACGTGGGAACAGCCGCCGTTGTTGTCCAAGCA	3885
	GCTACGAGTGCCTGTGC	3886
	GCACAGGCACTCGTAGC	3887
Hypercholesterolaemia Cys317Ser gTGC-AGC	TGCTTGGACAACAACGGCGGCTGTTCCCACGTCTGCAATGAC CTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCTTCCA GCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCCGGG	3888
	CCCGGAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGA AGCCGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCA TTGCAGACGTGGGAACAGCCGCCGTTGTTGTCCAAGCA	3889
	GCTACGAGTGCCTGTGC	3890
	GCACAGGCACTCGTAGC	3891
Hypercholesterolaemia Pro320Arg CCC-CGC	ACAACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCG GCTACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCC CAGCGAAGATGCGAAGGTGATTTCCGGGTGGGACTGAG	3892
	CTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTGGGCC ACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCGAT CTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTTGT	3893
	CCTGTGCCCCGACGGCT	3894
	AGCCGTCGGGGCACAGG	3895
Hypercholesterolaemia Asp321Asn cGAC-AAC	AACGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGC TACGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCA GCGAAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCC	3896
	GGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTGGG CCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGCCG ATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCGTT	3897
	TGTGCCCCGACGGCTTC	3898
	GAAGCCGTCGGGGCACA	3899
Hypercholesterolaemia Asp321Glu GACg-GAG	CGGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTA CGAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCAGC GAAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCCCT	3900
	AGGGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCTG GGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTAGC CGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCCG	3901
	TGCCCCGACGGCTTCCA	3902
	TGGAAGCCGTCGGGGCA	3903
Hypercholesterolaemia Gly322Ser cGGC-AGC	GGCGGCTGTTCCCACGTCTGCAATGACCTTAAGATCGGCTAC GAGTGCCTGTGCCCCGACGGCTTCCAGCTGGTGGCCCAGCG AAGATGCGAAGGTGATTTCCGGGTGGGACTGAGCCCTG	3904
	CAGGGCTCAGTCCCACCCGGAAATCACCTTCGCATCTTCGCT GGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCACTCGTA GCCGATCTTAAGGTCATTGCAGACGTGGGAACAGCCGCC	3905
	GCCCCGACGGCTTCCAG	3906

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CTGGAAGCCGTCGGGGC	3907
Hypercholesterolaemia Gln324Term cCAG-TAG	TGTTCCCACGTCTGCAATGACCTTAAGATCGGCTACGAGTGC CTGTGCCCCGACGGCTTCCAGCTGGTGGCCCAGCGAAGATG CGAAGGTGATTTCCGGGTGGGACTGAGCCCTGGGCCCC	3908
	GGGGCCCAGGGCTCAGTCCCACCCGGAAATCACCTTCGCAT CTTCGCTGGGCCACCAGCTGGAAGCCGTCGGGGCACAGGCA CTCGTAGCCGATCTTAAGGTCATTGCAGACGTGGGAACA	3909
	ACGGCTTCCAGCTGGTG	3910
	CACCAGCTGGAAGCCGT	3911
Hypercholesterolaemia Arg329Pro CGA-CCA	ATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGGCT TCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCCGG GTGGGACTGAGCCCTGGGCCCCCTCTGCGCTTCCTGAC	3912
	GTCAGGAAGCGCAGAGGGGGCCCAGGGCTCAGTCCCACCC GGAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGC CGTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCAT	3913
	GGCCCAGCGAAGATGCG	3914
	CGCATCTTCGCTGGGCC	3915
Hypercholesterolaemia Arg329Term gCGA-TGA	AATGACCTTAAGATCGGCTACGAGTGCCTGTGCCCCGACGG CTTCCAGCTGGTGGCCCAGCGAAGATGCGAAGGTGATTTCC GGGTGGGACTGAGCCCTGGGCCCCCTCTGCGCTTCCTGA	3916
	TCAGGAAGCGCAGAGGGGGCCCAGGGCTCAGTCCCACCCG GAAATCACCTTCGCATCTTCGCTGGGCCACCAGCTGGAAGCC GTCGGGGCACAGGCACTCGTAGCCGATCTTAAGGTCATT	3917
	TGGCCCAGCGAAGATGC	3918
	GCATCTTCGCTGGGCCA	3919
Hypercholesterolaemia Glu336Lys tGAG-AAG	TCTAGCCATTGGGGAAGAGCCTCCCCACCAAGCCTCTTTCTC TCTCTTCCAGATATCGATGAGTGTCAGGATCCCGACACCTGC AGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACAAGT	3920
	ACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGGCTGCAG GTGTCGGGATCCTGACACTCATCGATATCTGGAAGAGAGAGA AAGAGGCTTGGTGGGGAGGCTCTTCCCCAATGGCTAGA	3921
	ATATCGATGAGTGTCAG	3922
	CTGACACTCATCGATAT	3923
Hypercholesterolaemia Gln338Term tCAG-TAG	CATTGGGGAAGAGCCTCCCCACCAAGCCTCTTTCTCTCTCTT CCAGATATCGATGAGTGTCAGGATCCCGACACCTGCAGCCAG CTCTGCGTGAACCTGGAGGGTGGCTACAAGTGCCAGT	3924
	ACTGGCACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGG CTGCAGGTGTCGGGATCCTGACACTCATCGATATCTGGAAGA GAGAGAAAGAGGCTTGGTGGGGAGGCTCTTCCCCAATG	3925
	ATGAGTGTCAGGATCCC	3926
	GGGATCCTGACACTCAT	3927

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Hypercholesterolaemia Cys343Arg cTGC-CGC	TCCCCACCAAGCCTCTTTCTCTCTCTTCCAGATATCGATGAGT GTCAGGATCCCGACACCTGCAGCCAGCTCTGCGTGAACCTG GAGGGTGGCTACAAGTGCCAGTGTGAGGAAGGCTTCC	3928
	GGAAGCCTTCCTCACACTGGCACTTGTAGCCACCCTCCAGGT TCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTGACACTCA TCGATATCTGGAAGAGAGAGAAAGAGGCTTGGTGGGGA	3929
	CCGACACCTGCAGCCAG	3930
	CTGGCTGCAGGTGTCGG	3931
Hypercholesterolaemia Gln345Arg CAG-CGG	CAAGCCTCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGA TCCCGACACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTG GCTACAAGTGCCAGTGTGAGGAAGGCTTCCAGCTGGA	3932
	TCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTAGCCACCC TCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTG ACACTCATCGATATCTGGAAGAGAGAGAAAGAGGCTTG	3933
	CTGCAGCCAGCTCTGCG	3934
	CGCAGAGCTGGCTGCAG	3935
Hypercholesterolaemia Cys347Tyr TGC-TAC	TCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGATCCCGA CACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACA AGTGCCAGTGTGAGGAAGGCTTCCAGCTGGACCCCCA	3936
	TGGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTAG CCACCCTCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGGG ATCCTGACACTCATCGATATCTGGAAGAGAGAGAAAGA	3937
	CCAGCTCTGCGTGAACC	3938
	GGTTCACGCAGAGCTGG	3939
Hypercholesterolaemia Cys347Arg cTGC-CGC	CTCTTTCTCTCTCTTCCAGATATCGATGAGTGTCAGGATCCCG ACACCTGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTAC AAGTGCCAGTGTGAGGAAGGCTTCCAGCTGGACCCCC	3940
	GGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCACTTGTAG CCACCCTCCAGGTTCACGCAGAGCTGGCTGCAGGTGTCGGG ATCCTGACACTCATCGATATCTGGAAGAGAGAGAAAGAG	3941
	GCCAGCTCTGCGTGAAC	3942
	GTTCACGCAGAGCTGGC	3943
Hypercholesterolaemia Gly352Asp GGT-GAT	CAGATATCGATGAGTGTCAGGATCCCGACACCTGCAGCCAGC TCTGCGTGAACCTGGAGGGTGGCTACAAGTGCCAGTGTGAG GAAGGCTTCCAGCTGGACCCCCACACGAAGGCCTGCAA	3944
	TTGCAGGCCTTCGTGTGGGGGTCCAGCTGGAAGCCTTCCTC ACACTGGCACTTGTAGCCACCCTCCAGGTTCACGCAGAGCTG GCTGCAGGTGTCGGGATCCTGACACTCATCGATATCTG	3945
	CCTGGAGGGTGGCTACA	3946
	TGTAGCCACCCTCCAGG	3947
Hypercholesterolaemia Tyr354Cys TAC-TGC	TCGATGAGTGTCAGGATCCCGACACCTGCAGCCAGCTCTGC GTGAACCTGGAGGGTGGCTACAAGTGCCAGTGTGAGGAAGG CTTCCAGCTGGACCCCCACACGAAGGCCTGCAAGGCTGT	3948

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ACAGCCTTGCAGGCCTTCGTGTGGGGGTCCAGCTGGAAGCC TTCCTCACACTGGCACTTGTAGCCACCCTCCAGGTTCACGCA GAGCTGGCTGCAGGTGTCGGGATCCTGACACTCATCGA	3949
	GGGTGGCTACAAGTGCC	3950
	GGCACTTGTAGCCACCC	3951
Hypercholesterolaemia Cys358Arg gTGT-CGT	CAGGATCCCGACACCTGCAGCCAGCTCTGCGTGAACCTGGA GGGTGGCTACAAGTGCCAGTGTGAGGAAGGCTTCCAGCTGG ACCCCCACACGAAGGCCTGCAAGGCTGTGGGTGAGCACG	3952
	CGTGCTCACCCACAGCCTTGCAGGCCTTCGTGTGGGGGTCC AGCTGGAAGCCTTCCTCACACTGGCACTTGTAGCCACCCTCC AGGTTCACGCAGAGCTGGCTGCAGGTGTCGGGATCCTG	3953
	AGTGCCAGTGTGAGGAA	3954
	TTCCTCACACTGGCACT	3955
Hypercholesterolaemia Gln363Term cCAG-TAG	TGCAGCCAGCTCTGCGTGAACCTGGAGGGTGGCTACAAGTG CCAGTGTGAGGAAGGCTTCCAGCTGGACCCCCACACGAAGG CCTGCAAGGCTGTGGGTGAGCACGGGAAGGCGGCGGGTG	3956
	CACCCGCCGCCTTCCCGTGCTCACCCACAGCCTTGCAGGCC TTCGTGTGGGGGTCCAGCTGGAAGCCTTCCTCACACTGGCA CTTGTAGCCACCCTCCAGGTTCACGCAGAGCTGGCTGCA	3957
	AAGGCTTCCAGCTGGAC	3958
	GTCCAGCTGGAAGCCTT	3959

EXAMPLE 22

UDP-glucuronosyltransferase - UGT1

Mutations in the human UGT1 gene result in a range of disease syndromes, ranging from relatively common diseases such as Gilbert's syndrome, which effects up to 7% of the population, to rare disorders such as Crigler-Najjar syndrome. Symptoms of these diseases are the result of diminished bilirubin conjugation and typically present with jaundice or, when mild, as an incidental finding during routing laboratory analysis. Severe cases of Crigler-Najjar syndrome are caused by an absence of UGT1 activity and the majority of these patients die in the neonatal period. The only known treatment is liver transplant. The attached table discloses the correcting oligonucleotide base sequences for the UGT1 oligonucleotides of the invention.

Table 29

UGT1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Crigler-Najjar syndrome 2 Leu15Arg CTG-CGG	GCAGGAGCAAAGGCGCCATGGCTGTGGAGTCCCAGGGCGG ACGCCCACTTGTCCTGGGCCTGCTGCTGTGTGTGCTGGGCC CAGTGGTGTCCCATGCTGGGAAGATACTGTTGATCCCAGT	3960
	ACTGGGATCAACAGTATCTTCCCAGCATGGGACACCACTGGG CCCAGCACACACAGCAGCAGGCCCAGGACAAGTGGGCGTCC GCCCTGGGACTCCACAGCCATGGCGCCTTTGCTCCTGC	3961
	CCTGGGCCTGCTGCTGT	3962
	ACAGCAGCAGGCCCAGG	3963
Crigler-Najjar syndrome 1 Gln49Term CAG-TAG	GGGAAGATACTGTTGATCCCAGTGGATGGCAGCCACTGGCT GAGCATGCTTGGGGCCATCCAGCAGCTGCAGCAGAGGGGAC ATGAAATAGTTGTCCTAGCACCTGACGCCTCGTTGTACA	3964
	TGTACAACGAGGCGTCAGGTGCTAGGACAACTATTTCATGTC CCCTCTGCTGCAGCTGCTGGATGGCCCCAAGCATGCTCAGC CAGTGGCTGCCATCCACTGGGATCAACAGTATCTTCCC	3965
	GGGCCATCCAGCAGCTG	3966
	CAGCTGCTGGATGGCCC	3967
Crigler-Najjar syndrome 1 Gly71Arg GGA-AGA	CAGCAGAGGGGACATGAAATAGTTGTCCTAGCACCTGACGC CTCGTTGTACATCAGAGACGGAGCATTTTACACCTTGAAGAC GTACCCTGTGCCATTCCAAAGGGAGGATGTGAAAGAGT	3968
	ACTCTTTCACATCCTCCCTTTGGAATGGCACAGGGTACGTCTT CAAGGTGTAAAATGCTCCGTCTCTGATGTACAACGAGGCGTC AGGTGCTAGGACAACTATTTCATGTCCCCTCTGCTG	3969
	TCAGAGACGGAGCATTT	3970
	AAATGCTCCGTCTCTGA	3971
Gilbert syndrome Pro229Gln CCG-CAG	GGGTGAAGAACATGCTCATTGCCTTTTCACAGAACTTTCTGTG CGACGTGGTTTATTCCCCGTATGCAACCCTTGCCTCAGAATTC CTTCAGAGAGAGGTGACTGTCCAGGACCTATTGAG	3972
	CTCAATAGGTCCTGGACAGTCACCTCTCTCTGAAGGAATTCT GAGGCAAGGGTTGCATACGGGGAATAAACCACGTCGCACAG AAAGTTCTGTGAAAAGGCAATGAGCATGTTCTTCACCC	3973
	TTATTCCCCGTATGCAA	3974
	TTGCATACGGGGAATAA	3975
Crigler-Najjar syndrome 1 Cys280Term TGC-TGA	TGTGAAGGATTACCCTAGGCCCATCATGCCCAATATGGTTTTT GTTGGTGGAATCAACTGCCTTCACCAAAATCCACTATCCCAG GTGTGTATTGGAGTGGGACTTTTACATGCGTATATT	3976

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AATATACGCATGTAAAAGTCCCACTCCAATACACACCTGGGAT AGTGGATTTTGGTGAAGGCAGTTGATTCCACCAACAAAAACC ATATTGGGCATGATGGGCCTAGGGTAATCCTTCACA	3977
	ATCAACTGCCTTCACCA	3978
	TGGTGAAGGCAGTTGAT	3979
Crigler-Najjar syndrome 1 Ala292Val GCC-GTC	ATCAAAGAATATGAGAAAAAATTAACTGAAAATTTTTCTTCTGG CTCTAGGAATTTGAAGCCTACATTAATGCTTCTGGAGAACATG GAATTGTGGTTTTCTCTTTGGGATCAATGGTCTC	3980
	GAGACCATTGATCCCAAAGAGAAAACCACAATTCCATGTTCTC CAGAAGCATTAATGTAGGCTTCAAATTCCTAGAGCCAGAAGA AAAATTTTCAGTTAATTTTTTCTCATATTCTTTGAT	3981
	ATTTGAAGCCTACATTA	3982
	TAATGTAGGCTTCAAAT	3983
Crigler-Najjar syndrome 1 Gly308Glu GGA-GAA	AGGAATTTGAAGCCTACATTAATGCTTCTGGAGAACATGGAAT TGTGGTTTTCTCTTTGGGATCAATGGTCTCAGAAATTCCAGAG AAGAAAGCTATGGCAATTGCTGATGCTTTGGGCAA	3984
	TTGCCCAAAGCATCAGCAATTGCCATAGCTTTCTTCTCTGGAA TTTCTGAGACCATTGATCCCAAAGAGAAAACCACAATTCCATG TTCTCCAGAAGCATTAATGTAGGCTTCAAATTCCT	3985
	CTCTTTGGGATCAATGG	3986
	CCATTGATCCCAAAGAG	3987
Crigler-Najjar syndrome 1 Gln331Term CAG-TAG	GTCTCAGAAATTCCAGAGAAGAAAGCTATGGCAATTGCTGAT GCTTTGGGCAAAATCCCTCAGACAGTAAGAAGATTCTATACC ATGGCCTCATATCTATTTTCACAGGAGCGCTAATCCC	3988
	GGGATTAGCGCTCCTGTGAAAATAGATATGAGGCCATGGTAT AGAATCTTCTTACTGTCTGAGGGATTTTGCCCAAAGCATCAGC AATTGCCATAGCTTTCTTCTCTGGAATTTCTGAGAC	3989
	AAATCCCTCAGACAGTA	3990
	TACTGTCTGAGGGATTT	3991
Crigler-Najjar syndrome 1 Trp335Term TGG-TGA	TCTAATCATATTATGTTCTTTCTTTACGTTCTGCTCTTTTTGCC CCTCCCAGGTCCTGTGGCGGTACACTGGAACCCGACCATCG AATCTTGCGAACAACACGATACTTGTTAAGTGGCTA	3992
	TAGCCACTTAACAAGTATCGTGTTGTTCGCAAGATTCGATGGT CGGGTTCCAGTGTACCGCCACAGGACCTGGGAGGGGCAAAA AGAGCAGAACGTAAAGAAAGAACATAATATGATTAGA	3993
	GTCCTGTGGCGGTACAC	3994
	GTGTACCGCCACAGGAC	3995

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Crigler-Najjar syndrome 1 Gln357Arg CAA-CGA	ACACTGGAACCCGACCATCGAATCTTGCGAACAACACGATAC TTGTTAAGTGGCTACCCCAAAACGATCTGCTTGGTATGTTGG GCGGATTGGATGTATAGGTCAAACCAGGGTCAAATTA	3996
	TAATTTGACCCTGGTTTGACCTATACATCCAATCCGCCCAACA TACCAAGCAGATCGTTTTGGGGTAGCCACTTAACAAGTATCG TGTTGTTCGCAAGATTCGATGGTCGGGTTCCAGTGT	3997
	GCTACCCCAAAACGATC	3998
	GATCGTTTTGGGGTAGC	3999
Crigler-Najjar syndrome 1 Gln357Term CAA-TAA	TACACTGGAACCCGACCATCGAATCTTGCGAACAACACGATA CTTGTTAAGTGGCTACCCCAAAACGATCTGCTTGGTATGTTG GGCGGATTGGATGTATAGGTCAAACCAGGGTCAAATT	4000
	AATTTGACCCTGGTTTGACCTATACATCCAATCCGCCCAACAT ACCAAGCAGATCGTTTTGGGGTAGCCACTTAACAAGTATCGT GTTGTTCGCAAGATTCGATGGTCGGGTTCCAGTGTA	4001
	GGCTACCCCAAAACGAT	4002
	ATCGTTTTGGGGTAGCC	4003
Gilbert syndrome Arg367Gly CGT-GGT	AACTCAGAGATGTAACTGCTGACATCCTCCCTATTTTGCATCT CAGGTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTT CCCATGGTGTTTATGAAAGCATATGCAATGGCGTTC	4004
	GAACGCCATTGCATATGCTTTCATAAACACCATGGGAACCAG CATGGGTGATAAAGGCACGGGTCATCGGGTGACCTGAGATG CAAAATAGGGAGGATGTCAGCAGTTACATCTCTGAGTT	4005
	CGATGACCCGTGCCTTT	4006
	AAAGGCACGGGTCATCG	4007
Crigler-Najjar syndrome 1 Ala368Thr GCC-ACC	TCAGAGATGTAACTGCTGACATCCTCCCTATTTTGCATCTCAG GTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTTCCC ATGGTGTTTATGAAAGCATATGCAATGGCGTTCCCA	4008
	TGGGAACGCCATTGCATATGCTTTCATAAACACCATGGGAAC CAGCATGGGTGATAAAGGCACGGGTCATCGGGTGACCTGAG ATGCAAAATAGGGAGGATGTCAGCAGTTACATCTCTGA	4009
	TGACCCGTGCCTTTATC	4010
	GATAAAGGCACGGGTCA	4011
Crigler-Najjar syndrome 1 Ser375Phe TCC-TTC	CCTCCCTATTTTGCATCTCAGGTCACCCGATGACCCGTGCCT TTATCACCCATGCTGGTTCCCATGGTGTTTATGAAAGCATATG CAATGGCGTTCCCATGGTGATGATGCCCTTGTTTGG	4012
	CCAAACAAGGGCATCATCACCATGGGAACGCCATTGCATATG CTTTCATAAACACCATGGGAACCAGCATGGGTGATAAAGGCA CGGGTCATCGGGTGACCTGAGATGCAAAATAGGGAGG	4013
	TGCTGGTTCCCATGGTG	4014
	CACCATGGGAACCAGCA	4015

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Crigler-Najjar syndrome 1 Ser381Arg AGC-AGG	AGGTCACCCGATGACCCGTGCCTTTATCACCCATGCTGGTTC CCATGGTGTTTATGAAAGCATATGCAATGGCGTTCCCATGGT GATGATGCCCTTGTTTGGTGATCAGATGGACAATGCA	4016
	TGCATTGTCCATCTGATCACCAAACAAGGGCATCATCACCAT GGGAACGCCATTGCATATGCTTTCATAAACACCATGGGAACC AGCATGGGTGATAAAGGCACGGGTCATCGGGTGACCT	4017
	TATGAAAGCATATGCAA	4018
	TTGCATATGCTTTCATA	4019
Crigler-Najjar syndrome 1 Ala401Pro GCA-CCA	AGCATATGCAATGGCGTTCCCATGGTGATGATGCCCTTGTTT GGTGATCAGATGGACAATGCAAAGCGCATGGAGACTAAGGG AGCTGGAGTGACCCTGAATGTTCTGGAAATGACTTCTG	4020
	CAGAAGTCATTTCCAGAACATTCAGGGTCACTCCAGCTCCCT TAGTCTCCATGCGCTTTGCATTGTCCATCTGATCACCAAACAA GGGCATCATCACCATGGGAACGCCATTGCATATGCT	4021
	TGGACAATGCAAAGCGC	4022
	GCGCTTTGCATTGTCCA	4023
Crigler-Najjar syndrome 1 Lys428Glu AAA-GAA	GGAGCTGGAGTGACCCTGAATGTTCTGGAAATGACTTCTGAA GATTTAGAAAATGCTCTAAAAGCAGTCATCAATGACAAAAGGT AAGAAAGAAGATACAGAAGAATACTTTGGTCATGGC	4024
	GCCATGACCAAAGTATTCTTCTGTATCTTCTTTCTTACCTTTTG TCATTGATGACTGCTTTTAGAGCATTTTCTAAATCTTCAGAAGT CATTTCCAGAACATTCAGGGTCACTCCAGCTCC	4025
	ATGCTCTAAAAGCAGTC	4026
	GACTGCTTTTAGAGCAT	4027
Crigler-Najjar syndrome 1 Tyr486Asp TAC-GAC	ATGAGGCACAAGGGCGCGCCACACCTGCGCCCCGCAGCCCA CGACCTCACCTGGTACCAGTACCATTCCTTGGACGTGATTGG TTTCCTCTTGGCCGTCGTGCTGACAGTGGCCTTCATCA	4028
	TGATGAAGGCCACTGTCAGCACGACGGCCAAGAGGAAACCA ATCACGTCCAAGGAATGGTACTGGTACCAGGTGAGGTCGTG GGCTGCGGGGCGCAGGTGTGGCGCGCCCTTGTGCCTCAT	4029
	GGTACCAGTACCATTCC	4030
	GGAATGGTACTGGTACC	4031
Crigler-Najjar syndrome 1 Ser488Phe TCC-TTC	ACAAGGGCGCGCCACACCTGCGCCCCGCAGCCCACGACCTC ACCTGGTACCAGTACCATTCCTTGGACGTGATTGGTTTCCTCT TGGCCGTCGTGCTGACAGTGGCCTTCATCACCTTTAA	4032
	TTAAAGGTGATGAAGGCCACTGTCAGCACGACGGCCAAGAG GAAACCAATCACGTCCAAGGAATGGTACTGGTACCAGGTGAG GTCGTGGGCTGCGGGGCGCAGGTGTGGCGCGCCCTTGT	4033
	GTACCATTCCTTGGACG	4034
	CGTCCAAGGAATGGTAC	4035

EXAMPLE 23

Alzheimer's Disease - Amyloid precursor protein (APP)

Over the past few decades Alzheimer's disease (AD), once considered a rare disorder, has become recognized as a major public health problem. Although there is no agreement on the exact prevalence of Alzheimer's disease, in part due to difficulties of diagnosis, studies consistently point to an exponential rise in prevalence of this disease with age. After age 65, the percentage of affected people approximately doubles with every decade of life, regardless of definition. Among people age 85 or older, studies suggest that 25 to 35 percent have dementia, including Alzheimer's disease; one study reports that 47.2 percent of people over age 85 have Alzheimer's disease, exclusive of other dementias.

Alzheimer's disease progressively destroys memory, reason, judgment, language, and, eventually, the ability to carry out even the simplest tasks. Anatomic changes associated with Alzheimer's disease begin in the entorhinal cortex, proceed to the hippocampus, and then gradually spread to other regions, particularly the cerebral cortex. Chief among such anatomic changes are the presence of characteristic extracellular plaques and internal neurofibrillary tangles.

At least four genes have been identified to date that contribute to development of Alzheimer's disease: AD1 is caused by mutations in the amyloid precursor gene (APP); AD2 is associated with a particular allele of APOE (see Example 20); AD3 is caused by mutation in a gene encoding a 7-transmembrane domain protein, presenilin-1 (PSEN1), and AD4 is caused by mutation in a gene that encodes a similar 7-transmembrane domain protein, presenilin-2 (PSEN2). The attached table discloses the correcting oligonucleotide base sequences for the APP oligonucleotides of the invention.

Table 30

APP Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Glu665Asp GAG-GAC	CTGCATACTTTAATTATGATGTAATACAGGTTCTGGGTTGACA AATATCAAGACGGAGGA**G**ATCTCTGAAGTGAAGATGGATGCA GAATTCCGACATGACTCAGGATATGAAGTTCATCAT	4036
	ATGATGAACTTCATATCCTGAGTCATGTCGGAATTCTGCATCC ATCTTCACTTCAGAGAT**C**TCCTCCGTCTTGATATTTGTCAACC CAGAACCTGTATTACATCATAATTAAAGTATGCAG	4037
	ACGGAGGA**G**ATCTCTGA	4038
	TCAGAGAT**C**TCCTCCGT	4039

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Ala692Gly GCA-GGA	ATTATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTT TCAAGGTGTTCTTTGCAGAAGATGTGGGTTCAAACAAAGGTG CAATCATTGGACTCATGGTGGGCGGTGTTGTCAT	4040
	ATGACAACACCGCCCACCATGAGTCCAATGATTGCACCTTTG TTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAAATT AAGAAAAAGAATTTTAATTTCTAAATGCAATATAAT	4041
	GTTCTTTGCAGAAGATG	4042
	CATCTTCTGCAAAGAAC	4043
Alzheimer disease Glu693Gln GAA-CAA	TATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTTTC AAGGTGTTCTTTGCAGAAGATGTGGGTTCAAACAAAGGTGCA ATCATTGGACTCATGGTGGGCGGTGTTGTCATAG	4044
	CTATGACAACACCGCCCACCATGAGTCCAATGATTGCACCTT TGTTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAAA TTAAGAAAAAGAATTTTAATTTCTAAATGCAATATA	4045
	TCTTTGCAGAAGATGTG	4046
	CACATCTTCTGCAAAGA	4047
Alzheimer disease Glu693Gly GAA-GGA	ATATTGCATTTAGAAATTAAAATTCTTTTTCTTAATTTGTTTTCA AGGTGTTCTTTGCAGAAGATGTGGGTTCAAACAAAGGTGCAA TCATTGGACTCATGGTGGGCGGTGTTGTCATAGC	4048
	GCTATGACAACACCGCCCACCATGAGTCCAATGATTGCACCT TTGTTTGAACCCACATCTTCTGCAAAGAACACCTTGAAAACAA ATTAAGAAAAAGAATTTTAATTTCTAAATGCAATAT	4049
	CTTTGCAGAAGATGTGG	4050
	CCACATCTTCTGCAAAG	4051
Alzheimer disease Ala713Thr GCG-ACG	GAAGATGTGGGTTCAAACAAAGGTGCAATCATTGGACTCATG GTGGGCGGTGTTGTCATAGCGACAGTGATCGTCATCACCTTG GTGATGCTGAAGAAGAAACAGTACACATCCATTCATC	4052
	GATGAATGGATGTGTACTGTTTCTTCTTCAGCATCACCAAGGT GATGACGATCACTGTCGCTATGACAACACCGCCCACCATGAG TCCAATGATTGCACCTTTGTTTGAACCCACATCTTC	4053
	TTGTCATAGCGACAGTG	4054
	CACTGTCGCTATGACAA	4055
Schizophrenia Ala713Val GCG-GTG	AAGATGTGGGTTCAAACAAAGGTGCAATCATTGGACTCATGG TGGGCGGTGTTGTCATAGCGACAGTGATCGTCATCACCTTGG TGATGCTGAAGAAGAAACAGTACACATCCATTCATCA	4056
	TGATGAATGGATGTGTACTGTTTCTTCTTCAGCATCACCAAGG TGATGACGATCACTGTCGCTATGACAACACCGCCCACCATGA GTCCAATGATTGCACCTTTGTTTGAACCCACATCTT	4057
	TGTCATAGCGACAGTGA	4058
	TCACTGTCGCTATGACA	4059
Alzheimer disease Val715Met GTG-ATG	GTGGGTTCAAACAAAGGTGCAATCATTGGACTCATGGTGGGC GGTGTTGTCATAGCGACAGTGATCGTCATCACCTTGGTGATG CTGAAGAAGAAACAGTACACATCCATTCATCATGGTG	4060

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACCATGATGAATGGATGTGTACTGTTTCTTCTTCAGCATCAC CAAGGTGATGACGATCACTGTCGCTATGACAACACCGCCCAC CATGAGTCCAATGATTGCACCTTTGTTTGAACCCAC	4061
	TAGCGACAGTGATCGTC	4062
	GACGATCACTGTCGCTA	4063
Alzheimer disease Ile716Val ATC-GTC	GGTTCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGT GTTGTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTG AAGAAGAAACAGTACACATCCATTCATCATGGTGTGG	4064
	CCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAGCAT CACCAAGGTGATGACGATCACTGTCGCTATGACAACACCGCC CACCATGAGTCCAATGATTGCACCTTTGTTTGAACC	4065
	CGACAGTGATCGTCATC	4066
	GATGACGATCACTGTCG	4067
Alzheimer disease Val717Gly GTC-GGC	CAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTTG TCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAGA AGAAACAGTACACATCCATTCATCATGGTGTGGTGGA	4068
	TCCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCA GCATCACCAAGGTGATGACGATCACTGTCGCTATGACAACAC CGCCCACCATGAGTCCAATGATTGCACCTTTGTTTG	4069
	AGTGATCGTCATCACCT	4070
	AGGTGATGACGATCACT	4071
Alzheimer disease Val717Ile GTC-ATC	TCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTT GTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAG AAGAAACAGTACACATCCATTCATCATGGTGTGGTGG	4072
	CCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAG CATCACCAAGGTGATGACGATCACTGTCGCTATGACAACACC GCCCACCATGAGTCCAATGATTGCACCTTTGTTTGA	4073
	CAGTGATCGTCATCACC	4074
	GGTGATGACGATCACTG	4075
Alzheimer disease Val717Phe GTC-TTC	TCAAACAAAGGTGCAATCATTGGACTCATGGTGGGCGGTGTT GTCATAGCGACAGTGATCGTCATCACCTTGGTGATGCTGAAG AAGAAACAGTACACATCCATTCATCATGGTGTGGTGG	4076
	CCACCACACCATGATGAATGGATGTGTACTGTTTCTTCTTCAG CATCACCAAGGTGATGACGATCACTGTCGCTATGACAACACC GCCCACCATGAGTCCAATGATTGCACCTTTGTTTGA	4077
	CAGTGATCGTCATCACC	4078
	GGTGATGACGATCACTG	4079
Alzheimer disease Leu723Pro CTG-CCG	TTGGACTCATGGTGGGCGGTGTTGTCATAGCGACAGTGATCG TCATCACCTTGGTGATGCTGAAGAAGAAACAGTACACATCCA TTCATCATGGTGTGGTGGAGGTAGGTAAACTTGACTG	4080
	CAGTCAAGTTTACCTACCTCCACCACACCATGATGAATGGAT GTGTACTGTTTCTTCTTCAGCATCACCAAGGTGATGACGATCA CTGTCGCTATGACAACACCGCCCACCATGAGTCCAA	4081

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	GGTGATGCTGAAGAAGA	4082
	TCTTCTTCAGCATCACC	4083

EXAMPLE 24

Alzheimer's Disease - presenilin-1 (PSEN1)

The attached table discloses the correcting oligonucleotide base sequences for the PSEN1 oligonucleotides of the invention.

Table 31

PSEN1 Mutations and Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Ala79Val GCC-GTC	CCCGGCAGGTGGTGGAGCAAGATGAGGAAGAAGATGAGGAG CTGACATTGAAATATGGCGCCAAGCATGTGATCATGCTCTTTG TCCCTGTGACTCTCTGCATGGTGGTGGTCGTGGCTAC	4084
	GTAGCCACGACCACCACCATGCAGAGAGTCACAGGGACAAA GAGCATGATCACATGCTTGGCGCCATATTTCAATGTCAGCTC CTCATCTTCTTCCTCATCTTGCTCCACCACCTGCCGGG	4085
	ATATGGCGCCAAGCATG	4086
	CATGCTTGGCGCCATAT	4087
Alzheimer disease Val82Leu tGTG-CTG	GTGGTGGAGCAAGATGAGGAAGAAGATGAGGAGCTGACATT GAAATATGGCGCCAAGCATGTGATCATGCTCTTTGTCCCTGT GACTCTCTGCATGGTGGTGGTCGTGGCTACCATTAAGT	4088
	ACTTAATGGTAGCCACGACCACCACCATGCAGAGAGTCACAG GGACAAAGAGCATGATCACATGCTTGGCGCCATATTTCAATG TCAGCTCCTCATCTTCTTCCTCATCTTGCTCCACCAC	4089
	CCAAGCATGTGATCATG	4090
	CATGATCACATGCTTGG	4091
Alzheimer disease Val96Phe gGTC-TTC	AAATATGGCGCCAAGCATGTGATCATGCTCTTTGTCCCTGTG ACTCTCTGCATGGTGGTGGTCGTGGCTACCATTAAGTCAGTC AGCTTTTATACCCGGAAGGATGGGCAGCTGTACGTAT	4092
	ATACGTACAGCTGCCCATCCTTCCGGGTATAAAAGCTGACTG ACTTAATGGTAGCCACGACCACCACCATGCAGAGAGTCACAG GGACAAAGAGCATGATCACATGCTTGGCGCCATATTT	4093
	TGGTGGTGGTCGTGGCT	4094
	AGCCACGACCACCACCA	4095

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Phe105Leu TTTt-TTG	CTTTGTCCCTGTGACTCTCTGCATGGTGGTGGTCGTGGCTAC CATTAAGTCAGTCAGCTTTTATACCCGGAAGGATGGGCAGCT GTACGTATGAGTTTTGTTTTATTATTCTCAAAGCCAG	4096
	CTGGCTTTGAGAATAATAAAACAAAACTCATACGTACAGCTGC CCATCCTTCCGGGTATAAAAGCTGACTGACTTAATGGTAGCC ACGACCACCACCATGCAGAGAGTCACAGGGACAAAG	4097
	GTCAGCTTTTATACCCG	4098
	CGGGTATAAAAGCTGAC	4099
Alzheimer disease Thr116Asn ACC-AAC	TGGTGATCTCCATTAACACTGACCTAGGGCTTTTGTGTTTGTT TTATTGTAGAATCTATACCCCATTCACAGAAGATACCGAGACT GTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGC	4100
	GCATTCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCTCG GTATCTTCTGTGAATGGGGTATAGATTCTACAATAAAACAAAC ACAAAAGCCCTAGGTCAGTGTTAATGGAGATCACCA	4101
	AATCTATACCCCATTCA	4102
	TGAATGGGGTATAGATT	4103
Alzheimer disease Pro117Leu CCA-CTA	TGATCTCCATTAACACTGACCTAGGGCTTTTGTGTTTGTTTTAT TGTAGAATCTATACCCCATTCACAGAAGATACCGAGACTGTG GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGC	4104
	GCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTC TCGGTATCTTCTGTGAATGGGGTATAGATTCTACAATAAAACA AACACAAAAGCCCTAGGTCAGTGTTAATGGAGATCA	4105
	CTATACCCCATTCACAG	4106
	CTGTGAATGGGGTATAG	4107
Alzheimer disease Glu120Asp GAAg-GAT	TAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCT ATACCCCATTCACAGAAGATACCGAGACTGTGGGCCAGAGA GCCCTGCACTCAATTCTGAATGCTGCCATCATGATC	4108
	GATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTG GCCCACAGTCTCGGTATCTTCTGTGAATGGGGTATAGATTCT ACAATAAAACAAACACAAAAGCCCTAGGTCAGTGTTA	4109
	TTCACAGAAGATACCGA	4110
	TCGGTATCTTCTGTGAA	4111
Alzheimer disease Glu120Asp GAAg-GAC	TAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCT ATACCCCATTCACAGAAGATACCGAGACTGTGGGCCAGAGA GCCCTGCACTCAATTCTGAATGCTGCCATCATGATC	4112
	GATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTG GCCCACAGTCTCGGTATCTTCTGTGAATGGGGTATAGATTCT ACAATAAAACAAACACAAAAGCCCTAGGTCAGTGTTA	4113
	TTCACAGAAGATACCGA	4114
	TCGGTATCTTCTGTGAA	4115
Alzheimer disease Glu120Lys aGAA-AAA	ATTAACACTGACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAAT CTATACCCCATTCACAGAAGATACCGAGACTGTGGGCCAGAG AGCCCTGCACTCAATTCTGAATGCTGCCATCATGA	4116

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGC CCACAGTCTCGGTATCTTCTGTGAATGGGGTATAGATTCTACA ATAAAACAAACACAAAAGCCCTAGGTCAGTGTTAAT	4117
	CATTCACAGAAGATACC	4118
	GGTATCTTCTGTGAATG	4119
Alzheimer disease Glu123Lys cGAG-AAG	GACCTAGGGCTTTTGTGTTTGTTTTATTGTAGAATCTATACCC CATTCACAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTG CACTCAATTCTGAATGCTGCCATCATGATCAGTGTCA	4120
	TGACACTGATCATGATGGCAGCATTCAGAATTGAGTGCAGGG CTCTCTGGCCCACAGTCTCGGTATCTTCTGTGAATGGGGTAT AGATTCTACAATAAAACAAACACAAAAGCCCTAGGTC	4121
	AAGATACCGAGACTGTG	4122
	CACAGTCTCGGTATCTT	4123
Alzheimer disease Asn135Asp gAAT-GAT	TATACCCCATTCACAGAAGATACCGAGACTGTGGGCCAGAGA GCCCTGCACTCAATTCTGAATGCTGCCATCATGATCAGTGTC ATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTATA	4124
	TATACAGAACCACCAGGAGGATAGTCATGACAACAATGACAC TGATCATGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCT GGCCCACAGTCTCGGTATCTTCTGTGAATGGGGTATA	4125
	CAATTCTGAATGCTGCC	4126
	GGCAGCATTCAGAATTG	4127
Alzheimer disease Met139Ile ATGa-ATA	AGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCAA TTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGAC TATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTAT	4128
	ATAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCAT GACAACAATGACACTGATCATGATGGCAGCATTCAGAATTGA GTGCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCT	4129
	GCCATCATGATCAGTGT	4130
	ACACTGATCATGATGGC	4131
Alzheimer disease Met139Lys ATG-AAG	CAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCA ATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGA CTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTA	4132
	TAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATG ACAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAG TGCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTG	4133
	TGCCATCATGATCAGTG	4134
	CACTGATCATGATGGCA	4135
Alzheimer disease Met139Thr ATG-ACG	CAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTCA ATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATGA CTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCTA	4136
	TAGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATG ACAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAG TGCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTG	4137
	TGCCATCATGATCAGTG	4138

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CACTGATCATGATGGCA	4139
Alzheimer disease Met139Val cATG-GTG	ACAGAAGATACCGAGACTGTGGGCCAGAGAGCCCTGCACTC AATTCTGAATGCTGCCATCATGATCAGTGTCATTGTTGTCATG ACTATCCTCCTGGTGGTTCTGTATAAATACAGGTGCT	4140
	AGCACCTGTATTTATACAGAACCACCAGGAGGATAGTCATGA CAACAATGACACTGATCATGATGGCAGCATTCAGAATTGAGT GCAGGGCTCTCTGGCCCACAGTCTCGGTATCTTCTGT	4141
	CTGCCATCATGATCAGT	4142
	ACTGATCATGATGGCAG	4143
Alzheimer disease Ile143Phe cATT-TTT	GAGACTGTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGCT GCCATCATGATCAGTGTCATTGTTGTCATGACTATCCTCCTGG TGGTTCTGTATAAATACAGGTGCTATAAGGTGAGCA	4144
	TGCTCACCTTATAGCACCTGTATTTATACAGAACCACCAGGAG GATAGTCATGACAACAATGACACTGATCATGATGGCAGCATT CAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCTC	4145
	TCAGTGTCATTGTTGTC	4146
	GACAACAATGACACTGA	4147
Alzheimer disease Ile143Thr ATT-ACT	AGACTGTGGGCCAGAGAGCCCTGCACTCAATTCTGAATGCTG CCATCATGATCAGTGTCATTGTTGTCATGACTATCCTCCTGGT GGTTCTGTATAAATACAGGTGCTATAAGGTGAGCAT	4148
	ATGCTCACCTTATAGCACCTGTATTTATACAGAACCACCAGGA GGATAGTCATGACAACAATGACACTGATCATGATGGCAGCAT TCAGAATTGAGTGCAGGGCTCTCTGGCCCACAGTCT	4149
	CAGTGTCATTGTTGTCA	4150
	TGACAACAATGACACTG	4151
Alzheimer disease Met146Ile ATGa-ATA	CCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGAT CAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTAT AAATACAGGTGCTATAAGGTGAGCATGAGACACAGA	4152
	TCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGA ACCACCAGGAGGATAGTCATGACAACAATGACACTGATCATG ATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGG	4153
	GTTGTCATGACTATCCT	4154
	AGGATAGTCATGACAAC	4155
Alzheimer disease Met146Ile ATGa-ATC	CCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGAT CAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTAT AAATACAGGTGCTATAAGGTGAGCATGAGACACAGA	4156
	TCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGA ACCACCAGGAGGATAGTCATGACAACAATGACACTGATCATG ATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGG	4157
	GTTGTCATGACTATCCT	4158
	AGGATAGTCATGACAAC	4159

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Met146Leu cATG-TTG	GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATG ATCAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGT ATAAATACAGGTGCTATAAGGTGAGCATGAGACACA	4160
	TGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGAAC CACCAGGAGGATAGTCATGACAACAATGACACTGATCATGAT GGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCC	4161
	TTGTTGTCATGACTATC	4162
	GATAGTCATGACAACAA	4163
Alzheimer disease Met146Val cATG-GTG	GGCCAGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATG ATCAGTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGT ATAAATACAGGTGCTATAAGGTGAGCATGAGACACA	4164
	TGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACAGAAC CACCAGGAGGATAGTCATGACAACAATGACACTGATCATGAT GGCAGCATTCAGAATTGAGTGCAGGGCTCTCTGGCC	4165
	TTGTTGTCATGACTATC	4166
	GATAGTCATGACAACAA	4167
Alzheimer disease Thr147Ile ACT-ATT	AGAGAGCCCTGCACTCAATTCTGAATGCTGCCATCATGATCA GTGTCATTGTTGTCATGACTATCCTCCTGGTGGTTCTGTATAA ATACAGGTGCTATAAGGTGAGCATGAGACACAGATC	4168
	GATCTGTGTCTCATGCTCACCTTATAGCACCTGTATTTATACA GAACCACCAGGAGGATAGTCATGACAACAATGACACTGATCA TGATGGCAGCATTCAGAATTGAGTGCAGGGCTCTCT	4169
	TGTCATGACTATCCTCC	4170
	GGAGGATAGTCATGACA	4171
Alzheimer disease His163Arg CAT-CGT	CTTTTTAAGGGTTGTGGGACCTGTTAATTATATTGAAATGCTTT CTTTTCTAGGTCATCCATGCCTGGCTTATTATATCATCTCTATT GTTGCTGTTCTTTTTTTCATTCATTTACTTGGG	4172
	CCCAAGTAAATGAATGAAAAAAAGAACAGCAACAATAGAGAT GATATAATAAGCCAGGCATGGATGACCTAGAAAAGAAAGCAT TTCAATATAATTAACAGGTCCCACAACCCTTAAAAAG	4173
	GGTCATCCATGCCTGGC	4174
	GCCAGGCATGGATGACC	4175
Alzheimer disease His163Tyr cCAT-TAT	ACTTTTTAAGGGTTGTGGGACCTGTTAATTATATTGAAATGCT TTCTTTTCTAGGTCATCCATGCCTGGCTTATTATATCATCTCTA TTGTTGCTGTTCTTTTTTTCATTCATTTACTTGG	4176
	CCAAGTAAATGAATGAAAAAAAGAACAGCAACAATAGAGATG ATATAATAAGCCAGGCATGGATGACCTAGAAAAGAAAGCATTT CAATATAATTAACAGGTCCCACAACCCTTAAAAAGT	4177
	AGGTCATCCATGCCTGG	4178
	CCAGGCATGGATGACCT	4179
Alzheimer disease Trp165Cys TGGc-TGC	AGGGTTGTGGGACCTGTTAATTATATTGAAATGCTTTCTTTTC TAGGTCATCCATGCCTGGCTTATTATATCATCTCTATTGTTGCT GTTCTTTTTTTCATTCATTTACTTGGGGTAAGTT	4180

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	AACTTACCCCAAGTAAATGAATGAAAAAAAGAACAGCAACAAT AGAGATGATATAATAAGCCAGGCATGGATGACCTAGAAAAGA AAGCATTTCAATATAATTAACAGGTCCCACAACCCT	4181
	CATGCCTGGCTTATTAT	4182
	ATAATAAGCCAGGCATG	4183
Alzheimer disease Ser169Leu TCA-TTA	ACCTGTTAATTATATTGAAATGCTTTCTTTTCTAGGTCATCCAT GCCTGGCTTATTATATCATCTCTATTGTTGCTGTTCTTTTTTTC ATTCATTTACTTGGGGTAAGTTGTGAAATTTTT	4184
	AAAAATTTCACAACTTACCCCAAGTAAATGAATGAAAAAAAGA ACAGCAACAATAGAGATGATATAATAAGCCAGGCATGGATGA CCTAGAAAAGAAAGCATTTCAATATAATTAACAGGT	4185
	TATTATATCATCTCTAT	4186
	ATAGAGATGATATAATA	4187
Alzheimer disease Leu171Pro CTA-CCA	TAATTATATTGAAATGCTTTCTTTTCTAGGTCATCCATGCCTGG CTTATTATATCATCTCTATTGTTGCTGTTCTTTTTTTCATTCATT TACTTGGGGTAAGTTGTGAAATTTTTGGTCTG	4188
	CAGACCAAAAATTTCACAACTTACCCCAAGTAAATGAATGAAA AAAAGAACAGCAACAATAGAGATGATATAATAAGCCAGGCAT GGATGACCTAGAAAAGAAAGCATTTCAATATAATTA	4189
	ATCATCTCTATTGTTGC	4190
	GCAACAATAGAGATGAT	4191
Alzheimer disease Leu173Trp TTG-TGG	TATTGAAATGCTTTCTTTTCTAGGTCATCCATGCCTGGCTTATT ATATCATCTCTATTGTTGCTGTTCTTTTTTTCATTCATTTACTTG GGGTAAGTTGTGAAATTTTTGGTCTGTCTTTC	4192
	GAAAGACAGACCAAAAATTTCACAACTTACCCCAAGTAAATGA ATGAAAAAAAGAACAGCAACAATAGAGATGATATAATAAGCCA GGCATGGATGACCTAGAAAAGAAAGCATTTCAATA	4193
	TCTATTGTTGCTGTTCT	4194
	AGAACAGCAACAATAGA	4195
Alzheimer disease Gly209Arg gGGA-AGA	TATAACGTTGCTGTGGACTACATTACTGTTGCACTCCTGATCT GGAATTTTGGTGTGGTGGGAATGATTTCCATTCACTGGAAAG GTCCACTTCGACTCCAGCAGGCATATCTCATTATGA	4196
	TCATAATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTCC AGTGAATGGAAATCATTCCCACCACACCAAAATTCCAGATCA GGAGTGCAACAGTAATGTAGTCCACAGCAACGTTATA	4197
	GTGTGGTGGGAATGATT	4198
	AATCATTCCCACCACAC	4199
Alzheimer disease Gly209Val GGA-GTA	ATAACGTTGCTGTGGACTACATTACTGTTGCACTCCTGATCTG GAATTTTGGTGTGGTGGGAATGATTTCCATTCACTGGAAAGG TCCACTTCGACTCCAGCAGGCATATCTCATTATGAT	4200

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	ATCATAATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTC CAGTGAATGGAAATCATTCCCACCACACCAAAATTCCAGATCA GGAGTGCAACAGTAATGTAGTCCACAGCAACGTTAT	4201
	TGTGGTGGGAATGATTT	4202
	AAATCATTCCCACCACA	4203
Alzheimer disease Ile213Thr ATT-ACT	TGGACTACATTACTGTTGCACTCCTGATCTGGAATTTTGGTGT GGTGGGAATGATTTCCATTCACTGGAAAGGTCCACTTCGACT CCAGCAGGCATATCTCATTATGATTAGTGCCCTCAT	4204
	ATGAGGGCACTAATCATAATGAGATATGCCTGCTGGAGTCGA AGTGGACCTTTCCAGTGAATGGAAATCATTCCCACCACACCA AAATTCCAGATCAGGAGTGCAACAGTAATGTAGTCCA	4205
	GATTTCCATTCACTGGA	4206
	TCCAGTGAATGGAAATC	4207
Alzheimer disease Leu219Pro CTT-CCT	CACTCCTGATCTGGAATTTTGGTGTGGTGGGAATGATTTCCAT TCACTGGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCAT TATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAA	4208
	TTGATAAACACCAGGGCCATGAGGGCACTAATCATAATGAGA TATGCCTGCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAA ATCATTCCCACCACACCAAAATTCCAGATCAGGAGTG	4209
	AGGTCCACTTCGACTCC	4210
	GGAGTCGAAGTGGACCT	4211
Alzheimer disease Ala231Thr tGCC-ACC	ATTTCCATTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCA TATCTCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCA AGTACCTCCCTGAATGGACTGCGTGGCTCATCTTGG	4212
	CCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTGATAA ACACCAGGGCCATGAGGGCACTAATCATAATGAGATATGCCT GCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAAAT	4213
	TGATTAGTGCCCTCATG	4214
	CATGAGGGCACTAATCA	4215
Alzheimer disease Ala231Val GCC-GTC	TTTCCATTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCAT ATCTCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAA GTACCTCCCTGAATGGACTGCGTGGCTCATCTTGGC	4216
	GCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTGATA AACACCAGGGCCATGAGGGCACTAATCATAATGAGATATGCC TGCTGGAGTCGAAGTGGACCTTTCCAGTGAATGGAAA	4217
	GATTAGTGCCCTCATGG	4218
	CCATGAGGGCACTAATC	4219
Alzheimer disease Met233Thr ATG-ACG	TTCACTGGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCA TTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTACCT CCCTGAATGGACTGCGTGGCTCATCTTGGCTGTGAT	4220
	ATCACAGCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTA CTTGATAAACACCAGGGCCATGAGGGCACTAATCATAATGAG ATATGCCTGCTGGAGTCGAAGTGGACCTTTCCAGTGAA	4221
	TGCCCTCATGGCCCTGG	4222

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	CCAGGGCCATGAGGGCA	4223
Alzheimer disease Leu235Pro CTG-CCG	GGAAAGGTCCACTTCGACTCCAGCAGGCATATCTCATTATGA TTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTACCTCCCTG AATGGACTGCGTGGCTCATCTTGGCTGTGATTTCAGT	4224
	ACTGAAATCACAGCCAAGATGAGCCACGCAGTCCATTCAGGG AGGTACTTGATAAACACCAGGGCCATGAGGGCACTAATCATA ATGAGATATGCCTGCTGGAGTCGAAGTGGACCTTTCC	4225
	CATGGCCCTGGTGTTTA	4226
	TAAACACCAGGGCCATG	4227
Alzheimer disease Ala246Glu GCG-GAG	TCATTATGATTAGTGCCCTCATGGCCCTGGTGTTTATCAAGTA CCTCCCTGAATGGACTGCGTGGCTCATCTTGGCTGTGATTTC AGTATATGGTAAAACCCAAGACTGATAATTTGTTTG	4228
	CAAACAAATTATCAGTCTTGGGTTTTACCATATACTGAAATCA CAGCCAAGATGAGCCACGCAGTCCATTCAGGGAGGTACTTG ATAAACACCAGGGCCATGAGGGCACTAATCATAATGA	4229
	ATGGACTGCGTGGCTCA	4230
	TGAGCCACGCAGTCCAT	4231
Alzheimer disease Leu250Ser TTG-TCG	GTGCCCTCATGGCCCTGGTGTTTATCAAGTACCTCCCTGAAT GGACTGCGTGGCTCATCTTGGCTGTGATTTCAGTATATGGTA AAACCCAAGACTGATAATTTGTTTGTCACAGGAATGC	4232
	GCATTCCTGTGACAAACAAATTATCAGTCTTGGGTTTTACCAT ATACTGAAATCACAGCCAAGATGAGCCACGCAGTCCATTCAG GGAGGTACTTGATAAACACCAGGGCCATGAGGGCAC	4233
	GCTCATCTTGGCTGTGA	4234
	TCACAGCCAAGATGAGC	4235
Alzheimer disease Ala260Val GCT-GTT	AGTTTAGCCCATACATTTTATTAGATGTCTTTTATGTTTTTCTTT TTCTAGATTTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCG TATGCTGGTTGAAACAGCTCAGGAGAGAAATGA	4236
	TCATTTCTCTCCTGAGCTGTTTCAACCAGCATACGAAGTGGAC CTTTCGGACACAAAACAGCCACTAAATCTAGAAAAAGAAAAAC ATAAAAGACATCTAATAAAATGTATGGGCTAAACT	4237
	TTTAGTGGCTGTTTTGT	4238
	ACAAAACAGCCACTAAA	4239
Alzheimer disease Leu262Phe TTGt-TTC	CCCATACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGA TTTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTG GTTGAAACAGCTCAGGAGAGAAATGAAACGCTT	4240
	AAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCATACGA AGTGGACCTTTCGGACACAAAACAGCCACTAAATCTAGAAAA AGAAAAACATAAAAGACATCTAATAAAATGTATGGG	4241
	GCTGTTTTGTGTCCGAA	4242
	TTCGGACACAAAACAGC	4243

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Cys263Arg gTGT-CGT	CCATACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGAT TTAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTG GTTGAAACAGCTCAGGAGAGAAATGAAACGCTTT	4244
	AAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCATACG AAGTGGACCTTTCGGACACAAAACAGCCACTAAATCTAGAAA AAGAAAAACATAAAAGACATCTAATAAAATGTATGG	4245
	CTGTTTTGTGTCCGAAA	4246
	TTTCGGACACAAAACAG	4247
Alzheimer disease Pro264Leu CCG-CTG	ACATTTTATTAGATGTCTTTTATGTTTTTCTTTTTCTAGATTTAG TGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTG AAACAGCTCAGGAGAGAAATGAAACGCTTTTTCC	4248
	GGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCA TACGAAGTGGACCTTTCGGACACAAAACAGCCACTAAATCTA GAAAAAGAAAAACATAAAAGACATCTAATAAAATGT	4249
	TTTGTGTCCGAAAGGTC	4250
	GACCTTTCGGACACAAA	4251
Alzheimer disease Arg269Gly tCGT-GGT	GTCTTTTATGTTTTTCTTTTTCTAGATTTAGTGGCTGTTTTGTG TCCGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGA GAGAAATGAAACGCTTTTTCCAGCTCTCATTTACT	4252
	AGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGAG CTGTTTCAACCAGCATACGAAGTGGACCTTTCGGACACAAAA CAGCCACTAAATCTAGAAAAAGAAAAACATAAAAGAC	4253
	GTCCACTTCGTATGCTG	4254
	CAGCATACGAAGTGGAC	4255
Alzheimer disease Arg269His CGT-CAT	TCTTTTATGTTTTTCTTTTTCTAGATTTAGTGGCTGTTTTGTGTC CGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGA GAAATGAAACGCTTTTTCCAGCTCTCATTTACTC	4256
	GAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGA GCTGTTTCAACCAGCATACGAAGTGGACCTTTCGGACACAAA ACAGCCACTAAATCTAGAAAAAGAAAAACATAAAAGA	4257
	TCCACTTCGTATGCTGG	4258
	CCAGCATACGAAGTGGA	4259
Alzheimer disease Arg278Thr AGA-ACA	TAGTGGCTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGG TTGAAACAGCTCAGGAGAGAAATGAAACGCTTTTTCCAGCTC TCATTTACTCCTGTAAGTATTTGAGAATGATATTGAA	4260
	TTCAATATCATTCTCAAATACTTACAGGAGTAAATGAGAGCTG GAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACCAGCAT ACGAAGTGGACCTTTCGGACACAAAACAGCCACTA	4261
	TCAGGAGAGAAATGAAA	4262
	TTTCATTTCTCTCCTGA	4263
Alzheimer disease Glu280Ala GAA-GCA	CTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAAC AGCTCAGGAGAGAAATGAAACGCTTTTTCCAGCTCTCATTTAC TCCTGTAAGTATTTGAGAATGATATTGAATTAGTA	4264

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TACTAATTCAATATCATTCTCAAATACTTACAGGAGTAAATGAG AGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACC AGCATACGAAGTGGACCTTTCGGACACAAAACAG	4265
	GAGAAATGAAACGCTTT	4266
	AAAGCGTTTCATTTCTC	4267
Alzheimer disease Glu280Gly GAA-GGA	CTGTTTTGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAAC AGCTCAGGAGAGAAATGAAACGCTTTTTCCAGCTCTCATTTAC TCCTGTAAGTATTTGAGAATGATATTGAATTAGTA	4268
	TACTAATTCAATATCATTCTCAAATACTTACAGGAGTAAATGAG AGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTTTCAACC AGCATACGAAGTGGACCTTTCGGACACAAAACAG	4269
	GAGAAATGAAACGCTTT	4270
	AAAGCGTTTCATTTCTC	4271
Alzheimer disease Leu282Arg CTT-CGT	TGTGTCCGAAAGGTCCACTTCGTATGCTGGTTGAAACAGCTC AGGAGAGAAATGAAACGCTTTTTCCAGCTCTCATTTACTCCTG TAAGTATTTGAGAATGATATTGAATTAGTAATCAGT	4272
	ACTGATTACTAATTCAATATCATTCTCAAATACTTACAGGAGTA AATGAGAGCTGGAAAAAGCGTTTCATTTCTCTCCTGAGCTGTT TCAACCAGCATACGAAGTGGACCTTTCGGACACA	4273
	TGAAACGCTTTTTCCAG	4274
	CTGGAAAAAGCGTTTCA	4275
Alzheimer disease Ala285Val GCT-GTT	AAGGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGAGAA ATGAAACGCTTTTTCCAGCTCTCATTTACTCCTGTAAGTATTTG AGAATGATATTGAATTAGTAATCAGTGTAGAATTT	4276
	AAATTCTACACTGATTACTAATTCAATATCATTCTCAAATACTT ACAGGAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCTC CTGAGCTGTTTCAACCAGCATACGAAGTGGACCTT	4277
	TTTTCCAGCTCTCATTT	4278
	AAATGAGAGCTGGAAAA	4279
Alzheimer disease Leu286Val tCTC-GTC	GGTCCACTTCGTATGCTGGTTGAAACAGCTCAGGAGAGAAAT GAAACGCTTTTTCCAGCTCTCATTTACTCCTGTAAGTATTTGA GAATGATATTGAATTAGTAATCAGTGTAGAATTTAT	4280
	ATAAATTCTACACTGATTACTAATTCAATATCATTCTCAAATAC TTACAGGAGTAAATGAGAGCTGGAAAAAGCGTTTCATTTCTCT CCTGAGCTGTTTCAACCAGCATACGAAGTGGACC	4281
	TTCCAGCTCTCATTTAC	4282
	GTAAATGAGAGCTGGAA	4283
Alzheimer disease Gly384Ala GGA-GCA	GTGACCAACTTTTTAATATTTGTAACCTTTCCTTTTTAGGGGGA GTAAAACTTGGATTGGGAGATTTCATTTTCTACAGTGTTCTGG TTGGTAAAGCCTCAGCAACAGCCAGTGGAGACTG	4284
	CAGTCTCCACTGGCTGTTGCTGAGGCTTTACCAACCAGAACA CTGTAGAAAATGAAATCTCCCAATCCAAGTTTTACTCCCCCTA AAAAGGAAAGGTTACAAATATTAAAAAGTTGGTCAC	4285

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TGGATTGGGAGATTTCA	4286
	TGAAATCTCCCAATCCA	4287
Alzheimer disease Ser390Ile AGT-ATT	TTTGTAACCTTTCCTTTTTAGGGGGAGTAAAACTTGGATTGGG AGATTTCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCA ACAGCCAGTGGAGACTGGAACACAACCATAGCCTG	4288
	CAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTGCTGAG GCTTTACCAACCAGAACACTGTAGAAAATGAAATCTCCCAATC CAAGTTTTACTCCCCCTAAAAAGGAAAGGTTACAAA	4289
	TTTCTACAGTGTTCTGG	4290
	CCAGAACACTGTAGAAA	4291
Alzheimer disease Leu392Val tCTG-GTG	AACCTTTCCTTTTTAGGGGGAGTAAAACTTGGATTGGGAGATT TCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCAACAGC CAGTGGAGACTGGAACACAACCATAGCCTGTTTCG	4292
	CGAAACAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTG CTGAGGCTTTACCAACCAGAACACTGTAGAAAATGAAATCTC CCAATCCAAGTTTTACTCCCCCTAAAAAGGAAAGGTT	4293
	ACAGTGTTCTGGTTGGT	4294
	ACCAACCAGAACACTGT	4295
Alzheimer disease Asn405Ser AAC-AGC	ATTTCATTTTCTACAGTGTTCTGGTTGGTAAAGCCTCAGCAAC AGCCAGTGGAGACTGGAACACAACCATAGCCTGTTTCGTAGC CATATTAATTGTAAGTATACACTAATAAGAATGTGT	4296
	ACACATTCTTATTAGTGTATACTTACAATTAATATGGCTACGAA ACAGGCTATGGTTGTGTTCCAGTCTCCACTGGCTGTTGCTGA GGCTTTACCAACCAGAACACTGTAGAAAATGAAAT	4297
	AGACTGGAACACAACCA	4298
	TGGTTGTGTTCCAGTCT	4299
Alzheimer disease Ala409Thr aGCC-ACC	TACAGTGTTCTGGTTGGTAAAGCCTCAGCAACAGCCAGTGGA GACTGGAACACAACCATAGCCTGTTTCGTAGCCATATTAATTG TAAGTATACACTAATAAGAATGTGTCAGAGCTCTTA	4300
	TAAGAGCTCTGACACATTCTTATTAGTGTATACTTACAATTAAT ATGGCTACGAAACAGGCTATGGTTGTGTTCCAGTCTCCACTG GCTGTTGCTGAGGCTTTACCAACCAGAACACTGTA	4301
	CAACCATAGCCTGTTTC	4302
	GAAACAGGCTATGGTTG	4303
Alzheimer disease Cys410Tyr TGT-TAT	GTGTTCTGGTTGGTAAAGCCTCAGCAACAGCCAGTGGAGACT GGAACACAACCATAGCCTGTTTCGTAGCCATATTAATTGTAAG TATACACTAATAAGAATGTGTCAGAGCTCTTAATGT	4304
	ACATTAAGAGCTCTGACACATTCTTATTAGTGTATACTTACAAT TAATATGGCTACGAAACAGGCTATGGTTGTGTTCCAGTCTCCA CTGGCTGTTGCTGAGGCTTTACCAACCAGAACAC	4305
	CATAGCCTGTTTCGTAG	4306
	CTACGAAACAGGCTATG	4307

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Ala426Pro tGCC-CCC	TGTGAATGTGTGTCTTTCCCATCTTCTCCACAGGGTTTGTGCC TTACATTATTACTCCTTGCCATTTTCAAGAAAGCATTGCCAGC TCTTCCAATCTCCATCACCTTTGGGCTTGTTTTCT	4308
	AGAAAACAAGCCCAAAGGTGATGGAGATTGGAAGAGCTGGC AATGCTTTCTTGAAAATGGCAAGGAGTAATAATGTAAGGCACA AACCCTGTGGAGAAGATGGGAAAGACACACATTCACA	4309
	TACTCCTTGCCATTTTC	4310
	GAAAATGGCAAGGAGTA	4311
Alzheimer disease Pro436Gln CCA-CAA	AGGGTTTGTGCCTTACATTATTACTCCTTGCCATTTTCAAGAA AGCATTGCCAGCTCTTCCAATCTCCATCACCTTTGGGCTTGTT TTCTACTTTGCCACAGATTATCTTGTACAGCCTTT	4312
	AAAGGCTGTACAAGATAATCTGTGGCAAAGTAGAAAACAAGC CCAAAGGTGATGGAGATTGGAAGAGCTGGCAATGCTTTCTTG AAAATGGCAAGGAGTAATAATGTAAGGCACAAACCCT	4313
	AGCTCTTCCAATCTCCA	4314
	TGGAGATTGGAAGAGCT	4315
Alzheimer disease Pro436Ser tCCA-TCA	CAGGGTTTGTGCCTTACATTATTACTCCTTGCCATTTTCAAGA AAGCATTGCCAGCTCTTCCAATCTCCATCACCTTTGGGCTTGT TTTCTACTTTGCCACAGATTATCTTGTACAGCCTT	4316
	AAGGCTGTACAAGATAATCTGTGGCAAAGTAGAAAACAAGCC CAAAGGTGATGGAGATTGGAAGAGCTGGCAATGCTTTCTTGA AAATGGCAAGGAGTAATAATGTAAGGCACAAACCCTG	4317
	CAGCTCTTCCAATCTCC	4318
	GGAGATTGGAAGAGCTG	4319

EXAMPLE 25

Alzheimer's Disease - presenilin-2 (PSEN2)

The attached table discloses the correcting oligonucleotide base sequences for the PSEN2 oligonucleotides of the invention.

Table 32

PSEN2 Mutations And Genome-Correcting Oligos

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
Alzheimer disease Arg62His CGC-CAC	GATGTGGTTTCCCACAGAGAAGCCAGGAGAACGAGGAGGAC GGTGAGGAGGACCCTGACCGCTATGTCTGTAGTGGGGTTCC CGGGCGGCCGCCAGGCCTGGAGGAAGAGCTGACCCTCAA	4320
	TTGAGGGTCAGCTCTTCCTCCAGGCCTGGCGGCCGCCCGGG AACCCCACTACAGACATAGCGGTCAGGGTCCTCCTCACCGTC CTCCTCGTTCTCCTGGCTTCTCTGTGGGAAACCACATC	4321
	CCCTGACCGCTATGTCT	4322
	AGACATAGCGGTCAGGG	4323
Alzheimer disease Thr122Pro cACG-CCG	GCCTCGAGGAGCAGTCAGGGCCGGGAGCATCAGCCCTTTGC CTTCTCCCTCAGCATCTACACGACATTCACTGAGGACACACC CTCGGTGGGCCAGCGCCTCCTCAACTCCGTGCTGAACA	4324
	TGTTCAGCACGGAGTTGAGGAGGCGCTGGCCCACCGAGGGT GTGTCCTCAGTGAATGTCGTGTAGATGCTGAGGGAGAAGGCA AAGGGCTGATGCTCCCGGCCCTGACTGCTCCTCGAGGC	4325
	GCATCTACACGACATTC	4326
	GAATGTCGTGTAGATGC	4327
Alzheimer disease Asn141Ile AAC-ATC	ACACGCCATTCACTGAGGACACACCCTCGGTGGGCCAGCGC CTCCTCAACTCCGTGCTGAACACCCTCATCATGATCAGCGTC ATCGTGGTTATGACCATCTTCTTGGTGGTGCTCTACAA	4328
	TTGTAGAGCACCACCAAGAAGATGGTCATAACCACGATGACG CTGATCATGATGAGGGTGTTCAGCACGGAGTTGAGGAGGCG CTGGCCCACCGAGGGTGTGTCCTCAGTGAATGGCGTGT	4329
	CGTGCTGAACACCCTCA	4330
	TGAGGGTGTTCAGCACG	4331
Alzheimer disease Met239Ile ATGg-ATA	CCACTGGAAGGGCCCTCTGGTGCTGCAGCAGGCCTACCTCAT CATGATCAGTGCGCTCATGGCCCTAGTGTTCATCAAGTACCTC CCAGAGTGGTCCGCGTGGGTCATCCTGGGCGCCATC	4332
	GATGGCGCCCAGGATGACCCACGCGGACCACTCTGGGAGGT ACTTGATGAACACTAGGGCCATGAGCGCACTGATCATGATGA GGTAGGCCTGCTGCAGCACCAGAGGGCCCTTCCAGTGG	4333
	GCGCTCATGGCCCTAGT	4334
	ACTAGGGCCATGAGCGC	4335
Alzheimer disease Met239Val cATG-GTG	ATCCACTGGAAGGGCCCTCTGGTGCTGCAGCAGGCCTACCTC ATCATGATCAGTGCGCTCATGGCCCTAGTGTTCATCAAGTAC CTCCCAGAGTGGTCCGCGTGGGTCATCCTGGGCGCCA	4336
	TGGCGCCCAGGATGACCCACGCGGACCACTCTGGGAGGTAC TTGATGAACACTAGGGCCATGAGCGCACTGATCATGATGAGG TAGGCCTGCTGCAGCACCAGAGGGCCCTTCCAGTGGAT	4337
	GTGCGCTCATGGCCCTA	4338

Clinical Phenotype & Mutation	Correcting Oligos	SEQ ID NO:
	TAGGGCCATGAGCGCAC	4339

EXAMPLE 26

Plant Cells

The oligonucleotides of the invention can also be used to repair or direct a mutagenic event in plants and animal cells. Although little information is available on plant mutations amongst natural cultivars, the oligonucleotides of the invention can be used to produce "knock out" mutations by modification of specific amino acid codons to produce stop codons (e.g., a CAA codon specifying Gln can be modified at a specific site to TAA; a AAG codon specifying Lys can be modified to UAG at a specific site; and a CGA codon for Arg can be modified to a UGA codon at a specific site). Such base pair changes will terminate the reading frame and produce a defective truncated protein, shortened at the site of the stop codon. Alternatively, frameshift additions or deletions can be directed into the genome at a specific sequence to interrupt the reading frame and produce a garbled downstream protein. Such stop or frameshift mutations can be introduced to determine the effect of knocking out the protein in either plant or animal cells.

All publications and patent applications cited in this specification are herein incorporated by reference as if each individual publication or patent application were specifically and individually indicated to be incorporated by reference. Although the foregoing invention has been described in some detail by way of illustration and example for purposes of clarity of understanding, it will be readily apparent to those of ordinary skill in the art in light of the teachings of this invention that certain changes and modifications may be made thereto without departing from the spirit or scope of the appended claims.

WHAT IS CLAIMED IS:

1. An oligonucleotide for targeted alteration(s) of genetic sequence, comprising a single-stranded oligonucleotide having a DNA domain, said DNA domain having at least one mismatch with respect to the genetic sequence to be altered, and further comprising chemical modifications within the oligonucleotide, said targeted alteration(s) occurring more frequently than alteration(s) of the genetic sequence by a double-stranded double hairpin chimeric oligonucleotide containing RNA and DNA nucleotides.

2. The oligonucleotide according to claim one that comprises at least one phosphorothioate linkage within the oligonucleotide.

3. The oligonucleotide according to claim one that comprises a 2'-O-methyl analog.

4. The oligonucleotide according to claim one that comprises a locked nucleotide analog.

5. The oligonucleotide according to claim one that comprises a combination of at least two modifications selected from the group of a phosphorothioate linkage, a 2'-O-methyl analog, a locked nucleotide analog and a ribonucleotide.

6. The oligonucleotide according to any one of claims 1 to 5 that comprises at least one unmodified ribonucleotide.

7. The oligonucleotide according to any one of claims 1 to 6, wherein the sequence of said oligonucleotide is selected from the group consisting of SEQ ID NOS: 1-4339.

8. A method of targeted alteration of genetic material, comprising combining the target genetic material with an oligonucleotide according to any one of claims 1 to 7 in the presence of purified proteins.

9. A method of targeted alteration of genetic material, comprising administering to a cell extract an oligonucleotide of any one of claims 1 to 7.

10. A method of targeted alteration of genetic material, comprising administering to a cell an oligonucleotide of any one of claims 1 to 7.

11. A method of targeted alteration of genetic sequence in a subject, comprising administering to the subject an oligonucleotide of any one of claims 1 to 7.

12. A method of targeted alteration of genetic sequence, comprising combining target genetic material with an oligonucleotide according to any one of claims 1 to 7, said target genetic material being a non-transcribed DNA strand of a duplex DNA.

13. The genetic material obtained by any one of the methods of claim 8, 9 or claim 10.

14. A cell comprising the genetic material of claim 13.

15. A non-human organism comprising the cell according to claim 14.

16. A pharmaceutical composition comprising the oligonucleotide according to any one of claims 1 to 7.

17. A method of targeted chromosomal genomic alteration, comprising administering the pharmaceutical composition of claim 16 to a subject.

18. A non-human organism produced by the method of claim 11 or claim 17.

19. A method of optimizing an oligonucleotide for targeted alteration of a genetic sequence, which comprises:

(a) comparing the efficiency of alteration of a targeted genetic sequence by an oligonucleotide of any one of claims 1 to 7 with the efficiency of alteration of the same targeted genetic sequence by a second oligonucleotide, said second oligonucleotide selected from the group of (1) an oligonucleotide that is fully complementary to the target and lacks the mismatch, (2) a fully modified phosphorothiolated oligonucleotide, (3) a fully modified 2'-O-methylated oligonucleotide and (4) a chimeric double-stranded double hairpin containing RNA and DNA nucleotides.

20. The method of claim 19 in which the alteration is produced in a cell extract.

21. The method of claim 20 in which the cell extract is selected from the group of a fungal cell extract, a plant cell extract, a rodent cell extract, a primate cell extract and a human cell extract.

22. The method of claim 19 in which the alteration is produced in a cell.

23. The method of claim 21 in which the cell is selected from the group of a fungal cell, a plant cell, a rodent cell, a primate cell and a human cell.

24. A kit comprising the oligonucleotide according to any one of claims 1 to 7 and a second oligonucleotide selected from the group of (1) an oligonucleotide that is fully complementary to the target and lacks the mismatch, (2) a fully modified phosphorothiolated oligonucleotide, (3) a fully modified 2-O-methylated oligonucleotide and (4) a chimeric double stranded double hairpin containing RNA and DNA nucleotides.