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(54) Titre : PROCEDES DE MODULATION DE L'AUTOPHAGIE PAR LA MODULATION DE PRODUITS GENIQUES RENFORCANT L'AUTOPHAGIE

(54) Title: METHODS FOR MODULATION OF AUTOPHAGY THROUGH THE MODULATION OF AUTOPHAGY-ENHANCING GENE PRODUCTS

(57) Abrégé/Abstract:
The present disclosure relates to methods for the modulation of autophagy and the treatment of autophgy-related diseases, including cancer, neurodegenerative diseases and pancreatitis.

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(54) Title: METHODS FOR MODULATION OF AUTOPHAGY THROUGH THE MODULATION OF AUTOPHAGY-ENHANCING GENE PRODUCTS

(57) Abstract: The present disclosure relates to methods for the modulation of autophagy and the treatment of autophgy-related diseases, including cancer, neurodegenerative diseases and pancreatitis.

METHODS FOR MODULATION OF AUTOPHAGY THROUGH THE MODULATION OF AUTOPHAGY-ENHANCING GENE PRODUCTS

RELATED APPLICATIONS

This application claims the benefit of priority to United States Provisional Patent Application serial number 61/247251, filed September 30, 2009 and United States Provisional Patent Application serial number 61/247309, filed September 30, 2009; which are hereby incorporated by reference in their entirety.

GOVERNMENT SUPPORT

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BACKGROUND

Autophagy is a catabolic process that mediates the turnover of intracellular constituents in a lysosome-dependent manner (Levine and Klionsky, (2004) Dev Cell 6, 463-377). Autophagy is initiated by the formation of an isolation membrane, which expands to engulf a portion of the cytoplasm to form a double membrane vesicle called the autophagosome. The autophagosome then fuses with a lysosome to form an autolysosome, where the captured material and the inner membrane are degraded by lysosomal hydrolases. Autophagy is therefore critical for the clearance of large protein complexes and defective organelles, and plays an important role in cellular growth, survival and homeostasis.

Autophagy has been primarily studied in unicellular eukaryotes, where it is known to be critical for survival of starvation conditions. When a unicellular eukaryote is cultured under conditions of nutrient deprivation, products of autophagic degradation, such as amino acids, fatty acids and nucleotides, can be used by the cell as structural components and as sources of energy (Levine and Klionsky, (2004) Dev Cell 6, 463-377; Levine and Kroemer, (2008), Cell *132*, 27-42).

Cells in complex, multicellular eukaryotes, such as mammals, rarely experience nutrient deprivation under normal physiological conditions. However, when such cells undergo nutrient deprivation or cellular stress, autophagy is often upregulated, which enhances cell survival. Because of their rapid growth and genetic instability, cancer cells are more reliant on autophagy for survival and growth than untransformed cells (Ding *et al.*, (2009), Mol. Cancer Ther., *8*(7), 2036-2045). Additionally, autophagy is frequently

activated as a survival mechanism in cancer cells in response to the cellular stress caused by chemotherapeutic agents. Autophagy inhibitors therefore can act as anti-cancer therapeutic agents either alone or in combination with other cancer treatments (Maiuri *et al.*, (2007) Nat. Rev. Cell Biol. *8*, 741-752; Amaravadi *et al.,* (2007) J. Clin. Invest. *117*, 326-336).

In addition to its role in responding to cellular stress, autophagy is an important intracellular mechanism for the maintenance of cellular homeostasis through the turnover of malfunctioning, aged or damaged proteins and organelles (Levine and Kroemer, (2008), Cell *132*, 27-42). As a result, reduced levels of autophagy contribute to neurodegeneration by increasing the accumulation of misfolded proteins (Hara *et al.,* (2006), Nature, *441*, 885-889; Komatsu *et al.*, (2006), Nature, *441*, 880-884). Upregulation of autophagy has been demonstrated to reduce both the levels of aggregated proteins and the symptoms of neurodegenerative diseases (Rubinsztein *et al.,* (2007), Nat. Rev. Drug Discov. *6,* 304-312). Agents that enhance cellular autophagy therefore can act as therapeutic agents for the prevention or treatment of neurodegenerative diseases.

In addition to cancer and neurodegeneration, modulation of autophagy is a therapeutic strategy in a wide variety of additional diseases and disorders. For example, several liver diseases, cardiac diseases and muscle diseases are correlated with the accumulation of misfolded protein aggregates. In such diseases, agents that increase cellular autophagy may enhance the clearance of disease-causing aggregates and thereby contribute to treatment and reduce disease severity (Levine and Kroemer, (2008), Cell, *132*, 27-42). Additionally, elevated levels of autophagy have also been observed in pancreatic diseases, and have been demonstrated to be an early event in the progression of acute pancreatitis (Fortunato and Kroemer, (2009), Autophagy, *5*(6)). Inhibitors of autophagy may, therefore, function as therapeutic agents in the treatment of pancreatitis.

There is therefore abundant evidence indicating that modulation of autophagy is a useful approach for the treatment of a wide range of diseases and disorders. However, because the genes and pathways responsible for the regulation of mammalian autophagy are poorly understood, there are few validated autophagy regulators that can serve as targets for the development of new therapeutic agents and methods for the treatment of such diseases. Accordingly, there is great need for new methods for the modulation of autophagy and treatment of autophagy-associated diseases.

SUMMARY

The present invention provides novel methods for the modulation of autophagy and the treatment of autophagy-related diseases, including cancer, neurodegenerative diseases, liver diseases, muscle diseases and pancreatitis. In order to identify the methods of the present invention, a high-throughput image-based genome-wide screen of a human siRNA library was used to identify 236 autophagy-related genes. These genes were extensively characterized using a combination of high-throughput assays, low-throughput assays and bioinformatics analysis. Based on the results of these studies, biological and pharmaceutical agents useful in the modulation of these genes and their gene products were identified and novel methods for the modulation of autophagy and the treatment of autophagy-related diseases were developed.

In some embodiments, the invention relates to methods of inducing autophagy in a cell comprising contacting the cell with an agent that inhibits the activity of a product of an autophagy-inhibiting gene of the invention. In certain embodiments, the autophagy-inhibiting gene is selected from the genes listed in Table 1, Table 3, Table 5, Table 7, Figure 14, Figure 15, Figure 39, Figure 44, and/or Figure 55. In other embodiments, the autophagy-inhibiting gene is TRPM3, TMPRSS5, IRAK3, ADMR, FGFR1, UNC13B, PTGER2, AGER, BGN, GABBR2, PPARD, GHSR, BAIAIP2, SORCS2, PAQR6, EPHA6, TRHR, C5AR1, BAI3, TLR3, PTPRH, ADRA1A, UTS2R, RORC, CHRND, TACR2, P2RX1, PLXNA2, PTPRU, FCER1A, CD300C, TNFRSF19L CLCF1, LIF, FGF2, SDF1 or IGF. In certain aspects of the invention, the agent is an antibody, a siRNA molecule, a shRNA molecule, and/or an antisense RNA molecule. In other aspects, the agent is TK1258, PF 04494700, PMX53, Tamsulosin, Doxazosin, Prazosin hydrochloride, alfuzosin hydrochloride, Urotensin II, Mecamylamine hydrochloride, ISIS 3521, Gemcitabine, LY900003, MK-5108, U73122 or D609.

Certain embodiments of the invention relate to methods of inhibiting autophagy in a cell comprising contacting the cell with an agent that inhibits the activity of a product of an autophagy-enhancing gene of the invention. In some embodiments, the autophagy-enhancing gene is selected from the genes listed in Table 2, Table 4 and/or Table 6. In other embodiments, the autophagy enhancing gene is TPR, GPR18, RelA or NFκB. In certain embodiments the agent is an antibody, a siRNA molecule, a shRNA molecule, and/or an antisense RNA molecule.

In certain aspects, the invention relates to methods of inhibiting autophagy in a cell comprising contacting the cell with an agent that enhances the activity of a product of an autophagy-inhibiting gene of the invention. In some embodiments, the autophagy-inhibiting gene is selected from the genes listed in Table 1, Table 3, Table 5, Table 7, Figure 14, Figure 15, Figure 39, Figure 44, and/or Figure 55. In other embodiments, the autophagy-inhibiting gene is TRPM3, TMPRSS5, IRAK3, ADMR, FGFR1, UNC13B, PTGER2, AGER, BGN, GABBR2, PPARD, GHSR, BAIAIP2, SORCS2, PAQR6, EPHA6, TRHR, C5AR1, BAI3, TLR3, PTPRH, ADRA1A, UTS2R, RORC, CHRND, TACR2, P2RX1, PLXNA2, PTPRU, FCER1A, CD300C, TNFRSF19L CLCF1, LIF, FGF2, SDF1 or IGF. In certain embodiments the agent is an antibody. In some embodiments the agent is FGF-1, acidic FGF-1, XRP0038, RhaFGF, GW501516, Ibutamoren Mesylate, KP-102LN, EP1572, TRH, S-0373, Poly-ICR, CQ-07001 or cryptotanshinone. In some embodiments the agent is a growth factor. In other embodiments, the growth factor is CLCF1, LIF, FGF2, SDF1 or IGF1.

Some embodiments of the invention relate to methods of inducing autophagy in a cell comprising contacting the cell with an agent that enhances the activity of a product of an autophagy-enhancing gene of the invention. In some embodiments, the autophagy-enhancing gene is selected from the genes listed in Table 2, Table 4 and/or Table 6. In other embodiments, the autophagy enhancing gene is TPR, GPR18, RelA or NFκB. In certain embodiments the agent is an antibody.

In some embodiments, the invention relates to methods of treating a neurodegenerative disease and/or a proteinopathy in a subject comprising administering to the subject an agent that inhibits the activity of a product of an autophagy-inhibiting gene of the invention. In certain embodiments, the autophagy-inhibiting gene is selected from the genes listed in Table 1, Table 3, Table 5, Table 7, Figure 14, Figure 15, Figure 39, Figure 44, and/or Figure 55. In other embodiments, the autophagy-inhibiting gene is TRPM3, TMPRSS5, IRAK3, ADMR, FGFR1, UNC13B, PTGER2, AGER, BGN, GABBR2, PPARD, GHSR, BAIAIP2, SORCS2, PAQR6, EPHA6, TRHR, C5AR1, BAI3, TLR3, PTPRH, ADRA1A, UTS2R, RORC, CHRND, TACR2, P2RX1, PLXNA2, PTPRU, FCER1A, CD300C, TNFRSF19L CLCF1, SDF1, LIF, FGF2 or IGF. In some embodiments, the agent is an antibody, a siRNA molecule, a shRNA molecule, and/or an antisense RNA molecule. In other embodiments, the agent is TK1258, PF 04494700, PMX53, Tamsulosin, Doxazosin, Prazosin hydrochloride, alfuzosin hydrochloride,

Urotensin II, Mecamylamine hydrochloride, ISIS 3521, Gemcitabine, LY900003, MK-5108, U73122 or D609.

Some embodiments of the invention relate to methods of treating a neurodegenerative disease and/or a proteinopathy in a subject comprising administering to the subject an agent that enhances the activity of a product of an autophagy-enhancing gene of the invention. In some embodiments, the autophagy-enhancing gene is selected from the genes listed in Table 2, Table 4 and/or Table 6. In other embodiments, the autophagy enhancing gene is TPR, GPR18, RelA or NFκB. In certain embodiments the agent is an antibody.

In certain embodiments, the neurodegenerative disease is Adrenal Leukodystrophy, alcoholism, Alexander's disease, Alper's disease, Alzheimer's disease, Amyotrophic lateral sclerosis, ataxia telangiectasia, Batten disease, bovine spongiform encephalopathy, Canavan disease, cerebral palsy, cockayne syndrome, corticobasal degeneration, Creutzfeldt-Jakob disease, familial fatal insomnia, frontotemporal lobar degeneration, Huntington's disease, HIV-associated dementia, Kennedy's disease, Krabbe's disease, Lewy body dementia, neuroborreliosis, Machado-Joseph disease, multiple system atrophy, multiple sclerosis, narcolepsy, Niemann Pick disease, Parkinson's disease, Pelizaeus-Merzbacher disease, Pick's disease, primary lateral sclerosis, prion diseases, progressive supranuclear palsy, Refsum's disease, Sandhoff disease, Schilder's disease, subacute combined degeneration of spinal cord secondary to pernicious anaemia, Spielmeyer-Vogt-Sjogren-Batten disease, spinocerebellar ataxia, spinal muscular atrophy, Steele-Richardson-Olszewski disease, Tabes dorsalis, toxic encephalopathy and combinations of these diseases. In some embodiments, the proteinopathy is α1-antitrypsin deficiency, sporadic inclusion body myositis, limb girdle muscular dystrophy type 2B and Miyoshi myopathy Alzheimer's disease, Parkinson's disease, Lewy Body Dementia, ALS, Huntington's disease, spinocerebellar ataxias, spinobulbar musclular atrophy and combinations of these diseases.

Certain embodiments of the invention relate to methods of treating cancer or pancreatitis in a subject comprising administering to the subject an agent that inhibits the activity of a product of an autophagy-enhancing gene of the invention. In some embodiments, the autophagy-enhancing gene is selected from the genes listed in Table 2, Table 4 and/or Table 6. In other embodiments, the autophagy enhancing gene is TPR, GPR18, RelA or NFκB. In certain embodiments the agent is an antibody, a siRNA molecule, a shRNA molecule, and/or an antisense RNA molecule.

In certain aspects, the invention relates to methods of treating cancer or pancreatitis in a subject comprising administering to the subject an agent that enhances the activity of a product of an autophagy-inhibiting gene of the invention. In some embodiments, the autophagy-inhibiting gene is selected from the genes listed in Table 1, Table 3, Table 5, Table 7, Figure 14, Figure 15, Figure 39, Figure 44, and/or Figure 55. In other embodiments, the autophagy-inhibiting gene is TRPM3, TMPRSS5, IRAK3, ADMR, FGFR1, UNC13B, PTGER2, AGER, BGN, GABBR2, PPARD, GHSR, BAIAIP2, SORCS2, PAQR6, EPHA6, TRHR, C5AR1, BAI3, TLR3, PTPRH, ADRA1A, UTS2R, RORC, CHRND, TACR2, P2RX1, PLXNA2, PTPRU, FCER1A, CD300C, TNFRSF19L CLCF1, SDF1, LIF, FGF2 or IGF. In certain embodiments the agent is an antibody. In some embodiments the agent is FGF-1, acidic FGF-1, XRP0038, RhaFGF, GW501516, Ibutamoren Mesylate, KP-102LN, EP1572, TRH, S-0373, Poly-ICR, CQ-07001 or cryptotanshinone. In some embodiments the agent is a growth factor. In more specific embodiments, the growth factor is CLCF1, LIF, FGF2, SDF1 or IGF1.

In some embodiments, the methods of treating cancer further comprise known cancer treatment therapies such as the administration of a chemotherapeutic agent and/or radiation therapy. In certain embodiments the chemotherapeutic agent is altretamine, asparaginase, BCG, bleomycin sulfate, busulfan, camptothecin, carboplatin, carmusine, chlorambucil, cisplatin, claladribine, 2-chlorodeoxyadenosine, cyclophosphamide, cytarabine, dacarbazine imidazole carboxamide, dactinomycin, daunorubicin - dunomycin, dexamethosone, doxurubicin, etoposide, floxuridine, fluorouracil, fluoxymesterone, flutamide, fludarabine, goserelin, hydroxyurea, idarubicin HCL, ifosfamide, interferon α, interferon α 2a, interferon α 2b, interfereon α n3, irinotecan, leucovorin calcium, leuprolide, levamisole, lomustine, megestrol, melphalan, L-sarcosylin, melphalan hydrochloride, MESNA, mechlorethamine, methotrexate, mitomycin, mitoxantrone, mercaptopurine, paclitaxel, plicamycin, prednisone, procarbazine, streptozocin, tamoxifen, 6-thioguanine, thiotepa, topotecan, vinblastine, vincristine or vinorelbine tartrate.

Other embodiments of the invention relate to methods of determining whether an agent is an autophagy inhibitor comprising the step of contacting a cell with the agent, wherein the cell expresses a heterologous autophagy-enhancing gene of the invention, whereby a reduction in autophagy in the cell indicates that the agent is an autophagy inhibitor. In certain aspects, the agent is a small molecule, an antibody, or an inhibitory RNA molecule.

Certain embodiments of the invention relate to methods of determining whether an agent is an autophagy inhibitor, the method comprising the step of contacting a cell with the agent, wherein the expression of an autophagy-inhibiting gene of the invention is inhibited in the cell, whereby a reduction in autophagy in the cell indicates that the agent is an autophagy inhibitor. In certain aspects, the agent is a small molecule, an antibody, or an inhibitory RNA molecule. In some embodiments the cell contains a mutation to the autophagy-related gene. In other embodiments the autophagy-related gene is inhibited by an inhibitory RNA or small molecule.

BRIEF DESCRIPTION OF THE DRAWINGS

Figure 1A shows fluorescent microscope images depicting the localization of GFP expressed in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR or Atg5. **Figure 1B** shows the results of a western blot performed using antibodies specific for either LC3 or tubulin and lysates of H4 cells that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR or Atg5.

Figure 2 shows the quantification of the level of autophagosome-associated GFP in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR or Atg5. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant.

Figure 3 shows the gene symbols, Unigene ID numbers, Genbank accession numbers and names of the autophagy-modulating genes of the invention.

Figure 4 shows a schematic diagram depicting a selection of the screens and characterization assays used to identify and characterize the autophagy-modulating genes of the invention.

Figure 5 shows the quantification of a series of in-cell-western blot assays that measure mTORC1 activity. The asterisks indicate that the difference between the indicated samples and the ntRNA control samples is statistically significant.

Figure 6 shows the gene symbols, Unigene ID numbers, Genbank accession numbers and names of the genes for which the inhibition of their product results in reduced expression of mTORC.

Figure 7 shows the gene symbols, Unigene ID numbers, and names of the genes for which the inhibition of their product results in both reduced expression of mTORC and down-regulation of autophagy in the presence of rapamycin.

Figure 8A shows fluorescent microscope images depicting the localization of RFP expressed in H4 cells that stably express Lamp1-RFP and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR. **Figure 8B** shows the quantification of the level of autophagosome-associated RFP in H4 cells that stably express LC3-GFP and that were transfected with non-targeting control siRNA (ntRNA) or siRNA against mTOR or Atg5. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant.

Figure 9 shows the gene symbols, Unigene ID numbers, Genbank accession numbers and names of the genes for which the inhibition of their product result in a significant change in the levels of autophagosome-associated Lamp1-RFP in Lamp1-RFP expressing cells.

Figure 10A shows fluorescent microscope images depicting the localization of dsRed expressed in H4 cells that stably express FYVE-dsRed and that were transfected with siRNA against Vprs34 or mTOR. **Figure 10B** shows the quantification of the level of autophagosome-associated dsRed in H4 cells that stably express FYVE-dsRed and that were transfected with siRNA against Vprs34 or mTOR. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant. **Figure 10C** shows the quantification of the level of autophagosome-associated dsRed in H4 cells that stably express FYVE-dsRed and that were transfected with siRNA against Raptor or mTOR.

Figure 11 shows the gene symbols, Unigene ID numbers, Genbank accession numbers and names of the genes for which the inhibition of their product results in a significant change in the levels of PtdIns3P levels.

Figure 12 shows a Venn diagram depicting the subdivision of genes for which the inhibition of their products led to the induction of autophagy into functional categories based on their dependence on type III PI3 kinase activity, lysosomal function and mTORC1 activity.

Figure 13 shows the relative average viability of wild-type H4 cells transfected with autophagy-related gene targeting siRNAs (H4) compared to Bcl-2 expressing H4 cells

transfected with autophagy-related gene targeting siRNAs (H4 + Bcl-2). The asterisks indicate statistical significance.

Figure 14 shows the relative viability, gene symbols, Unigene ID numbers, and names of the genes for which the inhibition of their product results in enhancement of autophagy in Bcl-2 expressing cells.

Figure 15 shows the relative viability, gene symbols, Unigene ID numbers, and names of the genes for which the inhibition of their product results in enhancement of autophagy wild-type, but not in Bcl-2 expressing cells.

Figure 16 shows the quantification of in-cell western assays demonstrating an increase in the levels of GRP78 and GRP94 in H4 cells treated with tunicamycin. The asterisks indicate statistical significance.

Figure 17 shows the gene symbols, Unigene ID numbers, and names of the genes for which the inhibition of their product results in enhancement of autophagy and changes in Endoplasmic Reticulum (ER) stress levels.

Figure 18 shows a western blot depicting Bcl-2 expression in H4 LC3-GFP and H4 FYVE-dsRed cells following infection with pBabe-Bcl-2 retrovirus and puromycin selection.

Figure 19A shows the quantification of the level of autophagosome-associated GFP in H4 cells that stably express LC3-GFP and Bcl-2 and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant. **Figure 19B** shows the quantification of the level of autophagosome-associated dsRed in H4 cells that stably express FYVE-dsRed and Bcl-2 and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA against mTOR. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant. **Figure 19C** shows the quantification of the level of autophagosome-associated dsRed in H4 cells that stably express FYVE-dsRed and that were transfected with siRNA against autophagy-related gene products that either do not express Bcl-2 (H4) or express Bcl-2 (H4 + Bcl-2). The asterisks indicate that the difference between the indicated levels is statistically significant.

Figure 20 shows the subdivision of autophagy-related genes for which knock-down was able to induce autophagy under conditions of low PtdIns3P into functional categories

based on their ability to up-regulate type III PI3 kinase activity or to alter lysosomal function.

Figure 21A shows how selected autophagy-related gene products of the invention are associated with specific protein complexes. **Figure 21B** shows how selected autophagy-related gene products of the invention are associated with a network of transcription factors and chromatin modifying enzymes.

Figure 22 shows how selected autophagy-related gene products of the invention interact with core autophagic machinery.

Figure 23 shows how selected autophagy-related gene products of the invention interact within axon-guidance regulatory pathways.

Figure 24 shows how selected autophagy-related gene products of the invention interact within actin-cytoskeleton regulatory pathways.

Figure 25A shows the subdivision of the autophagy-related genes of the invention into molecular function categories. **Figure 25B** shows the further subdivision of the autophagy-related genes of the invention that are categorized as receptors in Figure 25A into receptor categories.

Figure 26 shows the molecular function categories, gene symbols, Unigene ID numbers and gene names of autophagy-related genes of the invention.

Figure 27A shows the subdivision of the autophagy-related genes of the invention into biological process categories. **Figure 27B** shows the further subdivision of the autophagy-related genes of the invention that are categorized as mediators of signal transduction in Figure 27A into signal transduction categories.

Figure 28 shows the quantification of autophagosome associated GFP in H4 LC3-GFP cells grown in the presence of the indicated growth factors (IGF1, FGF2, LIF, CLCF1 and SDF1). The asterisk indicates that the difference between the indicated level and that of the untreated cells is statistically significant.

Figure 29 shows fluorescent microscope images depicting the localization of GFP expressed in H4 cells that stably express LC3-GFP and that were either untreated under conditions of nutrient deprivation (untreated), untreated under normal growth conditions (serum), or treated with CLCF1, LIF, FGF2 or IGF1 under conditions of nutrient deprivation (CLCF1, LIF, FGF2 and IGF, respectively).

Figure 30 shows that cytokines are able to suppress autophagy in the absence and presence of rapamycin. H4 cells were grown in serum-free medium, followed by addition

of 100ng/mL IGF1 (**A**), 50ng/mL FGF2 (**B**), 50ng/mL LIF (**C**) or 50ng/mL CLCF1 (**D**) and 10μg/mL E64d (**E**). Where indicated, cells were pre-treated with 50 nM rapamycin 1 hour prior to the addition of cytokines. Levels of autophagy were assessed by western blot using antibody against LC3; mTORC1 activity was evaluated with antibodies against phospho-S6 (Ser235/236, P-S6) and phospho-S6 kinase (Thr389, P-S6K). Quantification of LC3 II/tubulin ratio is shown.

Figure 31A shows the quantification of autophagosome associated GFP in H4 LC3-GFP cells grown in the presence of 5, 20, 100 or 200 ng/ml of TNFα or the presence of rapamycin. The asterisks indicate that the difference between the indicated level and that of the untreated cells is statistically significant. **Figure 31B** shows western blots depicting the levels of p62 in H4 cells that were either untreated under conditions of nutrient deprivation (-), untreated under normal growth conditions (serum), treated with rapamycin (Rap), or treated with 5 ng/ml of TNFα under conditions of nutrient deprivation.

Figure 32 shows fluorescent microscope images depicting the localization of GFP expressed in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or four distinct siRNAs specific for RelA.

Figure 33 shows the quantification of the level of autophagosome-associated GFP in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or four distinct siRNAs specific for RelA. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant.

Figure 34A shows the results of semi-quantitative RT-PCR detecting the level of RelA mRNA H4 cells that were transfected with non-targeting, control siRNA (ntRNA) or one of four distinct siRNAs specific for RelA. **Figure 34B** shows the results a western blot detecting the level of p65 in H4 cells that were transfected with non-targeting, control siRNA (ntRNA), one of four distinct siRNAs specific for RelA, or a pool of the four RelA specific siRNAs.

Figure 35A shows western blots depicting the levels of RelA and LC3 in wild-type H4 cells (wt) and $RelA^{-/-}$ and $NF\kappa B^{-/-}$ double knock-out (DKO) H4 cells. **Figure 35B** shows western blots depicting the levels of RelA, p62 and LC3 in H4 cells that have been transfected with siRNAs specific for RelA, non-targeting siRNA (nt), mTor or Atg5.

Figure 36A shows FACS histograms depicting the levels of reactive oxygen species in wild-type H4 cells and $RelA^{-/-}$ and $NF\kappa B^{-/-}$ double knock-out (DKO) H4 cells under

normal growth conditions (mock) and conditions of nutrient deprivation (starvation). **Figure 36B** shows the quantification of the data depicted in Figure 36A. **Figure 36C** shows the quantification of the levels of reactive oxygen species in H4 cells transfected with non-targeting, control siRNA (ntRNA) or siRNAs specific for RelA grown under normal (+ serum) or starvation (HBSS) conditions.

Figure 37 shows the quantification of the level of autophagosome-associated GFP in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or siRNAs specific for RelA grown under conditions of nutrient deprivation and either in the presence of antioxidant (NAC) or absence of antioxidant.

Figure 38 shows the gene symbols, Unigene ID numbers and prediction basis for the autophagy-related genes of the invention whose products are predicted to be localized to the mitochondria.

Figure 39 shows the gene symbols, Unigene ID numbers and names of autophagy-related genes of the invention with known connections to oxidative damage or the regulation of reactive oxygen species.

Figure 40A shows western blots depicting the levels of SOD1, p62 and LC3 in H4 cells that were transfected with non-targeting, control siRNA (nt) or siRNA specific for SOD1. **Figure 40B** shows fluorescent microscope images depicting the levels of reactive oxygen species in cells transfected with non-targeting, control siRNA (nt) or siRNA specific for SOD1 or treated with 100 mM TBHP. **Figure 40C** shows the quantification of the levels of reactive oxygen species in cells transfected with non-targeting, control siRNA (nt) or siRNA specific for SOD1. The asterisks indicate that the difference between the indicated level and that of the ntRNA transfected cells is statistically significant.

Figure 41 shows the quantification of the level of autophagosome-associated GFP in H4 cells that stably express LC3-GFP and that were transfected with non-targeting, control siRNA (ntRNA) or siRNA specific for mTOR or SOD1 either in the presence of antioxidant (NAC) or absence of antioxidant (-).

Figure 42 shows the gene symbol, Unigene ID number and name of genes for which the inhibition of their product results in enhancement of autophagy in the absence but not in the presence of antioxidant.

Figure 43 shows the quantification of the average type III PI3 kinase activity following inhibition of the products of the autophagy-related genes of the invention able (yes) or unable (no) to induce autophagy in the presence of antioxidant (NAC).

Figure 44 shows the gene symbol, Unigene ID number and name of genes for which the inhibition of their product results in enhancement of autophagy in the presence of antioxidant.

Figure 45 shows an enrichment analysis of canonical pathways (MSigDB) among the hit genes relative to all genes examined in the screen. A p-value<0.05 (hyper geometric distribution) is considered significant. Only categories with at least five genes are displayed.

Figure 46 shows that down-regulation of autophagy by 50ng/mL FGF2 is prevented by addition of MEK inhibitor UO126. H4 cells were grown in serum-free media, levels of autophagy were assessed in the presence of 10μg/mL E64d, with antibodies against LC3, inhibition MEK with phospho-ERK 1/2, phospho-RSK and phospho-S6 (Ser235/236). Quantification of LC3 II/tubulin ratio is shown.

Figure 47 shows, an enrichment analysis of cis-regulatory elements/transcription factor (TF)-binding sites in the promoters of the hit genes, using motif-based gene sets from MSigDB and TF-binding sites defined in the TRANSFAC database. SRF sites are highlighted.

Figure 48 shows a western-blot depicting the phosphorylation of Stat3 following treatment with 50ng/mL CLCF1.

Figure 49 shows that the down-regulation of autophagy by 50ng/mL LIF is prevented by siRNA mediated knock-down of Stat3. H4 cells were transfected with indicated siRNAs for 72h, than cells were treated as described for Figure 46. Protein levels and phosphorylation of Stat3 are shown.

Figure 50 shows that suppression of autophagy by 100ng/mL IGF1 is prevented by Akt inhibitor VIII. Cells were treated as described for Figure 46. Akt activity was assessed with antibodies against phospho-Foxo3a and phospho-rpS6.

Figure 51 shows a clustering analysis of mRNA expression levels of select autophagy hit genes in young (≤ 40 years-old) or old (≥ 70 years old) human brain samples.

Figure 52 shows a correlation matrix for the data presented in Figure 45.

Figure 53 shows a clustering analysis (dChip) of mRNA expression levels of select autophagy hit genes in young (≤ 40 years-old) or old (≥ 70 years old) human brain samples.

Figure 54 shows a correlation matrix for autophagy-related genes of the invention with the most significant age-dependent regulation.

Figure 55 shows the gene symbol, Unigene ID number, fold change and p value of autophagy-related genes of the invention that are differentially regulated in human brains during aging.

Figure 56 shows the expression levels of autophagy-related genes of the invention during aging.

Figure 57 shows that differential gene expression leads to up regulation of autophagy in Alzheimer's disease. Forrest plots of Normalized Enrichment Score (NES) estimates with standard deviation for the screen hit gene sets are shown. **Figure 57A** shows a GSEA analysis of overall screen hit gene expression in different regions of AD brain as compared to unaffected age-matched controls. **Figures 57B and 57C** show GSEA analysis of hit genes determined to function as negative (**B**) or positive (**C**) regulators of autophagy flux. The size of a square is inversely proportional to the respective SD.

Figure 58 shows a comparison of the levels of LC3-II accumulation in the presence or absence of 10 μM E64d following treatment of H4 cells with 5 μM Aβ.

Figure 59 shows that Aβ induces accumulation of PtdIns3P. FYVE-dsRed cells were prepared as described in Figure 58, fixed and imaged. Where indicated the type III PI3 kinase inhibitor 3MA (10mM) was added for 8 hours prior to fixation.

Figure 60 shows that the induction of the type III PI3 kinase activity by Aβ is suppressed in the presence of antioxidant. Cells were prepared as described in Figure 59 and treated with or without antioxidant NAC.

Figure 61 shows that the induction of autophagy by Aβ is dependent on the type III PI3 kinase activity. H4 GFP-LC3 cells were treated and imaged as described for Figure 59.

Figure 62 shows that the induction of autophagy by Aβ is dependent on the type III PI3 kinase activity. H4 cells were transfected with siRNA against the type III PI3 kinase subunit Vps34 or non-targeting control siRNA and than treated as described in Figure 59. Autophagy and lysosomal changes were determined using antibodies against LC3 and Lamp 2, respectively.

Figure 63 shows the chemical structures of select small molecule agents that modulate activity of autophagy-related genes of the invention.

Figure 64 shows the Genbank accession numbers, names, gene symbols and mRNA sequences of the autophagy-related genes of the invention.

DETAILED DESCRIPTION

Autophagy is a lysosome-dependent catabolic process that mediates turnover of cellular components and protects multicellular eukaryotes from a wide range of diseases. In order to develop new methods for the modulation of autophagy and the treatment of autophagy-related diseases, a high-throughput image-based genome-wide screen of a human siRNA library was performed to identify genes involved in autophagy modulation and regulation. This screen led to the identification of 236 autophagy-related genes that, when knocked-down, led to either an increase or a decrease in levels of autophagy under normal nutrient conditions. The autophagy-related genes of the invention are listed in Figure 3. These genes were extensively characterized using a combination of high-throughput assays, low-throughput assays and bioinformatics analysis. Based on the results of these studies, biological and pharmaceutical agents useful in the modulation of these genes and their gene products were identified and novel methods for the modulation of autophagy and the treatment of autophagy-related diseases were identified. The present invention, therefore, provides novel methods for the modulation of autophagy and the treatment of autophagy-related diseases, including cancer, neurodegenerative diseases, liver diseases, muscle diseases and pancreatitis.

1. Definitions

In order for the present invention to be more readily understood, certain terms and phrases are defined below and throughout the specification.

The articles "*a*" and "*an*" are used herein to refer to one or to more than one (*i.e.*, to at least one) of the grammatical object of the article. By way of example, "an element" means one element or more than one element.

As used herein, the term "*administering*" means providing a pharmaceutical agent or composition to a subject, and includes, but is not limited to, administering by a medical professional and self-administering.

As used herein, the term "*agent*" refers to an entity capable of having a desired biological effect on a subject or cell. A variety of therapeutic agents is known in the art and may be identified by their effects. Examples of therapeutic agents of biological origin include growth factors, hormones, and cytokines. A variety of therapeutic agents is known in the art and may be identified by their effects. Examples include small molecules (*e.g.,* drugs), antibodies, peptides, proteins (*e.g.,* cytokines, hormones, soluble receptors and nonspecific-proteins), oligonucleotides (*e.g.,* peptide-coding DNA and RNA, double-stranded RNA and antisense RNA) and peptidomimetics.

As used herein, the term "*antibody*" includes full-length antibodies and any antigen binding fragment (*i.e.*, "antigen-binding portion") or single chain thereof. The term "antibody" includes, but is not limited to, a glycoprotein comprising at least two heavy (H) chains and two light (L) chains inter-connected by disulfide bonds, or an antigen binding portion thereof. Antibodies may be polyclonal or monoclonal; xenogeneic, allogeneic, or syngeneic; or modified forms thereof (*e.g.*, humanized, chimeric).

As used herein, the phrase "*antigen-binding portion*" of an antibody, refers to one or more fragments of an antibody that retain the ability to specifically bind to an antigen. The antigen-binding function of an antibody can be performed by fragments of a full-length antibody. Examples of binding fragments encompassed within the term "antigen-binding portion" of an antibody include (i) a Fab fragment, a monovalent fragment consisting of the V_H, V_L, CL and CH1 domains; (ii) a $F(ab')_2$ fragment, a bivalent fragment comprising two Fab fragments linked by a disulfide bridge at the hinge region; (iii) a Fd fragment consisting of the V_H and CH1 domains; (iv) a Fv fragment consisting of the V_H and V_L domains of a single arm of an antibody, (v) a dAb fragment (Ward *et al.*, (1989) Nature 341:544 546), which consists of a V_H domain; and (vi) an isolated complementarity determining region (CDR) or (vii) a combination of two or more isolated CDRs which may optionally be joined by a synthetic linker. Furthermore, although the two domains of the Fv fragment, V_H and V_L, are coded for by separate genes, they can be joined, using recombinant methods, by a synthetic linker that enables them to be made as a single protein chain in which the V_H and V_L regions pair to form monovalent molecules (known as single chain Fv (scFv); see *e.g.*, Bird *et al.* (1988) Science 242:423 426; and Huston *et al.* (1988) Proc. Natl. Acad. Sci. USA 85:5879 5883). Such single chain antibodies are also intended to be encompassed within the term "antigen-binding portion" of an antibody. These antibody fragments are obtained using conventional techniques known to those with skill in the art, and the fragments are screened for utility in the same manner as are intact antibodies.

As used herein, the term "*cancer*" includes, but is not limited to, solid tumors and blood borne tumors. The term cancer includes diseases of the skin, tissues, organs, bone, cartilage, blood and vessels. The term "cancer" further encompasses both primary and metastatic cancers.

As used herein, the phrases "*gene product*" and "*product of a gene*" refers to a substance encoded by a gene and able to be produced, either directly or indirectly, through

the transcription of the gene. The phrases "gene product" and "product of a gene" include RNA gene products (*e.g.* mRNA), DNA gene products (*e.g.* cDNA) and polypeptide gene products (*e.g.* proteins).

As used herein, the phrase "*enhancing the activity*" of a gene product refers to an increase in a particular activity associated with the gene product. Examples of enhanced activity include, but are not limited to, increased translation of mRNA, increased signal transduction by polypeptides or proteins and increased catalysis by enzymes. Enhancement of activity can occur, for example, through an increased amount of activity performed by individual gene products, through an increase number of gene products performing the activity, or a through any combination thereof. If a gene product enhances a biological process (*e.g.* autophagy), "enhancing the activity" of such a gene product will generally enhance the process. Conversely, if a gene product functions as an inhibitor of a biological process, "enhancing the activity" of such a gene product will generally inhibit the process.

As used herein, the phrase "*inhibiting the activity*" of a gene product refers to a decrease in a particular activity associated with the gene product. Examples of inhibited activity include, but are not limited to, decreased translation of mRNA, decreased signal transduction by polypeptides or proteins and decreased catalysis by enzymes. Inhibition of activity can occur, for example, through a reduced amount of activity performed by individual gene products, through a decreased number of gene products performing the activity, or a through any combination thereof. If a gene product enhances a biological process (*e.g.* autophagy), "inhibiting the activity" of such a gene product will generally inhibit the process. Conversely, if a gene product functions as an inhibitor of a biological process, "inhibiting the activity" of such a gene product will generally enhance the process.

As used herein, the term "*isolated*" refers to the state in which substances (*e.g.,* polypeptides or polynucleotides) are free or substantially free of material with which they are naturally associated such as other polypeptides or polynucleotides with which they are found in their natural environment or the environment in which they are prepared (*e.g.,* cell culture). Polypeptides or polynucleotides can be formulated with diluents or adjuvants and still be considered "isolated" - for example, polypeptides or polynucleotides can be mixed with pharmaceutically acceptable carriers or diluents when used in diagnosis or therapy.

As used herein, the term "*modulation*" refers to up regulation (*i.e.*, activation or stimulation), down regulation (*i.e.*, inhibition or suppression) of a biological activity, or the two in combination or apart.

As used herein, the phrases "*neurodegenerative disorder*" and "*neurodegenerative disease*" refers to a wide range of diseases and/or disorders of the central and peripheral nervous system, such as neuropathologies, and includes but is not limited to, Parkinson's disease, Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS), denervation atrophy, otosclerosis, stroke, dementia, multiple sclerosis, Huntington's disease, encephalopathy associated with acquired immunodeficiency disease (AIDS), and other diseases associated with neuronal cell toxicity and cell death.

As used herein, the phrase "*pharmaceutically acceptable*" refers to those agents, compounds, materials, compositions, and/or dosage forms which are, within the scope of sound medical judgment, suitable for use in contact with the tissues of human beings and animals without excessive toxicity, irritation, allergic response, or other problem or complication, commensurate with a reasonable benefit/risk ratio.

As used herein, the phrase "*pharmaceutically-acceptable carrier*" means a pharmaceutically-acceptable material, composition or vehicle, such as a liquid or solid filler, diluent, excipient, or solvent encapsulating material, involved in carrying or transporting an agent from one organ, or portion of the body, to another organ, or portion of the body. Each carrier must be "acceptable" in the sense of being compatible with the other ingredients of the formulation and not injurious to the patient. Some examples of materials which can serve as pharmaceutically-acceptable carriers include: (1) sugars, such as lactose, glucose and sucrose; (2) starches, such as corn starch and potato starch; (3) cellulose, and its derivatives, such as sodium carboxymethyl cellulose, ethyl cellulose and cellulose acetate; (4) powdered tragacanth; (5) malt; (6) gelatin; (7) talc; (8) excipients, such as cocoa butter and suppository waxes; (9) oils, such as peanut oil, cottonseed oil, safflower oil, sesame oil, olive oil, corn oil and soybean oil; (10) glycols, such as propylene glycol; (11) polyols, such as glycerin, sorbitol, mannitol and polyethylene glycol; (12) esters, such as ethyl oleate and ethyl laurate; (13) agar; (14) buffering agents, such as magnesium hydroxide and aluminum hydroxide; (15) alginic acid; (16) pyrogen-free water; (17) isotonic saline; (18) Ringer's solution; (19) ethyl alcohol; (20) pH buffered solutions; (21) polyesters, polycarbonates and/or polyanhydrides; and (22) other non-toxic compatible substances employed in pharmaceutical formulations.

As used herein, the phrase "*pharmaceutically-acceptable salts*" refers to the relatively non-toxic, inorganic and organic salts of compounds.

As used herein, the term "*subject*" means a human or non-human animal selected for treatment or therapy.

As used herein, the phrase "*subject suspected of having*" means a subject exhibiting one or more clinical indicators of a disease or condition. In certain embodiments, the disease or condition is cancer, a neurodegenerative disorder or pancreatitis.

As used herein, the phrase "*subject in need thereof*" means a subject identified as in need of a therapy or treatment of the invention.

As used herein, the phrase "*therapeutic effect*" refers to a local or systemic effect in animals, particularly mammals, and more particularly humans, caused by an agent. The phrases "*therapeutically-effective amount*" and "*effective amount*" mean the amount of an agent that produces some desired effect in at least a sub-population of cells. A therapeutically effective amount includes an amount of an agent that produces some desired local or systemic effect at a reasonable benefit/risk ratio applicable to any treatment. For example, certain agents used in the methods of the present invention may be administered in a sufficient amount to produce a reasonable benefit/risk ratio applicable to such treatment.

As used herein, the term "*treating*" a disease in a subject or "*treating*" a subject having or suspected of having a disease refers to subjecting the subject to a pharmaceutical treatment, *e.g.*, the administration of an agent, such that at least one symptom of the disease is decreased or prevented from worsening.

2. Autophagy-related genes

The autophagy-related genes of the present invention can be divided into genes whose products inhibit autophagy (or autophagy-inhibiting genes, listed in Table 1) and genes whose products enhance autophagy (or autophagy-enhancing genes, listed in Table 2).

Agents that modulate the activity of products of autophagy-inhibiting genes are useful in the treatment of autophagy-related diseases. Agents that inhibit the activity of the products of autophagy-inhibiting genes result in elevated autophagy levels and are therefore useful in methods of enhancing autophagy and the treatment of autophagy-related diseases that are responsive to elevated levels of autophagy, such as neurodegenerative diseases and proteinopathies. On the other hand, agents that enhance the activity of products of autophagy-inhibiting genes result in reduced autophagy levels, and are therefore useful in

methods of inhibition of autophagy and the treatment of autophagy-related diseases that are responsive to autophagy inhibition, such as cancer and pancreatitis.

Table 1. Autophagy-inhibiting genes.

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1
CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
XPO1	7514	NM_003400	exportin 1 (CRM1 homolog, yeast)
KIAA0133	9816	XM_375851	KIAA0133
ADMR	11318	NM_007264	adrenomedullin receptor
OGDH	4967	NM_002541	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
DDX24	57062	NM_020414	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
NUPR1	26471	NM_012385	nuclear protein 1
FXYD2	486	NM_001680	FXYD domain containing ion transport regulator 2
TRHR	7201	NM_003301	thyrotropin-releasing hormone receptor
SUV39H1	6839	NM_003173	suppressor of variegation 3-9 homolog 1 (Drosophila)
FCER1A	2205	NM_002001	Fc fragment of IgE, high affinity I, receptor for; alp polypeptide
PTPRU	10076	NM_005704	protein tyrosine phosphatase, receptor type, U
GPX2	2877	NM_002083	glutathione peroxidase 2 (gastrointestinal)
PRKCA	5578	NM_002737	protein kinase C, alpha
EP300	2033	NM_001429	E1A binding protein p300
LOC388959	388959	XM_373989	hypothetical LOC388959
NTN2L	4917	NM_006181	netrin 2-like (chicken)
DOCK8	81704	NM_203447	dedicator of cytokinesis 8

MAP3K7IP1	10454	NM_006116	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
PLAGL2	5326	NM_002657	pleiomorphic adenoma gene-like 2
NUDT1	4521	NM_002452	nudix (nucleoside diphosphate linked moiety X)-type motif 1
RELN	5649	NM_005045	reelin
PNKD	25953	NM_015488	paroxysmal nonkinesiogenic dyskinesia
RIPK1	8737	NM_003804	receptor (TNFRSF)-interacting serine-threonine kinase 1
GNG5	2787	NM_005274	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	NM_001277	choline kinase alpha
C5AR1	728	NM_001736	complement component 5a receptor 1
SCOTIN	51246	NM_016479	scotin
PIGY	84992	NM_032906	phosphatidylinositol glycan anchor biosynthesis, class Y
NAGK	55577	NM_017567	N-acetylglucosamine kinase
RAGE	5891	NM_014226	renal tumor antigen
USP24	23358	XM_165973	ubiquitin specific peptidase 24
AURKA	6790	NM_003600	aurora kinase A
PLDN	26258	NM_012388	pallidin homolog (mouse)
TLR3	7098	NM_003265	toll-like receptor 3
PPARD	5467	NM_006238	peroxisome proliferator-activated receptor delta
HRC	3270	NM_002152	histidine rich calcium binding protein
NNMT	4837	NM_006169	nicotinamide N-methyltransferase
COPB2	9276	NM_004766	coatomer protein complex, subunit beta 2 (beta prime)
CDK5RAP3	80279	NM_025197	CDK5 regulatory subunit associated protein 3
NLK	51701	NM_016231	nemo-like kinase
PFKL	5211	NM_002626	phosphofructokinase, liver
RNPEPL1	57140	NM_018226	arginyl aminopeptidase (aminopeptidase B)-like 1
EPHA6	203806	XM_114973	EPH receptor A6
CDCA8	55143	NM_018101	cell division cycle associated 8
CKAP5	9793	NM_014756	cytoskeleton associated protein 5

ZBTB16	7704	NM_006006	zinc finger and BTB domain containing 16
GABBR2	9568	NM_005458	gamma-aminobutyric acid (GABA) B receptor, 2
PTMA	5757	NM_002823	prothymosin, alpha (gene sequence 28)
PTCRA	171558	NM_138296	pre T-cell antigen receptor alpha
RORC	6097	NM_005060	RAR-related orphan receptor C
GNAI1	2770	NM_002069	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
UTS2R	2837	NM_018949	urotensin 2 receptor
MATN3	4148	NM_002381	matrilin 3
NPTX1	4884	NM_002522	neuronal pentraxin I
SP140	11262	NM_007237	SP140 nuclear body protein
SMARCD1	6602	NM_003076	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1
TRIM69	140691	NM_080745	tripartite motif-containing 69
CDKN2D	1032	NM_001800	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4)
PAK6	56924	NM_020168	p21(CDKN1A)-activated kinase 6
TACR2	6865	NM_001057	tachykinin receptor 2
MMP17	4326	NM_016155	matrix metallopeptidase 17 (membrane-inserted)
MUC3A	4584	XM_374502	mucin 3A, cell surface associated
PRKCZ	5590	NM_002744	protein kinase C, zeta
TNFRSF17	608	NM_001192	tumor necrosis factor receptor superfamily, memb 17
GTF2IRD2	84163	NM_173537	GTF2I repeat domain containing 2
TRPM3	80036	NM_020952 NM_000190,	transient receptor potential cation channel, subfamily M, member 3
HMBS	3145	NM_176954	hydroxymethylbilane synthase
CYP27A1	1593	NM_000784	cytochrome P450, family 27, subfamily A, polypeptide 1
FBXL20	84961	NM_032875	F-box and leucine-rich repeat protein 20
CD300C	10871	NM_006678	CD300c molecule
PSD	5662	NM_002779	pleckstrin and Sec7 domain containing

FRAG1	27315	NM_014489	FGF receptor activating protein 1
PCGF1	84759	NM_032673	polycomb group ring finger 1
SIX2	10736	NM_016932	sine oculis homeobox homolog 2 (Drosophila)
CLCN1	1180	NM_000083	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
EVL	51466	NM_016337	Enah/Vasp-like
TOM1	10043	NM_005488	target of myb1 (chicken)
BAIAP2	10458	NM_006340	BAI1-associated protein 2
ZFY	7544	NM_003411	zinc finger protein, Y-linked
UBE2D1	7321	NM_003338	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
KRT18	3875	NM_000224	keratin 18
GJA4	2701	NM_002060	gap junction protein, alpha 4, 37kDa
SF3A2	8175	NM_007165	splicing factor 3a, subunit 2, 66kDa
TRNT1	51095	NM_016000	tRNA nucleotidyl transferase, CCA-adding, 1
RANGAP1	5905	NM_002883	Ran GTPase activating protein 1
CCT4	10575	NM_006430	chaperonin containing TCP1, subunit 4 (delta)
TSPAN4	7106	NM_003271	tetraspanin 4
PTGER2	5732	NM_000956	prostaglandin E receptor 2 (subtype EP2), 53kDa
GTPBP4	23560	NM_012341	GTP binding protein 4
ADRA1A	148	NM_000680	adrenergic, alpha-1A-, receptor
PHB2	11331	NM_007273	prohibitin 2
TNFRSF19L	84957	NM_032871	tumor necrosis factor receptor superfamily, memb 19-like
COL14A1	7373	XM_044622	collagen, type XIV, alpha 1 (undulin)
CD79A	973	NM_001783	CD79a molecule, immunoglobulin-associated alpl
F12	2161	NM_000505	coagulation factor XII (Hageman factor)
ASMT	438	NM_004043	acetylserotonin O-methyltransferase
GRK6	2870	NM_002082	G protein-coupled receptor kinase 6
GNRH2	2797	NM_001501	gonadotropin-releasing hormone 2
SDHB	6390	NM_003000	succinate dehydrogenase complex, subunit B, iro sulfur (Ip)
THBS2	7058	NM_003247	thrombospondin 2

HIVEP2	3097	NM_145975, NM_006734	human immunodeficiency virus type I enhancer binding protein 2
WASF1	8936	NM_003931	WAS protein family, member 1
SSPN	8082	NM_005086	sarcospan (Kras oncogene-associated gene)
ITGAV	3685	NM_002210	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
PLXNA2	5362	XM_372810	plexin A2
IGF1	3479	NM_000618	insulin-like growth factor 1 (somatomedin C)
NCR3	259197	NM_147130	natural cytotoxicity triggering receptor 3
TH	7054	NM_000360	tyrosine hydroxylase
HMGCL	3155	NM_177229, NM_000191	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
CENPJ	55835	NM_018451	centromere protein J
FABP1	2168	NM_001443	fatty acid binding protein 1, liver
PRKAA2	5563	NM_006252	protein kinase, AMP-activated, alpha 2 catalytic subunit
CASP1	834	NM_001223	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
CAPN1	823	NM_005186	calpain 1, (mu/I) large subunit
MCCC1	56922	NM_020166	methylcrotonoyl-Coenzyme A carboxylase 1 (alph
RAB7A	7879	NM_004637	RAB7A, member RAS oncogene family
DBX1	120237	XM_061930	developing brain homeobox 1
KIAA0196	9897	NM_014846	KIAA0196
HLA-DRB1	3123	NM_002124, NM_172672	major histocompatibility complex, class II, DR beta 1
MMACHC	25974	XM_032397	methylmalonic aciduria (cobalamin deficiency) cbl type, with homocystinuria
TGFBI	7045	NM_000358	transforming growth factor, beta-induced, 68kDa
PPFIA4	8497	XM_046751	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
SORCS2	57537	NM_020777	sortilin-related VPS10 domain containing receptor
BAI3	577	NM_001704	brain-specific angiogenesis inhibitor 3
RFX1	5989	NM_002918	regulatory factor X, 1 (influences HLA class II expression)

IRAK3	11213	NM_007199	interleukin-1 receptor-associated kinase 3
PA2G4	5036	NM_006191	proliferation-associated 2G4, 38kDa
GCM2	9247	NM_004752	glial cells missing homolog 2 (Drosophila)
CHRND	1144	NM_000751	cholinergic receptor, nicotinic, delta
USP54	159195	NM_152586	ubiquitin specific peptidase 54
HNRPU	3192	NM_004501	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
NUTF2	10204	NM_005796	nuclear transport factor 2
HNRPK	3190	NM_002140	heterogeneous nuclear ribonucleoprotein K
ARCN1	372	NM_001655	archain 1
TRAF1	7185	NM_005658	TNF receptor-associated factor 1
TUBB2A	7280	NM_001069	tubulin, beta 2A
ATG16L2	89849	XM_058426	ATG16 autophagy related 16-like 2 (S. cerevisiae
ARSE	415	NM_000047	arylsulfatase E (chondrodysplasia punctata 1)
SIDT1	54847	NM_017699	SID1 transmembrane family, member 1
GNG11	2791	NM_004126	guanine nucleotide binding protein (G protein), gamma 11
NAT9	26151	NM_015654	N-acetyltransferase 9
MMP10	4319	NM_002425	matrix metallopeptidase 10 (stromelysin 2)
HOXD11	3237	NM_021192	homeobox D11
POLR3G	10622	NM_006467	polymerase (RNA) III (DNA directed) polypeptide (32kD)
TACC2	10579	NM_006997	transforming, acidic coiled-coil containing protein :
FGF2	2247	NM_002006	fibroblast growth factor 2 (basic)
BGN	633	NM_001711	biglycan
C11orf68	83638	NM_031450	chromosome 11 open reading frame 68
QSCN6	5768	NM_002826	quiescin Q6
TRIM8	81603	NM_030912	tripartite motif-containing 8
GJA3	2700	NM_021954, NM_029726	gap junction protein, alpha 3, 46kDa
TMPRSS5	80975	NM_030770	transmembrane protease, serine 5 (spinesin)
TAF2	6873	NM_003184	TAF2 RNA polymerase II, TATA box binding prote (TBP)-associated factor, 150kDa

OA48-18	10414	NM_006107	acid-inducible phosphoprotein
MKLN1	4289	NM_013255	muskelin 1, intracellular mediator containing kelch motifs
USP19	10869	XM_496642	ubiquitin specific peptidase 19
SETDB1	9869	NM_012432	SET domain, bifurcated 1
SLC25A19	60386	NM_021734	solute carrier family 25 (mitochondrial thiamine pyrophosphate carrier), member 19
PTPRH	5794	NM_002842	protein tyrosine phosphatase, receptor type, H
INTS4	92105	NM_033547	integrator complex subunit 4
COPE	11316	NM_007263	coatomer protein complex, subunit epsilon
PRKAG3	53632	NM_017431	protein kinase, AMP-activated, gamma 3 non-catalytic subunit
BPGM	669	NM_001724	2,3-bisphosphoglycerate mutase
PRAF2	11230	NM_007213	PRA1 domain family, member 2
NFIL3	4783	NM_005384	nuclear factor, interleukin 3 regulated
CXCL12	6387	NM_000609	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
PLCH2	9651	XM_371214	phospholipase C, eta 2
CHID1	66005	NM_023947	chitinase domain containing 1
CEND1	51286	NM_016564	cell cycle exit and neuronal differentiation 1
AMH	268	NM_000479	anti-Mullerian hormone
HIST2H3C	126961	NM_021059	histone cluster 2, H3c
CNKSR2	22866	NM_014927	connector enhancer of kinase suppressor of Ras 2
MYL3	4634	NM_000258	myosin, light chain 3, alkali; ventricular, skeletal, slow
SORBS3	10174	NM_005775	sorbin and SH3 domain containing 3
PFDN2	5202	NM_012394	prefoldin subunit 2
SOD1	6647	NM_000454	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult))
RBBP8	5932	NM_002894	retinoblastoma binding protein 8
PROSC	11212	NM_007198	proline synthetase co-transcribed homolog (bacterial)
TRIP6	7205	NM_003302	thyroid hormone receptor interactor 6
TNF	7124	NM_000594	tumor necrosis factor (TNF superfamily, member 2

HSFY2	159119	NM_153716	heat shock transcription factor, Y linked 2
SCAMP4	113178	NM_079834	secretory carrier membrane protein 4
TRPA1	8989	NM_007332	transient receptor potential cation channel, subfamily A, member 1
HNRPM	4670	NM_005968	heterogeneous nuclear ribonucleoprotein M
C2orf13	200558	NM_173545	chromosome 2 open reading frame 13
AGER	177	NM_001136	advanced glycosylation end product-specific receptor
GFER	2671	NM_005262	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae)
ERH	2079	NM_004450	enhancer of rudimentary homolog (Drosophila)
PAQR6	79957	NM_024897	progestin and adipoQ receptor family member VI
UNC13B	10497	NM_006377	unc-13 homolog B (C. elegans)
EGLN2	112398	NM_053046	egl nine homolog 2 (C. elegans)
FGFR1	2260	NM_000604	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome)
CARKL	23729	NM_013276	carbohydrate kinase-like
SEMA4B	10509	NM_020210	sema domain, immunoglobulin domain (Ig), transmembrane domain (TM) and short cytoplasm domain, (semaphorin) 4B
TUBGCP6	85378	NM_020461	tubulin, gamma complex associated protein 6
ICT1	3396	NM_001545, NM_016879	immature colon carcinoma transcript 1
WFDC2	10406	NM_006103	WAP four-disulfide core domain 2
CPNE6	9362	NM_006032	copine VI (neuronal)
CAMKV	79012	NM_024046	CaM kinase-like vesicle-associated
LOC285643	285643	XM_209695	KIF4B
C18orf8	29919	NM_013326	chromosome 18 open reading frame 8
LOR	4014	NM_000427	loricrin
ADM	133	NM_001124	adrenomedullin
LIF	3976	NM_002309	leukemia inhibitory factor (cholinergic differentiatic factor)
KIF11	3832	NM_004523	kinesin family member 11
FANCC	2176	NM_000136	Fanconi anemia, complementation group C

NOXO1	124056	NM_144603	NADPH oxidase organizer 1
UBE1L2	55236	NM_018227	ubiquitin-activating enzyme E1-like 2
P2RX1	5023	NM_002558	purinergic receptor P2X, ligand-gated ion channel
NPTN	27020	NM_012428	neuroplastin
STAT3	6774	NM_003150	signal transducer and activator of transcription 3 (acute-phase response factor)
PDCD5	9141	NM_004708	programmed cell death 5

Agents that modulate the activity of products of autophagy-enhancing genes are also useful in the treatment of autophagy-related diseases. For example, agents that inhibit the activity of products of autophagy-enhancing genes result in reduced autophagy levels and are therefore useful in methods of inhibition of autophagy and the treatment of autophagy-related diseases that are responsive to autophagy inhibition, such as cancer and pancreatitis. Agents that enhance the activity of products of autophagy-enhancing genes result in elevated autophagy levels and are therefore useful in methods of enhancement of autophagy and the treatment of autophagy-related diseases that are responsive to elevated levels of autophagy, such as neurodegenerative diseases and proteinopathies.

Table 2. Autophagy-enhancing genes.

Gene Symbol	**Gene ID**	**Genbank Acc. No.**	**Gene Name**
SMYD3	64754	NM_022743	SET and MYND domain containing 3
TCEB3	6924	NM_003198	transcription elongation factor B (SIII), polypeptide 3 (110kDa, elongin A)
CATSPER4	378807	XM_371237	cation channel, sperm associated 4
MEGF10	84466	NM_032446	multiple EGF-like-domains 10
KIF5C	3800	XM_377774	kinesin family member 5C
ATG7	10533	NM_006395	ATG7 autophagy related 7 homolog (S. cerevisiae)
RELA	5970	NM_021975	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer B-cells 3, p65 (avian)
GAB1	2549	NM_002039	GRB2-associated binding protein 1
LOC285647	285647	XM_209700	suppressor of defective silencing 3 pseudogene

GPR18	2841	NM_005292, NM_145948	G protein-coupled receptor 18
MBP	4155	NM_002385	myelin basic protein
PDCL	5082	NM_005388	phosducin-like
STIM1	6786	NM_003156	stromal interaction molecule 1
NFKB1	4790	NM_003998	nuclear factor of kappa light polypeptide gene enhancer B-cells 1 (p105)
TPR	7175	NM_003292	translocated promoter region (to activated MET oncogene)
PGGT1B	5229	NM_005023	protein geranylgeranyltransferase type I, beta subunit
ATG5	9474	NM_004849	ATG5 autophagy related 5 homolog (S. cerevisiae)

Thus, certain embodiments of the present invention relate to methods of enhancing autophagy and/or treating neurodegenerative diseases and/or proteinopathies through the inhibition of the activity of products of the autophagy-inhibiting genes listed in Table 1 or the enhancement of the activity of products of the autophagy-enhancing genes listed in Table 2. Other embodiments of the present invention relate to methods of inhibiting autophagy and/or treating cancer or pancreatitis through the enhancement of the activity of products of the autophagy-inhibiting genes listed in Table 1 or the inhibition of the activity of products of the autophagy-enhancing genes listed in Table 2.

Other embodiments of the present invention relate to methods of enhancing autophagy and/or treating neurodegenerative diseases and/or proteinopathies through the inhibition of the activity of products of the autophagy-inhibiting genes listed in Table 3 or the enhancement of the activity of products of the autophagy-enhancing genes listed in Table 4. Other embodiments of the present invention relate to methods of inhibiting autophagy and/or treating cancer or pancreatitis through the enhancement of the activity of products of the autophagy-inhibiting genes listed in Table 3 or the inhibition of the activity of products of the autophagy-enhancing genes listed in Table 4.

Table 3. Autophagy-inhibiting genes.

Gene Symbol	**Gene ID**	**Genbank Acc. No.**	**Gene Name**
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1

CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
KIAA0133	9816	XM_375851	KIAA0133
ADMR	11318	NM_007264	adrenomedullin receptor
OGDH	4967	NM_002541	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
DDX24	57062	NM_020414	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
NUPR1	26471	NM_012385	nuclear protein 1
FXYD2	486	NM_001680	FXYD domain containing ion transport regulator 2
TRHR	7201	NM_003301	thyrotropin-releasing hormone receptor
SUV39H1	6839	NM_003173	suppressor of variegation 3-9 homolog 1 (Drosophila)
FCER1A	2205	NM_002001	Fc fragment of IgE, high affinity I, receptor for; alp polypeptide
PTPRU	10076	NM_005704	protein tyrosine phosphatase, receptor type, U
GPX2	2877	NM_002083	glutathione peroxidase 2 (gastrointestinal)
EP300	2033	NM_001429	E1A binding protein p300
LOC388959	388959	XM_373989	hypothetical LOC388959
NTN2L	4917	NM_006181	netrin 2-like (chicken)
DOCK8	81704	NM_203447	dedicator of cytokinesis 8
MAP3K7IP1	10454	NM_006116	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
PLAGL2	5326	NM_002657	pleiomorphic adenoma gene-like 2
NUDT1	4521	NM_002452	nudix (nucleoside diphosphate linked moiety X)-type motif 1
RELN	5649	NM_005045	reelin
PNKD	25953	NM_015488	paroxysmal nonkinesiogenic dyskinesia
GNG5	2787	NM_005274	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	NM_001277	choline kinase alpha
C5AR1	728	NM_001736	complement component 5a receptor 1

SCOTIN	51246	NM_016479	scotin
PIGY	84992	NM_032906	phosphatidylinositol glycan anchor biosynthesis, class Y
NAGK	55577	NM_017567	N-acetylglucosamine kinase
RAGE	5891	NM_014226	renal tumor antigen
USP24	23358	XM_165973	ubiquitin specific peptidase 24
AURKA	6790	NM_003600	aurora kinase A
PLDN	26258	NM_012388	pallidin homolog (mouse)
PPARD	5467	NM_006238	peroxisome proliferator-activated receptor delta
HRC	3270	NM_002152	histidine rich calcium binding protein
NNMT	4837	NM_006169	nicotinamide N-methyltransferase
COPB2	9276	NM_004766	coatomer protein complex, subunit beta 2 (beta prime)
CDK5RAP3	80279	NM_025197	CDK5 regulatory subunit associated protein 3
NLK	51701	NM_016231	nemo-like kinase
PFKL	5211	NM_002626	phosphofructokinase, liver
RNPEPL1	57140	NM_018226	arginyl aminopeptidase (aminopeptidase B)-like 1
EPHA6	203806	XM_114973	EPH receptor A6
CDCA8	55143	NM_018101	cell division cycle associated 8
CKAP5	9793	NM_014756	cytoskeleton associated protein 5
ZBTB16	7704	NM_006006	zinc finger and BTB domain containing 16
GABBR2	9568	NM_005458	gamma-aminobutyric acid (GABA) B receptor, 2
PTMA	5757	NM_002823	prothymosin, alpha (gene sequence 28)
PTCRA	171558	NM_138296	pre T-cell antigen receptor alpha
RORC	6097	NM_005060	RAR-related orphan receptor C
GNAI1	2770	NM_002069	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
UTS2R	2837	NM_018949	urotensin 2 receptor
MATN3	4148	NM_002381	matrilin 3
NPTX1	4884	NM_002522	neuronal pentraxin I
SP140	11262	NM_007237	SP140 nuclear body protein
SMARCD1	6602	NM_003076	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d,

			member 1
PAK6	56924	NM_020168	p21(CDKN1A)-activated kinase 6
TACR2	6865	NM_001057	tachykinin receptor 2
MMP17	4326	NM_016155	matrix metallopeptidase 17 (membrane-inserted)
MUC3A	4584	XM_374502	mucin 3A, cell surface associated
PRKCZ	5590	NM_002744	protein kinase C, zeta
TNFRSF17	608	NM_001192	tumor necrosis factor receptor superfamily, memb 17
GTF2IRD2	84163	NM_173537	GTF2I repeat domain containing 2
TRPM3	80036	NM_020952	transient receptor potential cation channel, subfamily M, member 3
HMBS	3145	NM_000190, NM_176954	hydroxymethylbilane synthase
CYP27A1	1593	NM_000784	cytochrome P450, family 27, subfamily A, polypeptide 1
FBXL20	84961	NM_032875	F-box and leucine-rich repeat protein 20
CD300C	10871	NM_006678	CD300c molecule
PSD	5662	NM_002779	pleckstrin and Sec7 domain containing
FRAG1	27315	NM_014489	FGF receptor activating protein 1
PCGF1	84759	NM_032673	polycomb group ring finger 1
SIX2	10736	NM_016932	sine oculis homeobox homolog 2 (Drosophila)
CLCN1	1180	NM_000083	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
EVL	51466	NM_016337	Enah/Vasp-like
TOM1	10043	NM_005488	target of myb1 (chicken)
BAIAP2	10458	NM_006340	BAI1-associated protein 2
ZFY	7544	NM_003411	zinc finger protein, Y-linked
UBE2D1	7321	NM_003338	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
GJA4	2701	NM_002060	gap junction protein, alpha 4, 37kDa
SF3A2	8175	NM_007165	splicing factor 3a, subunit 2, 66kDa
TRNT1	51095	NM_016000	tRNA nucleotidyl transferase, CCA-adding, 1
RANGAP1	5905	NM_002883	Ran GTPase activating protein 1
CCT4	10575	NM_006430	chaperonin containing TCP1, subunit 4 (delta)

TSPAN4	7106	NM_003271	tetraspanin 4
PTGER2	5732	NM_000956	prostaglandin E receptor 2 (subtype EP2), 53kDa
GTPBP4	23560	NM_012341	GTP binding protein 4
ADRA1A	148	NM_000680	adrenergic, alpha-1A-, receptor
PHB2	11331	NM_007273	prohibitin 2
TNFRSF19L	84957	NM_032871	tumor necrosis factor receptor superfamily, memb 19-like
COL14A1	7373	XM_044622	collagen, type XIV, alpha 1 (undulin)
CD79A	973	NM_001783	CD79a molecule, immunoglobulin-associated alpl
F12	2161	NM_000505	coagulation factor XII (Hageman factor)
ASMT	438	NM_004043	acetylserotonin O-methyltransferase
GRK6	2870	NM_002082	G protein-coupled receptor kinase 6
GNRH2	2797	NM_001501	gonadotropin-releasing hormone 2
SDHB	6390	NM_003000	succinate dehydrogenase complex, subunit B, iro sulfur (Ip)
THBS2	7058	NM_003247	thrombospondin 2
HIVEP2	3097	NM_145975, NM_006734	human immunodeficiency virus type I enhancer binding protein 2
WASF1	8936	NM_003931	WAS protein family, member 1
SSPN	8082	NM_005086	sarcospan (Kras oncogene-associated gene)
ITGAV	3685	NM_002210	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
PLXNA2	5362	XM_372810	plexin A2
NCR3	259197	NM_147130	natural cytotoxicity triggering receptor 3
TH	7054	NM_000360	tyrosine hydroxylase
HMGCL	3155	NM_177229, NM_000191	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
CENPJ	55835	NM_018451	centromere protein J
FABP1	2168	NM_001443	fatty acid binding protein 1, liver
CASP1	834	NM_001223	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
MCCC1	56922	NM_020166	methylcrotonoyl-Coenzyme A carboxylase 1 (alph
DBX1	120237	XM_061930	developing brain homeobox 1
KIAA0196	9897	NM_014846	KIAA0196

HLA-DRB1	3123	NM_002124, NM_172672	major histocompatibility complex, class II, DR beta 1
MMACHC	25974	XM_032397	methylmalonic aciduria (cobalamin deficiency) cbl type, with homocystinuria
TGFBI	7045	NM_000358	transforming growth factor, beta-induced, 68kDa
PPFIA4	8497	XM_046751	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
SORCS2	57537	NM_020777	sortilin-related VPS10 domain containing receptor
BAI3	577	NM_001704	brain-specific angiogenesis inhibitor 3
RFX1	5989	NM_002918	regulatory factor X, 1 (influences HLA class II expression)
IRAK3	11213	NM_007199	interleukin-1 receptor-associated kinase 3
PA2G4	5036	NM_006191	proliferation-associated 2G4, 38kDa
GCM2	9247	NM_004752	glial cells missing homolog 2 (Drosophila)
CHRND	1144	NM_000751	cholinergic receptor, nicotinic, delta
USP54	159195	NM_152586	ubiquitin specific peptidase 54
HNRPU	3192	NM_004501	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
NUTF2	10204	NM_005796	nuclear transport factor 2
HNRPK	3190	NM_002140	heterogeneous nuclear ribonucleoprotein K
ARCN1	372	NM_001655	archain 1
TRAF1	7185	NM_005658	TNF receptor-associated factor 1
TUBB2A	7280	NM_001069	tubulin, beta 2A
ATG16L2	89849	XM_058426	ATG16 autophagy related 16-like 2 (S. cerevisiae
ARSE	415	NM_000047	arylsulfatase E (chondrodysplasia punctata 1)
SIDT1	54847	NM_017699	SID1 transmembrane family, member 1
GNG11	2791	NM_004126	guanine nucleotide binding protein (G protein), gamma 11
NAT9	26151	NM_015654	N-acetyltransferase 9
MMP10	4319	NM_002425	matrix metallopeptidase 10 (stromelysin 2)
HOXD11	3237	NM_021192	homeobox D11
POLR3G	10622	NM_006467	polymerase (RNA) III (DNA directed) polypeptide (32kD)

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TRIM8	81603	NM_030912	tripartite motif-containing 8
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TMPRSS5	80975	NM_030770	transmembrane protease, serine 5 (spinesin)
TAF2	6873	NM_003184	TAF2 RNA polymerase II, TATA box binding protein (TBP)-associated factor, 150kDa
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MKLN1	4289	NM_013255	muskelin 1, intracellular mediator containing kelch motifs
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SETDB1	9869	NM_012432	SET domain, bifurcated 1
SLC25A19	60386	NM_021734	solute carrier family 25 (mitochondrial thiamine pyrophosphate carrier), member 19
PTPRH	5794	NM_002842	protein tyrosine phosphatase, receptor type, H
INTS4	92105	NM_033547	integrator complex subunit 4
COPE	11316	NM_007263	coatomer protein complex, subunit epsilon
PRKAG3	53632	NM_017431	protein kinase, AMP-activated, gamma 3 non-catalytic subunit
BPGM	669	NM_001724	2,3-bisphosphoglycerate mutase
PRAF2	11230	NM_007213	PRA1 domain family, member 2
NFIL3	4783	NM_005384	nuclear factor, interleukin 3 regulated
CXCL12	6387	NM_000609	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
PLCH2	9651	XM_371214	phospholipase C, eta 2
CHID1	66005	NM_023947	chitinase domain containing 1
CEND1	51286	NM_016564	cell cycle exit and neuronal differentiation 1
HIST2H3C	126961	NM_021059	histone cluster 2, H3c
CNKSR2	22866	NM_014927	connector enhancer of kinase suppressor of Ras 2
MYL3	4634	NM_000258	myosin, light chain 3, alkali; ventricular, skeletal, slow

SORBS3	10174	NM_005775	sorbin and SH3 domain containing 3
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RBBP8	5932	NM_002894	retinoblastoma binding protein 8
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CARKL	23729	NM_013276	carbohydrate kinase-like
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TUBGCP6	85378	NM_020461	tubulin, gamma complex associated protein 6
ICT1	3396	NM_001545, NM_016879	immature colon carcinoma transcript 1
WFDC2	10406	NM_006103	WAP four-disulfide core domain 2
CPNE6	9362	NM_006032	copine VI (neuronal)
CAMKV	79012	NM_024046	CaM kinase-like vesicle-associated
LOC285643	285643	XM_209695	KIF4B
C18orf8	29919	NM_013326	chromosome 18 open reading frame 8

LOR	4014	NM_000427	loricrin
ADM	133	NM_001124	adrenomedullin
KIF11	3832	NM_004523	kinesin family member 11
FANCC	2176	NM_000136	Fanconi anemia, complementation group C
NOXO1	124056	NM_144603	NADPH oxidase organizer 1
UBE1L2	55236	NM_018227	ubiquitin-activating enzyme E1-like 2
P2RX1	5023	NM_002558	purinergic receptor P2X, ligand-gated ion channel
NPTN	27020	NM_012428	neuroplastin
PDCD5	9141	NM_004708	programmed cell death 5

Table 4. Autophagy-enhancing genes.

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
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MEGF10	84466	NM_032446	multiple EGF-like-domains 10
KIF5C	3800	XM_377774	kinesin family member 5C
RELA	5970	NM_021975	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer B-cells 3, p65 (avian)
GAB1	2549	NM_002039	GRB2-associated binding protein 1
LOC285647	285647	XM_209700	suppressor of defective silencing 3 pseudogene
GPR18	2841	NM_005292, NM_145948	G protein-coupled receptor 18
PDCL	5082	NM_005388	phosducin-like
STIM1	6786	NM_003156	stromal interaction molecule 1
NFKB1	4790	NM_003998	Nuclear factor of kappa light polypeptide gene enhancer in B-cells 1
TPR	7175	NM_003292	translocated promoter region (to activated MET oncogene)
PGGT1B	5229	NM_005023	protein geranylgeranyltransferase type I, beta subunit

The products of the autophagy-related genes of the invention can be classified into a number of non-mutually exclusive categories. For example, certain gene products of the present invention can be classified as oxidoreductases, receptors, proteases, ligases, kinases, synthases, synthetases, chaperones, hydrolases, membrane traffic proteins, calcium binding proteins and/or regulatory molecules. The classification of selected autophagy-inhibiting gene products is listed in Table 5, while the classification of selected autophagy-enhancing gene products is listed in Table 6. Since certain types of agents are better suited for the modulation of the activity of a specific class of gene product, in some embodiments the present invention is directed towards the modulation of one or more class of autophagy-related gene product.

Table 5. Classification of certain autophagy-inhibiting gene products.

Gene Symbol	Gene Name	Class
CYP27A1	cytochrome P450, family 27, subfamily A, polypeptide 1;CYP27A1	Oxidoreductase
SDHB	succinate dehydrogenase complex, subunit B, iron sulfur (Ip);SDHB	Oxidoreductase
OGDH	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide);OGDH	Oxidoreductase
QSCN6	quiescin Q6;QSCN6	Oxidoreductase
EGLN2	egl nine homolog 2 (C. elegans);EGLN2	Oxidoreductase
TH	tyrosine hydroxylase;TH	Oxidoreductase
COX5A	cytochrome c oxidase subunit Va;COX5A	Oxidoreductase
SOD1	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult));SOD1	Oxidoreductase
GPX2	glutathione peroxidase 2 (gastrointestinal);GPX2	Oxidoreductase
GFER	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae);GFER	Oxidoreductase
TRPM3	transient receptor potential cation channel, subfamily M, member 3;TRPM3	Receptor
TMPRSS5	transmembrane protease, serine 5 (spinesin);TMPRSS5	Receptor

IRAK3	interleukin-1 receptor-associated kinase 3;IRAK3	Receptor
ADMR(Also Known as GPR182)	adrenomedullin receptor;ADMR	Receptor
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	Receptor
UNC13B	unc-13 homolog B (C. elegans);UNC13B	Receptor
PTGER2	prostaglandin E receptor 2 (subtype EP2), 53kDa;PTGER2	Receptor
AGER	advanced glycosylation end product-specific receptor;AGER	Receptor
BGN	biglycan;BGN	Receptor
GABBR2	gamma-aminobutyric acid (GABA) B receptor, 2;GABBR2	Receptor
PPARD	peroxisome proliferator-activated receptor delta;PPARD	Receptor
GHSR	growth hormone secretagogue receptor;GHSR	Receptor
BAIAP2	BAI1-associated protein 2;BAIAP2	Receptor
SORCS2	sortilin-related VPS10 domain containing receptor 2;SORCS2	Receptor
PAQR6	progestin and adipoQ receptor family member VI;PAQR6	Receptor
EPHA6	EPH receptor A6;EPHA6	Receptor
TRHR	thyrotropin-releasing hormone receptor;TRHR	Receptor
C5AR1	complement component 5a receptor 1;C5AR1	Receptor
BAI3	brain-specific angiogenesis inhibitor 3;BAI3	Receptor
TLR3	toll-like receptor 3;TLR3	Receptor
PTPRH	protein tyrosine phosphatase, receptor type, H;PTPRH	Receptor
ADRA1A	adrenergic, alpha-1A-, receptor;ADRA1A	Receptor
UTS2R	urotensin 2 receptor;UTS2R	Receptor

RORC	RAR-related orphan receptor C;RORC	Receptor
CHRND	cholinergic receptor, nicotinic, delta;CHRND	Receptor
TACR2	tachykinin receptor 2;TACR2	Receptor
P2RX1	purinergic receptor P2X, ligand-gated ion channel, 1;P2RX1	Receptor
PLXNA2	plexin A2;PLXNA2	Receptor
PTPRU	protein tyrosine phosphatase, receptor type, U;PTPRU	Receptor
FCER1A	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide;FCER1A	Receptor
CD300C	CD300c molecule;CD300C	Receptor
TNFRSF19L(Also known as RELT)	tumor necrosis factor receptor superfamily, member 19-like;TNFRSF19L	Receptor
TMPRSS5	transmembrane protease, serine 5 (spinesin);TMPRSS5	Protease
USP19	ubiquitin specific peptidase 19;USP19	Protease
RNPEPL1	arginyl aminopeptidase (aminopeptidase B)-like 1;RNPEPL1	Protease
MMP10	matrix metallopeptidase 10 (stromelysin 2);MMP10	Protease
RELN	reelin;RELN	Protease
F12	coagulation factor XII (Hageman factor);F12	Protease
CASP1	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase);CASP1	Protease
MMP17	matrix metallopeptidase 17 (membrane-inserted);MMP17	Protease
CAPN1	calpain 1, (mu/I) large subunit;CAPN1	Protease
TRIM8	tripartite motif-containing 8;TRIM8	Ligase
UBE1L2(Also known as UBA6)	ubiquitin-activating enzyme E1-like 2;UBE1L2	Ligase
MCCC1	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha);MCCC1	Ligase

TRIM69	tripartite motif-containing 69;TRIM69	Ligase
UBE2D1	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast);UBE2D1	Ligase
HMGCL	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria);HMGCL	Lyase
PAK6	p21(CDKN1A)-activated kinase 6;PAK6	Kinase
CHKA	choline kinase alpha;CHKA	Kinase
RAGE	renal tumor antigen;RAGE	Kinase
IHPK3(Also known as IP6K3)	inositol hexaphosphate kinase 3;IHPK3	Kinase
CAMKV	CaM kinase-like vesicle-associated;CAMKV	Kinase
PRKAA2	protein kinase, AMP-activated, alpha 2 catalytic subunit;PRKAA2	Kinase
PRKCZ	protein kinase C, zeta;PRKCZ	Kinase
PRKCA	protein kinase C, alpha;PRKCA	Kinase
CARKL(Also known as SHPK)	carbohydrate kinase-like;CARKL	Kinase
PFKL	phosphofructokinase, liver;PFKL	Kinase
NLK	nemo-like kinase;NLK	Kinase
AURKA	aurora kinase A;AURKA	Kinase
PROSC	proline synthetase co-transcribed homolog (bacterial);PROSC	Synthase & synthetase
CCT4	chaperonin containing TCP1, subunit 4 (delta);CCT4	Chaperone
PFDN2	prefoldin subunit 2;PFDN2	Chaperone
CHID1	chitinase domain containing 1;CHID1	Hydrolase
ARSE	arylsulfatase E (chondrodysplasia punctata 1);ARSE	Hydrolase
PLCH2	phospholipase C, eta 2;PLCH2	Hydrolase
HMBS	hydroxymethylbilane synthase;HMBS	Hydrolase
PNKD	paroxysmal nonkinesiogenic dyskinesia;PNKD	Hydrolase

NUDT1	nudix (nucleoside diphosphate linked moiety X)-type motif 1;NUDT1	Hydrolase
COPB2	coatomer protein complex, subunit beta 2 (beta prime);COPB2	Membrane traffic protein
ARCN1	archain 1;ARCN1	Membrane traffic protein
CPNE6	copine VI (neuronal);CPNE6	Membrane traffic protein
COPE	coatomer protein complex, subunit epsilon;COPE	Membrane traffic protein
HRC	histidine rich calcium binding protein;HRC	Calcium binding protein
MYL3	myosin, light chain 3, alkali; ventricular, skeletal, slow;MYL3	Calcium binding protein
RANGAP1	Ran GTPase activating protein 1;RANGAP1	Regulatory molecule
GTPBP4	GTP binding protein 4;GTPBP4	Regulatory molecule
TRIP6	thyroid hormone receptor interactor 6;TRIP6	Regulatory molecule
CNKSR2	connector enhancer of kinase suppressor of Ras 2;CNKSR2	Regulatory molecule
PSD	pleckstrin and Sec7 domain containing;PSD	Regulatory molecule
DOCK8	dedicator of cytokinesis 8;DOCK8	Regulatory molecule
THBS2	thrombospondin 2;THBS2	Regulatory molecule
GNAI1	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1;GNAI1	Regulatory molecule
FRAG1	FGF receptor activating protein 1;unassigned	Regulatory molecule
RAB7A	RAB7, member RAS oncogene family;RAB7	Regulatory molecule
CDKN2D	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4);CDKN2D	Regulatory molecule
GNG5	guanine nucleotide binding protein (G protein), gamma 5;GNG5	Regulatory molecule

GNG11	guanine nucleotide binding protein (G protein), gamma 11;GNG11	Regulatory molecule
PDCD5	programmed cell death 5;PDCD5	Regulatory molecule
WFDC2	WAP four-disulfide core domain 2;WFDC2	Regulatory molecule

Table 6. Classification of certain autophagy-enhancing gene products.

Gene Symbol	Gene Name	Class
TPR	translocated promoter region (to activated MET oncogene);TPR	Receptor
GPR18	G protein-coupled receptor 18;GPR18	Receptor
PDCL	phosducin-like;PDCL	Regulatory molecule

3. Modulators of autophagy-related gene products.

Certain embodiments of the present invention relate to methods of modulating autophagy or treating autophagy-related diseases (*e.g.* neurodegenerative disease, liver disease, muscle disease, cancer, pancreatitis). These methods involve administering an agent that modulates the activity of one or more autophagy-related gene products of the invention. In certain embodiments, methods of the invention include treatment of autophagy-related diseases by administering to a subject an agent which decreases the activity of one or more products of the genes listed in Tables 1-4. In other embodiments, methods of the invention include treatment of autophagy-related diseases by administering to a subject an agent which increases the activity of one or more products of the genes listed in Tables 1-4. Agents which may be used to modulate the activity of a gene product listed in Tables 1-4, and to thereby treat or prevent an autophagy-related disease, include antibodies (*e.g.,* conjugated antibodies), proteins, peptides, small molecules, RNA interfering agents, *e.g.,* siRNA molecules, ribozymes, and antisense oligonucleotides.

Any agent that modulates the activity of an autophagy-related gene product of the invention can be used to practice certain methods of the invention. Such agents can be those described herein, those known in the art, or those identified through routine screening assays (*e.g.* the screening assays described herein).

In some embodiments, assays used to identify agents useful in the methods of the present invention include a reaction between the autophagy-related gene product and one or more assay components. The other components may be either a test compound (*e.g.* the potential agent), or a combination of test compounds and a natural binding partner of the autophagy-related gene product. Agents identified *via* such assays, such as those described herein, may be useful, for example, for modulating autophagy and treating autophagy-related diseases.

Agents useful in the methods of the present invention may be obtained from any available source, including systematic libraries of natural and/or synthetic compounds. Agents may also be obtained by any of the numerous approaches in combinatorial library methods known in the art, including: biological libraries; peptoid libraries (libraries of molecules having the functionalities of peptides, but with a novel, non-peptide backbone which are resistant to enzymatic degradation but which nevertheless remain bioactive; see, *e.g.,* Zuckermann *et al.*, 1994, *J. Med. Chem.* 37:2678-85); spatially addressable parallel solid phase or solution phase libraries; synthetic library methods requiring deconvolution; the 'one-bead one-compound' library method; and synthetic library methods using affinity chromatography selection. The biological library and peptoid library approaches are limited to peptide libraries, while the other four approaches are applicable to peptide, non-peptide oligomer or small molecule libraries of compounds (Lam, 1997, *Anticancer Drug Des.* 12:145).

Examples of methods for the synthesis of molecular libraries can be found in the art, for example in: DeWitt *et al.* (1993) *Proc. Natl. Acad. Sci. U.S.A.* 90:6909; Erb *et al.* (1994) *Proc. Natl. Acad. Sci. USA* 91:11422; Zuckermann *et al.* (1994). *J. Med. Chem.* 37:2678; Cho *et al.* (1993) *Science* 261:1303; Carrell *et al.* (1994) *Angew. Chem. Int. Ed. Engl.* 33:2059; Carell *et al.* (1994) *Angew. Chem. Int. Ed. Engl.* 33:2061; and in Gallop *et al.* (1994) *J. Med. Chem.* 37:1233.

Libraries of agents may be presented in solution (*e.g.,* Houghten, 1992, *Biotechniques* 13:412-421), or on beads (Lam, 1991, *Nature* 354:82-84), chips (Fodor, 1993, *Nature* 364:555-556), bacteria and/or spores, (Ladner, USP 5,223,409), plasmids (Cull *et al*, 1992, *Proc Natl Acad Sci USA* 89:1865-1869) or on phage (Scott and Smith, 1990, *Science* 249:386-390; Devlin, 1990, *Science* 249:404-406; Cwirla *et al,* 1990, *Proc. Natl. Acad. Sci.* 87:6378-6382; Felici, 1991, *J. Mol. Biol.* 222:301-310; Ladner, *supra.*).

Agents useful in the methods of the present invention may be identified, for example, using assays for screening candidate or test compounds which are substrates of an autophagy-related gene product of the invention or biologically active portion thereof. In another embodiment, agents useful in the methods of the invention may be identified using assays for screening candidate or test compounds which bind to an autophagy-related gene product of the invention or a biologically active portion thereof. Determining the ability of the test compound to directly bind to an autophagy-related gene product can be accomplished, for example, by coupling the compound with a radioisotope or enzymatic label such that binding of the compound to the autophagy-related gene product can be determined by detecting the labeled compound in a complex. For example, compounds can be labeled with ^{125}I, ^{35}S, ^{14}C, or ^{3}H, either directly or indirectly, and the radioisotope detected by direct counting of radioemission or by scintillation counting. Alternatively, assay components can be enzymatically labeled with, for example, horseradish peroxidase, alkaline phosphatase, or luciferase, and the enzymatic label detected by determination of conversion of an appropriate substrate to product.

Agents useful in the methods of the invention may also be identified, for example, using assays that identify compounds which modulate (*e.g.,* affect either positively or negatively) interactions between an autophagy-related gene product and its substrates and/or binding partners. Such compounds can include, but are not limited to, molecules such as antibodies, peptides, hormones, oligonucleotides, nucleic acids, and analogs thereof. Such compounds may also be obtained from any available source, including systematic libraries of natural and/or synthetic compounds.

The basic principle of the assay systems used to identify compounds that modulate the interaction between the autophagy-related gene product and its binding partner involves preparing a reaction mixture containing the autophagy-related gene product and its binding partner under conditions and for a time sufficient to allow the two products to interact and bind, thus forming a complex. In order to test an agent for inhibitory activity, the reaction mixture is prepared in the presence and absence of the test compound. The test compound can be initially included in the reaction mixture, or can be added at a time subsequent to the addition of the autophagy-related gene product and its binding partner. Control reaction mixtures are incubated without the test compound or with a placebo. The formation of any complexes between the autophagy-related gene product and its binding partner is then detected. The formation of a complex in the control reaction, but less or no such formation

in the reaction mixture containing the test compound, indicates that the compound interferes with the interaction of the autophagy-related gene product and its binding partner. Conversely, the formation of more complex in the presence of the compound than in the control reaction indicates that the compound may enhance interaction of the autophagy-related gene product and its binding partner.

The assay for compounds that modulate the interaction of the autophagy-related gene product with its binding partner may be conducted in a heterogeneous or homogeneous format. Heterogeneous assays involve anchoring either the autophagy-related gene product or its binding partner onto a solid phase and detecting complexes anchored to the solid phase at the end of the reaction. In homogeneous assays, the entire reaction is carried out in a liquid phase. In either approach, the order of addition of reactants can be varied to obtain different information about the compounds being tested. For example, test compounds that interfere with the interaction between the autophagy-related gene products and the binding partners (*e.g.,* by competition) can be identified by conducting the reaction in the presence of the test substance, *i.e.,* by adding the test substance to the reaction mixture prior to or simultaneously with the autophagy-related gene product and its interactive binding partner. Alternatively, test compounds that disrupt preformed complexes, *e.g.,* compounds with higher binding constants that displace one of the components from the complex, can be tested by adding the test compound to the reaction mixture after complexes have been formed. The various formats are briefly described below.

In a heterogeneous assay system, either the autophagy-related gene product or its binding partner is anchored onto a solid surface or matrix, while the other corresponding non-anchored component may be labeled, either directly or indirectly. In practice, microtitre plates are often utilized for this approach. The anchored species can be immobilized by a number of methods, either non-covalent or covalent, that are typically well known to one who practices the art. Non-covalent attachment can often be accomplished simply by coating the solid surface with a solution of the autophagy-related gene product or its binding partner and drying. Alternatively, an immobilized antibody specific for the assay component to be anchored can be used for this purpose.

In related assays, a fusion protein can be provided which adds a domain that allows one or both of the assay components to be anchored to a matrix. For example, glutathione-S-transferase/marker fusion proteins or glutathione-S-transferase/binding partner can be

adsorbed onto glutathione sepharose beads (Sigma Chemical, St. Louis, MO) or glutathione derivatized microtiter plates, which are then combined with the test compound or the test compound and either the non-adsorbed autophagy-related gene product or its binding partner, and the mixture incubated under conditions conducive to complex formation (*e.g.,* physiological conditions). Following incubation, the beads or microtiter plate wells are washed to remove any unbound assay components, the immobilized complex assessed either directly or indirectly, for example, as described above. Alternatively, the complexes can be dissociated from the matrix, and the level of autophagy-related gene product binding or activity determined using standard techniques.

A homogeneous assay may also be used to identify modulators of autophagy-related gene products. This is typically a reaction, analogous to those mentioned above, which is conducted in a liquid phase in the presence or absence of the test compound. The formed complexes are then separated from unreacted components, and the amount of complex formed is determined. As mentioned for heterogeneous assay systems, the order of addition of reactants to the liquid phase can yield information about which test compounds modulate (inhibit or enhance) complex formation and which disrupt preformed complexes.

In such a homogeneous assay, the reaction products may be separated from unreacted assay components by any of a number of standard techniques, including but not limited to: differential centrifugation, chromatography, electrophoresis and immunoprecipitation. In differential centrifugation, complexes of molecules may be separated from uncomplexed molecules through a series of centrifugal steps, due to the different sedimentation equilibria of complexes based on their different sizes and densities (see, for example, Rivas, G., and Minton, A.P., *Trends Biochem Sci* 1993 Aug;18(8):284-7). Standard chromatographic techniques may also be utilized to separate complexed molecules from uncomplexed ones. For example, gel filtration chromatography separates molecules based on size, and through the utilization of an appropriate gel filtration resin in a column format, for example, the relatively larger complex may be separated from the relatively smaller uncomplexed components. Similarly, the relatively different charge properties of the complex as compared to the uncomplexed molecules may be exploited to differentially separate the complex from the remaining individual reactants, for example through the use of ion-exchange chromatography resins. Such resins and chromatographic techniques are well known to one skilled in the art (see, *e.g.,* Heegaard, 1998, *J Mol. Recognit.* 11:141-148; Hage and Tweed, 1997, *J. Chromatogr. B. Biomed. Sci. Appl.,*

699:499-525). Gel electrophoresis may also be employed to separate complexed molecules from unbound species (see, *e.g.,* Ausubel *et al* (eds.), In: *Current Protocols in Molecular Biology*, J. Wiley & Sons, New York. 1999). In this technique, protein or nucleic acid complexes are separated based on size or charge, for example. In order to maintain the binding interaction during the electrophoretic process, nondenaturing gels in the absence of reducing agent are typically preferred, but conditions appropriate to the particular interactants will be well known to one skilled in the art. Immunoprecipitation is another common technique utilized for the isolation of a protein-protein complex from solution (see, *e.g.,* Ausubel *et al* (eds.), In: *Current Protocols in Molecular Biology*, J. Wiley & Sons, New York. 1999). In this technique, all proteins binding to an antibody specific to one of the binding molecules are precipitated from solution by conjugating the antibody to a polymer bead that may be readily collected by centrifugation. The bound assay components are released from the beads (through a specific proteolysis event or other technique well known in the art which will not disturb the protein-protein interaction in the complex), and a second immunoprecipitation step is performed, this time utilizing antibodies specific for the correspondingly different interacting assay component. In this manner, only formed complexes should remain attached to the beads. Variations in complex formation in both the presence and the absence of a test compound can be compared, thus offering information about the ability of the compound to modulate interactions between the autophagy-related gene product and its binding partner.

Modulators of autophagy-related gene product expression may also be identified, for example, using methods wherein a cell is contacted with a candidate compound and the expression of mRNA or protein, corresponding to an autophagy-related gene in the cell, is determined. The level of expression of mRNA or protein in the presence of the candidate compound is compared to the level of expression of mRNA or protein in the absence of the candidate compound. The candidate compound can then be identified as a modulator of autophagy-related gene product expression based on this comparison. For example, when expression of autophagy-related gene product is greater in the presence of the candidate compound than in its absence, the candidate compound is identified as a stimulator of marker mRNA or protein expression. Conversely, when expression of autophagy-related gene product is less in the presence of the candidate compound than in its absence, the candidate compound is identified as an inhibitor of marker mRNA or protein expression.

The level of autophagy-related gene product expression in the cells can be determined by methods described herein for detecting marker mRNA or protein.

Agents that inhibit the activity of autophagy-inhibiting gene products are useful, for example, in enhancing autophagy and in the treatment of neurodegenerative diseases. Examples of such inhibitors of autophagy-inhibiting gene products are listed in Table 7 and Figure 63.

Table 7. Agents that inhibit autophagy-inhibiting gene products.

Target Gene Symbol	Target Gene Name	Agent
TH	tyrosine hydroxylase;TH	alpha-methyl-para-tyrosine (Metyrosine)
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	TK1258 (CHIR258)
AGER	advanced glycosylation end product-specific receptor;AGER	PF 04494700 (TTP488)
C5AR1	complement component 5a receptor 1;C5AR1	PMX53
ADRA1A	adrenergic, alpha-1A-, receptor;ADRA1A	Tamsulosin
ADRA1A	adrenergic, alpha-1A-, receptor;ADRA1A	Doxazosin
ADRA1A	adrenergic, alpha-1A-, receptor;ADRA1A	Prazosin hydrochloride
ADRA1A	adrenergic, alpha-1A-, receptor;ADRA1A	alfuzosin hydrochloride
UTS2R	urotensin 2 receptor;UTS2R	Urotensin II
CHRND	cholinergic receptor, nicotinic, delta;CHRND	Galantamine (Galanthamine)
CHRND	cholinergic receptor, nicotinic, delta;CHRND	Mecamylamine hydrochloride (Inversine)
CASP1	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase);CASP1	Pralnacasan (VX-740, HMR 3480)
PRKCA	protein kinase C, alpha;PRKCA	ISIS 3521 (carboplatin,

		paclitaxel)
PRKCA	protein kinase C, alpha;PRKCA	Gemcitabine;
PRKCA	protein kinase C, alpha;PRKCA	LY900003
AURKA	aurora kinase A;AURKA	MK-5108
PLCH2	phospholipase C, eta 2;PLCH2	U73122
PLCH2	phospholipase C, eta 2;PLCH2	D609

Alternatively, agents that enhance the activity of autophagy-inhibiting gene products are useful, for example, in inhibiting autophagy and in the treatment of cancer and pancreatitis. Examples of such enhancers of autophagy-inhibiting gene products are listed in Table 8 and Figure 63.

Table 8. Agents that enhance autophagy-inhibiting gene products.

Target Gene Symbol	Target Gene Name	Agent
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	Cardio Vascu-Grow (FGF-1, CVBT-141)
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	Acidic FGF (aFGF);
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	XRP0038 (NV1FGF)
FGFR1	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1	Rh-aFGF
PPARD	peroxisome proliferator-activated receptor delta; PPARD	GW501516
GHSR	growth hormone secretagogue receptor;GHSR	Ibutamoren Mesylate (MK-0677)
GHSR	growth hormone secretagogue	KP-102LN

	receptor;GHSR	
GHSR	growth hormone secretagogue receptor;GHSR	EP1572 (ghrelin agonist)
TRHR	thyrotropin-releasing hormone receptor;TRHR	TRH
TRHR	thyrotropin-releasing hormone receptor;TRHR	S-0373 (KPS-0373)
TRHR	thyrotropin-releasing hormone receptor;TRHR	S-14820
TLR3	toll-like receptor 3;TLR3	Poly-ICR
TLR3	toll-like receptor 3;TLR3	CQ-07001
PRKAA2	protein kinase, AMP-activated, alpha 2 catalytic subunit;PRKAA2	cryptotanshinone

Further examples of agents that modulate the autophagy-related gene products listed in tables 1-4 can be found in, for example, U.S. Patent Numbers: 7,348,140; 6,982,265; 6,723,694; 6,617,311; 6,372,250; 6,334,998; 6,319,905; 6,312,949; 6,297,238; 6,228,835; 6,214,334; 6,096,778; 5,990,083; 5,834,457; 5,783,683; 5,681,747; 5,556,837; 5,464,614, each of which is hereby specifically incorporated by reference in its entirety. Examples of agents that modulate the autophagy-related gene products listed in tables 1-4 can also be found in, for example, U.S. Patent Application Publication Numbers: US2009/0137572; US2009/0136475; US2009/0105149; US2009/0088401; US2009/0087454; US2009/0087410; US2009/0075900; US2009/0074774; US2009/0074711; US2009/0074676; US2009/0069245; US2009/0068194; US2009/0068168; US2009/0060898; US2009/0047240; US2009/0042803; US2009/0029992; US2009/0011994; US2009/0005431; US2009/0005309; US2009/0004194; US2008/0319026; US2008/0312247; US2008/0300316; US2008/0300180; US2008/0299138; US2008/0280991; US2008/0280886; US2008/0268071; US2008/0262086; US2008/0255200; US2008/0255084; US2008/0255036; US2008/0242687; US2008/0241289; US2008/0234284; US2008/0234257; US2008/0221132; US2008/0194672; US2008/0194555; US2008/0187490; US2008/0171769; US2008/0167312; US2008/0146573; US2008/0132555; US2008/0125386; US2008/0124379; US2008/0103189; US2008/0051465;

US2008/0051383; US2008/0045588; US2008/0045561; US2008/0045558; US2008/0039473; US2008/0033056; US2008/0021036; US2008/0021029; US2008/0004300; US2007/0293525; US2007/0293494; US2007/0287734; US2007/0286853; US2007/0281965; US2007/0281894; US2007/0280886; US2007/0274981; US2007/0259891; US2007/0259827; US2007/0254877; US2007/0249519; US2007/0248605; US2007/0219235; US2007/0219114; US2007/0203064; US2007/0173440; US2007/0155820; US2007/0149622; US2007/0149580; US2007/0134273; US2007/0129389; US2007/0112031; US2007/0099964; US2007/0099952; US2007/0098716; US2007/0093480; US2007/0082929; US2007/0004765; US2007/0004654; US2006/0286102; US2006/0276381; US2006/0265767; US2006/0263368; US2006/0257867; US2006/0223742; US2006/0211752; US2006/0199796; US2006/0194821; US2006/0166871; US2006/0147456; US2006/0134128; US2006/0115475; US2006/0110746; US2006/0058255; US2006/0025566; US2006/0009454; US2006/0009452; US2006/0002866; US2005/0288316; US2005/0288243; US2005/0250719; US2005/0249751; US2005/0246794; US2005/0227921; US2005/0222171; US2005/0197341; US2005/0187237; US2005/0182006; US2005/0175581; US2005/0171182; US2005/0164298; US2005/0153955; US2005/0153878; US2005/0148511; US2005/0143381; US2005/0119273; US2005/0106142; US2005/0096363; US2005/0070493; US2005/0043233; US2005/0043221; US2005/0038049; US2005/0015263; US2005/0009870; US2004/0266777; US2004/0261190; US2004/0248965; US2004/0248884; US2004/0242559; US2004/0241797; US2004/0229250; US2004/0220270; US2004/0204368; US2004/0192629; US2004/0186157; US2004/0132648; US2004/0091919; US2004/0072836; US2004/0063708; US2004/0063707; US2004/0057950; US2003/0225098; US2003/0220246; US2003/0211967; US2003/0199525; US2003/0187001; US2003/0186844; US2003/0166574; US2003/0166573; US2003/0166001; US2003/0153752; US2003/0077298; US2003/0069430; US2003/0059455; US2003/0040612; US2009/0099069; US2008/0312413; US2008/0280845; US2008/0248462; US2008/0248462; US2008/0213250; US2008/0145313; US2008/0021080; US2008/0021036; US2008/0004309; US2007/0298124; US2007/0298104; US2007/0281986; US2007/0264195; US2007/0232556; US2007/0190149; US2007/0111934;

US2007/0071675; US2007/0021360; US2007/0010658; US2006/0235034; US2006/0233799; US2006/0160737; US2006/0128696; US2006/0121042; US2006/0039904; US2006/0019882; US2005/0272655; US2005/0197293; US2004/0247592; US2004/0204356; US2004/0132023; US2004/0116669; US2004/0072836; US2004/0048895; US2004/0022765; US2003/0165485; US2003/0162964; US2003/0153503; US2003/0125276; US2003/0114657; US2003/0091569; US2003/0078199; US2002/0137095; US2001/0006793; US2001/0002393; US2002/0183319; and US2002/0156081, each of which is hereby specifically incorporated by reference in its entirety.

4. Oligonucleotide inhibitors of autophagy-related gene products

In certain embodiments of the present invention, oligonucleotide inhibitors of autophagy-related RNA gene products are used to modulate autophagy and to treat autophagy-related diseases. Oligonucleotide inhibitors include, but are not limited to, antisense molecules, siRNA molecules, shRNA molecules, ribozymes and triplex molecules. Such molecules are known in the art and the skilled artisan would be able to create oligonucleotide inhibitors for any of the autophagy-related genes of the invention using routine methods.

Antisense molecules, siRNA or shRNA molecules, ribozymes or triplex molecules may be contacted with a cell or administered to an organism. Alternatively, constructs encoding such molecules may be contacted with or introduced into a cell or organism. Antisense constructs, antisense oligonucleotides, RNA interference constructs or siRNA duplex RNA molecules can be used to interfere with expression of a protein of interest, *e.g.,* an autophagy-related gene of the present invention. Typically at least 15, 17, 19, or 21 nucleotides of the complement of the mRNA sequence are sufficient for an antisense molecule. Typically at least 15, 19, 21, 22, or 23 nucleotides of a target sequence are sufficient for an RNA interference molecule. In some embodiments, an RNA interference molecule will have a 2 nucleotide 3' overhang. If the RNA interference molecule is expressed in a cell from a construct, for example from a hairpin molecule or from an inverted repeat of the desired autophagy-related gene sequence, then the endogenous cellular machinery may create the overhangs. siRNA molecules can be prepared by chemical synthesis, in vitro transcription, or digestion of long dsRNA by Rnase III or Dicer. These can be introduced into cells by transfection, electroporation, intracellular infection or

other methods known in the art. See, for example: Hannon, GJ, 2002, RNA Interference, Nature 418: 244-251; Bernstein E *et al.*, 2002, The rest is silence. RNA 7: 1509-1521; Hutvagner G *et al.*, RNAi: Nature abhors a double-strand. Cur. Open. Genetics & Development 12: 225-232; Brummelkamp, 2002, A system for stable expression of short interfering RNAs in mammalian cells. Science 296: 550-553; Lee NS, Dohjima T, Bauer G, Li H, Li M-J, Ehsani A, Salvaterra P, and Rossi J. (2002). Expression of small interfering RNAs targeted against HIV-1 rev transcripts in human cells. Nature Biotechnol. 20:500-505; Miyagishi M, and Taira K. (2002). U6-promoter-driven siRNAs with four uridine 3' overhangs efficiently suppress targeted gene expression in mammalian cells. Nature Biotechnol. 20:497-500; Paddison PJ, Caudy AA, Bernstein E, Hannon GJ, and Conklin DS. (2002). Short hairpin RNAs (shRNAs) induce sequence-specific silencing in mammalian cells. Genes & Dev. 16:948-958; Paul CP, Good PD, Winer I, and Engelke DR. (2002). Effective expression of small interfering RNA in human cells. Nature Biotechnol. 20:505-508; Sui G, Soohoo C, Affar E-B, Gay F, Shi Y, Forrester WC, and Shi Y. (2002). A DNA vector-based RNAi technology to suppress gene expression in mammalian cells. Proc. Natl. Acad. Sci. USA 99(6):5515-5520; Yu J-Y, DeRuiter SL, and Turner DL. (2002). RNA interference by expression of short-interfering RNAs and hairpin RNAs in mammalian cells. Proc. Natl. Acad. Sci. USA 99(9):6047-6052, PCT publications WO2006/066048 and WO2009/029688, US published application US2009/0123426, each of which is incorporated by reference in its entirety.

Antisense or RNA interference molecules can be delivered in vitro to cells or in vivo, *e.g.,* to tumors or diseased tissues of a mammal. Typical delivery means known in the art can be used. For example, delivery to a tumor can be accomplished by intratumoral injections. Other modes of delivery can be used without limitation, including: intravenous, intramuscular, intraperitoneal, intraarterial, local delivery during surgery, endoscopic, subcutaneous, and per os. Vectors can be selected for desirable properties for any particular application. Vectors can be viral, bacterial or plasmid. Adenoviral vectors are useful in this regard. Tissue-specific, cell-type specific, or otherwise regulatable promoters can be used to control the transcription of the inhibitory polynucleotide molecules. Non-viral carriers such as liposomes or nanospheres can also be used.

In the present methods, a RNA interference molecule or an RNA interference encoding oligonucleotide can be administered to the subject, for example, as naked RNA, in combination with a delivery reagent, and/or as a nucleic acid comprising sequences that

express the siRNA or shRNA molecules. In some embodiments the nucleic acid comprising sequences that express the siRNA or shRNA molecules are delivered within vectors, *e.g.* plasmid, viral and bacterial vectors. Any nucleic acid delivery method known in the art can be used in the present invention. Suitable delivery reagents include, but are not limited to, *e.g*, the Mirus Transit TKO lipophilic reagent; lipofectin; lipofectamine; cellfectin; polycations (*e.g.,* polylysine), atelocollagen, nanoplexes and liposomes.

The use of atelocollagen as a delivery vehicle for nucleic acid molecules is described in Minakuchi *et al.* Nucleic Acids Res., 32(13):e109 (2004); Hanai *et al.* Ann NY Acad Sci., 1082:9-17 (2006); and Kawata *et al.* Mol Cancer Ther., 7(9):2904-12 (2008); each of which is incorporated herein in their entirety.

In some embodiments of the invention, liposomes are used to deliver an inhibitory oligonucleotide to a subject. Liposomes suitable for use in the invention can be formed from standard vesicle-forming lipids, which generally include neutral or negatively charged phospholipids and a sterol, such as cholesterol. The selection of lipids is generally guided by consideration of factors such as the desired liposome size and half-life of the liposomes in the blood stream. A variety of methods are known for preparing liposomes, for example, as described in Szoka *et al.* (1980), Ann. Rev. Biophys. Bioeng. 9:467; and U.S. Pat. Nos. 4,235,871, 4,501,728, 4,837,028, and 5,019,369, the entire disclosures of which are herein incorporated by reference.

The liposomes for use in the present methods can comprise a ligand molecule that targets the liposome to cancer cells, pancreatic cells or neurons. Ligands which bind to receptors prevalent in cancer cells, pancreatic cells or neurons, such as monoclonal antibodies that bind to cell-type specific antigens, are preferred.

The liposomes for use in the present methods can also be modified so as to avoid clearance by the mononuclear macrophage system ("MMS") and reticuloendothelial system ("RES"). Such modified liposomes have opsonization-inhibition moieties on the surface or incorporated into the liposome structure. In an embodiment, a liposome of the invention can comprise both opsonization-inhibition moieties and a ligand.

Opsonization-inhibiting moieties for use in preparing the liposomes of the invention are typically large hydrophilic polymers that are bound to the liposome membrane. As used herein, an opsonization inhibiting moiety is "bound" to a liposome membrane when it is chemically or physically attached to the membrane, *e.g.*, by the intercalation of a lipid-soluble anchor into the membrane itself, or by binding directly to active groups of

membrane lipids. These opsonization-inhibiting hydrophilic polymers form a protective surface layer that significantly decreases the uptake of the liposomes by the MMS and RES; *e.g.*, as described in U.S. Pat. No. 4,920,016, the entire disclosure of which is herein incorporated by reference.

Opsonization inhibiting moieties suitable for modifying liposomes are preferably water-soluble polymers with a number-average molecular weight from about 500 to about 40,000 daltons, and more preferably from about 2,000 to about 20,000 daltons. Such polymers include polyethylene glycol (PEG) or polypropylene glycol (PPG) derivatives; *e.g.*, methoxy PEG or PPG, and PEG or PPG stearate; synthetic polymers such as polyacrylamide or poly N-vinyl pyrrolidone; linear, branched, or dendrimeric polyamidoamines; polyacrylic acids; polyalcohols, *e.g.*, polyvinylalcohol and polyxylitol to which carboxylic or amino groups are chemically linked, as well as gangliosides, such as ganglioside GM1. Copolymers of PEG, methoxy PEG, or methoxy PPG, or derivatives thereof, are also suitable. In addition, the opsonization inhibiting polymer can be a block copolymer of PEG and either a polyamino acid, polysaccharide, polyamidoamine, polyethyleneamine, or polynucleotide. The opsonization inhibiting polymers can also be natural polysaccharides containing amino acids or carboxylic acids, *e.g.*, galacturonic acid, glucuronic acid, mannuronic acid, hyaluronic acid, pectic acid, neuraminic acid, alginic acid, carrageenan; aminated polysaccharides or oligosaccharides (linear or branched); or carboxylated polysaccharides or oligosaccharides, *e.g.*, reacted with derivatives of carbonic acids with resultant linking of carboxylic groups. Preferably, the opsonization-inhibiting moiety is a PEG, PPG, or derivatives thereof. Liposomes modified with PEG or PEG-derivatives are sometimes called "PEGylated liposomes."

The opsonization inhibiting moiety can be bound to the liposome membrane by any one of numerous well-known techniques. For example, an N-hydroxysuccinimide ester of PEG can be bound to a phosphatidyl-ethanolamine lipid-soluble anchor, and then bound to a membrane. Similarly, a dextran polymer can be derivatized with a stearylamine lipid-soluble anchor via reductive amination using $Na(CN)BH_3$ and a solvent mixture, such as tetrahydrofuran and water in a 30:12 ratio at 60°C.

Liposomes modified with opsonization-inhibition moieties remain in the circulation much longer than unmodified liposomes. For this reason, such liposomes are sometimes called "stealth" liposomes. Stealth liposomes are known to accumulate in tissues fed by porous or "leaky" microvasculature. Thus, tissue characterized by such microvasculature

defects, for example solid tumors, will efficiently accumulate these liposomes; see Gabizon, *et al.* (1988), Proc. Natl. Acad. Sci., USA, 18:6949-53. In addition, the reduced uptake by the RES lowers the toxicity of stealth liposomes by preventing significant accumulation of the liposomes in the liver and spleen.

5. Antibodies specific for autophagy-related gene products

Because of their ability to bind to a particular target with high specificity, antibodies specific for polypeptide autophagy-related gene products are able to either inhibit or enhance the activities of such gene products and thereby inhibit or enhance autophagy. For example, in some embodiments, an antibody specific for a receptor can inhibit the activity of the receptor by blocking its interaction with an activating ligand. Likewise, antibodies specific for a soluble ligand (*e.g.* a cytokine or growth factor) or a membrane-bound ligand can inhibit the activity of a receptor that is capable of binding to the ligand by inhibiting the binding of the ligand to the receptor. In other embodiments, antibodies specific for a receptor can be used to cross-link and thereby activate the receptor. Though antibodies are particularly useful in inhibiting or enhancing the activity extracellular proteins (*e.g.,* receptors and/or ligands), the use of intracellular antibodies to inhibit protein function in a cell is also known in the art (see *e.g.*, Carlson, J. R. (1988) *Mol. Cell. Biol.* 8:2638-2646; Biocca, S. *et al.* (1990) *EMBO J.* 9:101-108; Werge, T. M. *et al.* (1990) *FEBS Lett.* 274:193-198; Carlson, J. R. (1993) *Proc. Natl. Acad. Sci. USA* 90:7427-7428; Marasco, W. A. *et al.* (1993) *Proc. Natl. Acad. Sci. USA* 90:7889-7893; Biocca, S. *et al.* (1994) *Biotechnology (NY)* 12:396-399; Chen, S-Y. *et al.* (1994) *Hum. Gene Ther.* 5:595-601; Duan, L *et al.* (1994) *Proc. Natl. Acad. Sci. USA* 91:5075-5079; Chen, S-Y. *et al.* (1994) *Proc. Natl. Acad. Sci. USA* 91:5932-5936; Beerli, R. R. *et al.* (1994) *J. Biol. Chem.* 269:23931-23936; Beerli, R. R. *et al.* (1994) *Biochem. Biophys. Res. Commun.* 204:666-672; Mhashilkar, A. M. *et al.* (1995) *EMBO J.* 14:1542-1551; Richardson, J. H. *et al.* (1995) *Proc. Natl. Acad. Sci. USA* 92:3137-3141; PCT Publication No. WO 94/02610 by Marasco *et al.*; and PCT Publication No. WO 95/03832 by Duan *et al.*). Therefore, antibodies specific for peptide products of autophagy-related genes are useful as biological agents for the methods of the present invention.

Antibodies that specifically bind to a peptide product of an autophagy-related gene can be produced using a variety of known techniques, such as the standard somatic cell hybridization technique described by Kohler and Milstein, Nature 256: 495 (1975).

Additionally, other techniques for producing monoclonal antibodies known in the art can also be employed, *e.g.*, viral or oncogenic transformation of B lymphocytes, phage display technique using libraries of human antibody genes.

Polyclonal antibodies can be prepared by immunizing a suitable subject with a polypeptide immunogen. The polypeptide antibody titer in the immunized subject can be monitored over time by standard techniques, such as with an enzyme linked immunosorbent assay (ELISA) using immobilized polypeptide. If desired, the antibody directed against the antigen can be isolated from the mammal (*e.g.*, from the blood) and further purified by well known techniques, such as protein A chromatography to obtain the IgG fraction. At an appropriate time after immunization, *e.g.*, when the antibody titers are highest, antibody-producing cells can be obtained from the subject and used to prepare monoclonal antibodies.

Any of the many well known protocols used for fusing lymphocytes and immortalized cell lines can be applied for the purpose of generating monoclonal antibodies specific against the products of autophagy-related genes (see, *e.g.*, Galfre, G. *et al.* (1977) *Nature* 266:55052; Gefter *et al.* (1977) *supra*; Lerner (1981) *supra*; Kenneth (1980) *supra*). Moreover, the ordinary skilled worker will appreciate that there are many variations of such methods which also would be useful. Typically, an immortal cell line (*e.g.*, a myeloma cell line) is derived from the same mammalian species as the lymphocytes. For example, murine hybridomas can be made by fusing lymphocytes from a mouse immunized with an immunogenic preparation of the present invention with an immortalized mouse cell line. An example of an appropriate mouse cell lines are mouse myeloma cell lines that are sensitive to culture medium containing hypoxanthine, aminopterin and thymidine ("HAT medium"). Any of a number of myeloma cell lines can be used as a fusion partner according to standard techniques, *e.g.*, the P3-NS1/1-Ag4-1, P3-x63-Ag8.653 or Sp2/O-Ag14 myeloma lines. These myeloma lines are available from the American Type Culture Collection (ATCC), Rockville, Md. Typically, HAT-sensitive mouse myeloma cells are fused to mouse splenocytes using polyethylene glycol ("PEG"). Hybridoma cells resulting from the fusion are then selected using HAT medium, which kills unfused and unproductively fused myeloma cells (unfused splenocytes die after several days because they are not transformed). Hybridoma cells producing a monoclonal antibody of the invention are detected by screening the hybridoma culture supernatants for antibodies that bind a given polypeptide, *e.g.*, using a standard ELISA assay.

As an alternative to preparing monoclonal antibody-secreting hybridomas, a monoclonal antibody specific for one of the above described autophagy-related gene products can be identified and isolated by screening a recombinant combinatorial immunoglobulin library (*e.g.*, an antibody phage or yeast display library) with the appropriate autophagy-related gene product to thereby isolate immunoglobulin library members that bind the autophagy-related gene product. Kits for generating and screening phage display libraries are commercially available (*e.g.*, the Pharmacia *Recombinant Phage Antibody System*, Catalog No. 27-9400-01; and the Stratagene *SurfZAP*^{TM} *Phage Display Kit*, Catalog No. 240612), and methods for screening phage and yeast display libraries are known in the art. Examples of methods and reagents particularly amenable for use in generating and screening an antibody display library can be found in, for example, Ladner *et al.* U.S. Patent No. 5,223,409; Kang *et al.* International Publication No. WO 92/18619; Dower *et al.* International Publication No. WO 91/17271; Winter *et al.* International Publication WO 92/20791; Markland *et al.* International Publication No. WO 92/15679; Breitling *et al.* International Publication WO 93/01288; McCafferty *et al.* International Publication No. WO 92/01047; Garrard *et al.* International Publication No. WO 92/09690; Ladner *et al.* International Publication No. WO 90/02809; Fuchs *et al.* (1991) *Biotechnology (NY)* 9:1369-1372; Hay *et al.* (1992) *Hum. Antibod. Hybridomas* 3:81-85; Huse *et al.* (1989) *Science* 246:1275-1281; Griffiths *et al.* (1993) *EMBO J.* 12:725-734; Hawkins *et al.* (1992) *J. Mol. Biol.* 226:889-896; Clarkson *et al.* (1991) *Nature* 352:624-628; Gram *et al.* (1992) *Proc. Natl. Acad. Sci. USA* 89:3576-3580; Garrard *et al.* (1991) *Biotechnology (NY)* 9:1373-1377; Hoogenboom *et al.* (1991) *Nucleic Acids Res.* 19:4133-4137; Barbas *et al.* (1991) *Proc. Natl. Acad. Sci. USA* 88:7978-7982; and McCafferty *et al.* (1990) *Nature* 348:552-554.

In addition, chimeric and humanized antibodies against autophagy-related gene products can be made according to standard protocols such as those disclosed in US patent 5,565,332. In another embodiment, antibody chains or specific binding pair members can be produced by recombination between vectors comprising nucleic acid molecules encoding a fusion of a polypeptide chain of a specific binding pair member and a component of a replicable generic display package and vectors containing nucleic acid molecules encoding a second polypeptide chain of a single binding pair member using techniques known in the art, *e.g.*, as described in US patents 5,565,332, 5,871,907, or 5,733,743.

In another embodiment, human monoclonal antibodies directed against autophagy-related gene product can be generated using transgenic or transchromosomal mice carrying parts of the human immune system rather than the mouse system. In one embodiment, transgenic mice, referred to herein as "humanized mice," which contain a human immunoglobulin gene miniloci that encodes unrearranged human heavy and light chain variable region immunoglobulin sequences, together with targeted mutations that inactivate or delete the endogenous μ and κ chain loci (Lonberg, N. *et al.* (1994) Nature 368(6474): 856 859). The mice may also contain human heavy chain constant region immunoglobulin sequences. Accordingly, the mice express little or no mouse IgM or κ, and in response to immunization, the introduced human heavy and light chain variable region transgenes undergo class switching and somatic mutation to generate high affinity human variable region antibodies (Lonberg, N. *et al.* (1994), supra; reviewed in Lonberg, N. (1994) Handbook of Experimental Pharmacology 113:49 101; Lonberg, N. and Huszar, D. (1995) Intern. Rev. Immunol. Vol. 13: 65 93, and Harding, F. and Lonberg, N. (1995) Ann. N. Y Acad. Sci 764:536 546). These mice can be used to generate fully human monoclonal antibodies using the techniques described above or any other technique known in the art. The preparation of humanized mice is described in Taylor, L. *et al.* (1992) Nucleic Acids Research 20:6287 6295; Chen, J. *et al.* (1993) International Immunology 5: 647 656; Tuaillon *et al.* (1993) Proc. Natl. Acad. Sci USA 90:3720 3724; Choi *et al.* (1993) Nature Genetics 4:117 123; Chen, J. *et al.* (1993) EMBO J. 12: 821 830; Tuaillon *et al.* (1994) J. Immunol. 152:2912 2920; Lonberg *et al.*, (1994) Nature 368(6474): 856 859; Lonberg, N. (1994) Handbook of Experimental Pharmacology 113:49 101; Taylor, L. *et al.* (1994) International Immunology 6: 579 591; Lonberg, N. and Huszar, D. (1995) Intern. Rev. Immunol. Vol. 13: 65 93; Harding, F. and Lonberg, N. (1995) Ann. N.Y. Acad. Sci 764:536 546; Fishwild, D. *et al.* (1996) Nature Biotechnology 14: 845 851. See further, U.S. Pat. Nos. 5,545,806; 5,569,825; 5,625,126; 5,633,425; 5,789,650; 5,877,397; 5,661,016; 5,814,318; 5,874,299; and 5,770,429; all to Lonberg and Kay, and GenPharm International; U.S. Pat. No. 5,545,807 to Surani *et al.*

6. Pharmaceutical compositions

The invention provides pharmaceutical compositions comprising modulators of autophagy-related gene products. In one aspect, the present invention provides pharmaceutically acceptable compositions which comprise a therapeutically-effective

amount of one or more of the agents described above, formulated together with one or more pharmaceutically acceptable carriers (additives) and/or diluents. In another aspect, the agents of the invention can be administered as such, or administered in mixtures with pharmaceutically acceptable carriers and can also be administered in conjunction with other agents. Conjunctive therapy thus includes sequential, simultaneous and separate, or co-administration of one or more agent of the invention, wherein the therapeutic effects of the first administered has not entirely disappeared when the subsequent compound is administered.

As described in detail below, the pharmaceutical compositions of the present invention may be specially formulated for administration in solid or liquid form, including those adapted for the following: (1) oral administration, for example, drenches (aqueous or non-aqueous solutions or suspensions), tablets, *e.g.*, those targeted for buccal, sublingual, and systemic absorption, boluses, powders, granules, pastes for application to the tongue; (2) parenteral administration, for example, by subcutaneous, intramuscular, intravenous or epidural injection as, for example, a sterile solution or suspension, or sustained-release formulation; (3) topical application, for example, as a cream, ointment, or a controlled-release patch or spray applied to the skin; (4) intravaginally or intrarectally, for example, as a pessary, cream or foam; (5) sublingually; (6) ocularly; (7) transdermally; or (8) nasally.

As set out above, in certain embodiments, agents of the invention may be compounds containing a basic functional group, such as amino or alkylamino, and are, thus, capable of forming pharmaceutically-acceptable salts with pharmaceutically-acceptable acids. These salts can be prepared *in situ* in the administration vehicle or the dosage form manufacturing process, or through a separate reaction of a purified compound of the invention in its free base form with a suitable organic or inorganic acid, and isolating the salt thus formed during subsequent purification. Representative salts include the hydrobromide, hydrochloride, sulfate, bisulfate, phosphate, nitrate, acetate, valerate, oleate, palmitate, stearate, laurate, benzoate, lactate, phosphate, tosylate, citrate, maleate, fumarate, succinate, tartrate, napthylate, mesylate, glucoheptonate, lactobionate, and laurylsulphonate salts and the like (see, for example, Berge *et al.* (1977) "Pharmaceutical Salts", *J. Pharm. Sci.* 66:1-19).

The pharmaceutically acceptable salts of the subject compounds include the conventional nontoxic salts or quaternary ammonium salts of the compounds, *e.g.*, from non-toxic organic or inorganic acids. For example, such conventional nontoxic salts

include those derived from inorganic acids such as hydrochloride, hydrobromic, sulfuric, sulfamic, phosphoric, nitric, and the like; and the salts prepared from organic acids such as acetic, propionic, succinic, glycolic, stearic, lactic, malic, tartaric, citric, ascorbic, palmitic, maleic, hydroxymaleic, phenylacetic, glutamic, benzoic, salicyclic, sulfanilic, 2-acetoxybenzoic, fumaric, toluenesulfonic, methanesulfonic, ethane disulfonic, oxalic, isothionic, and the like.

In other cases, the agents of the present invention may be compounds containing one or more acidic functional groups and, thus, are capable of forming pharmaceutically-acceptable salts with pharmaceutically-acceptable bases. These salts can likewise be prepared *in situ* in the administration vehicle or the dosage form manufacturing process, or by separately reacting the purified compound in its free acid form with a suitable base, such as the hydroxide, carbonate or bicarbonate of a pharmaceutically-acceptable metal cation, with ammonia, or with a pharmaceutically-acceptable organic primary, secondary or tertiary amine. Representative alkali or alkaline earth salts include the lithium, sodium, potassium, calcium, magnesium, and aluminum salts and the like. Representative organic amines useful for the formation of base addition salts include ethylamine, diethylamine, ethylenediamine, ethanolamine, diethanolamine, piperazine and the like (see, for example, Berge *et al.*, *supra*).

Wetting agents, emulsifiers and lubricants, such as sodium lauryl sulfate and magnesium stearate, as well as coloring agents, release agents, coating agents, sweetening, flavoring and perfuming agents, preservatives and antioxidants can also be present in the compositions.

Examples of pharmaceutically-acceptable antioxidants include: (1) water soluble antioxidants, such as ascorbic acid, cysteine hydrochloride, sodium bisulfate, sodium metabisulfite, sodium sulfite and the like; (2) oil-soluble antioxidants, such as ascorbyl palmitate, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), lecithin, propyl gallate, alpha-tocopherol, and the like; and (3) metal chelating agents, such as citric acid, ethylenediamine tetraacetic acid (EDTA), sorbitol, tartaric acid, phosphoric acid, and the like.

The formulations of the agents of the invention may be presented in unit dosage form and may be prepared by any methods well known in the art of pharmacy. The amount of active ingredient which can be combined with a carrier material to produce a single dosage form will vary depending upon the host being treated and the particular mode of

administration. The amount of active ingredient which can be combined with a carrier material to produce a single dosage form will generally be that amount of the agent which produces a therapeutic effect.

In certain embodiments, a formulation of the present invention comprises an excipient, including, but not limited to, cyclodextrins, liposomes, micelle forming agents, *e.g.*, bile acids, and polymeric carriers, *e.g.*, polyesters and polyanhydrides; and an agent of the present invention. In certain embodiments, an aforementioned formulation renders orally bioavailable a agent of the present invention.

Methods of preparing these formulations or compositions may include the step of bringing into association an agent of the present invention with the carrier and, optionally, one or more accessory ingredients.

Liquid dosage forms for oral administration of the compounds of the invention include pharmaceutically acceptable emulsions, microemulsions, solutions, suspensions, syrups and elixirs. In addition to the active ingredient, the liquid dosage forms may contain inert diluents commonly used in the art, such as, for example, water or other solvents, solubilizing agents and emulsifiers, such as ethyl alcohol, isopropyl alcohol, ethyl carbonate, ethyl acetate, benzyl alcohol, benzyl benzoate, propylene glycol, 1,3-butylene glycol, oils (in particular, cottonseed, groundnut, corn, germ, olive, castor and sesame oils), glycerol, tetrahydrofuryl alcohol, polyethylene glycols and fatty acid esters of sorbitan, and mixtures thereof.

Besides inert diluents, the oral compositions can also include adjuvants such as wetting agents, emulsifying and suspending agents, sweetening, flavoring, coloring, perfuming and preservative agents.

Suspensions, in addition to the active compounds, may contain suspending agents as, for example, ethoxylated isostearyl alcohols, polyoxyethylene sorbitol and sorbitan esters, microcrystalline cellulose, aluminum metahydroxide, bentonite, agar-agar and tragacanth, and mixtures thereof.

Formulations of the invention suitable for oral administration may be in the form of capsules, cachets, pills, tablets, lozenges (using a flavored basis, usually sucrose and acacia or tragacanth), powders, granules, or as a solution or a suspension in an aqueous or non-aqueous liquid, or as an oil-in-water or water-in-oil liquid emulsion, or as an elixir or syrup, or as pastilles (using an inert base, such as gelatin and glycerin, or sucrose and acacia) and/or as mouth washes and the like, each containing a predetermined amount of a

compound of the present invention as an active ingredient. A compound of the present invention may also be administered as a bolus, electuary or paste.

In solid dosage forms of the invention for oral administration (capsules, tablets, pills, dragees, powders, granules and the like), the active ingredient is mixed with one or more pharmaceutically-acceptable carriers, such as sodium citrate or dicalcium phosphate, and/or any of the following: (1) fillers or extenders, such as starches, lactose, sucrose, glucose, mannitol, and/or silicic acid; (2) binders, such as, for example, carboxymethylcellulose, alginates, gelatin, polyvinyl pyrrolidone, sucrose and/or acacia; (3) humectants, such as glycerol; (4) disintegrating agents, such as agar-agar, calcium carbonate, potato or tapioca starch, alginic acid, certain silicates, and sodium carbonate; (5) solution retarding agents, such as paraffin; (6) absorption accelerators, such as quaternary ammonium compounds; (7) wetting agents, such as, for example, cetyl alcohol, glycerol monostearate, and non-ionic surfactants; (8) absorbents, such as kaolin and bentonite clay; (9) lubricants, such a talc, calcium stearate, magnesium stearate, solid polyethylene glycols, sodium lauryl sulfate, and mixtures thereof; and (10) coloring agents. In the case of capsules, tablets and pills, the pharmaceutical compositions may also comprise buffering agents. Solid compositions of a similar type may also be employed as fillers in soft and hard-shelled gelatin capsules using such excipients as lactose or milk sugars, as well as high molecular weight polyethylene glycols and the like.

A tablet may be made by compression or molding, optionally with one or more accessory ingredients. Compressed tablets may be prepared using binder (for example, gelatin or hydroxypropylmethyl cellulose), lubricant, inert diluent, preservative, disintegrant (for example, sodium starch glycolate or cross-linked sodium carboxymethyl cellulose), surface-active or dispersing agent. Molded tablets may be made by molding in a suitable machine a mixture of the powdered compound moistened with an inert liquid diluent.

The tablets, and other solid dosage forms of the pharmaceutical compositions of the present invention, such as dragees, capsules, pills and granules, may optionally be scored or prepared with coatings and shells, such as enteric coatings and other coatings well known in the pharmaceutical-formulating art. They may also be formulated so as to provide slow or controlled release of the active ingredient therein using, for example, hydroxypropylmethyl cellulose in varying proportions to provide the desired release profile, other polymer matrices, liposomes and/or microspheres. Compositions of the invention may also be

formulated for rapid release, *e.g.*, freeze-dried. They may be sterilized by, for example, filtration through a bacteria-retaining filter, or by incorporating sterilizing agents in the form of sterile solid compositions which can be dissolved in sterile water, or some other sterile injectable medium immediately before use. These compositions may also optionally contain opacifying agents and may be of a composition that they release the active ingredient(s) only, or preferentially, in a certain portion of the gastrointestinal tract, optionally, in a delayed manner. Examples of embedding compositions which can be used include polymeric substances and waxes. The active ingredient can also be in micro-encapsulated form, if appropriate, with one or more of the above-described excipients.

Formulations of the pharmaceutical compositions of the invention for rectal or vaginal administration may be presented as a suppository, which may be prepared by mixing one or more compounds of the invention with one or more suitable nonirritating excipients or carriers comprising, for example, cocoa butter, polyethylene glycol, a suppository wax or a salicylate, and which is solid at room temperature, but liquid at body temperature and, therefore, will melt in the rectum or vaginal cavity and release the active compound.

Formulations of the present invention which are suitable for vaginal administration also include pessaries, tampons, creams, gels, pastes, foams or spray formulations containing such carriers as are known in the art to be appropriate.

Dosage forms for the topical or transdermal administration of a compound of this invention include powders, sprays, ointments, pastes, creams, lotions, gels, solutions, patches and inhalants. The active compound may be mixed under sterile conditions with a pharmaceutically-acceptable carrier, and with any preservatives, buffers, or propellants which may be required.

The ointments, pastes, creams and gels may contain, in addition to an active compound of this invention, excipients, such as animal and vegetable fats, oils, waxes, paraffins, starch, tragacanth, cellulose derivatives, polyethylene glycols, silicones, bentonites, silicic acid, talc and zinc oxide, or mixtures thereof.

Powders and sprays can contain, in addition to a compound of this invention, excipients such as lactose, talc, silicic acid, aluminum hydroxide, calcium silicates and polyamide powder, or mixtures of these substances. Sprays can additionally contain customary propellants, such as chlorofluorohydrocarbons and volatile unsubstituted hydrocarbons, such as butane and propane.

Transdermal patches have the added advantage of providing controlled delivery of a compound of the present invention to the body. Such dosage forms can be made by dissolving or dispersing the compound in the proper medium. Absorption enhancers can also be used to increase the flux of the compound across the skin. The rate of such flux can be controlled by either providing a rate controlling membrane or dispersing the compound in a polymer matrix or gel.

Ophthalmic formulations, eye ointments, powders, solutions and the like, are also contemplated as being within the scope of this invention.

Pharmaceutical compositions of this invention suitable for parenteral administration comprise one or more compounds of the invention in combination with one or more pharmaceutically-acceptable sterile isotonic aqueous or nonaqueous solutions, dispersions, suspensions or emulsions, or sterile powders which may be reconstituted into sterile injectable solutions or dispersions just prior to use, which may contain sugars, alcohols, antioxidants, buffers, bacteriostats, solutes which render the formulation isotonic with the blood of the intended recipient or suspending or thickening agents.

Examples of suitable aqueous and nonaqueous carriers which may be employed in the pharmaceutical compositions of the invention include water, ethanol, polyols (such as glycerol, propylene glycol, polyethylene glycol, and the like), and suitable mixtures thereof, vegetable oils, such as olive oil, and injectable organic esters, such as ethyl oleate. Proper fluidity can be maintained, for example, by the use of coating materials, such as lecithin, by the maintenance of the required particle size in the case of dispersions, and by the use of surfactants.

In some cases, in order to prolong the effect of a drug, it is desirable to slow the absorption of the drug from subcutaneous or intramuscular injection. This may be accomplished by the use of a liquid suspension of crystalline or amorphous material having poor water solubility. The rate of absorption of the drug then depends upon its rate of dissolution which, in turn, may depend upon crystal size and crystalline form. Alternatively, delayed absorption of a parenterally-administered drug form is accomplished by dissolving or suspending the drug in an oil vehicle.

Injectable depot forms are made by forming microencapsule matrices of the subject compounds in biodegradable polymers such as polylactide-polyglycolide. Depending on the ratio of drug to polymer, and the nature of the particular polymer employed, the rate of drug release can be controlled. Examples of other biodegradable polymers include

poly(orthoesters) and poly(anhydrides). Depot injectable formulations are also prepared by entrapping the drug in liposomes or microemulsions which are compatible with body tissue.

Exemplary formulations comprising agents of the invention are determined based on various properties including, but not limited to, chemical stability at body temperature, functional efficiency time of release, toxicity and optimal dose.

The preparations of the present invention may be given orally, parenterally, topically, or rectally. They are of course given in forms suitable for each administration route. For example, they are administered in tablets or capsule form, by injection, inhalation, eye lotion, ointment, suppository, administration by injection, infusion or inhalation; topical by lotion or ointment; and rectal by suppositories.

Regardless of the route of administration selected, the compounds of the present invention, which may be used in a suitable hydrated form, and/or the pharmaceutical compositions of the present invention, are formulated into pharmaceutically-acceptable dosage forms by conventional methods known to those of skill in the art.

In certain embodiments, the above-described pharmaceutical compositions comprise one or more of the agents of the invention, a chemotherapeutic agent, and optionally a pharmaceutically acceptable carrier.

The term chemotherapeutic agent includes, without limitation, platinum-based agents, such as carboplatin and cisplatin; nitrogen mustard alkylating agents; nitrosourea alkylating agents, such as carmustine (BCNU) and other alkylating agents; antimetabolites, such as methotrexate; purine analog antimetabolites; pyrimidine analog antimetabolites, such as fluorouracil (5-FU) and gemcitabine; hormonal antineoplastics, such as goserelin, leuprolide, and tamoxifen; natural antineoplastics, such as taxanes (*e.g.*, docetaxel and paclitaxel), aldesleukin, interleukin-2, etoposide (VP-16), interferon α, and tretinoin (ATRA); antibiotic natural antineoplastics, such as bleomycin, dactinomycin, daunorubicin, doxorubicin, and mitomycin; and vinca alkaloid natural antineoplastics, such as vinblastine and vincristine.

Further, the following drugs may also be used in combination with a chemotherapetutic agent, even if not considered chemotherapeutic agents themselves: dactinomycin; daunorubicin HCl; docetaxel; doxorubicin HCl; epoetin α; etoposide (VP-16); ganciclovir sodium; gentamicin sulfate; interferon α; leuprolide acetate; meperidine HCl; methadone HCl; ranitidine HCl; vinblastin sulfate; and zidovudine (AZT). For

example, fluorouracil has recently been formulated in conjunction with epinephrine and bovine collagen to form a particularly effective combination.

Still further, the following listing of amino acids, peptides, polypeptides, proteins, polysaccharides, and other large molecules may also be used: interleukins 1 through 18, including mutants and analogues; interferons or cytokines, such as interferons α, β, and γ; hormones, such as luteinizing hormone releasing hormone (LHRH) and analogues and, gonadotropin releasing hormone (GnRH); growth factors, such as transforming growth factor-β (TGF-β), fibroblast growth factor (FGF), nerve growth factor (NGF), growth hormone releasing factor (GHRF), epidermal growth factor (EGF), fibroblast growth factor homologous factor (FGFHF), hepatocyte growth factor (HGF), and insulin growth factor (IGF); tumor necrosis factor-α & β (TNF-α & β); invasion inhibiting factor-2 (IIF-2); bone morphogenetic proteins 1-7 (BMP 1-7); somatostatin; thymosin- α -1; γ-globulin; superoxide dismutase (SOD); complement factors; anti-angiogenesis factors; antigenic materials; and pro-drugs.

Chemotherapeutic agents for use with the compositions and methods of treatment described herein include, but are not limited to alkylating agents such as thiotepa and cyclosphosphamide; alkyl sulfonates such as busulfan, improsulfan and piposulfan; aziridines such as benzodopa, carboquone, meturedopa, and uredopa; ethylenimines and methylamelamines including altretamine, triethylenemelamine, trietylenephosphoramide, triethiylenethiophosphoramide and trimethylolomelamine; acetogenins (especially bullatacin and bullatacinone); a camptothecin (including the synthetic analogue topotecan); bryostatin; callystatin; CC-1065 (including its adozelesin, carzelesin and bizelesin synthetic analogues); cryptophycins (particularly cryptophycin 1 and cryptophycin 8); dolastatin; duocarmycin (including the synthetic analogues, KW-2189 and CB1-TM1); eleutherobin; pancratistatin; a sarcodictyin; spongistatin; nitrogen mustards such as chlorambucil, chlornaphazine, cholophosphamide, estramustine, ifosfamide, mechlorethamine, mechlorethamine oxide hydrochloride, melphalan, novembichin, phenesterine, prednimustine, trofosfamide, uracil mustard; nitrosureas such as carmustine, chlorozotocin, fotemustine, lomustine, nimustine, and ranimnustine; antibiotics such as the enediyne antibiotics (*e.g.*, calicheamicin, especially calicheamicin gammaII and calicheamicin omegaI1; dynemicin, including dynemicin A; bisphosphonates, such as clodronate; an esperamicin; as well as neocarzinostatin chromophore and related chromoprotein enediyne antiobiotic chromophores, aclacinomysins, actinomycin, authrarnycin, azaserine,

bleomycins, cactinomycin, carabicin, caminomycin, carzinophilin, chromomycinis, dactinomycin, daunorubicin, detorubicin, 6-diazo-5-oxo-L-norleucine, doxorubicin (including morpholino-doxorubicin, cyanomorpholino-doxorubicin, 2-pyrrolino-doxorubicin and deoxydoxorubicin), epirubicin, esorubicin, idarubicin, marcellomycin, mitomycins such as mitomycin C, mycophenolic acid, nogalamycin, olivomycins, peplomycin, potfiromycin, puromycin, quelamycin, rodorubicin, streptonigrin, streptozocin, tubercidin, ubenimex, zinostatin, zorubicin; anti-metabolites such as methotrexate and 5-fluorouracil (5-FU); folic acid analogues such as denopterin, methotrexate, pteropterin, trimetrexate; purine analogs such as fludarabine, 6-mercaptopurine, thiamiprine, thioguanine; pyrimidine analogs such as ancitabine, azacitidine, 6-azauridine, carmofur, cytarabine, dideoxyuridine, doxifluridine, enocitabine, floxuridine; androgens such as calusterone, dromostanolone propionate, epitiostanol, mepitiostane, testolactone; anti-adrenals such as aminoglutethimide, mitotane, trilostane; folic acid replenisher such as frolinic acid; aceglatone; aldophosphamide glycoside; aminolevulinic acid; eniluracil; amsacrine; bestrabucil; bisantrene; edatraxate; defofamine; demecolcine; diaziquone; elformithine; elliptinium acetate; an epothilone; etoglucid; gallium nitrate; hydroxyurea; lentinan; lonidainine; maytansinoids such as maytansine and ansamitocins; mitoguazone; mitoxantrone; mopidanmol; nitraerine; pentostatin; phenamet; pirarubicin; losoxantrone; podophyllinic acid; 2-ethylhydrazide; procarbazine; PSK polysaccharide complex); razoxane; rhizoxin; sizofuran; spirogermanium; tenuazonic acid; triaziquone; 2,2',2''-trichlorotriethylamine; trichothecenes (especially T-2 toxin, verracurin A, roridin A and anguidine); urethan; vindesine; dacarbazine; mannomustine; mitobronitol; mitolactol; pipobroman; gacytosine; arabinoside ("Ara-C"); cyclophosphamide; thiotepa; taxoids, *e.g.*, paclitaxel and doxetaxel; chlorambucil; gemcitabine; 6-thioguanine; mercaptopurine; methotrexate; platinum coordination complexes such as cisplatin, oxaliplatin and carboplatin; vinblastine; platinum; etoposide (VP-16); ifosfamide; mitoxantrone; vincristine; vinorelbine; novantrone; teniposide; edatrexate; daunomycin; aminopterin; xeloda; ibandronate; irinotecan (*e.g.*, CPT-11); topoisomerase inhibitor RFS 2000; difluoromethylomithine (DMFO); retinoids such as retinoic acid; capecitabine; and pharmaceutically acceptable salts, acids or derivatives of any of the above.

In another embodiment, the composition of the invention may comprise other biologically active substances, including therapeutic drugs or pro-drugs, for example, other chemotherapeutic agents, scavenger compounds, antibiotics, anti-virals, anti-fungals, anti-

inflammatories, vasoconstrictors and anticoagulants, antigens useful for cancer vaccine applications or corresponding pro-drugs.

Exemplary scavenger compounds include, but are not limited to thiol-containing compounds such as glutathione, thiourea, and cysteine; alcohols such as mannitol, substituted phenols; quinones, substituted phenols, aryl amines and nitro compounds.

Various forms of the chemotherapeutic agents and/or other biologically active agents may be used. These include, without limitation, such forms as uncharged molecules, molecular complexes, salts, ethers, esters, amides, and the like, which are biologically active.

7. Therapeutic Methods of the invention

The present invention further provides novel therapeutic methods of treating autophagy-related diseases, including cancer, neurodegenerative diseases, liver diseases, muscle diseases and pancreatitis, comprising administering to a subject, (*e.g.,* a subject in need thereof), an effective amount of a modulator of an autophagy-related gene product of the invention.

A subject in need thereof may include, for example, a subject who has been diagnosed with a tumor, including a pre-cancerous tumor, a cancer, or a subject who has been treated, including subjects that have been refractory to previous treatment.

Autophagy has been implicated as playing a role in the axonal degeneration that occurs following nerve injury. For example, traumatic spinal cord injury results in a rapid increase of itraaxonal calcium levels, which results in an increase in neuronal autophagy and cell death (Knoferle *et al.,* (2009), PNAS, *107*, 6064-6069). Inhibition of either calcium flux or autophagy attenuates axonal degeneration. Notably, a number of calcium binding proteins were identified in the autophagy modulator screen of the instant invention (Table 5). Thus, in certain embodiments the invention relates to the treatment or prevention of axonal degeneration following neural trauma through the modulation of calcium-binding autophagy modulating gene products or through the modulation of other autophagy-related gene products.

The methods of the present invention may be used to treat any cancerous or pre-cancerous tumor. Cancers that may treated by methods and compositions of the invention include, but are not limited to, cancer cells from the bladder, blood, bone, bone marrow, brain, breast, colon, esophagus, gastrointestine, gum, head, kidney, liver, lung, nasopharynx, neck, ovary, prostate, skin, stomach, testis, tongue, or uterus. In addition, the

cancer may specifically be of the following histological type, though it is not limited to these: neoplasm, malignant; carcinoma; carcinoma, undifferentiated; giant and spindle cell carcinoma; small cell carcinoma; papillary carcinoma; squamous cell carcinoma; lymphoepithelial carcinoma; basal cell carcinoma; pilomatrix carcinoma; transitional cell carcinoma; papillary transitional cell carcinoma; adenocarcinoma; gastrinoma, malignant; cholangiocarcinoma; hepatocellular carcinoma; combined hepatocellular carcinoma and cholangiocarcinoma; trabecular adenocarcinoma; adenoid cystic carcinoma; adenocarcinoma in adenomatous polyp; adenocarcinoma, familial polyposis coli; solid carcinoma; carcinoid tumor, malignant; branchiolo-alveolar adenocarcinoma; papillary adenocarcinoma; chromophobe carcinoma; acidophil carcinoma; oxyphilic adenocarcinoma; basophil carcinoma; clear cell adenocarcinoma; granular cell carcinoma; follicular adenocarcinoma; papillary and follicular adenocarcinoma; nonencapsulating sclerosing carcinoma; adrenal cortical carcinoma; endometroid carcinoma; skin appendage carcinoma; apocrine adenocarcinoma; sebaceous adenocarcinoma; ceruminous adenocarcinoma; mucoepidermoid carcinoma; cystadenocarcinoma; papillary cystadenocarcinoma; papillary serous cystadenocarcinoma; mucinous cystadenocarcinoma; mucinous adenocarcinoma; signet ring cell carcinoma; infiltrating duct carcinoma; medullary carcinoma; lobular carcinoma; inflammatory carcinoma; paget's disease, mammary; acinar cell carcinoma; adenosquamous carcinoma; adenocarcinoma w/squamous metaplasia; thymoma, malignant; ovarian stromal tumor, malignant; thecoma, malignant; granulosa cell tumor, malignant; and roblastoma, malignant; sertoli cell carcinoma; leydig cell tumor, malignant; lipid cell tumor, malignant; paraganglioma, malignant; extra-mammary paraganglioma, malignant; pheochromocytoma; glomangiosarcoma; malignant melanoma; amelanotic melanoma; superficial spreading melanoma; malig melanoma in giant pigmented nevus; epithelioid cell melanoma; blue nevus, malignant; sarcoma; fibrosarcoma; fibrous histiocytoma, malignant; myxosarcoma; liposarcoma; leiomyosarcoma; rhabdomyosarcoma; embryonal rhabdomyosarcoma; alveolar rhabdomyosarcoma; stromal sarcoma; mixed tumor, malignant; mullerian mixed tumor; nephroblastoma; hepatoblastoma; carcinosarcoma; mesenchymoma, malignant; brenner tumor, malignant; phyllodes tumor, malignant; synovial sarcoma; mesothelioma, malignant; dysgerminoma; embryonal carcinoma; teratoma, malignant; struma ovarii, malignant; choriocarcinoma; mesonephroma, malignant; hemangiosarcoma; hemangioendothelioma, malignant; kaposi's sarcoma; hemangiopericytoma, malignant; lymphangiosarcoma;

osteosarcoma; juxtacortical osteosarcoma; chondrosarcoma; chondroblastoma, malignant; mesenchymal chondrosarcoma; giant cell tumor of bone; ewing's sarcoma; odontogenic tumor, malignant; ameloblastic odontosarcoma; ameloblastoma, malignant; ameloblastic fibrosarcoma; pinealoma, malignant; chordoma; glioma, malignant; ependymoma; astrocytoma; protoplasmic astrocytoma; fibrillary astrocytoma; astroblastoma; glioblastoma; oligodendroglioma; oligodendroblastoma; primitive neuroectodermal; cerebellar sarcoma; ganglioneuroblastoma; neuroblastoma; retinoblastoma; olfactory neurogenic tumor; meningioma, malignant; neurofibrosarcoma; neurilemmoma, malignant; granular cell tumor, malignant; malignant lymphoma; Hodgkin's disease; Hodgkin's lymphoma; paragranuloma; malignant lymphoma, small lymphocytic; malignant lymphoma, large cell, diffuse; malignant lymphoma, follicular; mycosis fungoides; other specified non-Hodgkin's lymphomas; malignant histiocytosis; multiple myeloma; mast cell sarcoma; immunoproliferative small intestinal disease; leukemia; lymphoid leukemia; plasma cell leukemia; erythroleukemia; lymphosarcoma cell leukemia; myeloid leukemia; basophilic leukemia; eosinophilic leukemia; monocytic leukemia; mast cell leukemia; megakaryoblastic leukemia; myeloid sarcoma; and hairy cell leukemia.

In certain embodiments, the methods of the present invention include the treatment of cancer comprising the administration of an autophagy-inhibiting agent of the present invention in combination with a chemotherapeutic agent. Such autophagy-inhibiting agents include agents that inhibit the activity of products of autophagy-enhancing genes (Table 2) and agents that enhance the activity of the products of autophagy-inhibiting genes (Table 1). Any chemotherapeutic agent is suitable for use in the methods of the instant invention, particularly chemotherapeutic agents that that induce cellular stress in cancer cells. Chemotherapeutic agents useful in the instant invention include, but are not limited to, to alkylating agents such as thiotepa and cyclosphosphamide; alkyl sulfonates such as busulfan, improsulfan and piposulfan; aziridines such as benzodopa, carboquone, meturedopa, and uredopa; ethylenimines and methylamelamines including altretamine, triethylenemelamine, trietylenephosphoramide, triethiylenethiophosphoramide and trimethylolomelamine; acetogenins (especially bullatacin and bullatacinone); a camptothecin (including the synthetic analogue topotecan); bryostatin; callystatin; CC-1065 (including its adozelesin, carzelesin and bizelesin synthetic analogues); cryptophycins (particularly cryptophycin 1 and cryptophycin 8); dolastatin; duocarmycin (including the synthetic analogues, KW-2189 and CB1-TM1); eleutherobin; pancratistatin; a sarcodictyin;

spongistatin; nitrogen mustards such as chlorambucil, chlornaphazine, cholophosphamide, estramustine, ifosfamide, mechlorethamine, mechlorethamine oxide hydrochloride, melphalan, novembichin, phenesterine, prednimustine, trofosfamide, uracil mustard; nitrosureas such as carmustine, chlorozotocin, fotemustine, lomustine, nimustine, and ranimnustine; antibiotics such as the enediyne antibiotics (*e.g.*, calicheamicin, especially calicheamicin gammaII and calicheamicin omegaI1; dynemicin, including dynemicin A; bisphosphonates, such as clodronate; an esperamicin; as well as neocarzinostatin chromophore and related chromoprotein enediyne antiobiotic chromophores, aclacinomysins, actinomycin, authrarnycin, azaserine, bleomycins, cactinomycin, carabicin, caminomycin, carzinophilin, chromomycinis, dactinomycin, daunorubicin, detorubicin, 6-diazo-5-oxo-L-norleucine, doxorubicin (including morpholino-doxorubicin, cyanomorpholino-doxorubicin, 2-pyrrolino-doxorubicin and deoxydoxorubicin), epirubicin, esorubicin, idarubicin, marcellomycin, mitomycins such as mitomycin C, mycophenolic acid, nogalamycin, olivomycins, peplomycin, potfiromycin, puromycin, quelamycin, rodorubicin, streptonigrin, streptozocin, tubercidin, ubenimex, zinostatin, zorubicin; anti-metabolites such as methotrexate and 5-fluorouracil (5-FU); folic acid analogues such as denopterin, methotrexate, pteropterin, trimetrexate; purine analogs such as fludarabine, 6-mercaptopurine, thiamiprine, thioguanine; pyrimidine analogs such as ancitabine, azacitidine, 6-azauridine, carmofur, cytarabine, dideoxyuridine, doxifluridine, enocitabine, floxuridine; androgens such as calusterone, dromostanolone propionate, epitiostanol, mepitiostane, testolactone; anti-adrenals such as aminoglutethimide, mitotane, trilostane; folic acid replenisher such as frolinic acid; aceglatone; aldophosphamide glycoside; aminolevulinic acid; eniluracil; amsacrine; bestrabucil; bisantrene; edatraxate; defofamine; demecolcine; diaziquone; elformithine; elliptinium acetate; an epothilone; etoglucid; gallium nitrate; hydroxyurea; lentinan; lonidainine; maytansinoids such as maytansine and ansamitocins; mitoguazone; mitoxantrone; mopidanmol; nitraerine; pentostatin; phenamet; pirarubicin; losoxantrone; podophyllinic acid; 2-ethylhydrazide; procarbazine; PSK polysaccharide complex); razoxane; rhizoxin; sizofuran; spirogermanium; tenuazonic acid; triaziquone; 2,2',2"-trichlorotriethylamine; trichothecenes (especially T-2 toxin, verracurin A, roridin A and anguidine); urethan; vindesine; dacarbazine; mannomustine; mitobronitol; mitolactol; pipobroman; gacytosine; arabinoside ("Ara-C"); cyclophosphamide; thiotepa; taxoids, *e.g.*, paclitaxel and doxetaxel; chlorambucil; gemcitabine; 6-thioguanine; mercaptopurine; methotrexate; platinum coordination complexes such as cisplatin,

oxaliplatin and carboplatin; vinblastine; platinum; etoposide (VP-16); ifosfamide; mitoxantrone; vincristine; vinorelbine; novantrone; teniposide; edatrexate; daunomycin; aminopterin; xeloda; ibandronate; irinotecan (*e.g.*, CPT-11); topoisomerase inhibitor RFS 2000; difluoromethylomithine (DMFO); retinoids such as retinoic acid; capecitabine; and pharmaceutically acceptable salts, acids or derivatives of any of the above.

In certain embodiments, the methods of the present invention include the treatment of cancer comprising the administration of an autophagy-inhibiting agent of the present invention in combination with radiation therapy. An optimized dose of radiation therapy may be given to a subject as a daily dose. Optimized daily doses of radiation therapy may be, for example, from about 0.25 to 0.5 Gy, about 0.5 to 1.0 Gy, about 1.0 to 1.5 Gy, about 1.5 to 2.0 Gy, about 2.0 to 2.5 Gy, and about 2.5 to 3.0 Gy. An exemplary daily dose may be, for example, from about 2.0 to 3.0 Gy. A higher dose of radiation may be administered, for example, if a tumor is resistant to lower doses of radiation. High doses of radiation may reach, for example, 4 Gy. Further, the total dose of radiation administered over the course of treatment may, for example, range from about 50 to 200 Gy. In an exemplary embodiment, the total dose of radiation administered over the course of treatment ranges, for example, from about 50 to 80 Gy. In certain embodiments, a dose of radiation may be given over a time interval of, for example, 1, 2, 3, 4, or 5 minutes, wherein the amount of time is dependent on the dose rate of the radiation source.

In certain embodiments, a daily dose of optimized radiation may be administered, for example, 4 or 5 days a week, for approximately 4 to 8 weeks. In an alternate embodiment, a daily dose of optimized radiation may be administered daily seven days a week, for approximately 4 to 8 weeks. In certain embodiments, a daily dose of radiation may be given a single dose. Alternately, a daily dose of radiation may given as a plurality of doses. In a further embodiment, the optimized dose of radiation may be a higher dose of radiation than can be tolerated by the patient on a daily base. As such, high doses of radiation may be administered to a patient, but in a less frequent dosing regimen.

The types of radiation that may be used in cancer treatment are well known in the art and include electron beams, high-energy photons from a linear accelerator or from radioactive sources such as cobalt or cesium, protons, and neutrons. An exemplary ionizing radiation is an x-ray radiation.

Methods to administer radiation are well known in the art. Exemplary methods include, but are not limited to, external beam radiation, internal beam radiation, and

radiopharmaceuticals. In external beam radiation, a linear accelerator is used to deliver high-energy x-rays to the area of the body affected by cancer. Since the source of radiation originates outside of the body, external beam radiation can be used to treat large areas of the body with a uniform dose of radiation. Internal radiation therapy, also known as brachytherapy, involves delivery of a high dose of radiation to a specific site in the body. The two main types of internal radiation therapy include interstitial radiation, wherein a source of radiation is placed in the effected tissue, and intracavity radiation, wherein the source of radiation is placed in an internal body cavity a short distance from the affected area. Radioactive material may also be delivered to tumor cells by attachment to tumor-specific antibodies. The radioactive material used in internal radiation therapy is typically contained in a small capsule, pellet, wire, tube, or implant. In contrast, radiopharmaceuticals are unsealed sources of radiation that may be given orally, intravenously or directly into a body cavity.

Radiation therapy may also include sterotactic surgery or sterotactic radiation therapy, wherein a precise amount of radiation can be delivered to a small tumor area using a linear accelerator or gamma knife and three dimensional conformal radiation therapy (3DCRT), which is a computer assisted therapy to map the location of the tumor prior to radiation treatment.

A subject in need thereof may also include, for example, a subject who has been diagnosed with a neurodegenerative disease or a subject who has been treated for a neurodegenerative disease, including subjects that have been refractory to the previous treatment.

The methods of the present invention may be used to treat any neurodegenerative disease. In certain embodiments, the neurodegenerative disease is a proteinopathy, or protein-folding disease. Examples of such proteinopathies include, but are not limited to, Alzheimer's disease, Parkinson's disease, Lewy Body Dementia, ALS, Huntington's disease, spinocerebellar ataxias and spinobulbar musclular atrophy. In other embodiments, the methods of the present invention can be used to treat any neurodegenerative disease. Neurodegenerative diseases treatable by the methods of the present invention include, but are not limited to, Adrenal Leukodystrophy, alcoholism, Alexander's disease, Alper's disease, Alzheimer's disease, Amyotrophic lateral sclerosis, ataxia telangiectasia, Batten disease, bovine spongiform encephalopathy, Canavan disease, cerebral palsy, cockayne syndrome, corticobasal degeneration, Creutzfeldt-Jakob disease, familial fatal insomnia,

frontotemporal lobar degeneration, Huntington's disease, HIV-associated dementia, Kennedy's disease, Krabbe's disease, Lewy body dementia, neuroborreliosis, Machado-Joseph disease, multiple system atrophy, multiple sclerosis, narcolepsy, Niemann Pick disease, Parkinson's disease, Pelizaeus-Merzbacher disease, Pick's disease, primary lateral sclerosis, prion diseases, progressive supranuclear palsy, Refsum's disease, Sandhoff disease, Schilder's disease, subacute combined degeneration of spinal cord secondary to pernicious anaemia, Spielmeyer-Vogt-Sjogren-Batten disease, spinocerebellar ataxia, spinal muscular atrophy, Steele-Richardson-Olszewski disease, Tabes dorsalis and toxic encephalopathy.

A subject in need thereof may also include, for example, a subject who has been diagnosed with a liver disease or a subject who has been treated for a liver disease, including subjects that have been refractory to previous treatment. In certain embodiments, the liver disease is a proteinopathy, or protein-folding disease. An example of such a proteinopathy is α1-antitrypsin deficiency.

A subject in need thereof may also include, for example, a subject who has been diagnosed with a muscle disease or a subject who has been treated for a muscle disease, including subjects that have been refractory to previous treatment. In certain embodiments, the muscle disease is a proteinopathy, or protein-folding disease. Examples of such a proteinopathies include, but are not limited to, deficiency sporadic inclusion body myositis, limb girdle muscular dystrophy type 2B and Miyoshi myopathy.

A subject in need thereof may also include, for example, a subject who has been diagnosed with a proteinopathy, including subjects that have been refractory to previous treatment. Examples of proteinopathies include, but are not limited to Alzheimer's disease, cerebral β-amyloid angiopathy, retinal ganglion cell degeneration, prion diseases (*e.g.* bovine spongiform encephalopathy, kuru, Creutzfeldt-Jakob disease, variant Creutzfeldt-Jakob disease, Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia) tauopathies (*e.g.* frontotemporal dementia, Alzheimer's disease, progressive supranuclear palsy, corticobasal degeration, frontotemporal lobar degeneration), frontemporal lobar degeneration, amyotrophic lateral sclerosis, Huntington's disease, familial British dementia, Familial Danish dementia, hereditary cerebral hemorrhage with amyloidosis (Iclandic), CADASIL, Alexander disease, Seipinopathies, familial amyloidotic neuropothy, senile systemic amyloidosis, serpinopathies, AL amyloidosis, AA amyloidosis, type II diabetes, aortic medial amyloidosis, ApoAI amyloidosis, ApoII amyloidosis, ApoAIV amyloidosis,

familial amyloidosis of the Finish type, lysozyme amyloidosis, fibrinogen amyloidosis, dialysis amyloidosis, inclusion body myositis/myopathy, cataracts, medullary thyroid carcinoma, cardiac atrial amyloidosis, pituitary prolactinoma, hereditary lattice corneal dystrophy, cutaneous lichen amyloidosis, corneal lactoferrin amyloidosis, corneal lactoferrin amyloidosis, pulmonary alveolar proteinosis, odontogenic tumor amylois, seminal vesical amyloid, cystric fibrosis, sickle cell disease and critical illness myopathy.

In some embodiments, the subject pharmaceutical compositions of the present invention will incorporate the substance or substances to be delivered in an amount sufficient to deliver to a patient a therapeutically effective amount of an incorporated therapeutic agent or other material as part of a prophylactic or therapeutic treatment. The desired concentration of the active agent will depend on absorption, inactivation, and excretion rates of the drug as well as the delivery rate of the compound. It is to be noted that dosage values may also vary with the severity of the condition to be alleviated. It is to be further understood that for any particular subject, specific dosage regimens should be adjusted over time according to the individual need and the professional judgment of the person administering or supervising the administration of the compositions. Typically, dosing will be determined using techniques known to one skilled in the art.

The dosage of the subject agent may be determined by reference to the plasma concentrations of the agent. For example, the maximum plasma concentration (Cmax) and the area under the plasma concentration-time curve from time 0 to infinity (AUC (0-4)) may be used. Dosages for the present invention include those that produce the above values for Cmax and AUC (0-4) and other dosages resulting in larger or smaller values for those parameters.

Actual dosage levels of the active ingredients in the pharmaceutical compositions of this invention may be varied so as to obtain an amount of the active ingredient which is effective to achieve the desired therapeutic response for a particular patient, composition, and mode of administration, without being toxic to the patient.

The selected dosage level will depend upon a variety of factors including the activity of the particular agent employed, the route of administration, the time of administration, the rate of excretion or metabolism of the particular compound being employed, the duration of the treatment, other drugs, compounds and/or materials used in combination with the particular compound employed, the age, sex, weight, condition,

general health and prior medical history of the patient being treated, and like factors well known in the medical arts.

A physician or veterinarian having ordinary skill in the art can readily determine and prescribe the effective amount of the pharmaceutical composition required. For example, the physician or veterinarian could prescribe and/or administer doses of the agents of the invention employed in the pharmaceutical composition at levels lower than that required in order to achieve the desired therapeutic effect and gradually increase the dosage until the desired effect is achieved.

In general, a suitable daily dose of an agent of the invention will be that amount of the agent which is the lowest dose effective to produce a therapeutic effect. Such an effective dose will generally depend upon the factors described above.

If desired, the effective daily dose of the agent may be administered as two, three, four, five, six or more sub-doses administered separately at appropriate intervals throughout the day, optionally, in unit dosage forms.

The precise time of administration and amount of any particular agent that will yield the most effective treatment in a given patient will depend upon the activity, pharmacokinetics, and bioavailability of a particular agent, physiological condition of the patient (including age, sex, disease type and stage, general physical condition, responsiveness to a given dosage and type of medication), route of administration, and the like. The guidelines presented herein may be used to optimize the treatment, *e.g.*, determining the optimum time and/or amount of administration, which will require no more than routine experimentation consisting of monitoring the subject and adjusting the dosage and/or timing.

While the subject is being treated, the health of the subject may be monitored by measuring one or more of the relevant indices at predetermined times during a 24-hour period. All aspects of the treatment, including supplements, amounts, times of administration and formulation, may be optimized according to the results of such monitoring. The patient may be periodically reevaluated to determine the extent of improvement by measuring the same parameters, the first such reevaluation typically occurring at the end of four weeks from the onset of therapy, and subsequent reevaluations occurring every four to eight weeks during therapy and then every three months thereafter. Therapy may continue for several months or even years, with a minimum of one month being a typical length of therapy for humans. Adjustments, for example, to the amount(s)

of agent administered and to the time of administration may be made based on these reevaluations.

Treatment may be initiated with smaller dosages which are less than the optimum dose of the compound. Thereafter, the dosage may be increased by small increments until the optimum therapeutic effect is attained. In addition, the combined use an agent that modulates a autotrophy-associated gene product and a second agent, *e.g.* another agent useful for the treatment of the autophagy-related disease, may reduce the required dosage for any individual agent because the onset and duration of effect of the different compounds and/or agents may be complimentary.

EXAMPLES

Materials and Methods

Cell lines and Culture Conditions

H4 human neuroblastoma cells were cultured under standard tissue culture conditions in DMEM media supplemented with 10% normal calf serum, penicillin/streptomycin, sodium pyruvate (Invitrogen) and, where appropriate, 0.4-1.2 mg/mL G418. LC3-GFP and FYVE-dsRed H4 cells were generated as described in Zhang *et al.,* PNAS, *102*, 15545-15550 (2007). To create a stable line expressing Lamp1, H4 cells were transfected with Lamp1-RFP plasmid using TransIT LT1 reagent (Mirus), followed by selection with 0.4 mg/mL G418. Bcl-2 expressing cell lines were created by infecting LC3-GFP and FYVE-dsRed H4 cells with pBabe-Bcl-2 retrovirus, followed by selection with 1 μg/mL puromycin.

For the cytokine assays, cells were seeded at 0.5×10^5 in full medium in either 24-well (western) or 96-well (LC3-GFP quantification) plates. After 24 hours, cells were washed in PBS and serum-free OptiMEM medium (Invitrogen) was added along with the indicated growth factors and/or cytokines for an additional 24 hours. Growth factors and cytokines used include human TNFα (Cell Sciences), human LIF (GeneScript Corporation), human FGF2 (ProSpec), human IGF1 (ProSpec), human SDF1 (Prospec) and human CLCF1 (R&D Systems). To induce starvation, cells were cultured for 24 hours in full media, washed in PBS and cultured for additional 4 hours in HBSS bedia (Invitrogen). Where indicated, 2.5 mM N-acetyl-L-cysteine (NAC, Sigma) was added at the time of media change.

For antioxidant assays, cells were treated 24 hours after siRNA transfection with N-acetyl-L-cysteine (NAC, Sigma) at a concentration of 2.5 mM and cultured for additional

48 hours before fixation and image analysis (see below for details). For western blot analysis, lysosomal protease inhibitor E64d (Sigma) was added at a concentration of 10 μg/mL for the last 8-12 hours before cell lysis.

siRNA transfection

For the primary screens, an arrayed library of 21,121 siRNA pools covering the majority of the human genome were used (Dharmacon si*ARRAY* siRNA library (Human Genome, G-005000-05), Thermo Fisher Scientific, Lafayette, CO). Each pool contained of 4 unique oligonucleotides targeting different sequences from the same gene. Each assay plate also included the following controls: non-targeting siRNA, mTOR siRNA, ATG5 siRNA and PLK1 siRNA (a transfection efficiency control). siRNAs were transiently transfected in triplicate into H4 cells stably expressing a LC3-GFP reporter at a final concentration of 40 nM using reverse transfection with the HiPerfect reagent (Qiagen). HiPerfect was diluted 1:20 in DMEM and 8 μl of the mixture was added to wells of 384 well plates. The plates were centrifuged at 1,000 rpm, after which 2 μl of 1 μM arrayed siRNA pools were added to each well. After 30 minutes of incubation, 500 cells in 40μl of media were added to the wells. Cells were incubated for 72 hours under standard culture conditions, counterstained with 0.5 μM Hoechst 33342 (Invitrogen) for 1 hour and fixed by addition of 30 μl of 8% paraformaldehyde. After 30 minutes, cells were washed 3 times with PBS prior to analysis.

For secondary screens, a siRNA library was used in which the 4 siRNAs of each siRNA pool were separated into individual wells. The cells were transfected and treated as in the primary screen, except that siRNAs were used at a final concentration of 30 nM (1.5μL/well of 1uM stock) and HiPerfect was diluted 1:30 in OptiMEM (Invitrogen). The secondary screen transfections were done in 2 rounds: in the first one a 1:1 mixture of H4 cells stably expressing LC3-GFP with FYVE-dsRed was transfected in triplicate; in the second round a 1:1 mixture of H4 cells expressing LC3-GFP with Lamp1-RFP was transfected in duplicate. All tertiary characterization screens were done in duplicate using a mixture of LC3-GFP and FYVE-dsRed cells. Each assay plate included 10-12 wells of non-targeting siRNA as well as mTOR, ATG5, PLK1 and, depending on screen, Vps34 or SOD1 siRNA controls.

For low-throughput confirmation of screen hits, cells were transfected in 12- or 6-well plates using reverse transfection with 2 μl or 6 μl of HiPerfect per mL of media, 40 nM or 10 nM final siRNA concentration and cells at 5×10^4 or 2×10^5 cells/mL for H4 and

MCF7 cells, respectively. For RT-PCR and FACS analysis, cells were harvested after 72 hours. For western and imaging analysis, cells were split 24 hours after transfection into 24-well plates at 2.5 x 10^4 or 1 x 10^5 cells/ml and harvested after additional 48 hours.

Imaging and image quantification

For high-throughput screens, cells were imaged on an automated CellWoRx microscope (Applied Precision) at 10x magnification using 2 wavelengths (350nm to detect Hoechst, 488nm to detect LC3-GFP) for the primary screens and 3 wavelengths (350nm, 488nm and 550nm to detect Lamp1-RFP or FYVE-dsRed) for the secondary screens. All images were quantified using VHSscan and VHSview image analysis software (Cellomics). Total cell number, total LC3-GFP intensity/cell as well as number, area and intensity of LC3-GFP positive autophagosomes/cell were scored. All dead and mitotic cells were excluded from analysis based on nuclear intensity. The final autophagy score for each well was obtained by multiplying the total autophagosome intensity/cell by the number of autophagosomes/cell and dividing by the average cell intensity. This formula was empirically determined to accurately measure LC3-GFP translocation from cytosol into autophagosomes as reflected by consistently significant z-scores and p-values when using siRNAs against mTOR and Atg5 controls. FYVE-dsRed and Lamp1-RFP scores were obtained in a manner similar to LC3-GFP scores, except that for Lamp1-RFP, which measures total accumulation of the reporter rather than its translocation, division by the average cell intensity was omitted.

For low-throughput follow-up analysis, cells were grown on glass cover slips. Following fixation in 4% paraformaldehyde and counterstaining with Hoechst, cover slips were mounted in 50% glycerol, 0.1% n-propyl gallate/PBS. Cells were imaged at 40x magnification on a Nikon Eclipse E800 microscope. Cell numbers, cell area and intensity, as well as autophagosome number and intensity, were quantified using Metamorph software. Autophagy was scored as number of autophagosomes per cell.

In-cell-western assays

For quantitative analysis of mTORC1 signaling and induction of endoplasmic reticulum stress, in-cell-western analysis of rpS6 phosphorylation and KDEL (GRP78/GRP94) expression, respectively, were performed. H4 cells were cultured in 384-well plates and fixed and counterstained as described for the LC3-GFP assay. Following imaging, the cells were permeabilized in PBS containing 0.2% Tx-100 and stained with Alexa-680 NHS-ester, a non-specific lysine reactive probe used to measure relative cell

number, at 20 ng/mL for 15 minutes. Subsequently, the cells were washed with PBS containing 0.2% Tx-100 and incubated for 30 minutes in blocking buffer (LiCOR Blocking Buffer diluted 1:1 with PBS + 0.2% Tx-100). Cells were then incubated overnight with a rabbit-anti-rpS6 phospho-235/236 (Cell Signaling Technologies), or mouse-anti-KDEL (Stressgen) antibody diluted 1:1000 in blocking buffer. Following primary antibody staining, the cells were washed in PBS + 0.2% Tx-100 and stained with an IRDye-800-conjugated secondary antibody (LiCOR) diluted 1:1000 in blocking buffer. The plates were scanned on the Aerius infrared imaging system (LiCOR). The intensities of both, the rpS6 phospho-235/236 or KDEL staining, and of NHS-ester staining were integrated, and the normalized phospho-S6 or KDEL score were calculated by dividing phospho-rpS6 or KDEL intensity by NHS-ester intensity.

Statistical analysis

All screen data was normalized by conversion to logarithmic scale (log10). For primary screens, z-scores were calculated based on plate median (controls excluded) and Median Absolute Deviation (MAD), with z-score = (cell score - median plate score) / (plate MAD X 1.4826). The screen hits were than selected based on the median z-score of the 3 replica-plates with cutoffs set at z-score > 1.7 or < -1.9, which gives a p value of 0.02. The same method was used for the rpS6 and KDEL secondary screens except the assays were performed in duplicate. For LC3-GFP, FYVE-dsRed and Lamp1-RFP secondary screens z-scores were calculated based on non-targeting siRNA control mean and standard deviation. For secondary confirmation of hits in the LC3-GFP assay it was required that at least 2 out of 4 individual siRNA oligonucleotides for each gene had median z-scores > 1.5 or < -1.5 based on 5 replica plates and were consistent with the primary screen z-score. This resulted in $p < 0.01$. In all other secondary assays z-scores > 1.5 and < -1.5 were also considered significant. The final z-scores for confirmed genes were calculated based on average z-scores of all wells for oligonucleotides considered positive in the secondary LC3-GFP assay.

The correlation analysis between LC3-GFP and other secondary assays was performed based on individual assay well quadrant analysis: for each well a score of +1 was assigned if z-scores for both features were > 1.5 or both were < -1.5; a score of -1 if one z-score was > 1.5 while the other was < -1.5; a score of 0 if either z-score failed to reach the cut-off. The individual well scores were than summed up for each gene for all oligonucleotides considered significant in the LC3-GFP secondary assay and divided by the

total number of wells assayed for these oligonucleotides. A correlation between features was considered to be positive if the final score was ≥ 0.5, negative if it was ≤ -0.5.

Relative viability was calculated by dividing number of cells in each well based on Hoechst imaging by the average cell number in the plate. The reported viability for each hit gene reflects average viability of all wells for oligonucleotides positive in the secondary LC3-GFP assay. The number of positive oligonucleotides with average viability below 50% is also reported. The relative viability for +NAC and Bcl-2 tertiary assays was calculated by dividing number of cells in each well by the average cell numbers in matching control plates without NAC or Bcl-2, respectively.

Unless otherwise indicated, all remaining p values were calculated from a 2-tailed student t-test with equal variance. All error bars are standard error.

Western analysis

For western blots, cells were lysed in Lammeli sample buffer, resolved on a 10-12% SDS-PAGE gel and transferred to PVDF membrane. The following antibodies were used: LC3 (Novus), p62 (Pharmigen), phospho-S6K (Thr389), phospho-Akt (Ser473), phospho-Stat3 (Tyr705), RelA, Sod1, phospho-PTEN (Ser380/Thr382/383) (all Cell Signaling), Bcl-2 (Santa Cruz), all at 1:1000, phospho-S6 (Ser235/236) (Cell Signaling) and phospho-ERK 1/2 (Sigma) at 1:2000, tubulin (Sigma) at 1:5000. Where indicated, blots were quantified using NIH ImageJ64 software.

Semi-quantitative RT-PCR

Total RNA was prepared using RNeasy mini kits (Qiagen) according to the manufacturer's instructions. For cDNA synthesis, 1.25 μg of RNA was used in the SuperScript First-Strand Synthesis System for RT-PCR (Invitrogen) with oligo dT primers. The following primers were used in the RT-PCR reactions: RelA AGCGCATCCAGACCAACAACAACC and CCGCCGCAGCTGCATGGAGACC, AMPKα2 CACCTCGCCTGGGCAGTCACACC and ATTGGGGGCATAAACACAGCATAA, Sod1 GGTGCTGGTTTGCGTCGTAGTCTC and ACCAGTGTGCGGCCAATGATG, β actin GACCTGACAGACTACCTCAT and AGACAGCACTGTGTTGGCTA. PCR product was resolved on 2% agarose gels and quantified using NIH ImageJ64 software.

Quantification of cellular Reactive Oxygen Species (ROS) levels

ROS levels were quantified 72 hours after siRNA transfection using Image-iT LIVE Green ROS Detection Kit for microscopy (Molecular Probes) according to the

manufacturer's instructions. Images were acquired on a Nikon Eclipse E800 microscope at 40x magnification and quantified using Metamorph software. Alternatively, ROS levels were quantified following 4 hour starvation in HBSS. Cells were stained with 10 μM dihydroethidium for 20 min at 37 °C, washed twice in PBS and analyzed by flow cytometry.

Bioinformatics analysis

For enrichment analyses, siRNA screen hit genes were classified into functional categories such as biological process, molecular function (PANTHER classification system), cellular component (Gene Ontology (GO) classification system), canonical pathways (MSigDB) and transcription factor binding sites (MSigDB and TRANSFAC v7.4). To assess the statistical enrichment or over-representation of these categories for the hit genes relative to their representation in the global set of genes examined in the siRNA screen, P-values were computed using the hypergeometric probability distribution, which was implemented in the R language.

For the protein interaction network, the network was constructed by iteratively connecting interacting proteins, with data extracted from genome-wide interactome screens , from databases: HPRD , MINT , REACTOME and curated literature entries. For yeast interaction data, yeast proteins were mapped to human orthologs (reciprocal Blastp analysis and Homologene). The network uses graph theoretic representations, which abstract components (gene products) as nodes and relationships (interactions) between components as edges, implemented in the Perl programming language.

Analysis of hit gene expression during aging

Gene expression during aging analysis was based on Affymetrix HG-U133_Plus_2 microarray data of young (≤40 years old) and old (≥70 years old) human brain samples. Array normalization, expression value calculation and clustering analysis were performed using the dChip software. Hierarchical clustering analysis was used to group genes or samples with similar expression pattern. Two genes or samples with the closest distance were first merged into a super-gene or super-sample and connected by branches with length representing their distance, and were deleted from future merging. Then the next pair of genes or samples (super-genes or super-samples) with the smallest distance was than chosen to be merged. The process was repeated until all the genes and samples were merged into one cluster.

Example 1. A high-throughput image-based siRNA screen for genes involved in the regulation of autophagy

Human neuroblastoma H4 cells stably expressing the LC3-GFP reporter were used to identify genes involved in the regulation of autophagy in mammals. Under normal growth conditions, LC3-GFP in these cells exhibits a diffused cytosolic localization. When autophagy is induced in these cells, LC3-GFP is recruited from the cytosol and can be visualized in a punctate pattern corresponding to autophagosomes. In order to validate the system, cells were transfected with siRNA against either the essential autophagy mediator ATG5 or against mTOR, a suppressor of starvation-induced autophagy. Following 72 hours of incubation under normal nutritional conditions, cells were transfected with ATG5 siRNA. This led to significant down-regulation of autophagy as assessed by a reduction in the number and intensity of LC3-GFP positive autophagosomes (Figure 1A), as well as a decrease in LC3II to LC3I ratio on a western blot (Figure 1B). Conversely, expression of siRNA against mTOR, the catalytic subunit of mTORC1, led to an increase in the number and intensity of LC3-GFP positive autophagosomes (Figure 1A) and an increase in LC3II to LC3I ratio (Figure 1B). Quantification of the LC3-GFP images in 384-well format acquired on a high-throughput automated fluorescent microscope revealed that the changes in the levels of autophagy following ATG5 or mTOR siRNA transfection were statistically significant as compared to non-targeting, control siRNA (Figure 2).

This system was used to screen a human genome siRNA library containing siRNA pools targeting 21,121 genes, with each pool containing 4 independent siRNA oligonucleotides for each gene. The primary screen was performed in triplicate and resulted in the identification of 574 genes (2.7% of the all genes tested) which knock-down led to a median decrease in LC3-GFP positive autophagosome formation by at least 1.9 standard deviations (SD) or increase by at least 1.7 SD from the plate median.

The candidate genes identified in the primary screen were confirmed using a deconvolved library, in which the 4 siRNAs from each pool were evaluated separately. Of the 547 candidate genes, 236 (41%) were confirmed with at least 2 independent siRNA oligonucleotides resulting in median increase or decrease in the levels of autophagy by at least 1.5 SD as compared to non-targeting siRNA control (Figure 3, $p<0.05$). Knock-down of a majority of these hits (219, 93% of all confirmed genes, Table 1) led to the induction of autophagy, indicating that these genes were autophagy-inhibiting genes, while knockdown

of the remaining 17 hits led to the inhibition of autophagy, indicating that these genes were autophagy-enhancing genes (Table 2).

Example 2. A secondary high-throughput characterization of the candidate genes

In order to elucidate the molecular pathways involved in regulation of autophagy by the newly identified genes, additional high-throughput assays were developed and performed to characterize the hits (Figure 4). In one of these assays, the function of mTORC1, an essential mediator of starvation-induced autophagy was investigated. To determine which of the candidate genes regulate autophagy by altering mTORC1 activity, an in-cell-western assay was used to evaluate the phosphorylation status of a downstream target of mTORC1 signaling, the ribosomal S6 protein (rpS6). To validate this system, H4 cells were transfected with mTOR siRNA. A significant decrease in the levels of rpS6 phosphorylation in mTOR siRNA transfected cells as compared to non-targeting siRNA was observed (Figure 5). Using the in-cell-western assay it was determined that only 14 (6%) out of the 219 confirmed genes which knockdown led to the induction of autophagy were strongly correlated with down-regulation of mTORC1 activity, while nine genes (4%) were identified in which knockdown led to up-regulation of both autophagy and of mTORC1 activity (Figure 6).

In a follow up tertiary screen of the 17 confirmed genes which knock down resulted in suppression of autophagy, 35% of these genes were found to be able to down-regulate autophagy in the presence of rapamycin, a potent inhibitor of mTORC1, which indicates that such genes function downstream of mTORC1 (Figure 7).

Accumulation of LC3-GFP may be due to, for example, increased initiation of autophagy or a block in degradation of autophagosomes. In order to evaluate the shape and size of the lysosomal compartment, H4 cells stably expressing lysosomal protein Lamp1-RFP were used. Knock-down of mTOR led to re-distribution as well as a significant increase in the levels of Lamp1-RFP (Figure 8), suggesting that in addition to up-regulating autophagy, inhibition of mTOR also causes an expansion of the lysosomal compartment. Using this system it was determined that transfection of siRNAs against 78 genes (30%) led to a significant (+/- 1.5 SD) change in the levels of Lamp1-RFP, which positively correlated with the changes in the levels of autophagy, suggesting that these genes regulate autophagy by altering the lysosomal function (Figure 9).

The impact of the knock-down of the individual hits on the activity of the type III PI3 kinase, an important mediator of autophagy in both yeast and mammalian cells was also

determined. In order to identify genes that induce or suppress autophagy by altering type III PI3 kinase activity, H4 cells stably expressing FYVE-dsRed reporter, which specifically binds to the product of the type III PI3 kinase, PtdIns3P, were used. Accumulation of PtdIns3P caused by elevated type III PI3 kinase activity results in a punctate vesicular localization of this reporter. Transfection of siRNA against Vps34, the catalytic component of the kinase, significantly decreased FYVE-dsRed vesicle recruitment (Figures 10A and B). Consistent with the effects of rapamycin, knock-down of mTORC1 components mTOR and Raptor strongly increased FYVE-dsRed vesicular signal (Figure 10C). Using this system, it was also demonstrated that knock-down of 110 (47%) out of the 236 confirmed genes led to a significant (+/- 1.5 SD) alteration in PtdIns3P levels, which positively correlated with the change in LC3-GFP positive autophagosome formation (Figure 11), suggesting that these genes act upstream of the type III PI3 kinase in the regulation of autophagy. Agents that increase the levels of both LC3-GFP and FYVE-dsRed vesicle recruitment are among those likely to induce autophagic degradation.

To further sub-divide the 219 genes which knock-down induced autophagy, the hits belonging to each of the subgroups identified in the secondary characterization assays were compared (Figure 12). A substantial overlap between the hits with increased vesicular localization of FYVE-dsRed and those that accumulated Lamp1-RFP was demonstrated. Agents that inhibit the activity of this subset of genes are among those likely will simultaneously regulate the type III PI3 kinase, autophagy and lysosomal activity.

Example 3. Cell death and ER stress are not major contributors to the induction of the autophagy induced during the siRNA screen.

It was investigated whether the induction of autophagy observed during the siRNA screen reflected a general response to cellular stress following knock-down of an essential gene, rather than a specific function of that gene in the regulation of autophagy. Expression of Bcl-2 significantly improved average cell viability following siRNA transfection (Figures 13-15). With the exception of Kif 11 and integrin α5, knock-down of the 91 genes able to induce autophagy in cells expressing Bcl-2 failed to generate substantial loss of viability in these cells. This suggests that up regulation of autophagy following inhibition of these genes was not dependent on the induction of a cell death response. Of the genes which knock-down was unable to up regulate autophagy in cells expressing Bcl-2, 81 had high (over 85%) viability in wild type cells. Therefore, inhibition of the activity of 170 of

the 129 identified autophagy-inhibitor genes results in the induction of autophagy through a cell-death independent mechanism.

In addition to cell death, autophagy is often induced in response to various forms of cellular stress, including ER stress. In order to determine whether stimulation of autophagy in response to knock-down of our hit genes could be due to ER stress, in-cell-western assays assessing the expression levels of GRP78 and GRP94, specific markers of ER stress, were performed. Treatment with tunicamycin, a potent inducer of ER stress, led to a dose-dependent up-regulation of GRP78 and GRP94 (Figure 16), as well as to increase in autophagy. In 97% of the genes tested (182 out of 188 genes tested, Figure 17) there was no significant up-regulation of ER stress following knock-down of genes leading to the stimulation of autophagy. Therefore, ER stress is not a major contributor to the induction of the autophagy observed in the screen. The data therefore suggest that induction of autophagy following knock-down of the majority of the hits is due to the induction of a specific signaling event, rather than a part of a general cellular stress response induced by cell death or a result of a widespread ER stress.

Example 4. The effects of Bcl-2 on induction of autophagy

Beclin 1, the regulatory autophagy specific component of the type III PI3 kinase, was originally identified as a binding partner of the anti-apoptotic protein Bcl-2. Recently, in addition to its prominent function in regulation of apoptotic cell death, Bcl-2 has been suggested to negatively regulate autophagy through its interaction with beclin 1 and consequent inhibition of the type III PI3 kinase activity. In order to assess the function of Bcl-2, a tertiary characterization screen was performed to compare the induction of autophagy and the type III PI3 kinase activity in wild-type H4 cells and cells stably expressing Bcl-2 (Figure 18). As a control, it was demonstrated that knock-down of mTOR was able to significantly induce both LC3-GFP and FYVE-dsRed vesicle recruitment in the Bcl-2 expressing cells (Figure 19A and B). Consistent with the proposed negative regulation of type III PI3 kinase by Bcl-2, a significant decrease in average FYVE-dsRed induction following knock down of the hit genes in H4 cells expressing Bcl-2 as compared to wild type controls occurred (Figure 19C). Knock-down of 91 (42%) out of the 215 tested genes was able to induce translocation of LC3-GFP to autophagosomes in the presence of Bcl-2 (Figures 14 and 20). In 17 (19%) out of these 91 genes induction of autophagy was correlated with the increase in type III PI3 kinase activity as assessed by the vesicle recruitment of FYVE-dsRed, indicating that these genes are involved in additional

mechanisms that regulate production of PtdIns3P downstream of Bcl-2. On the other hand, knock-down of the remaining 74 genes was able to induce autophagy without additional activation of the type III PI3 kinase. Knock-down of 31 of these genes led to Lamp1-RFP accumulation in wild type H4 cells, indicating that, in these cases, a block in lysosomal degradation may contribute to the increase in autophagy in Bcl-2 expressing cells. No changes in the lysosomal function were observed for the remaining 43 genes. Thus the inhibitory effect of Bcl-2 on type III PI3 kinase is not always incompatible with the induction of autophagy, the activation of which can be accomplished without increase in PtdIns3P levels. Finally, knock-down of the remaining 124 (58%) genes was unable to induce accumulation of vesicular LC3-GFP in cells over expressing Bcl-2 (Figure 15).

Example 5. Bioinformatics network analysis of autophagy-related genes

In order to further elucidate the biological networks involved in regulation of autophagy, interactions between the hit genes were explored by mapping their direct physical interactions based on both mammalian and yeast data. Among the hits were included multiple members of several known protein complexes (Figure 21A), including 2 subunits of NF-κB (NFκB1 and RelA), 3 ribonucleoproteins involved in pre-mRNA processing (HNRPK, HNRPM and HNRPNU), 3 coatamer components (CopB2, CopE and Arcn1) and 2 AMPK subunits (AMPKα2 and AMPKγ3). Additionally, a large network of interacting transcription factors and chromatin modifying enzymes centered on p300 HAT and NFκB were identified (Figure 21B). The latter indicates that transcriptional regulation may play a critical role in the regulation of autophagy.

Interolog analysis (yeast-human orthologous mapping of protein-protein interactions) between the core autophagy components and the genes identified in the screen revealed that at least two of the hits, Xpo1 and OGDH, may physically interact with core autophagic machinery (Figure 22). Xpo1 is the mammalian homolog of yeast CRM1 and an essential component of nuclear export machinery. Its interaction with Beclin1 and Atg12 likely reflects its function in the nuclear export of these proteins. On the other hand, OGDH, a metabolic enzyme localized to the mitochondrial matrix, has been reported to have cytoprotective activity independent of the enzymatic activity of the associated complex , making it a candidate for the regulation of autophagy induced by mitochondrial damage.

In order to investigate the connection between autophagy, axon guidance and actin dynamics, a protein-protein interaction network anchored by the hit genes belonging to

these canonical pathways was generated (Figures 23 and 24). This analysis revealed two related networks encompassing, respectively, 27 and 61 of the hit genes.

These analyses indicate that autophagy can be modulated through the use of agents that modulate the activity of specific pathways and complexes identified herein as being associated with the regulation of autophagy.

Example 6. The use of cytokines in the modulation of autophagy

Molecular function analysis of the 236 confirmed hits using Gene Ontology (GO) revealed a highly significant enrichment in genes encoding kinases ($p=0.0006$), proteins with receptor activity ($p=7.7X10^{-5}$) and extracellular matrix proteins ($p=0.03$) (Figures 25 and 26). The latter categories indicate that the extracellular environment, including the presence of growth factors, hormones and cytokines, plays a role in the regulation of autophagy under normal nutritional conditions. The results of GO biological process analysis also demonstrated significant enrichment in signaling molecules ($p=2.8X10^{-7}$) (Figure 27A). In agreement with the proposed function of extracellular factors in regulation of autophagy, further subdivision of these signaling molecules revealed that the largest sub-group (49%) was involved in cell surface receptor signal transduction (Figure 27B).

Cells were treated with several of the cytokines and growth factors identified as hits in our screen. Based on the results of the characterization assays, knock-down of IGF1, FGF2, LIF, CLCF1 and the chemokine SDF1 (CXCL12) resulted in mTORC1 independent increase in initiation of autophagy. In agreement, treatment of H4 LC3-GFP cells grown in a serum-free medium with any of these cytokines led to a significant down-regulation of autophagy as measured by LC3-GFP translocation (Figures 28 and 29). This data was confirmed in multiple cell lines (H4, HEK293, HeLa and MCF7) by western blot (Figure 30). In agreement with the proposed function of cytokines in the regulation of autophagy, cells cultured in their absence displayed high basal levels of autophagy as assessed by accumulation of LC3II, which was partially suppressed by the addition of even single cytokines identified in the screen. Thus, the identified cytokines and growth factors are both necessary and sufficient for the regulation of autophagy.

In the screen described above, knock-down of the TNF gene led to an increase in the formation of LC3-GFP positive autophagosomes, indicating a negative role for this cytokine in the regulation of basal autophagy. In order to further investigate the role of TNFα in autophagy, H4 LC3-GFP cells grown in a defined medium were treated with increasing doses of TNFα. Low doses of TNFα led to down-regulation of autophagy,

while higher doses led to up-regulation of autophagy (Figure 31A). This was confirmed by western blot showing a significant accumulation of p62 following treatment with low levels of TNFα (Figure 31B). Since physiological levels of TNFα are very low, this suggests that this cytokine normally functions as a negative regulator of autophagy. On the other hand, increased concentrations of TNFα under pathological conditions lead to up-regulation of autophagy.

Example 7. The function of NF-κB in the regulation of autophagy

The canonical pathway analysis described above demonstrated enrichment of autophagy hits in the NF-κB ($p=8.7X10^{-6}$) and RelA ($p=1.2X10^{-6}$) pathways. As a validation of the screen, H4 LC3-GFP cells transfected with siRNAs against RelA were individually imaged. The levels of autophagy by quantifying translocation of LC3-GFP by fluorescence microscopy were assessed using an alternative low-throughput method. In agreement with our screen results treatment with all 4 oligonucleotides against RelA lead to strong down-regulation of number and intensity of autophagosomes (Figures 32 and 33). Confirming that the observed differences in the levels of autophagy were due to the knock-down of the target genes, a strong down-regulation of RelA at both mRNA (Figure 34A) and protein level (Figure 34B) was observed. In order to confirm that the findings regarding the function of NF-κB as a positive mediator of autophagy are not restricted to H4 cells, levels of autophagy in wild-type and double knock-out $RelA^{-/-}$; $NF\text{-}\kappa B^{-/-}$ (DKO) MEFs and in human breast cancer MCF7 cells transfected with either siRNA were compared against RelA or control non-targeting siRNA. Absence or down-regulation of RelA/NFκB expression led to suppression of autophagy as assessed by decrease in LC3 II and accumulation of p62 (Figure 35). These data confirm NFκB as a positive regulator of basal autophagy.

In contrast with the results described herein, NF-κB activation has been previously reported to *negatively* regulate autophagy associated with cell death induced in response to noxious stimuli such as nutrient starvation or death receptor ligation (Djavaheri-Mergy *et al.,* J. Biol. Chem *281*, 30373-30382 (2006)). Since reactive oxygen species (ROS) have been proposed to participate in the mediation of starvation-induced autophagy , it was hypothesized that, under conditions of nutrient deprivation, down regulation of autophagy may be the result of the attenuation of ROS production by NF-κB. Wild type and dKO MEFs and H4 LC3-GFP cells transfected with either non-targeting siRNA or siRNA

against RelA were subjected to nutrient starvation. Starvation of RelA/NF-κB deficient cells led to higher ROS accumulation than observed in wild type controls (Figure 36). The elevated induction of autophagy observed in response to starvation in RelA deficient H4 cells was attenuated in the presence of the antioxidant N-acetyl-L-cysteine (NAC) (Figure 37).

These data indicate that, while NF-κB plays a positive function in regulation of basal autophagy, its ability to attenuate ROS production can indirectly lead to decrease in the levels of autophagy observed under nutrient starvation condition. Thus, contrary to previous reports, NF-κB acts as an autophagy-enhancer under the non-starvation conditions most prevalent in multicellular organisms. Therefore, agents that inhibit the activity of the components of NF-κB (NFKB1 and RELA) act as inhibitors of autophagy and are useful for the treatment of cancer and/or pancreatitis.

Example 8. The function of reactive oxygen species (ROS) in regulation of autophagy

Genes that induce autophagy when knocked-down included SOD1 and GPx2, the major components of the ROS detoxification pathway, as well as several mitochondrial proteins, many of them involved in oxidative respiration and electron transport (Figure 38). Inhibition of the activity of any of these genes would be expected to lead to the up-regulation of the levels of ROS by either increasing their production or blocking their degradation. Furthermore, many additional screen hits have been reported to be involved in the regulation or to be regulated by ROS (Figure 39). In order to evaluate a possible role of ROS as a general mediator of autophagy, it was first confirmed that transfection of SOD1 siRNA led to both the induction of autophagy as well as elevated levels of ROS (Figure 40). Confirming a causal role of ROS, treatment with the antioxidant NAC significantly attenuated induction of autophagy caused by knock-down of Sod1 (Figure 41). Therefore, interference with normal cellular ROS homeostasis is sufficient for the induction of autophagy.

In order to determine if ROS may have a general signaling role during induction of autophagy, a tertiary characterization screen to compare levels of autophagy and type III PI3 kinase activity induced by knock-down of our hit genes in the presence and absence of NAC was performed. Knocking-down a group of the confirmed genes (117, or 54% of all genes tested) led to vesicular LC3-GFP accumulation in the absence but not the presence of the antioxidant, indicating that ROS were required for the induction of autophagy (Figure 42). Knock-down of these genes also largely failed to increase the accumulation of vesicle-

associated FYVE-dsRed in the presence of NAC (Figures 42 and 43). This indicates that ROS serve a general function in activation of the type III PI3 kinase, implicating them as important signaling molecules in the early steps of the autophagic pathway.

On the other hand, inhibition of the activity of the remaining 98 (46%) genes was able to induce accumulation of LC3-GFP in the presence of NAC, indicating that, in these cases, autophagy can be induced independently of ROS (Figure 44). Knock-down of these genes was also able to induce comparable average levels of vesicular FYVE-dsRed in the presence and absence of NAC (Figure 43). Thus, inhibition of the activity of this group of genes led to induction of the type III PI3 kinase through a mechanism independent of ROS.

Example 9. Growth-promoting pathways negatively regulate autophagy.

Bioinformatics analysis of the autophagy screen hits indicated significant enrichment for several canonical pathways known to mediate signaling from cell surface receptors (Figure 45). These pathways included the MAPK ($p=0.039$), Stat3 ($p=0.008$) and CXCR4 ($p=1.1X10^{-5}$) pathways regulated by the cytokines identified in the screen. FGF2 is known to activate the MAPK pathway and an increased level of phospho-ERK1/2 and phospho-RSK were observed following treatment with FGF2 (Figure 46). Confirming the essential function of the MAPK pathway, pre-treatment with UO126, an inhibitor of MEK, attenuated inhibition of autophagy following addition of FGF2 (Figure 46). Additionally, analysis of the promoter regions of all the hit genes revealed significant enrichment in consensus sites for several transcription factors (Figure 47), including 3 enriched sites for RSRFC4, a member of the serum response factor (SRF) family and a downstream target of MAPK signalling, suggesting additional involvement of transcriptional regulation by the MAPK pathway in control of autophagy under normal growth conditions.

Another hit gene pulled out of the screen as a negative regulator of autophagy was the transcription factor Stat3, a mediator of LIF and CLCF1 signaling. Indeed, treatment with either LIF or CLCF1 increased activating phosphorylation of Stat3 (Figures 48 and 49). Consistent with the essential function of Stat3, its siRNA mediated knock-down attenuated down-regulation of autophagy in response to LIF (Figure 49). Therefore, LIF and CLCF1 regulate autophagy through the Stat3 pathway.

In addition to activating mTORC1, Akt directly phosphorylates and inhibits Foxo3a, a transcription factor that positively regulates autophagy during muscle degeneration. Indeed, phosphorylation of both Akt and Foxo3a was increased following IGF-1 treatment in both the absence and presence of rapamycin (Figure 50). Inhibition of Akt by treatment

with Akt inhibitor VIII attenuated phosphorylation of both Foxo3a and the mTORC1 target S6 kinase, as well as prevented inhibition of autophagy by IGF1 (Figure 50). Therefore, under normal nutrient conditions IGF-1 regulates autophagy in a type I PI3 kinase/Akt dependent manner, likely through both the mTORC1 and Foxo3a pathways.

Example 10. The down regulation of autophagy during human aging

In order to specifically address the potential function of the autophagy-related genes in neurodegeneration associated with aging, the mRNA expression of the autophagy hit genes were analyzed in a set of young versus old human brain samples. Differential expression of a large subset of genes (Figures 51 and 52) was observed, including a groups of 32 genes significantly ($p<0.05$) up-regulated and 46 genes significantly down-regulated with age (Figure 53-55). Interestingly, gene ontology (GO) biological process analysis revealed that the age up regulated group was highly enriched in genes involved in mediation and regulation of the MAPK pathway ($p=1.6X10^{-4}$), the increased activity of which is predicted by our analysis to lead to the suppression of autophagy. Conversely, expression of the key autophagy genes, Atg5 and Atg7, was down regulated during aging (Figure 55). These data suggest that differential gene expression leads to the down regulation of autophagy in the brain during aging, which would contribute to development of chronic neurodegenerative diseases. Consistent with this hypothesis, further analysis in a more extensive set of samples, including those from middle-aged individuals, revealed that Atg5 and Atg7 were among a group of genes necessary for the mediation of autophagy in mammalian cells whose expression was gradually down-regulated in an age-dependent manner starting in the early sixties (Figure 56), which is often the earliest age of onset for the sporadic neurodegenerative diseases such as Alzheimer's Disease (AD). Therefore, age-dependent regulation of genes identified in our screen likely contributes to down-regulation of autophagy during normal human aging, and thus useful as therapeutic targets to prevent and treat age-related neurodegenerative diseases.

Example 11. Differential expression of autophagy regulators in Alzheimer's Disease brain samples

Accumulation of both ROS and autophagic vesicles (AV) are early features in AD. To determine if we can detect changes in the expression of genes involved in regulation of autophagy in this disease, the expression of the autophagy screen hit genes from six brain regions of 34 cases with AD and 14 age-matched normal controls were analyzed. An

overall significant under-expression of the hit genes in AD patient samples compared to controls specifically in the hippocampus and entorhinal cortex, the brain regions most affected by the disease, were observed (Figure 57A). Consistent trends were observed in other brain regions affected by AD (superior frontal gyrus, posterior cingulate, and medial temporal gyrus). Notably, in the visual cortex, a brain region relatively resistant to AD pathology, these changes were absent. Further sub-division of the hit genes revealed that in the entorhinal cortex negative regulators of autophagy flux were specifically negatively enriched (Figure 57B). A similar trend was also observed in other brain areas affected by AD. Conversely, positive regulators of autophagy were positively enriched in the entorhinal cortex (Figure 57C). Such differential expression patterns of autophagy regulators suggest up-regulation of autophagy in AD brains.

Example 12. ROS mediate autophagy in response to amyloid β

Amyloid β (Aβ) is the main pathogenic factor in AD. Whether induction of autophagy by Aβ was be mediated by ROS was examined. Following treatment of H4 cells with Aβ, increased levels of autophagy were observed (Figure 58). In order to determine if this was due to an increase in the initiation of autophagy or to a block in lysosomal degradation, the accumulation of LC3-II following Aβ treatment in the absence and presence of lysosomal protease inhibitor E64d was observed (Figure 58). Up to 8 hours after treatment, the accumulation of LC3 -II could be observed only in the presence of E64d. At 48 hrs after the addition of Aβ, the increased levels of LC3 -II were observed even without E64d, but were further increased in the presence of E64d, Additionally, increased conjugation of Atg12-Atg5 starting 4 hours after Aβ treatment was observed. Together these data indicate increased initiation of autophagy in response to Aβ.

The involvement of type III PI3 kinase in the induction of autophagy by Aβ was investigated. Accumulation of PtdIns3P was observed, which was suppressed in the presence of 3MA (Figure 59), confirming the involvement of the type III PI3 kinase. In agreement with a causal role of ROS, accumulation of PtdIns3P was suppressed in the presence of NAC (Figure 60). Finally, treatment with 3MA (Figure 61) or knock down of Vps34 (Figure 62) was able to attenuate induction of autophagy in response to Aβ.

EQUIVALENTS

The present invention provides, methods for the modulation of autophagy and the treatment of autophagy related diseases. While specific embodiments of the subject invention have been discussed, the above specification is illustrative and not restrictive.

Many variations of the invention will become apparent to those skilled in the art upon review of this specification. The appended claims are not intended to claim all such embodiments and variations, and the full scope of the invention should be determined by reference to the claims, along with their full scope of equivalents, and the specification, along with such variations.

All publications and patents mentioned herein are hereby incorporated by reference in their entirety as if each individual publication or patent was specifically and individually indicated to be incorporated by reference. In case of conflict, the present application, including any definitions herein, will control.

CLAIMS

What is claimed is

1. A method of inhibiting autophagy in a cell comprising contacting said cell with an agent that inhibits the activity of a product of a gene selected from the group consisting of the genes listed in Table 2.

2. The method of claim 1, wherein the gene is selected from the group consisting of the genes listed in Table 4.

3. The method of claim 1, wherein said gene is selected from the group consisting of the genes listed in Table 6.

4. The method of claim 1, wherein said agent is a siRNA, shRNA or antisense RNA molecule.

5. The method of claim 3, wherein said gene is TPR or GPR18.

6. The method of claim 5, wherein said agent is an antibody specific for the product of said gene.

7. The method of claim 1, wherein said gene is RelA or NFκB.

8. The method of claim 7, wherein said gene is RelA.

9. A method of inducing autophagy in a cell comprising contacting said cell with an agent that enhances the activity of a product of a gene selected from the group consisting of the genes listed in Table 2 (hits that decrease autophagy).

10. The method of claim 9, wherein said gene is selected from the group consisting of the genes listed in Table 4.

11. The method of claim 9, wherein said gene is selected from the group consisting of the genes listed in Table 6.

12. The method of claim 11, wherein said gene is TPR or GPR18.

13. The method of claim 12, wherein said agent is an antibody specific for the product of said gene.

14. The method of claim 9, wherein said gene is RelA or NFκB.

15. The method of claim 14, wherein said gene is RelA.

16. A method of treating a neurodegenerative disease in a subject comprising administering to said subject an agent that enhances the activity of a product of a gene selected from the group consisting of the genes listed in Table 2.

17. The method of claim 16, wherein said gene is selected from the group consisting of the genes listed in Table 4.

18. The method of claim 16, wherein said gene is selected from the group consisting of the genes listed in Table 6.

19. The method of claim 18, wherein said gene is TPR or GPR18.

20. The method of claim 19, wherein said agent is an antibody specific for the product of said gene.

21. The method of claim 16, wherein said gene is RelA or NFκB.

22. The method of claim 21, wherein said gene is RelA.

23. The method of claim 16, wherein said neurodegenerative disease is selected from the group consisting of Adrenal Leukodystrophy, alcoholism, Alexander's disease, Alper's disease, Alzheimer's disease, Amyotrophic lateral sclerosis, ataxia telangiectasia, Batten disease, bovine spongiform encephalopathy, Canavan disease, cerebral palsy, cockayne syndrome, corticobasal degeneration, Creutzfeldt-Jakob disease, familial fatal insomnia, frontotemporal lobar degeneration, Huntington's disease, HIV-associated dementia, Kennedy's disease, Krabbe's disease, Lewy body dementia, neuroborreliosis, Machado-Joseph disease, multiple system atrophy, multiple sclerosis, narcolepsy, Niemann Pick disease, Parkinson's disease, Pelizaeus-Merzbacher disease, Pick's disease, primary lateral sclerosis, prion diseases, progressive supranuclear palsy, Refsum's disease, Sandhoff disease, Schilder's disease, subacute combined degeneration of spinal cord secondary to pernicious anaemia, Spielmeyer-Vogt-Sjogren-Batten disease, spinocerebellar ataxia, spinal muscular atrophy, Steele-Richardson-Olszewski disease, Tabes dorsalis and toxic encephalopathy.

24. The method of claim 16, wherein said neurodegenerative disease is a proteinopathy.

25. The method of claim 24, wherein said proteinopathy is selected from the group consisting of Alzheimer’s disease, Parkinson’s disease, Lewy Body Dementia, ALS, Huntington’s disease, spinocerebellar ataxias and spinobulbar musclular atrophy.

26. A method of treating a disease in a subject comprising administering to said subject an agent that inhibits the activity of a product of a gene selected from the group consisting of the genes listed in Table 2 (hits that decrease autophagy), wherein said disease is cancer or pancreatitis.

27. The method of claim 26, wherein the gene is selected from the group consisting of the genes listed in Table 4.

28. The method of claim 26, wherein said gene is selected from the group consisting of the genes listed in Table 6.

29. The method of claim 26, wherein said agent is a siRNA, shRNA or antisense RNA molecule.

30. The method of claim 28, wherein said gene is TPR or GPR18.

31. The method of claim 30, wherein said agent is an antibody specific for the product of said gene.

32. The method of claim 26, wherein said gene is RelA or NFκB.

33. The method of claim 32, wherein said gene is RelA.

34. The method of claim 26, wherein said disease is cancer.

35. The method of claim 34, further comprising the administration of a chemotherapeutic agent.

36. The method of claim 35, wherein the chemotherapeutic agent is selected from the group consisting of: altretamine, asparaginase, BCG, bleomycin sulfate, busulfan, camptothecin, carboplatin, carmusine, chlorambucil, cisplatin, claladribine, 2-chlorodeoxyadenosine, cyclophosphamide, cytarabine, dacarbazine imidazole carboxamide, dactinomycin, daunorubicin - dunomycin, dexamethosone, doxurubicin, etoposide, floxuridine, fluorouracil, fluoxymesterone, flutamide, fludarabine, goserelin, hydroxyurea, idarubicin HCL, ifosfamide, interferon α, interferon α 2a, interferon α 2b, interfereon α n3, irinotecan, leucovorin calcium, leuprolide, levamisole, lomustine, megestrol, melphalan, L-sarcosylin, melphalan hydrochloride, MESNA, mechlorethamine, methotrexate, mitomycin, mitoxantrone, mercaptopurine, paclitaxel, plicamycin, prednisone, procarbazine, streptozocin, tamoxifen, 6-thioguanine, thiotepa, topotecan, vinblastine, vincristine and vinorelbine tartrate.

37. The method of claim 34, further comprising the administration of radiation therapy.

38. The method of claim 26, wherein said disease is pancreatitis.

39. A method of treating a proteinopathy in a subject comprising administering to said subject an agent that enhances the activity of a product of a gene selected from the group consisting of the genes listed in Table 2.

40. The method of claim 39, wherein said gene is selected from the group consisting of the genes listed in Table 4.

41. The method of claim 39, wherein said gene is selected from the group consisting of the genes listed in Table 6.

42. The method of claim 41, wherein said gene is TPR or GPR18.

43. The method of claim 42, wherein said agent is an antibody specific for the product of said gene.

44. The method of claim 39, wherein said gene is RelA or NFκB.

45. The method of claim 44, wherein said gene is RelA.

46. The method of claim 39, wherein said proteinopathy is selected from the group consisting of α1-antitrypsin deficiency, sporadic inclusion body myositis, limb girdle muscular dystrophy type 2B and Miyoshi myopathy Alzheimer's disease, Parkinson's disease, Lewy Body Dementia, ALS, Huntington's disease, spinocerebellar ataxias and spinobulbar musclular atrophy..

47. A method of determining whether an agent is an autophagy inhibitor, the method comprising the step of contacting a cell with the agent, wherein the cell expresses a heterologous autophagy-enhancing gene, wherein said autophagy enhancing gene is selected from the group consisting of the genes listed in Table 2, whereby a reduction in autophagy in the cell indicates that the agent is an autophagy inhibitor.

48. The method of claim 47, wherein the agent is a small molecule.

49. The method of claim 47, wherein the agent is an antibody.

50. The method of claim 47, wherein the agent is an inhibitory RNA molecule.

DEMANDE OU BREVET VOLUMINEUX

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CECI EST LE TOME 1 DE 2
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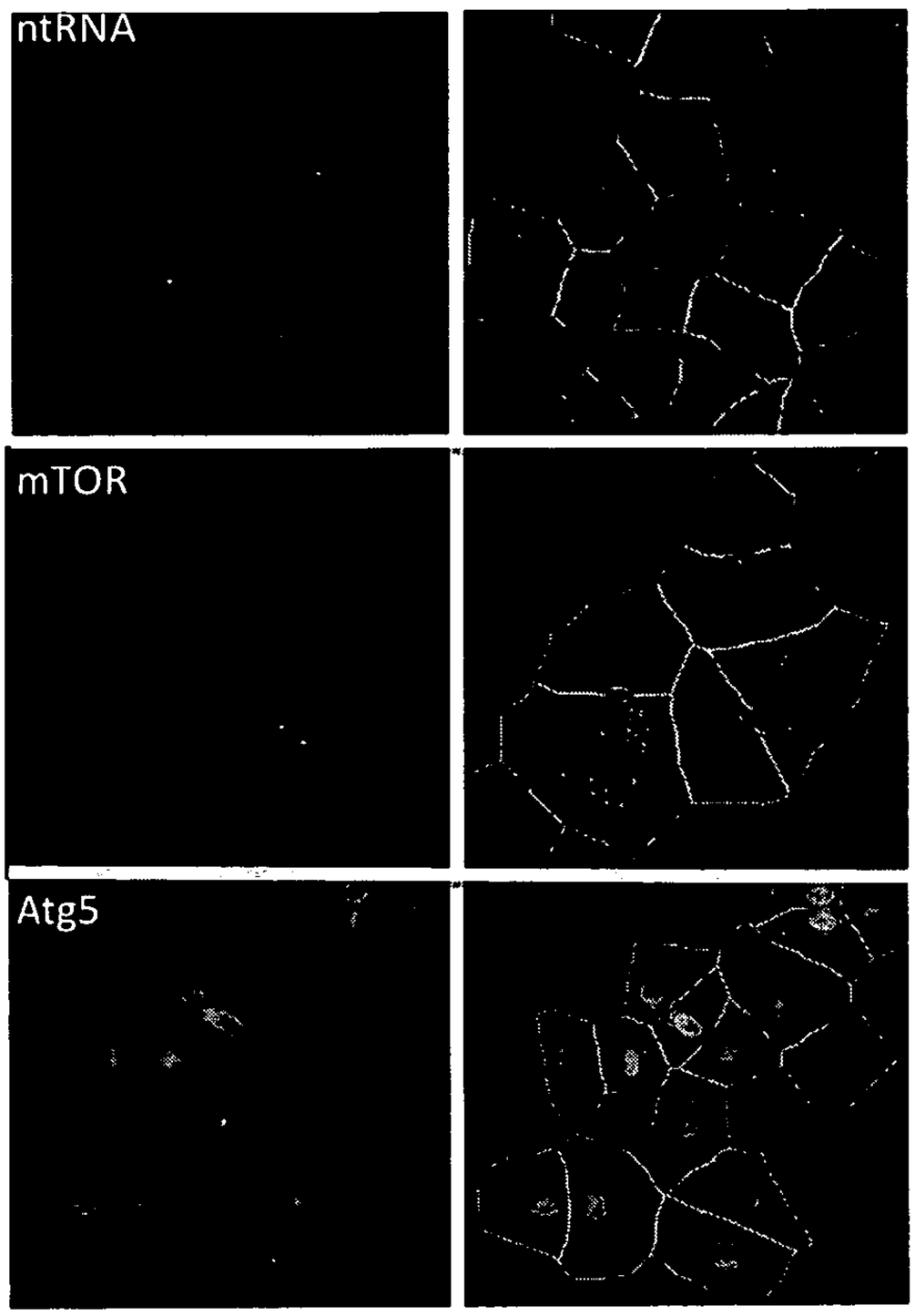
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Figure 1.

A.



B.

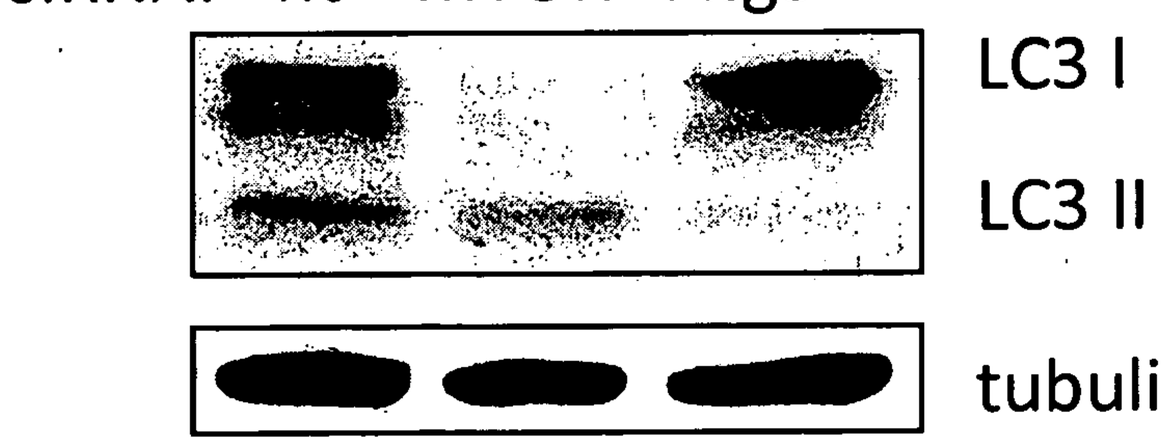
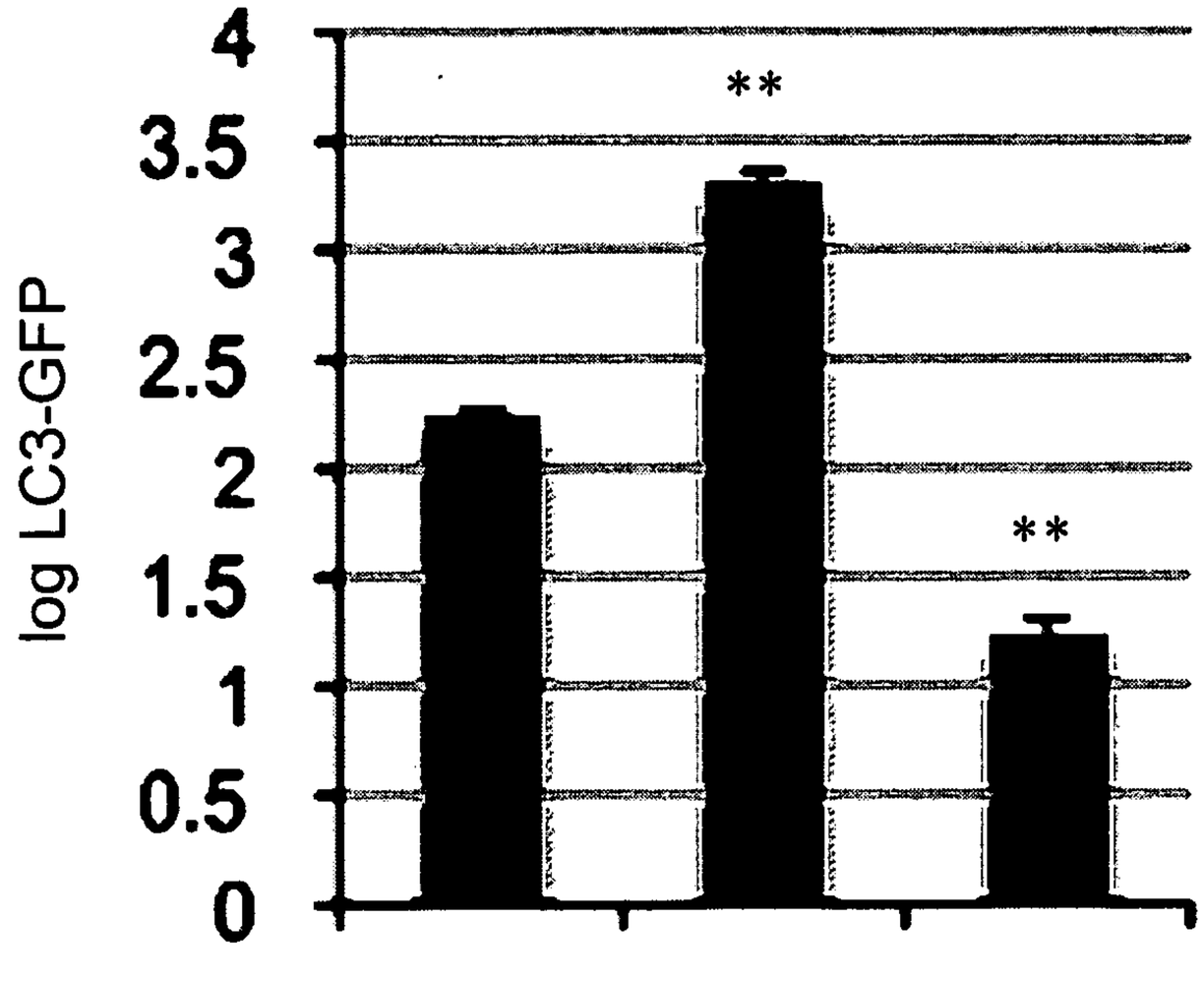


Figure 2.



	ntRNA	mTOR	Atg5
p:		$2.1X10^{-21}$	$5.2X10^{-19}$
z-score:		6.7	-6.4

Figure 3

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1
CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
XPO1	7514	NM_003400	exportin 1 (CRM1 homolog, yeast)
KIAA0133	9816	XM_375851	KIAA0133
ADMR	11318	NM_007264	adrenomedullin receptor
OGDH	4967	NM_002541	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
DDX24	57062	NM_020414	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
NUPR1	26471	NM_012385	nuclear protein 1
FXYD2	486	NM_001680	FXYD domain containing ion transport regulator 2
TRHR	7201	NM_003301	thyrotropin-releasing hormone receptor
SUV39H1	6839	NM_003173	suppressor of variegation 3-9 homolog 1 (Drosophila)
FCER1A	2205	NM_002001	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide
PTPRU	10076	NM_005704	protein tyrosine phosphatase, receptor type, U
GPX2	2877	NM_002083	glutathione peroxidase 2 (gastrointestinal)
PRKCA	5578	NM_002737	protein kinase C, alpha
EP300	2033	NM_001429	E1A binding protein p300
LOC388959	388959	XM_373989	hypothetical LOC388959
NTN2L	4917	NM_006181	netrin 2-like (chicken)
DOCK8	81704	NM_203447	dedicator of cytokinesis 8
MAP3K7IP1	10454	NM_006116	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
PLAGL2	5326	NM_002657	pleiomorphic adenoma gene-like 2
NUDT1	4521	NM_002452	nudix (nucleoside diphosphate linked moiety X)-type motif 1
RELN	5649	NM_005045	reelin
PNKD	25953	NM_015488	paroxysmal nonkinesiogenic dyskinesia
RIPK1	8737	NM_003804	receptor (TNFRSF)-interacting serine-threonine kinase 1
GNG5	2787	NM_005274	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	NM_001277	choline kinase alpha
C5AR1	728	NM_001736	complement component 5a receptor 1
SCOTIN	51246	NM_016479	scotin

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
PIGY	84992	NM_032906	phosphatidylinositol glycan anchor biosynthesis, class Y
NAGK	55577	NM_017567	N-acetylglucosamine kinase
RAGE	5891	NM_014226	renal tumor antigen
USP24	23358	XM_165973	ubiquitin specific peptidase 24
AURKA	6790	NM_003600	aurora kinase A
PLDN	26258	NM_012388	pallidin homolog (mouse)
TLR3	7098	NM_003265	toll-like receptor 3
PPARD	5467	NM_006238	peroxisome proliferator-activated receptor delta
HRC	3270	NM_002152	histidine rich calcium binding protein
NNMT	4837	NM_006169	nicotinamide N-methyltransferase
COPB2	9276	NM_004766	coatomer protein complex, subunit beta 2 (beta prime)
CDK5RAP3	80279	NM_025197	CDK5 regulatory subunit associated protein 3
NLK	51701	NM_016231	nemo-like kinase
PFKL	5211	NM_002626	phosphofructokinase, liver
RNPEPL1	57140	NM_018226	arginyl aminopeptidase (aminopeptidase B)-like 1
EPHA6	203806	XM_114973	EPH receptor A6
CDCA8	55143	NM_018101	cell division cycle associated 8
CKAP5	9793	NM_014756	cytoskeleton associated protein 5
ZBTB16	7704	NM_006006	zinc finger and BTB domain containing 16
GABBR2	9568	NM_005458	gamma-aminobutyric acid (GABA) B receptor, 2
PTMA	5757	NM_002823	prothymosin, alpha (gene sequence 28)
PTCRA	171558	NM_138296	pre T-cell antigen receptor alpha
RORC	6097	NM_005060	RAR-related orphan receptor C
GNAI1	2770	NM_002069	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
UTS2R	2837	NM_018949	urotensin 2 receptor
MATN3	4148	NM_002381	matrilin 3
NPTX1	4884	NM_002522	neuronal pentraxin I
SP140	11262	NM_007237	SP140 nuclear body protein
SMARCD1	6602	NM_003076	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1
TRIM69	140691	NM_080745	tripartite motif-containing 69
CDKN2D	1032	NM_001800	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4)
PAK6	56924	NM_020168	p21(CDKN1A)-activated kinase 6
TACR2	6865	NM_001057	tachykinin receptor 2
MMP17	4326	NM_016155	matrix metallopeptidase 17 (membrane-inserted)

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
MUC3A	4584	XM_374502	mucin 3A, cell surface associated
PRKCZ	5590	NM_002744	protein kinase C, zeta
TNFRSF17	608	NM_001192	tumor necrosis factor receptor superfamily, member 17
GTF2IRD2	84163	NM_173537	GTF2I repeat domain containing 2
TRPM3	80036	NM_020952	transient receptor potential cation channel, subfamily M, member 3
HMBS	3145	NM_000190, NM_176954	hydroxymethylbilane synthase
CYP27A1	1593	NM_000784	cytochrome P450, family 27, subfamily A, polypeptide 1
FBXL20	84961	NM_032875	F-box and leucine-rich repeat protein 20
CD300C	10871	NM_006678	CD300c molecule
PSD	5662	NM_002779	pleckstrin and Sec7 domain containing
FRAG1	27315	NM_014489	FGF receptor activating protein 1
PCGF1	84759	NM_032673	polycomb group ring finger 1
SIX2	10736	NM_016932	sine oculis homeobox homolog 2 (Drosophila)
CLCN1	1180	NM_000083	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
EVL	51466	NM_016337	Enah/Vasp-like
TOM1	10043	NM_005488	target of myb1 (chicken)
BAIAP2	10458	NM_006340	BAI1-associated protein 2
ZFY	7544	NM_003411	zinc finger protein, Y-linked
UBE2D1	7321	NM_003338	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
KRT18	3875	NM_000224	keratin 18
GJA4	2701	NM_002060	gap junction protein, alpha 4, 37kDa
SF3A2	8175	NM_007165	splicing factor 3a, subunit 2, 66kDa
TRNT1	51095	NM_016000	tRNA nucleotidyl transferase, CCA-adding, 1
RANGAP1	5905	NM_002883	Ran GTPase activating protein 1
CCT4	10575	NM_006430	chaperonin containing TCP1, subunit 4 (delta)
TSPAN4	7106	NM_003271	tetraspanin 4
PTGER2	5732	NM_000956	prostaglandin E receptor 2 (subtype EP2), 53kDa
GTPBP4	23560	NM_012341	GTP binding protein 4
ADRA1A	148	NM_000680	adrenergic, alpha-1A-, receptor
PHB2	11331	NM_007273	prohibitin 2
TNFRSF19L	84957	NM_032871	tumor necrosis factor receptor superfamily, member 19-like
COL14A1	7373	XM_044622	collagen, type XIV, alpha 1 (undulin)
CD79A	973	NM_001783	CD79a molecule, immunoglobulin-associated alpha
F12	2161	NM_000505	coagulation factor XII (Hageman factor)

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
ASMT	438	NM_004043	acetylserotonin O-methyltransferase
GRK6	2870	NM_002082	G protein-coupled receptor kinase 6
GNRH2	2797	NM_001501	gonadotropin-releasing hormone 2
SDHB	6390	NM_003000	succinate dehydrogenase complex, subunit B, iron sulfur (Ip)
THBS2	7058	NM_003247	thrombospondin 2
HIVEP2	3097	NM_145975, NM_006734	human immunodeficiency virus type I enhancer binding protein 2
WASF1	8936	NM_003931	WAS protein family, member 1
SSPN	8082	NM_005086	sarcospan (Kras oncogene-associated gene)
ITGAV	3685	NM_002210	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
PLXNA2	5362	XM_372810	plexin A2
IGF1	3479	NM_000618	insulin-like growth factor 1 (somatomedin C)
NCR3	259197	NM_147130	natural cytotoxicity triggering receptor 3
TH	7054	NM_000360	tyrosine hydroxylase
HMGCL	3155	NM_177229, NM_000191	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
CENPJ	55835	NM_018451	centromere protein J
FABP1	2168	NM_001443	fatty acid binding protein 1, liver
PRKAA2	5563	NM_006252	protein kinase, AMP-activated, alpha 2 catalytic subunit
CASP1	834	NM_001223	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
CAPN1	823	NM_005186	calpain 1, (mu/I) large subunit
MCCC1	56922	NM_020166	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha)
RAB7A	7879	NM_004637	RAB7A, member RAS oncogene family
DBX1	120237	XM_061930	developing brain homeobox 1
KIAA0196	9897	NM_014846	KIAA0196
HLA-DRB1	3123	NM_002124, NM_172672	major histocompatibility complex, class II, DR beta 1
MMACHC	25974	XM_032397	methylmalonic aciduria (cobalamin deficiency) cblC type, with homocystinuria
TGFBI	7045	NM_000358	transforming growth factor, beta-induced, 68kDa
PPFIA4	8497	XM_046751	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
SORCS2	57537	NM_020777	sortilin-related VPS10 domain containing receptor 2
BAI3	577	NM_001704	brain-specific angiogenesis inhibitor 3
RFX1	5989	NM_002918	regulatory factor X, 1 (influences HLA class II expression)
IRAK3	11213	NM_007199	interleukin-1 receptor-associated kinase 3
PA2G4	5036	NM_006191	proliferation-associated 2G4, 38kDa
GCM2	9247	NM_004752	glial cells missing homolog 2 (Drosophila)
CHRND	1144	NM_000751	cholinergic receptor, nicotinic, delta

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
USP54	159195	NM_152586	ubiquitin specific peptidase 54
HNRPU	3192	NM_004501	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
NUTF2	10204	NM_005796	nuclear transport factor 2
HNRPK	3190	NM_002140	heterogeneous nuclear ribonucleoprotein K
ARCN1	372	NM_001655	archain 1
TRAF1	7185	NM_005658	TNF receptor-associated factor 1
TUBB2A	7280	NM_001069	tubulin, beta 2A
ATG16L2	89849	XM_058426	ATG16 autophagy related 16-like 2 (S. cerevisiae)
ARSE	415	NM_000047	arylsulfatase E (chondrodysplasia punctata 1)
SIDT1	54847	NM_017699	SID1 transmembrane family, member 1
GNG11	2791	NM_004126	guanine nucleotide binding protein (G protein), gamma 11
NAT9	26151	NM_015654	N-acetyltransferase 9
MMP10	4319	NM_002425	matrix metallopeptidase 10 (stromelysin 2)
HOXD11	3237	NM_021192	homeobox D11
POLR3G	10622	NM_006467	polymerase (RNA) III (DNA directed) polypeptide G (32kD)
TACC2	10579	NM_006997	transforming, acidic coiled-coil containing protein 2
FGF2	2247	NM_002006	fibroblast growth factor 2 (basic)
BGN	633	NM_001711	biglycan
C11orf68	83638	NM_031450	chromosome 11 open reading frame 68
QSCN6	5768	NM_002826	quiescin Q6
TRIM8	81603	NM_030912	tripartite motif-containing 8
GJA3	2700	NM_021954, NM_029726	gap junction protein, alpha 3, 46kDa
TMPRSS5	80975	NM_030770	transmembrane protease, serine 5 (spinesin)
TAF2	6873	NM_003184	TAF2 RNA polymerase II, TATA box binding protein (TBP)-associated factor, 150kDa
OA48-18	10414	NM_006107	acid-inducible phosphoprotein
MKLN1	4289	NM_013255	muskelin 1, intracellular mediator containing kelch motifs
USP19	10869	XM_496642	ubiquitin specific peptidase 19
SETDB1	9869	NM_012432	SET domain, bifurcated 1
SLC25A19	60386	NM_021734	solute carrier family 25 (mitochondrial thiamine pyrophosphate carrier), member 19
PTPRH	5794	NM_002842	protein tyrosine phosphatase, receptor type, H
INTS4	92105	NM_033547	integrator complex subunit 4
COPE	11316	NM_007263	coatomer protein complex, subunit epsilon
PRKAG3	53632	NM_017431	protein kinase, AMP-activated, gamma 3 non-catalytic subunit
BPGM	669	NM_001724	2,3-bisphosphoglycerate mutase

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
PRAF2	11230	NM_007213	PRA1 domain family, member 2
NFIL3	4783	NM_005384	nuclear factor, interleukin 3 regulated
CXCL12	6387	NM_000609	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
PLCH2	9651	XM_371214	phospholipase C, eta 2
CHID1	66005	NM_023947	chitinase domain containing 1
CEND1	51286	NM_016564	cell cycle exit and neuronal differentiation 1
AMH	268	NM_000479	anti-Mullerian hormone
HIST2H3C	126961	NM_021059	histone cluster 2, H3c
CNKSR2	22866	NM_014927	connector enhancer of kinase suppressor of Ras 2
MYL3	4634	NM_000258	myosin, light chain 3, alkali; ventricular, skeletal, slow
SORBS3	10174	NM_005775	sorbin and SH3 domain containing 3
PFDN2	5202	NM_012394	prefoldin subunit 2
SOD1	6647	NM_000454	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult))
RBBP8	5932	NM_002894	retinoblastoma binding protein 8
PROSC	11212	NM_007198	proline synthetase co-transcribed homolog (bacterial)
TRIP6	7205	NM_003302	thyroid hormone receptor interactor 6
TNF	7124	NM_000594	tumor necrosis factor (TNF superfamily, member 2)
HSFY2	159119	NM_153716	heat shock transcription factor, Y linked 2
SCAMP4	113178	NM_079834	secretory carrier membrane protein 4
TRPA1	8989	NM_007332	transient receptor potential cation channel, subfamily A, member 1
HNRPM	4670	NM_005968	heterogeneous nuclear ribonucleoprotein M
C2orf13	200558	NM_173545	chromosome 2 open reading frame 13
AGER	177	NM_001136	advanced glycosylation end product-specific receptor
GFER	2671	NM_005262	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae)
ERH	2079	NM_004450	enhancer of rudimentary homolog (Drosophila)
PAQR6	79957	NM_024897	progestin and adipoQ receptor family member VI
UNC13B	10497	NM_006377	unc-13 homolog B (C. elegans)
EGLN2	112398	NM_053046	egl nine homolog 2 (C. elegans)
FGFR1	2260	NM_000604	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome)
CARKL	23729	NM_013276	carbohydrate kinase-like
SEMA4B	10509	NM_020210	sema domain, immunoglobulin domain (Ig), transmembrane domain (TM) and short cytoplasmic domain, (semaphorin) 4B
TUBGCP6	85378	NM_020461	tubulin, gamma complex associated protein 6
ICT1	3396	NM_001545, NM_016879	immature colon carcinoma transcript 1
WFDC2	10406	NM_006103	WAP four-disulfide core domain 2

Figure 3 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
CPNE6	9362	NM_006032	copine VI (neuronal)
CAMKV	79012	NM_024046	CaM kinase-like vesicle-associated
LOC285643	285643	XM_209695	KIF4B
C18orf8	29919	NM_013326	chromosome 18 open reading frame 8
LOR	4014	NM_000427	loricrin
ADM	133	NM_001124	adrenomedullin
LIF	3976	NM_002309	leukemia inhibitory factor (cholinergic differentiation factor)
KIF11	3832	NM_004523	kinesin family member 11
FANCC	2176	NM_000136	Fanconi anemia, complementation group C
NOXO1	124056	NM_144603	NADPH oxidase organizer 1
UBE1L2	55236	NM_018227	ubiquitin-activating enzyme E1-like 2
P2RX1	5023	NM_002558	purinergic receptor P2X, ligand-gated ion channel, 1
NPTN	27020	NM_012428	neuroplastin
STAT3	6774	NM_003150	signal transducer and activator of transcription 3 (acute-phase response factor)
PDCD5	9141	NM_004708	programmed cell death 5
SMYD3	64754	NM_022743	SET and MYND domain containing 3
TCEB3	6924	NM_003198	transcription elongation factor B (SIII), polypeptide 3 (110kDa, elongin A)
CATSPER4	378807	XM_371237	cation channel, sperm associated 4
MEGF10	84466	NM_032446	multiple EGF-like-domains 10
KIF5C	3800	XM_377774	kinesin family member 5C
ATG7	10533	NM_006395	ATG7 autophagy related 7 homolog (S. cerevisiae)
RELA	5970	NM_021975	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian)
GAB1	2549	NM_002039	GRB2-associated binding protein 1
LOC285647	285647	XM_209700	suppressor of defective silencing 3 pseudogene
GPR18	2841	NM_005292, NM_145948	G protein-coupled receptor 18
MBP	4155	NM_002385	myelin basic protein
PDCL	5082	NM_005388	phosducin-like
STIM1	6786	NM_003156	stromal interaction molecule 1
NFKB1	4790	NM_003998	nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 (p105)
TPR	7175	NM_003292	translocated promoter region (to activated MET oncogene)
PGGT1B	5229	NM_005023	protein geranylgeranyltransferase type I, beta subunit
ATG5	9474	NM_004849	ATG5 autophagy related 5 homolog (S. cerevisiae)

Figure 4

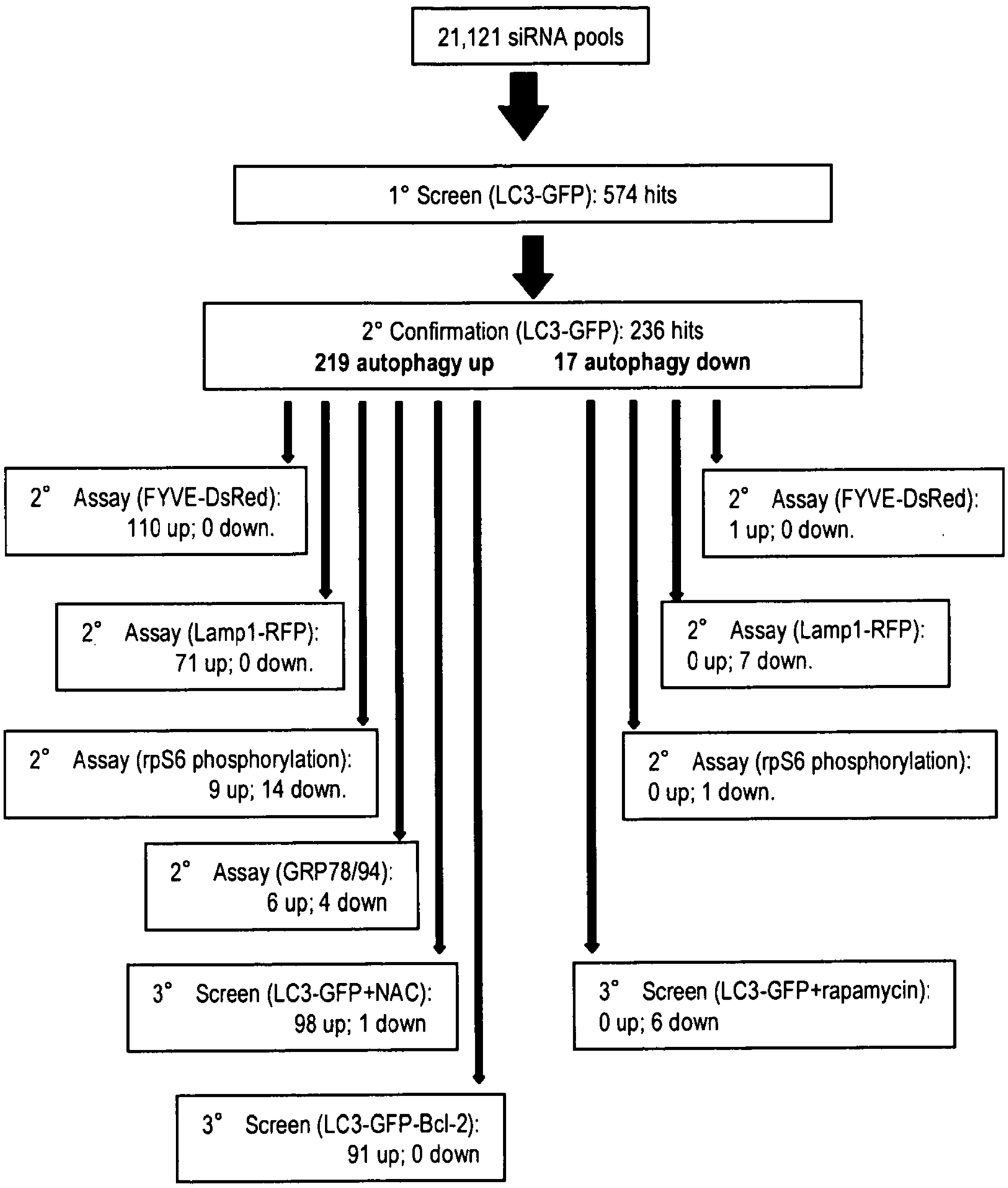

21,121 siRNA pools
1° Screen (LC3-GFP): 574 hits
2° Confirmation (LC3-GFP): 236 hits
219 autophagy up
17 autophagy down
2° Assay (FYVE-DsRed): 110 up; 0 down.
2° Assay (Lamp1-RFP): 71 up; 0 down.
2° Assay (rpS6 phosphorylation): 9 up; 14 down.
2° Assay (GRP78/94): 6 up; 4 down
3° Screen (LC3-GFP+NAC): 98 up; 1 down
3° Screen (LC3-GFP-Bcl-2): 91 up; 0 down
2° Assay (FYVE-DsRed): 1 up; 0 down.
2° Assay (Lamp1-RFP): 0 up; 7 down.
2° Assay (rpS6 phosphorylation): 0 up; 1 down.
3° Screen (LC3-GFP+rapamycin): 0 up; 6 down

Figure 5.

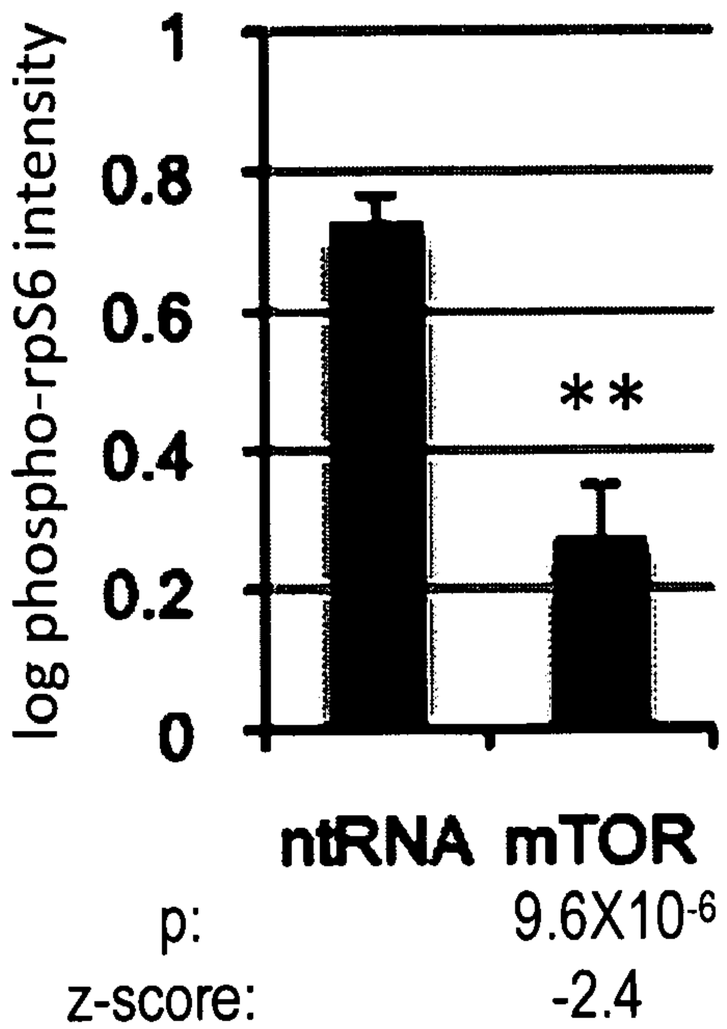

log phospho-rpS6 intensity
1
0.8
0.6
0.4
0.2
0
**
ntRNA
mTOR
p:
9.6X10-6
z-score:
-2.4

Figure 6.

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1
CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
COPB2	9276	NM_004766	coatomer protein complex, subunit beta 2 (beta prime)
CDK5RAP3	80279	NM_025197	CDK5 regulatory subunit associated protein 3
NLK	51701	NM_016231	nemo-like kinase
PFKL	5211	NM_002626	phosphofructokinase, liver
GTF2IRD2	84163	NM_173537	GTF2I repeat domain containing 2
HNRPU	3192	NM_004501	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
NUTF2	10204	NM_005796	nuclear transport factor 2
HNRPK	3190	NM_002140	heterogeneous nuclear ribonucleoprotein K
ARCN1	372	NM_001655	archain 1
NFKB1	4790	NM_003998	nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 (p105)
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
TRPM3	80036	NM_020952	transient receptor potential cation channel, subfamily M, member 3
HMBS	3145	NM_000190, NM_176954	hydroxymethylbilane synthase
TRAF1	7185	NM_005658	TNF receptor-associated factor 1
TUBB2A	7280	NM_001069	tubulin, beta 2A
ATG16L2	89849	XM_058426	ATG16 autophagy related 16-like 2 (S. cerevisiae)
ARSE	415	NM_000047	arylsulfatase E (chondrodysplasia punctata 1)
SIDT1	54847	NM_017699	SID1 transmembrane family, member 1

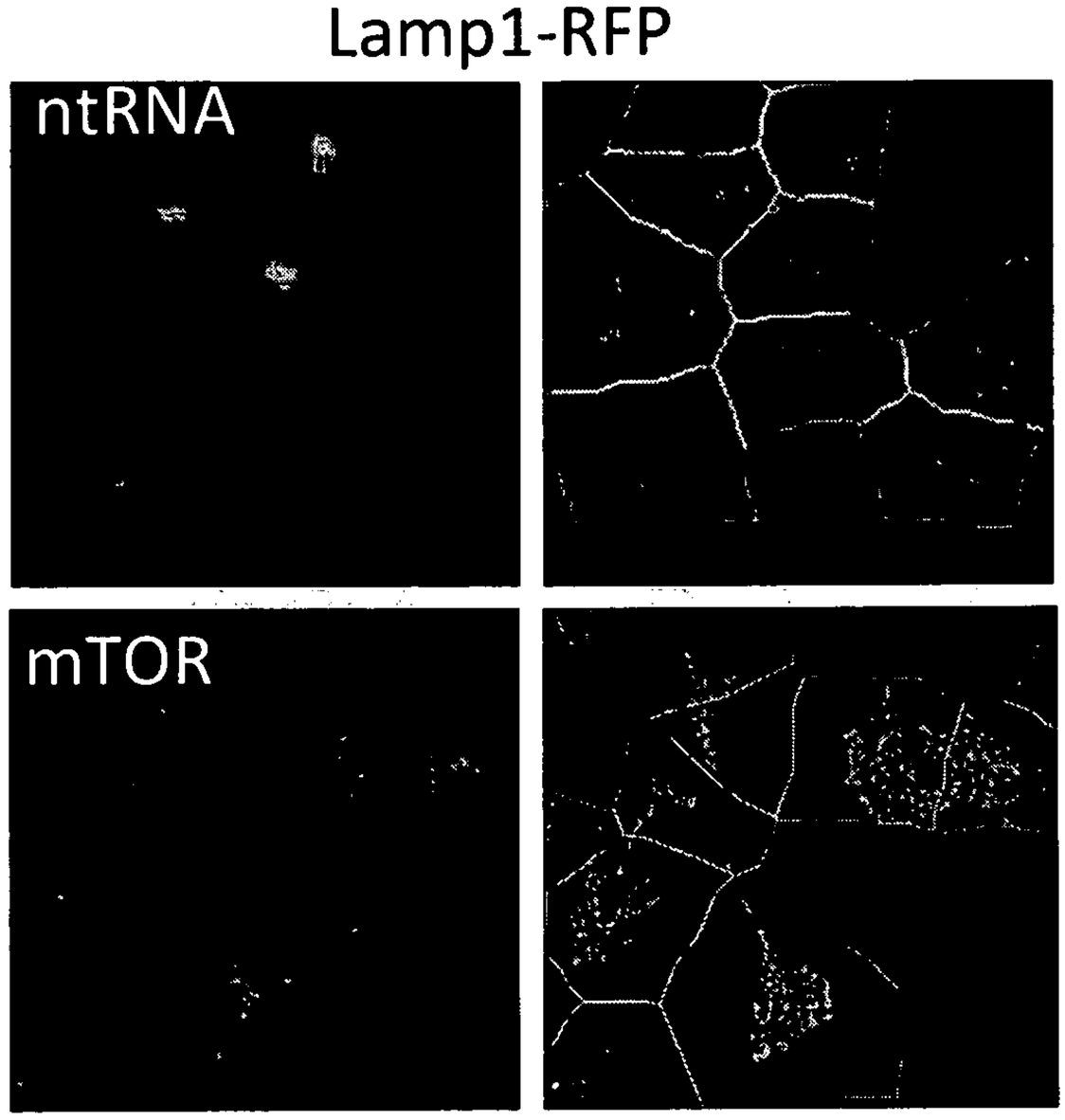
Figure 7.

Genes whose knock-down suppresses autophagy downstream of mTORC1.

Gene Symbol	Gene ID	Gene Name
KIF5C	3800	kinesin family member 5C
ATG7	10533	ATG7 autophagy related 7 homolog (S. cerevisiae)
NFKB1	4790	nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 (p105)
MEGF10	84466	multiple EGF-like-domains 10
ATG5	9474	ATG5 autophagy related 5 homolog (S. cerevisiae)
RELA	5970	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian)

Figure 8

A.


Lamp1-RFP
ntRNA
mTOR

B.

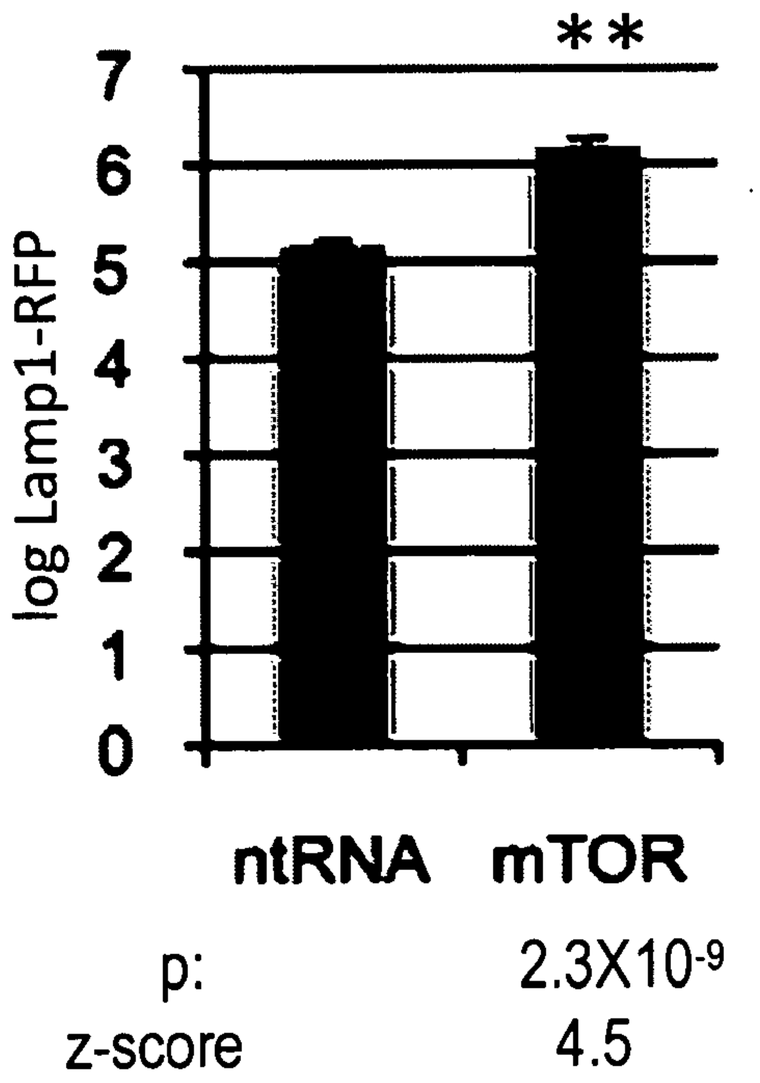

**
log Lamp1-RFP
0
1
2
3
4
5
6
7
ntRNA
mTOR
p:
2.3X10-9
z-score
4.5

Figure 9.

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
TCEB3	6924	NM_003198	transcription elongation factor B (SIII), polypeptide 3 (110kDa, elongin A)
CATSPER4	378807	XM_371237	cation channel, sperm associated 4
MEGF10	84466	NM_032446	multiple EGF-like-domains 10
KIF5C	3800	XM_377774	kinesin family member 5C
ATG7	10533	NM_006395	ATG7 autophagy related 7 homolog (S. cerevisiae)
RELA	5970	NM_021975	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian)
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1
CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
COPB2	9276	NM_004766	coatomer protein complex, subunit beta 2 (beta prime)
CDK5RAP3	80279	NM_025197	CDK5 regulatory subunit associated protein 3
NLK	51701	NM_016231	nemo-like kinase
PFKL	5211	NM_002626	phosphofructokinase, liver
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
XPO1	7514	NM_003400	exportin 1 (CRM1 homolog, yeast)
KIAA0133	9816	XM_375851	KIAA0133
ADMR	11318	NM_007264	adrenomedullin receptor
OGDH	4967	NM_002541	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
DDX24	57062	NM_020414	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
NUPR1	26471	NM_012385	nuclear protein 1
FXYD2	486	NM_001680	FXYD domain containing ion transport regulator 2
TRHR	7201	NM_003301	thyrotropin-releasing hormone receptor
SUV39H1	6839	NM_003173	suppressor of variegation 3-9 homolog 1 (Drosophila)
FCER1A	2205	NM_002001	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide
PTPRU	10076	NM_005704	protein tyrosine phosphatase, receptor type, U
GPX2	2877	NM_002083	glutathione peroxidase 2 (gastrointestinal)
PRKCA	5578	NM_002737	protein kinase C, alpha
EP300	2033	NM_001429	E1A binding protein p300
LOC388959	388959	XM_373989	hypothetical LOC388959
NTN2L	4917	NM_006181	netrin 2-like (chicken)
DOCK8	81704	NM_203447	dedicator of cytokinesis 8

Figure 9 (Continued)

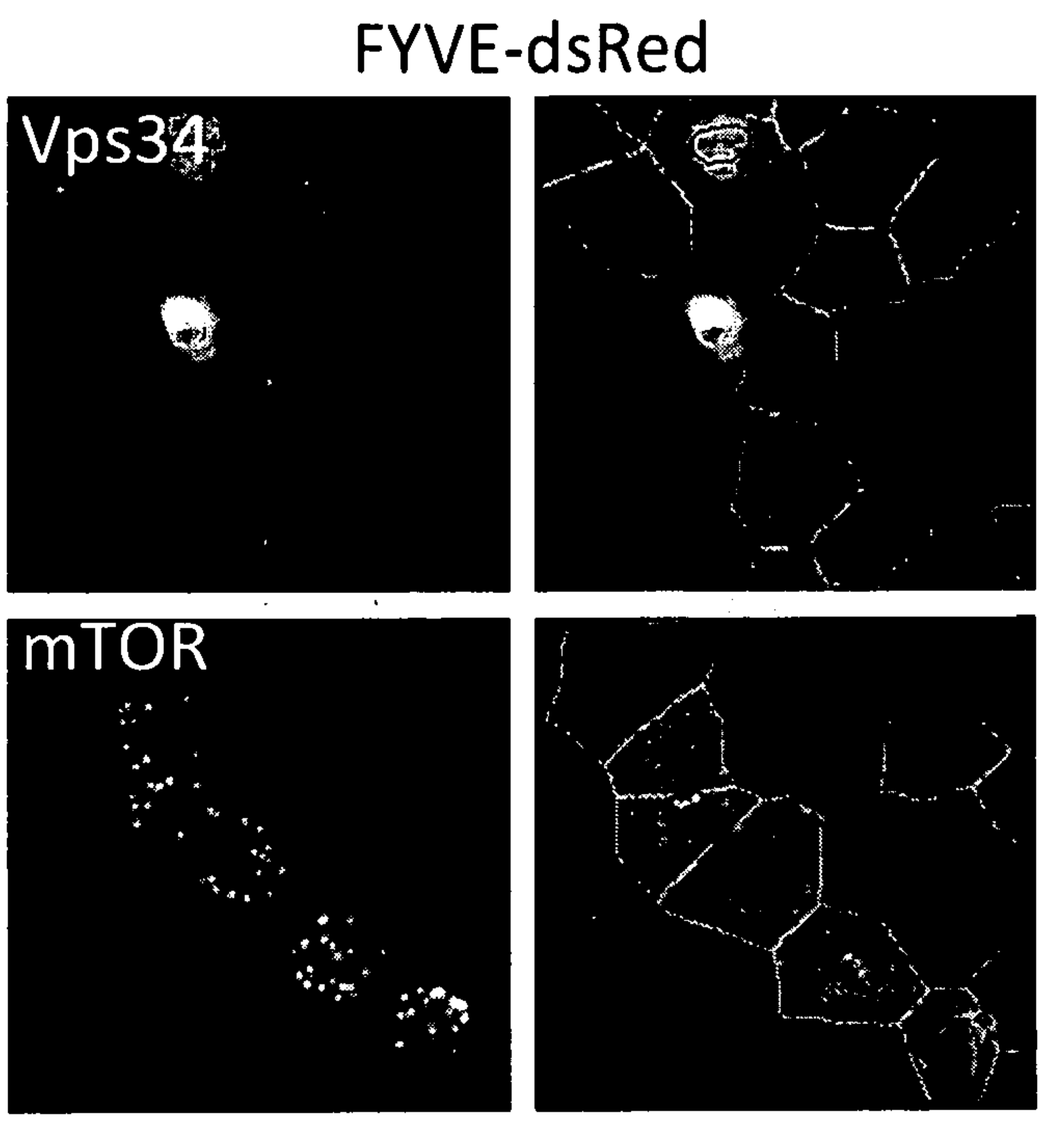
Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
MAP3K7IP1	10454	NM_006116	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
PLAGL2	5326	NM_002657	pleiomorphic adenoma gene-like 2
NUDT1	4521	NM_002452	nudix (nucleoside diphosphate linked moiety X)-type motif 1
RELN	5649	NM_005045	reelin
PNKD	25953	NM_015488	paroxysmal nonkinesiogenic dyskinesia
RIPK1	8737	NM_003804	receptor (TNFRSF)-interacting serine-threonine kinase 1
GNG5	2787	NM_005274	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	NM_001277	choline kinase alpha
C5AR1	728	NM_001736	complement component 5a receptor 1
SCOTIN	51246	NM_016479	scotin
PIGY	84992	NM_032906	phosphatidylinositol glycan anchor biosynthesis, class Y
NAGK	55577	NM_017567	N-acetylglucosamine kinase
RAGE	5891	NM_014226	renal tumor antigen
USP24	23358	XM_165973	ubiquitin specific peptidase 24
AURKA	6790	NM_003600	aurora kinase A
PLDN	26258	NM_012388	pallidin homolog (mouse)
TLR3	7098	NM_003265	toll-like receptor 3
PPARD	5467	NM_006238	peroxisome proliferator-activated receptor delta
HRC	3270	NM_002152	histidine rich calcium binding protein
NNMT	4837	NM_006169	nicotinamide N-methyltransferase
RNPEPL1	57140	NM_018226	arginyl aminopeptidase (aminopeptidase B)-like 1
EPHA6	203806	XM_114973	EPH receptor A6
CDCA8	55143	NM_018101	cell division cycle associated 8
CKAP5	9793	NM_014756	cytoskeleton associated protein 5
ZBTB16	7704	NM_006006	zinc finger and BTB domain containing 16
GABBR2	9568	NM_005458	gamma-aminobutyric acid (GABA) B receptor, 2
PTMA	5757	NM_002823	prothymosin, alpha (gene sequence 28)
PTCRA	171558	NM_138296	pre T-cell antigen receptor alpha
RORC	6097	NM_005060	RAR-related orphan receptor C
GNAI1	2770	NM_002069	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
UTS2R	2837	NM_018949	urotensin 2 receptor
MATN3	4148	NM_002381	matrilin 3
NPTX1	4884	NM_002522	neuronal pentraxin I
SP140	11262	NM_007237	SP140 nuclear body protein

Figure 9 (Continued)

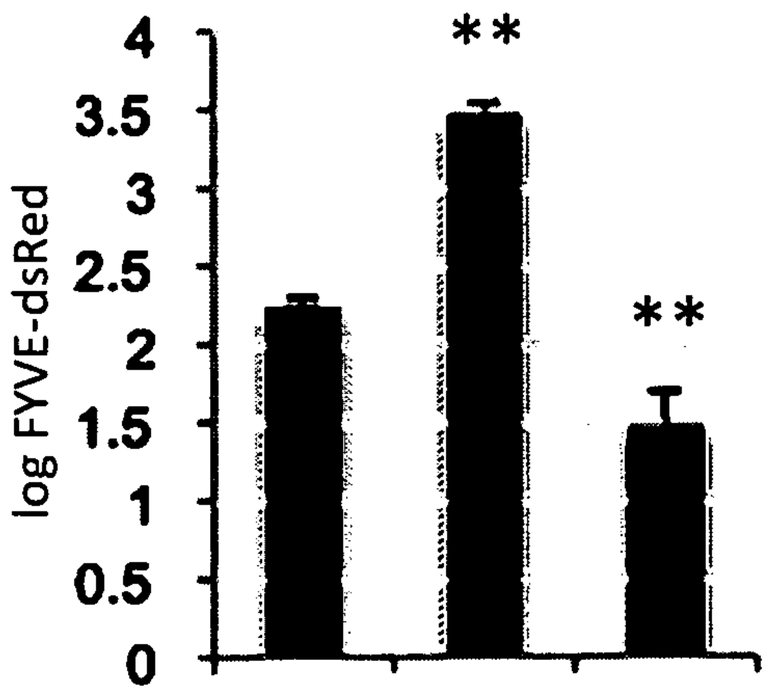
Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
SMARCD1	6602	NM_003076	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1
TRIM69	140691	NM_080745	tripartite motif-containing 69
CDKN2D	1032	NM_001800	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4)
PAK6	56924	NM_020168	p21(CDKN1A)-activated kinase 6
TACR2	6865	NM_001057	tachykinin receptor 2
MMP17	4326	NM_016155	matrix metallopeptidase 17 (membrane-inserted)
MUC3A	4584	XM_374502	mucin 3A, cell surface associated
PRKCZ	5590	NM_002744	protein kinase C, zeta
TNFRSF17	608	NM_001192	tumor necrosis factor receptor superfamily, member 17

Figure 10.

A.

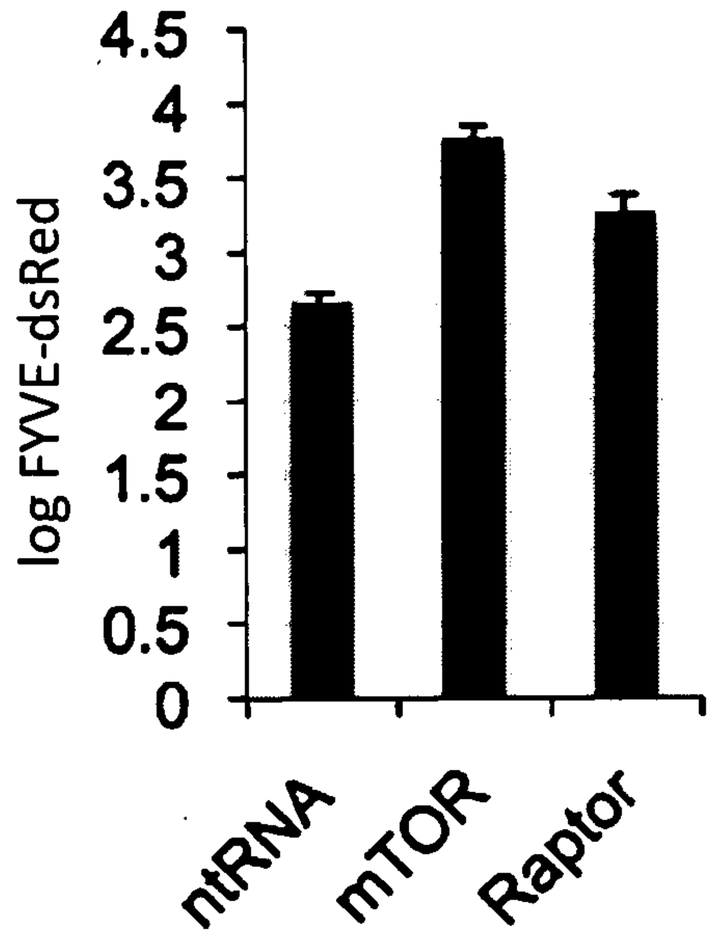


B.



	ntRNA	mTOR	Vps34
p:		$1.8X10^{-15}$	$3.7X10^{-6}$
z-score:		4.3	-2.7

C.



	ntRNA	mTOR	Raptor
p:		$4.2X10^{-10}$	0.012
z-score:		2.8	1.5

Figure 11.

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
MEGF10	84466	NM_032446	multiple EGF-like-domains 10
USP54	159195	NM_152586	ubiquitin specific peptidase 54
GHSR	2693	NM_004122	growth hormone secretagogue receptor
TINP1	10412	NM_014886	TGF beta-inducible nuclear protein 1
CHAF1B	8208	NM_005441	chromatin assembly factor 1, subunit B (p60)
COX5A	9377	NM_004255	cytochrome c oxidase subunit Va
IHPK3	117283	NM_054111	inositol hexaphosphate kinase 3
CENPE	1062	NM_001813	centromere protein E, 312kDa
CLCF1	23529	NM_013246	cardiotrophin-like cytokine factor 1
XPO1	7514	NM_003400	exportin 1 (CRM1 homolog, yeast)
KIAA0133	9816	XM_375851	KIAA0133
ADMR	11318	NM_007264	adrenomedullin receptor
OGDH	4967	NM_002541	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
DDX24	57062	NM_020414	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
NUPR1	26471	NM_012385	nuclear protein 1
FXYD2	486	NM_001680	FXYD domain containing ion transport regulator 2
TRHR	7201	NM_003301	thyrotropin-releasing hormone receptor
SUV39H1	6839	NM_003173	suppressor of variegation 3-9 homolog 1 (Drosophila)
FCER1A	2205	NM_002001	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide
PTPRU	10076	NM_005704	protein tyrosine phosphatase, receptor type, U
GPX2	2877	NM_002083	glutathione peroxidase 2 (gastrointestinal)
PRKCA	5578	NM_002737	protein kinase C, alpha
EP300	2033	NM_001429	E1A binding protein p300
LOC388959	388959	XM_373989	hypothetical LOC388959
NTN2L	4917	NM_006181	netrin 2-like (chicken)
DOCK8	81704	NM_203447	dedicator of cytokinesis 8
MAP3K7IP1	10454	NM_006116	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
PLAGL2	5326	NM_002657	pleiomorphic adenoma gene-like 2
NUDT1	4521	NM_002452	nudix (nucleoside diphosphate linked moiety X)-type motif 1
RELN	5649	NM_005045	reelin
PNKD	25953	NM_015488	paroxysmal nonkinesiogenic dyskinesia
RIPK1	8737	NM_003804	receptor (TNFRSF)-interacting serine-threonine kinase 1
GNG5	2787	NM_005274	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	NM_001277	choline kinase alpha

Figure 11 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
C5AR1	728	NM_001736	complement component 5a receptor 1
SCOTIN	51246	NM_016479	scotin
PIGY	84992	NM_032906	phosphatidylinositol glycan anchor biosynthesis, class Y
NAGK	55577	NM_017567	N-acetylglucosamine kinase
RAGE	5891	NM_014226	renal tumor antigen
USP24	23358	XM_165973	ubiquitin specific peptidase 24
AURKA	6790	NM_003600	aurora kinase A
PLDN	26258	NM_012388	pallidin homolog (mouse)
TLR3	7098	NM_003265	toll-like receptor 3
PPARD	5467	NM_006238	peroxisome proliferator-activated receptor delta
HRC	3270	NM_002152	histidine rich calcium binding protein
NNMT	4837	NM_006169	nicotinamide N-methyltransferase
GTF2IRD2	84163	NM_173537	GTF2I repeat domain containing 2
TRPM3	80036	NM_020952	transient receptor potential cation channel, subfamily M, member 3
HMBS	3145	NM_000190, NM_176954	hydroxymethylbilane synthase
CYP27A1	1593	NM_000784	cytochrome P450, family 27, subfamily A, polypeptide 1
FBXL20	84961	NM_032875	F-box and leucine-rich repeat protein 20
CD300C	10871	NM_006678	CD300c molecule
PSD	5662	NM_002779	pleckstrin and Sec7 domain containing
FRAG1	27315	NM_014489	FGF receptor activating protein 1
PCGF1	84759	NM_032673	polycomb group ring finger 1
SIX2	10736	NM_016932	sine oculis homeobox homolog 2 (Drosophila)
CLCN1	1180	NM_000083	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
EVL	51466	NM_016337	Enah/Vasp-like
TOM1	10043	NM_005488	target of myb1 (chicken)
BAIAP2	10458	NM_006340	BAI1-associated protein 2
ZFY	7544	NM_003411	zinc finger protein, Y-linked
UBE2D1	7321	NM_003338	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
KRT18	3875	NM_000224	keratin 18
GJA4	2701	NM_002060	gap junction protein, alpha 4, 37kDa
SF3A2	8175	NM_007165	splicing factor 3a, subunit 2, 66kDa
TRNT1	51095	NM_016000	tRNA nucleotidyl transferase, CCA-adding, 1
RANGAP1	5905	NM_002883	Ran GTPase activating protein 1
CCT4	10575	NM_006430	chaperonin containing TCP1, subunit 4 (delta)

Figure 11 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
TSPAN4	7106	NM_003271	tetraspanin 4
PTGER2	5732	NM_000956	prostaglandin E receptor 2 (subtype EP2), 53kDa
GTPBP4	23560	NM_012341	GTP binding protein 4
ADRA1A	148	NM_000680	adrenergic, alpha-1A-, receptor
PHB2	11331	NM_007273	prohibitin 2
TNFRSF19L	84957	NM_032871	tumor necrosis factor receptor superfamily, member 19-like
COL14A1	7373	XM_044622	collagen, type XIV, alpha 1 (undulin)
CD79A	973	NM_001783	CD79a molecule, immunoglobulin-associated alpha
F12	2161	NM_000505	coagulation factor XII (Hageman factor)
ASMT	438	NM_004043	acetylserotonin O-methyltransferase
GRK6	2870	NM_002082	G protein-coupled receptor kinase 6
GNRH2	2797	NM_001501	gonadotropin-releasing hormone 2
SDHB	6390	NM_003000	succinate dehydrogenase complex, subunit B, iron sulfur (Ip)
THBS2	7058	NM_003247	thrombospondin 2
HIVEP2	3097	NM_145975, NM_006734	human immunodeficiency virus type I enhancer binding protein 2
WASF1	8936	NM_003931	WAS protein family, member 1
SSPN	8082	NM_005086	sarcospan (Kras oncogene-associated gene)
ITGAV	3685	NM_002210	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
PLXNA2	5362	XM_372810	plexin A2
IGF1	3479	NM_000618	insulin-like growth factor 1 (somatomedin C)
NCR3	259197	NM_147130	natural cytotoxicity triggering receptor 3
TH	7054	NM_000360	tyrosine hydroxylase
HMGCL	3155	NM_177229, NM_000191	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
CENPJ	55835	NM_018451	centromere protein J
FABP1	2168	NM_001443	fatty acid binding protein 1, liver
PRKAA2	5563	NM_006252	protein kinase, AMP-activated, alpha 2 catalytic subunit
CASP1	834	NM_001223	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
CAPN1	823	NM_005186	calpain 1, (mu/I) large subunit
MCCC1	56922	NM_020166	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha)
RAB7A	7879	NM_004637	RAB7A, member RAS oncogene family
DBX1	120237	XM_061930	developing brain homeobox 1
KIAA0196	9897	NM_014846	KIAA0196
HLA-DRB1	3123	NM_002124, NM_172672	major histocompatibility complex, class II, DR beta 1
MMACHC	25974	XM_032397	methylmalonic aciduria (cobalamin deficiency) cblC type, with homocystinuria

Figure 11 (Continued)

Gene Symbol	Gene ID	Genbank Acc. No.	Gene Name
TGFBI	7045	NM_000358	transforming growth factor, beta-induced, 68kDa
PPFIA4	8497	XM_046751	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
SORCS2	57537	NM_020777	sortilin-related VPS10 domain containing receptor 2
BAI3	577	NM_001704	brain-specific angiogenesis inhibitor 3
RFX1	5989	NM_002918	regulatory factor X, 1 (influences HLA class II expression)
IRAK3	11213	NM_007199	interleukin-1 receptor-associated kinase 3
PA2G4	5036	NM_006191	proliferation-associated 2G4, 38kDa
GCM2	9247	NM_004752	glial cells missing homolog 2 (Drosophila)
CHRND	1144	NM_000751	cholinergic receptor, nicotinic, delta

Figure 12.

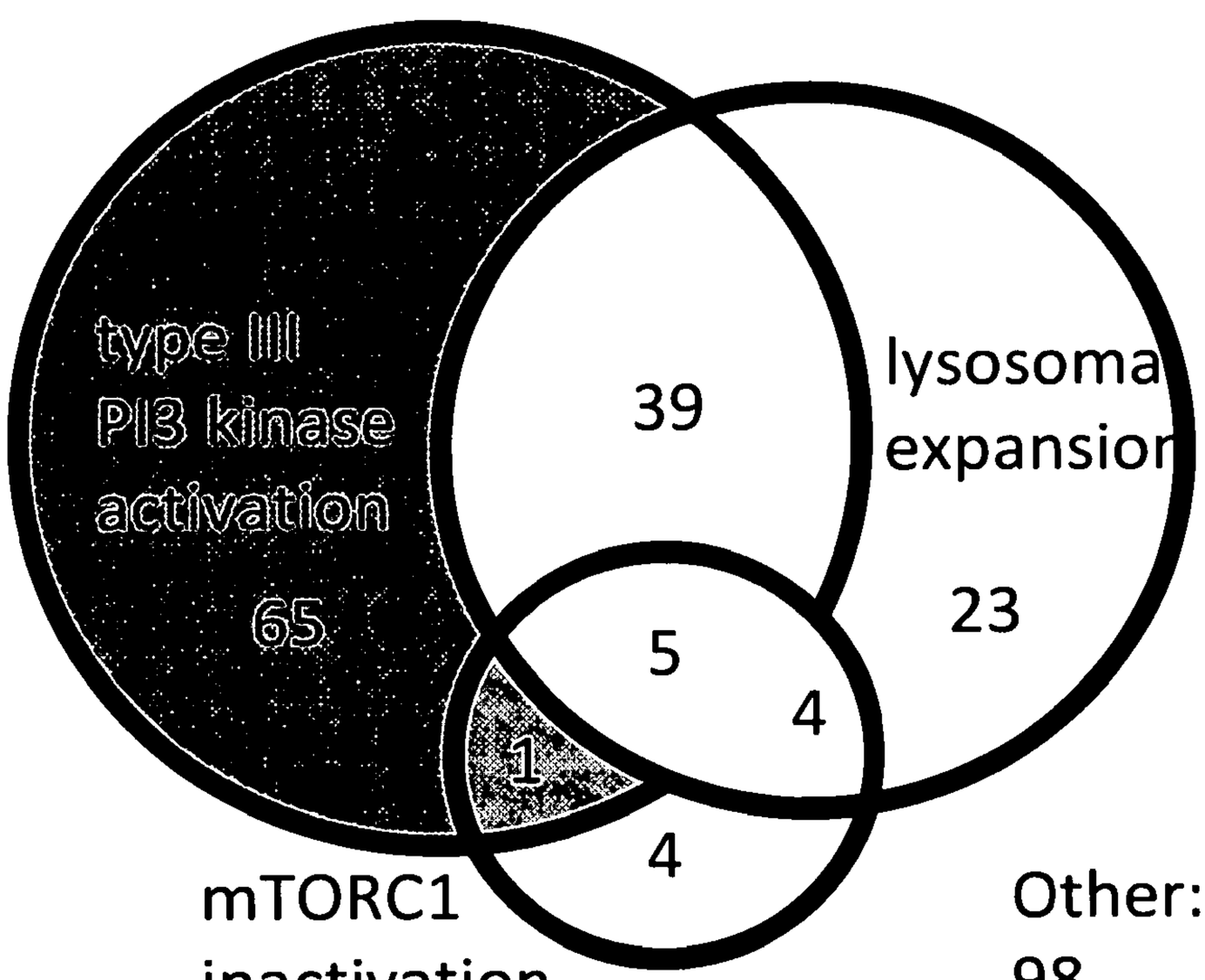

type III
PI3 kinase
activation
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39
lysosomal
expansion
23
5
4
1
4
mTORC1
inactivation
Other:
98

Figure 13.

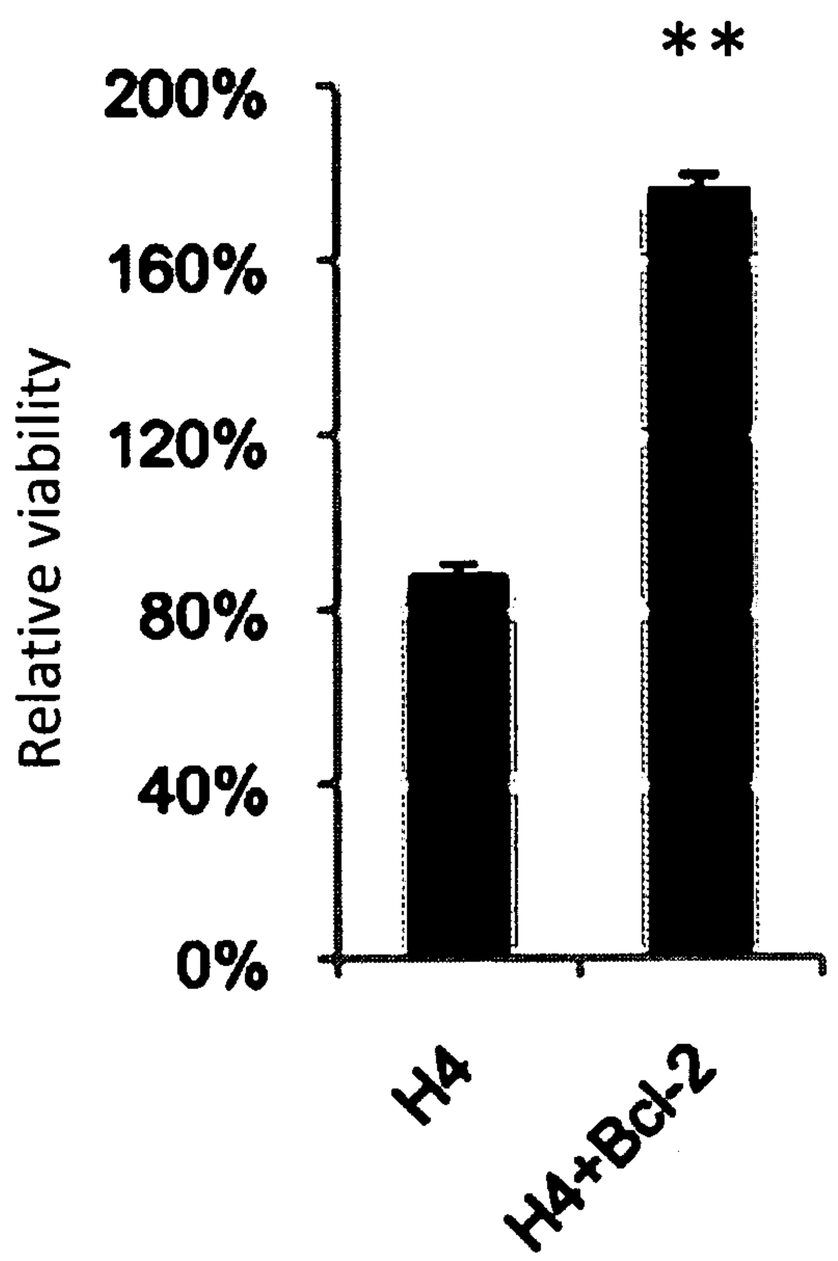


Figure 14.

viability	Gene Symbol	Gene ID	Gene Name
67%	KIF11	3832	kinesin family member 11
177%	PHB2	11331	prohibitin 2
136%	DDX24	57062	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
118%	ZFY	7544	zinc finger protein, Y-linked
132%	TINP1	10412	TGF beta-inducible nuclear protein 1
159%	OGDH	4967	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
136%	C5AR1	728	complement component 5a receptor 1
117%	EVL	51466	Enah/Vasp-like
230%	EP300	2033	E1A binding protein p300
118%	TLR3	7098	toll-like receptor 3
207%	FRAG1	27315	FGF receptor activating protein 1
176%	CENPE	1062	centromere protein E, 312kDa
118%	RIPK1	8737	receptor (TNFRSF)-interacting serine-threonine kinase 1
130%	HIVEP2	3097	human immunodeficiency virus type I enhancer binding protein 2
197%	NUDT1	4521	nudix (nucleoside diphosphate linked moiety X)-type motif 1
133%	GNAI1	2770	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
134%	NFIL3	4783	nuclear factor, interleukin 3 regulated
141%	ARCN1	372	archain 1
155%	TNFRSF17	608	tumor necrosis factor receptor superfamily, member 17
91%	CKAP5	9793	cytoskeleton associated protein 5
193%	HRC	3270	histidine rich calcium binding protein
134%	HNRPU	3192	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
253%	RAB7A	7879	RAB7A, member RAS oncogene family
207%	NPTX1	4884	neuronal pentraxin I
106%	COPB2	9276	coatomer protein complex, subunit beta 2 (beta prime)
118%	HOXD11	3237	homeobox D11
134%	XPO1	7514	exportin 1 (CRM1 homolog, yeast)
182%	HNRPK	3190	heterogeneous nuclear ribonucleoprotein K
75%	ITGAV	3685	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
235%	GNRH2	2797	gonadotropin-releasing hormone 2
129%	PRKCA	5578	protein kinase C, alpha
170%	SMARCD1	6602	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1
188%	CLCN1	1180	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
228%	TH	7054	tyrosine hydroxylase

Figure 14 (Continued)

viability	Gene Symbol	Gene ID	Gene Name
120%	CDK5RAP3	80279	CDK5 regulatory subunit associated protein 3
126%	SP140	11262	SP140 nuclear body protein
109%	GRK6	2870	G protein-coupled receptor kinase 6
146%	CDCA8	55143	cell division cycle associated 8
211%	GJA3	2700	gap junction protein, alpha 3, 46kDa
135%	CEND1	51286	cell cycle exit and neuronal differentiation 1
136%	SCOTIN	51246	scotin
139%	OA48-18	10414	acid-inducible phosphoprotein
140%	ADRA1A	148	adrenergic, alpha-1A-, receptor
109%	PFKL	5211	phosphofructokinase, liver
206%	TNFRSF19L	84957	tumor necrosis factor receptor superfamily, member 19-like
187%	CLCF1	23529	cardiotrophin-like cytokine factor 1
194%	TRAF1	7185	TNF receptor-associated factor 1
203%	SUV39H1	6839	suppressor of variegation 3-9 homolog 1 (Drosophila)
153%	SF3A2	8175	splicing factor 3a, subunit 2, 66kDa
225%	NNMT	4837	nicotinamide N-methyltransferase
188%	FXYD2	486	FXYD domain containing ion transport regulator 2
130%	GHSR	2693	growth hormone secretagogue receptor
163%	PLCH2	9651	phospholipase C, eta 2
119%	AURKA	6790	aurora kinase A
222%	CD300C	10871	CD300c molecule
201%	PTPRU	10076	protein tyrosine phosphatase, receptor type, U
141%	CHAF1B	8208	chromatin assembly factor 1, subunit B (p60)
148%	CXCL12	6387	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
164%	NAT9	26151	N-acetyltransferase 9
177%	LOC285643	285643	KIF4B
202%	KRT18	3875	keratin 18
207%	PTPRH	5794	protein tyrosine phosphatase, receptor type, H
214%	NCR3	259197	natural cytotoxicity triggering receptor 3
219%	SDHB	6390	succinate dehydrogenase complex, subunit B, iron sulfur (Ip)
117%	ZBTB16	7704	zinc finger and BTB domain containing 16
209%	RELN	5649	reelin
113%	PRKAG3	53632	protein kinase, AMP-activated, gamma 3 non-catalytic subunit
201%	C11orf68	83638	chromosome 11 open reading frame 68

Figure 14 (Continued)

viability	Gene Symbol	Gene ID	Gene Name
134%	GABBR2	9568	gamma-aminobutyric acid (GABA) B receptor, 2
137%	MMACHC	25974	methylmalonic aciduria (cobalamin deficiency) cblC type, with homocystinuria
190%	TRPM3	80036	transient receptor potential cation channel, subfamily M, member 3
150%	SEMA4B	10509	sema domain, immunoglobulin domain (Ig), transmembrane domain (TM) and short cytoplasmic domain, (semaphorin) 4B
122%	IHPK3	117283	inositol hexaphosphate kinase 3
137%	PLAGL2	5326	pleiomorphic adenoma gene-like 2
161%	PLXNA2	5362	plexin A2
226%	IGF1	3479	insulin-like growth factor 1 (somatomedin C)
165%	NUTF2	10204	nuclear transport factor 2
131%	GTF2IRD2	84163	GTF2I repeat domain containing 2
142%	PAK6	56924	p21(CDKN1A)-activated kinase 6
167%	KIAA0133	9816	KIAA0133
184%	NTN2L	4917	netrin 2-like (chicken)
144%	PTGER2	5732	prostaglandin E receptor 2 (subtype EP2), 53kDa
161%	TMPRSS5	80975	transmembrane protease, serine 5 (spinesin)
209%	PFDN2	5202	prefoldin subunit 2
229%	AGER	177	advanced glycosylation end product-specific receptor
231%	PSD	5662	pleckstrin and Sec7 domain containing
142%	SIX2	10736	sine oculis homeobox homolog 2 (Drosophila)
233%	PPFIA4	8497	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
143%	ADMR	11318	adrenomedullin receptor
208%	GJA4	2701	gap junction protein, alpha 4, 37kDa
233%	MAP3K7IP1	10454	mitogen-activated protein kinase kinase kinase 7 interacting protein 1

Figure 15.

viability	Gene Symbol	Gene ID	Gene Name
213%	CAPN1	823	calpain 1, (mu/I) large subunit
112%	NLK	51701	nemo-like kinase
120%	NAGK	55577	N-acetylglucosamine kinase
142%	IRAK3	11213	interleukin-1 receptor-associated kinase 3
136%	PRKAA2	5563	protein kinase, AMP-activated, alpha 2 catalytic subunit
166%	PLDN	26258	pallidin homolog (mouse)
204%	HMBS	3145	hydroxymethylbilane synthase
238%	RANGAP1	5905	Ran GTPase activating protein 1
234%	CHID1	66005	chitinase domain containing 1
237%	MMP10	4319	matrix metallopeptidase 10 (stromelysin 2)
221%	CD79A	973	CD79a molecule, immunoglobulin-associated alpha
125%	CAMKV	79012	CaM kinase-like vesicle-associated
151%	WASF1	8936	WAS protein family, member 1
212%	LIF	3976	leukemia inhibitory factor (cholinergic differentiation factor)
212%	HMGCL	3155	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
142%	GCM2	9247	glial cells missing homolog 2 (Drosophila)
232%	MCCC1	56922	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha)
231%	AMH	268	anti-Mullerian hormone
218%	ASMT	438	acetylserotonin O-methyltransferase
245%	CASP1	834	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
186%	MMP17	4326	matrix metallopeptidase 17 (membrane-inserted)
205%	RBBP8	5932	retinoblastoma binding protein 8
252%	TUBB2A	7280	tubulin, beta 2A
152%	PIGY	84992	phosphatidylinositol glycan anchor biosynthesis, class Y
231%	TRPA1	8989	transient receptor potential cation channel, subfamily A, member 1
222%	ADM	133	adrenomedullin
167%	POLR3G	10622	polymerase (RNA) III (DNA directed) polypeptide G (32kD)
145%	CPNE6	9362	copine VI (neuronal)
221%	PNKD	25953	paroxysmal nonkinesiogenic dyskinesia
153%	SSPN	8082	sarcospan (Kras oncogene-associated gene)
114%	TRHR	7201	thyrotropin-releasing hormone receptor
133%	CDKN2D	1032	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4)
153%	USP54	159195	ubiquitin specific peptidase 54
219%	GPX2	2877	glutathione peroxidase 2 (gastrointestinal)

Figure 15 (Continued)

viability	Gene Symbol	Gene ID	Gene Name
153%	NUPR1	26471	nuclear protein 1
226%	PA2G4	5036	proliferation-associated 2G4, 38kDa
137%	UTS2R	2837	urotensin 2 receptor
145%	GTPBP4	23560	GTP binding protein 4
167%	STAT3	6774	signal transducer and activator of transcription 3 (acute-phase response factor)
201%	SORCS2	57537	sortilin-related VPS10 domain containing receptor 2
259%	TRNT1	51095	tRNA nucleotidyl transferase, CCA-adding, 1
166%	EGLN2	112398	egl nine homolog 2 (C. elegans)
210%	BGN	633	biglycan
216%	PPARD	5467	peroxisome proliferator-activated receptor delta
149%	RNPEPL1	57140	arginyl aminopeptidase (aminopeptidase B)-like 1
199%	FCER1A	2205	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide
199%	PTMA	5757	prothymosin, alpha (gene sequence 28)
156%	RAGE	5891	renal tumor antigen
203%	PTCRA	171558	pre T-cell antigen receptor alpha
140%	GNG5	2787	guanine nucleotide binding protein (G protein), gamma 5
122%	CHKA	1119	choline kinase alpha
155%	RFX1	5989	regulatory factor X, 1 (influences HLA class II expression)
263%	HLA-DRB1	3123	major histocompatibility complex, class II, DR beta 1
161%	DOCK8	81704	dedicator of cytokinesis 8
160%	INTS4	92105	integrator complex subunit 4
163%	WFDC2	10406	WAP four-disulfide core domain 2
216%	SETDB1	9869	SET domain, bifurcated 1
172%	SOD1	6647	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult))
201%	FBXL20	84961	F-box and leucine-rich repeat protein 20
183%	NOXO1	124056	NADPH oxidase organizer 1
165%	TACR2	6865	tachykinin receptor 2
169%	TAF2	6873	TAF2 RNA polymerase II, TATA box binding protein (TBP)-associated factor, 150kDa
208%	CCT4	10575	chaperonin containing TCP1, subunit 4 (delta)
129%	PRKCZ	5590	protein kinase C; zeta
225%	RORC	6097	RAR-related orphan receptor C
144%	DBX1	120237	developing brain homeobox 1
217%	USP24	23358	ubiquitin specific peptidase 24
219%	CHRND	1144	cholinergic receptor, nicotinic, delta

Figure 15 (Continued)

viability	Gene Symbol	Gene ID	Gene Name
164%	COX5A	9377	cytochrome c oxidase subunit Va
169%	EPHA6	203806	EPH receptor A6
152%	CARKL	23729	carbohydrate kinase-like
164%	HNRPM	4670	heterogeneous nuclear ribonucleoprotein M
136%	F12	2161	coagulation factor XII (Hageman factor)
212%	THBS2	7058	thrombospondin 2
157%	CYP27A1	1593	cytochrome P450, family 27, subfamily A, polypeptide 1
196%	MYL3	4634	myosin, light chain 3, alkali; ventricular, skeletal, slow
247%	TNF	7124	tumor necrosis factor (TNF superfamily, member 2)
241%	TRIP6	7205	thyroid hormone receptor interactor 6
216%	FABP1	2168	fatty acid binding protein 1, liver
211%	QSCN6	5768	quiescin Q6
167%	COPE	11316	coatomer protein complex, subunit epsilon
172%	ATG16L2	89849	ATG16 autophagy related 16-like 2 (S. cerevisiae)
168%	PRAF2	11230	PRA1 domain family, member 2
211%	PCGF1	84759	polycomb group ring finger 1
189%	GFER	2671	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae)
199%	FANCC	2176	Fanconi anemia, complementation group C
167%	LOC388959	388959	hypothetical LOC388959
169%	HIST2H3C	126961	histone cluster 2, H3c
165%	TSPAN4	7106	tetraspanin 4
213%	FGF2	2247	fibroblast growth factor 2 (basic)
213%	CENPJ	55835	centromere protein J
173%	TRIM69	140691	tripartite motif-containing 69
253%	UNC13B	10497	unc-13 homolog B (C. elegans)
197%	COL14A1	7373	collagen, type XIV, alpha 1 (undulin)
205%	TOM1	10043	target of myb1 (chicken)
154%	PROSC	11212	proline synthetase co-transcribed homolog (bacterial)
152%	TACC2	10579	transforming, acidic coiled-coil containing protein 2
160%	C2orf13	200558	chromosome 2 open reading frame 13
230%	HSFY2	159119	heat shock transcription factor, Y linked 2
158%	FGFR1	2260	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome)
160%	PAQR6	79957	progestin and adipoQ receptor family member VI
195%	BPGM	669	2,3-bisphosphoglycerate mutase

Figure 15 (Continued)

viability	Gene Symbol	Gene ID	Gene Name
171%	KIAA0196	9897	KIAA0196
161%	BAI3	577	brain-specific angiogenesis inhibitor 3
172%	SCAMP4	113178	secretory carrier membrane protein 4
192%	C18orf8	29919	chromosome 18 open reading frame 8
166%	MKLN1	4289	muskelin 1, intracellular mediator containing kelch motifs
188%	MUC3A	4584	mucin 3A, cell surface associated
212%	SLC25A19	60386	solute carrier family 25 (mitochondrial thiamine pyrophosphate carrier), member 19
179%	UBE1L2	55236	ubiquitin-activating enzyme E1-like 2
212%	ARSE	415	arylsulfatase E (chondrodysplasia punctata 1)
216%	UBE2D1	7321	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
205%	TRIM8	81603	tripartite motif-containing 8
149%	ICT1	3396	immature colon carcinoma transcript 1
197%	ERH	2079	enhancer of rudimentary homolog (Drosophila)
239%	NPTN	27020	neuroplastin
153%	GNG11	2791	guanine nucleotide binding protein (G protein), gamma 11
155%	TUBGCP6	85378	tubulin, gamma complex associated protein 6
166%	SIDT1	54847	SID1 transmembrane family, member 1
211%	MATN3	4148	matrilin 3
236%	LOR	4014	loricrin
212%	BAIAP2	10458	BAI1-associated protein 2
197%	SORBS3	10174	sorbin and SH3 domain containing 3
218%	USP19	10869	ubiquitin specific peptidase 19

Figure 16.

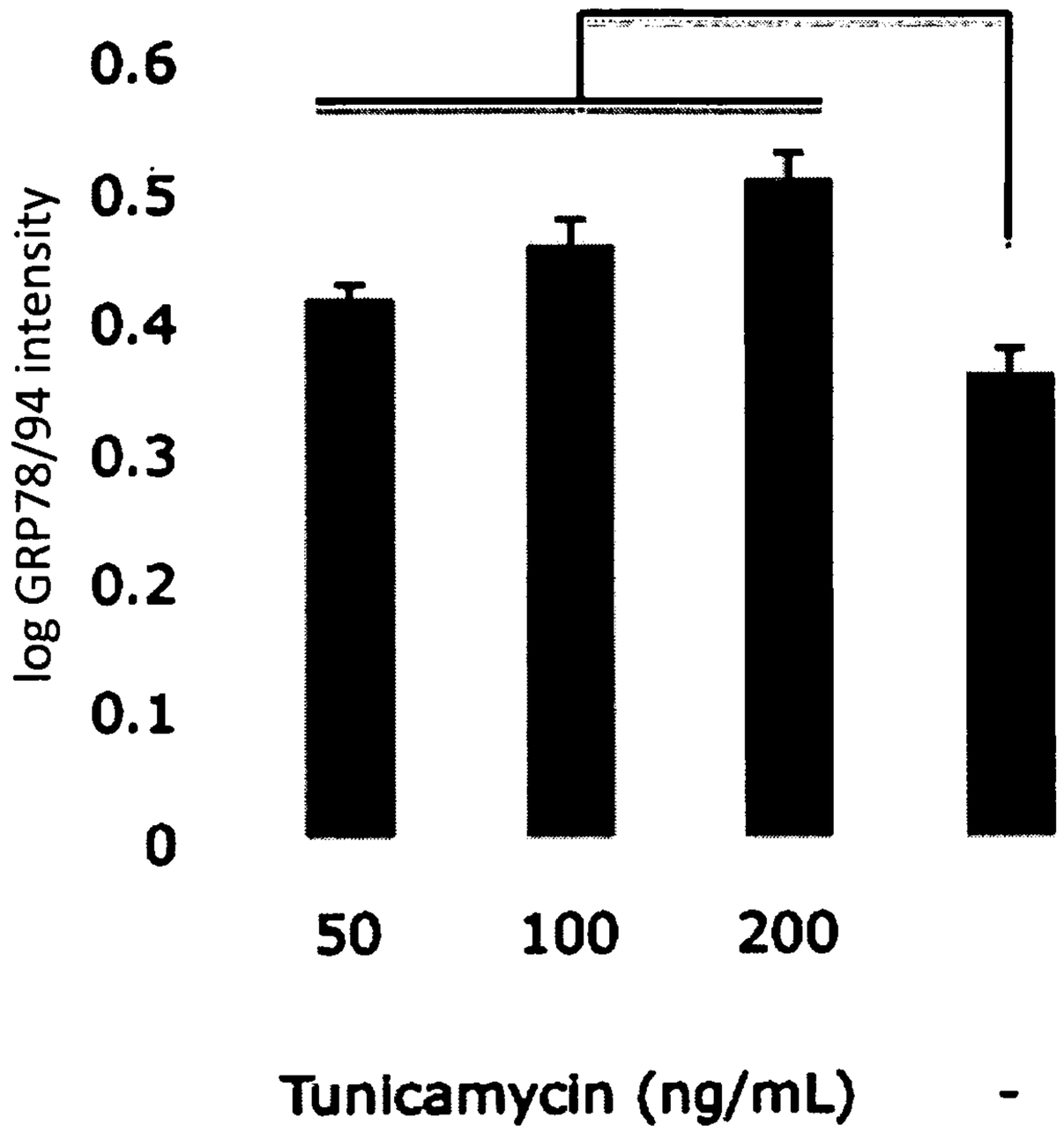

**
0.6
0.5
0.4
0.3
0.2
0.1
0
log GRP78/94 intensity
50
100
200
Tunicamycin (ng/mL)
-

Figure 17.

Genes whose knock-down leads to induction of autophagy and changes in ER stress levels.

Gene Symbol	Gene ID	Gene Name
SIDT1	54847	SID1 transmembrane family, member 1
C2orf13	200558	chromosome 2 open reading frame 13
DDX24	57062	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
HIST2H3C	126961	histone cluster 2, H3c
MMP17	4326	matrix metallopeptidase 17 (membrane-inserted)
SCAMP4	113178	secretory carrier membrane protein 4
PPARD	5467	peroxisome proliferator-activated receptor delta
COX5A	9377	cytochrome c oxidase subunit Va
NUDT1	4521	nudix (nucleoside diphosphate linked moiety X)-type motif 1
KIF11	3832	kinesin family member 11

Figure 18.

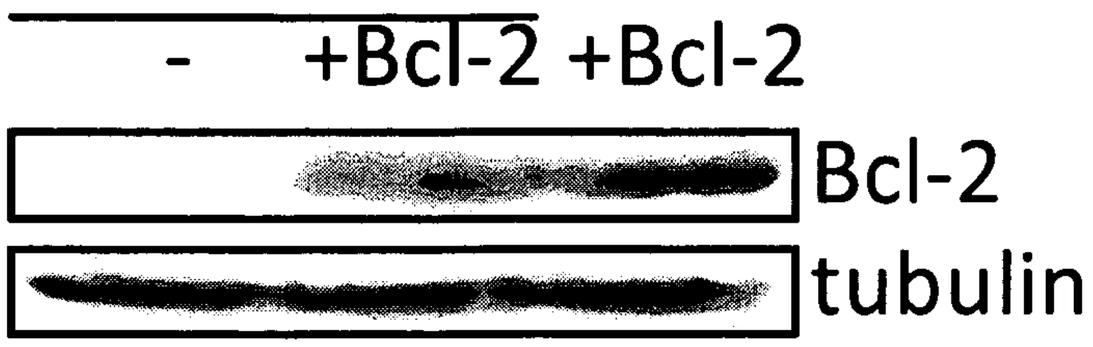
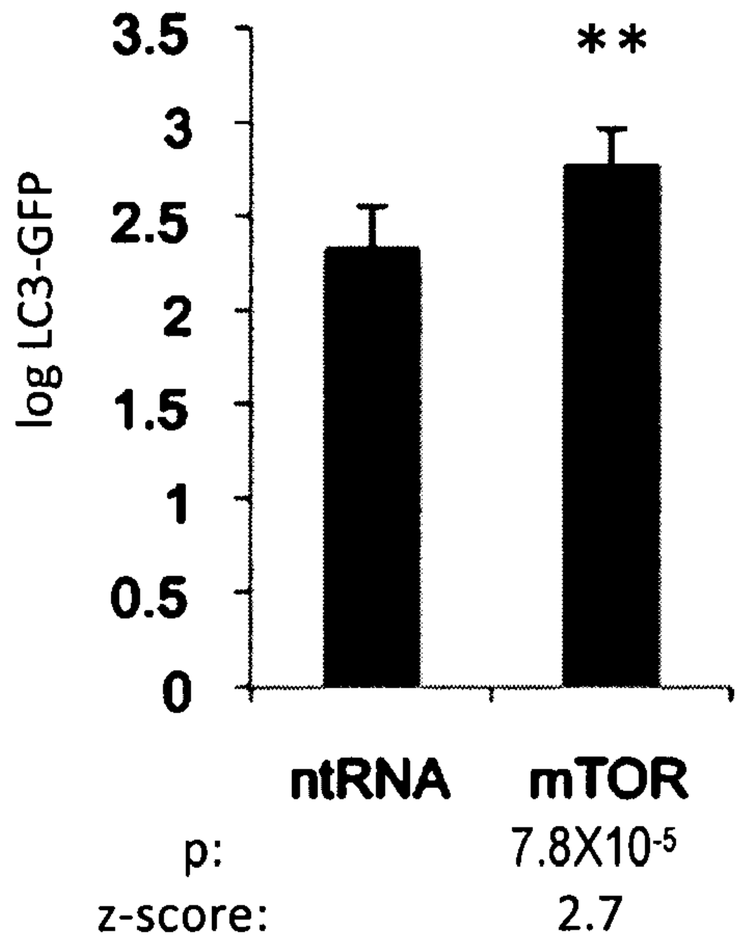


Figure 19.

A.


**
3.5
3
2.5
2
1.5
1
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0
log LC3-GFP
ntRNA
mTOR
p:
7.8X10-5
z-score:
2.7

B.

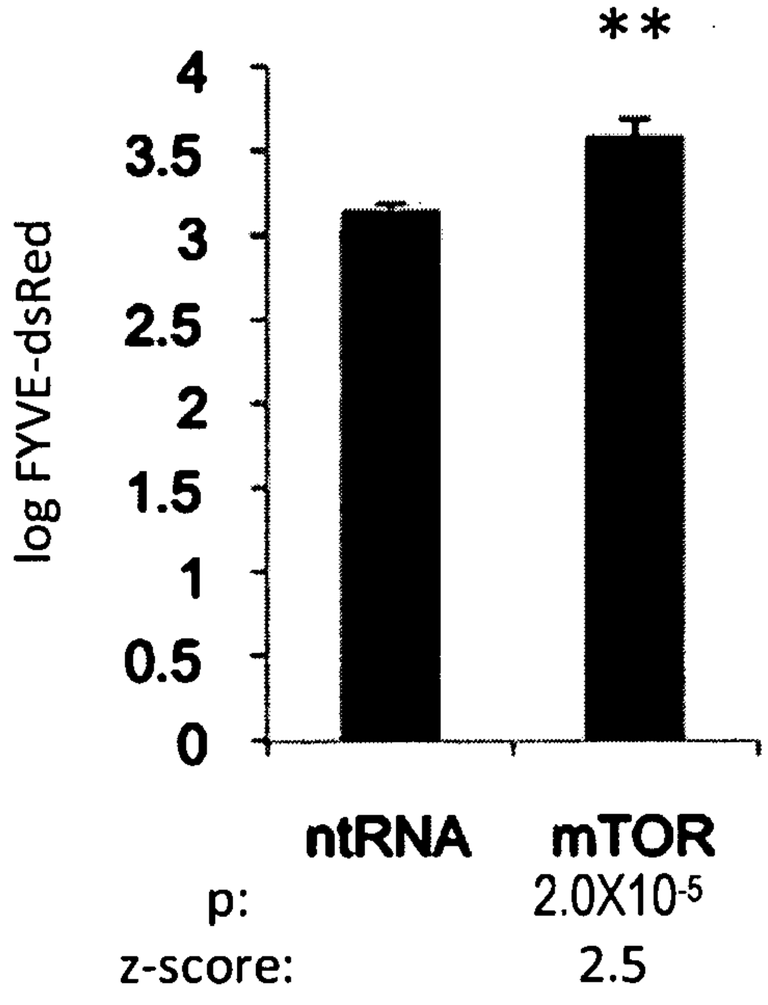

**
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2.5
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1.5
1
0.5
0
log FYVE-dsRed
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mTOR
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Figure 19 (Continued)

C.



Figure 20.

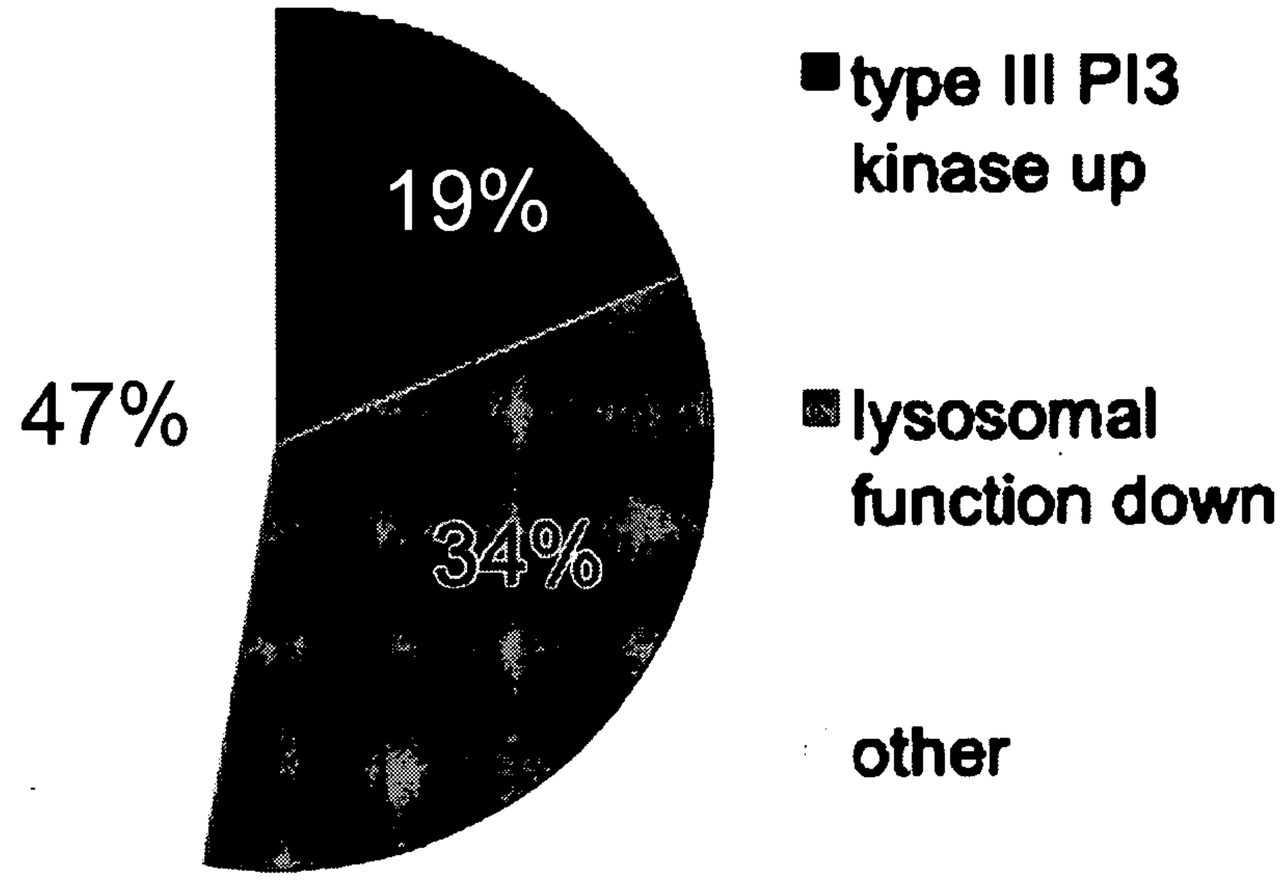
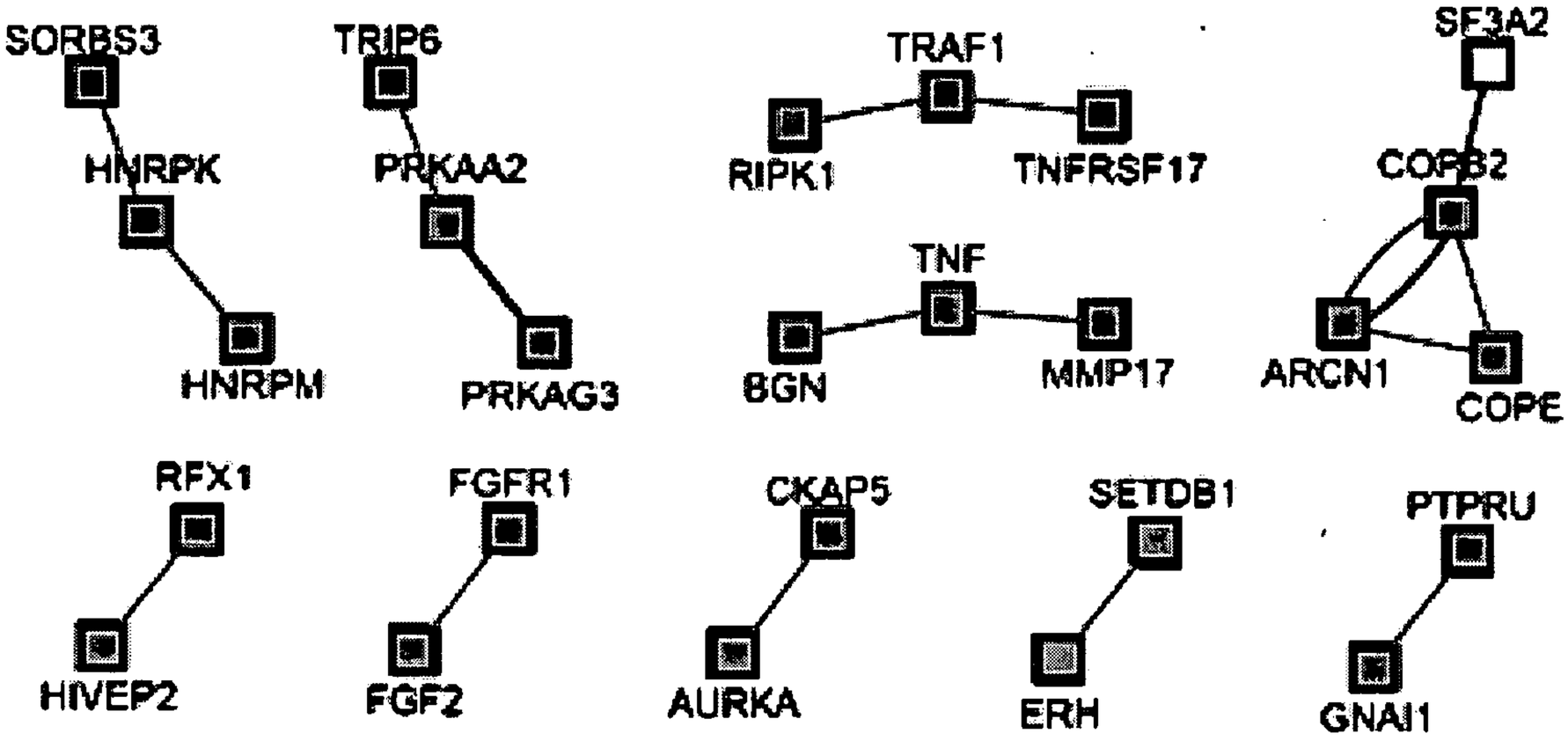

19%
47%
34%
type III PI3 kinase up
lysosomal function down
other

Figure 21.

A.


SORBS3
HNRPK
HNRPM
TRIP6
PRKAA2
PRKAG3
TRAF1
RIPK1
TNFRSF17
SF3A2
COPB2
TNF
BGN
MMP17
ARCN1
COPE
RFX1
HIVEP2
FGFR1
FGF2
CKAP5
AURKA
SETDB1
ERH
PTPRU
GNAI1

B.

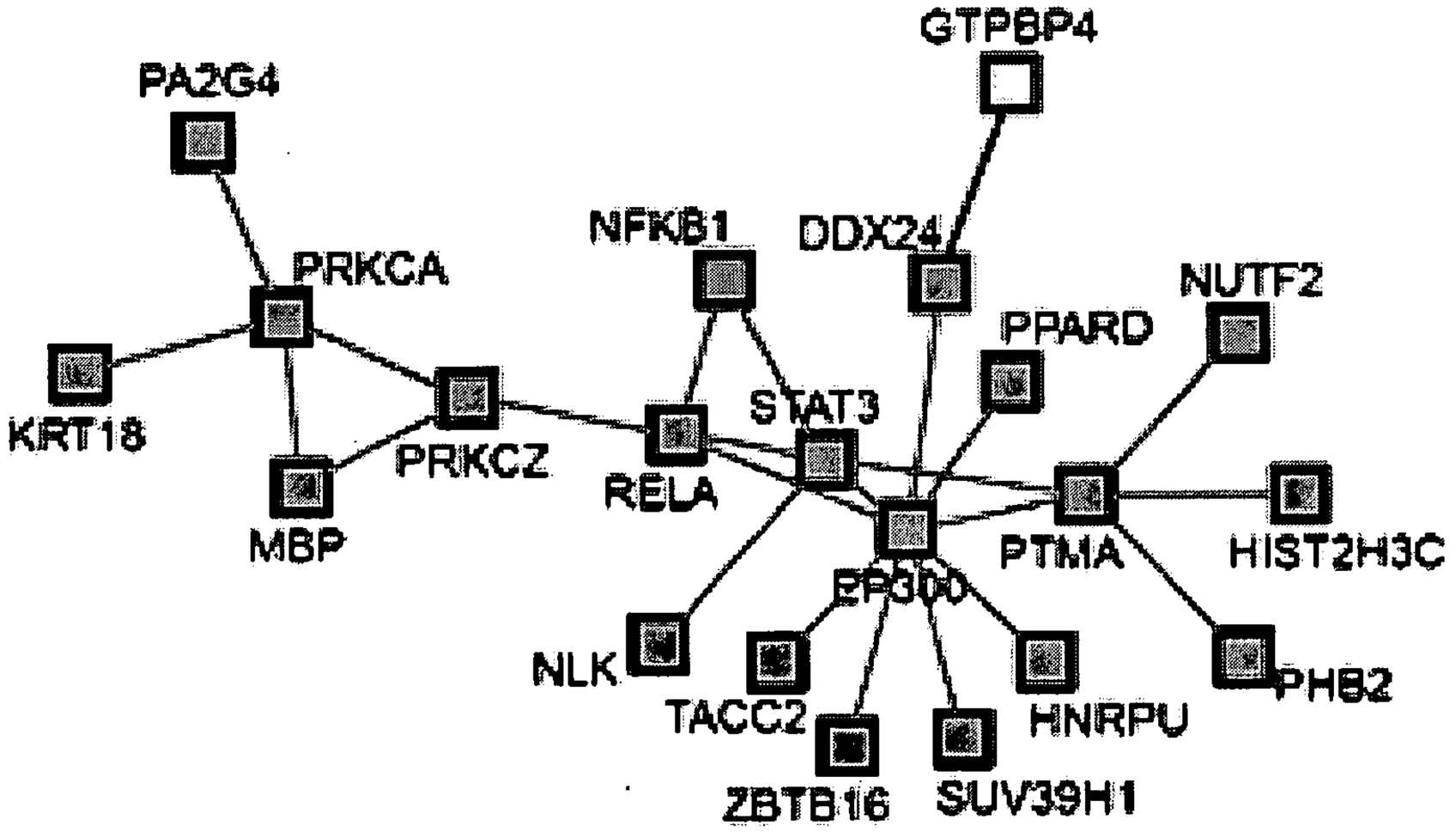

GTPBP4
PA2G4
PRKCA
KRT18
MBP
PRKCZ
NFKB1
RELA
DDX24
STAT3
PPARD
NUTF2
PTMA
HIST2H3C
EP300
NLK
TACC2
ZBTB16
SUV39H1
HNRPU
PHB2

Figure 22

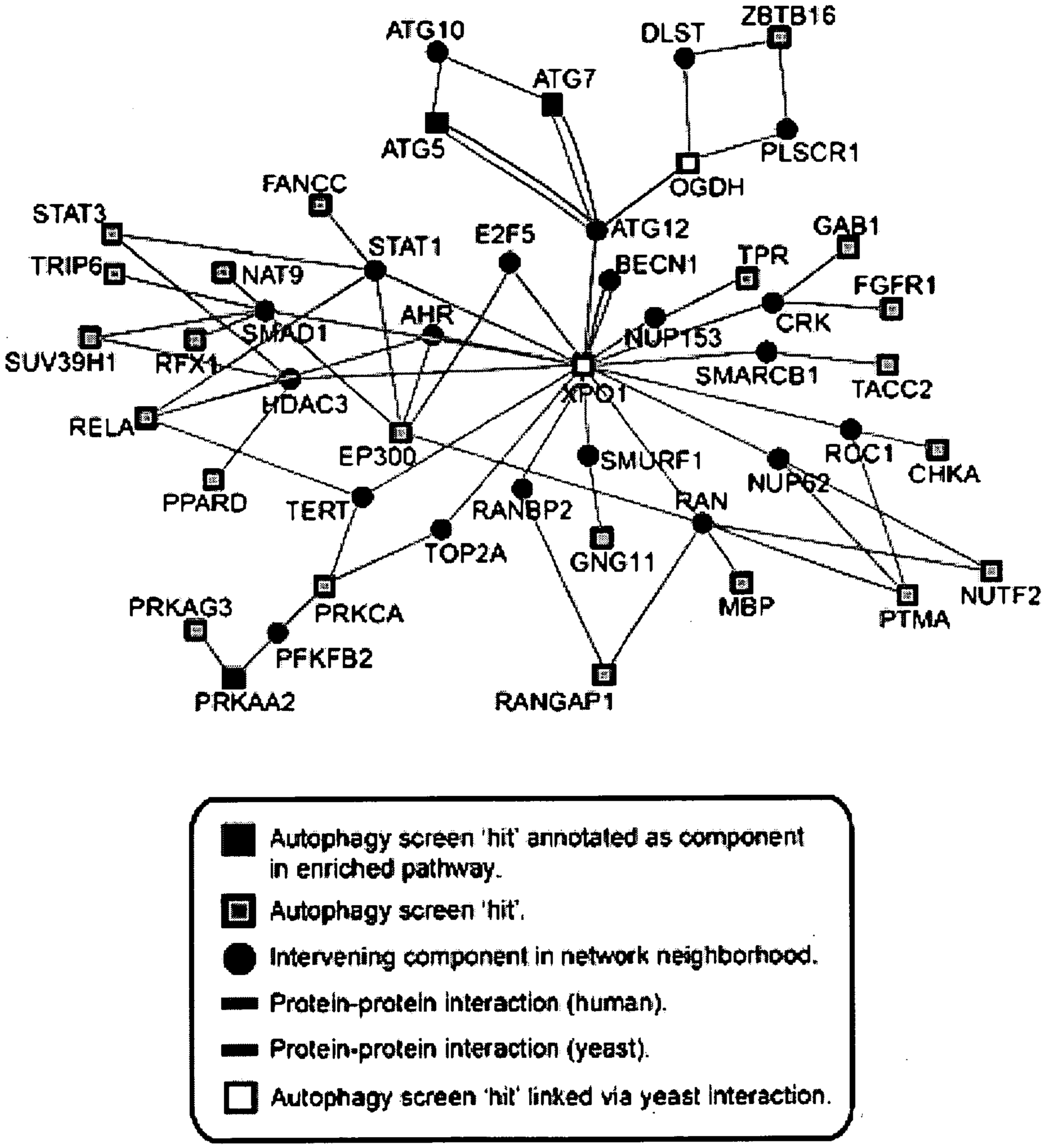

ATG10
ATG7
ATG5
FANCC
STAT3
STAT1
E2F5
ATG12
TRIP6
NAT9
BECN1
SMAD1
AHR
SUV39H1
RFX1
NUP153
HDAC3
XPO1
RELA
EP300
SMURF1
PPARD
TERT
RANBP2
RAN
TOP2A
GNG11
PRKAG3
PRKCA
PFKFB2
PRKAA2
RANGAP1
MBP
PTMA
NUTF2
DLST
ZBTB16
PLSCR1
OGDH
GAB1
TPR
FGFR1
CRK
SMARCB1
TACC2
ROC1
CHKA
NUP62
Autophagy screen 'hit' annotated as component in enriched pathway.
Autophagy screen 'hit'.
Intervening component in network neighborhood.
Protein-protein interaction (human).
Protein-protein interaction (yeast).
Autophagy screen 'hit' linked via yeast interaction.

Figure 23

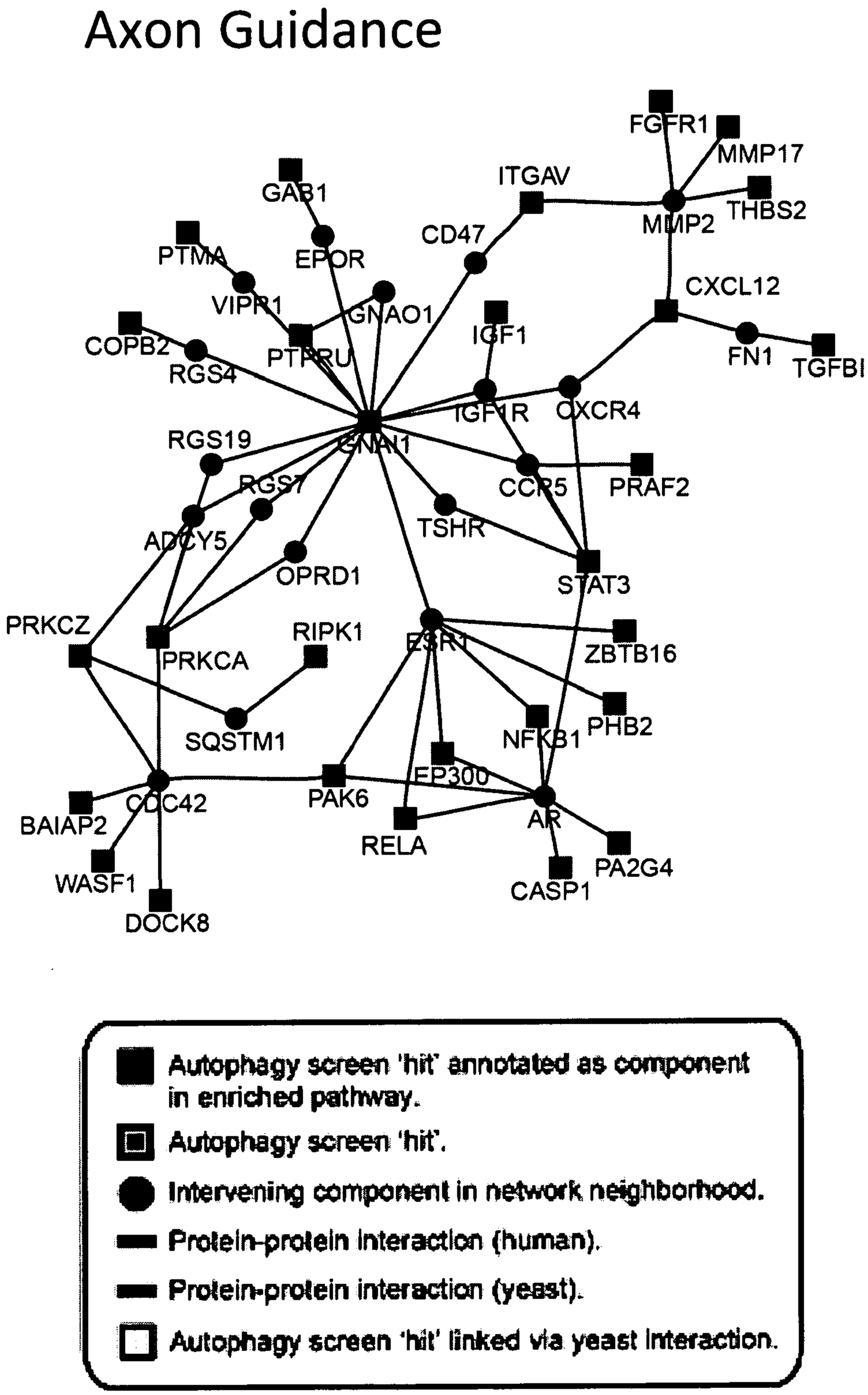

Axon Guidance
FGFR1
MMP17
ITGAV
GAB1
THBS2
MMP2
CD47
PTMA
EPOR
CXCL12
VIPR1
GNAO1
IGF1
COPB2
PTPRU
FN1
TGFBI
RGS4
IGF1R
CXCR4
RGS19
GNAI1
CCR5
PRAF2
RGS7
TSHR
ADCY5
OPRD1
STAT3
PRKCZ
RIPK1
ESR1
ZBTB16
PRKCA
PHB2
SQSTM1
NFKB1
EP300
CDC42
PAK6
BAIAP2
AR
RELA
PA2G4
WASF1
CASP1
DOCK8
Autophagy screen 'hit' annotated as component in enriched pathway.
Autophagy screen 'hit'.
Intervening component in network neighborhood.
Protein-protein interaction (human).
Protein-protein interaction (yeast).
Autophagy screen 'hit' linked via yeast interaction.

Figure 24.

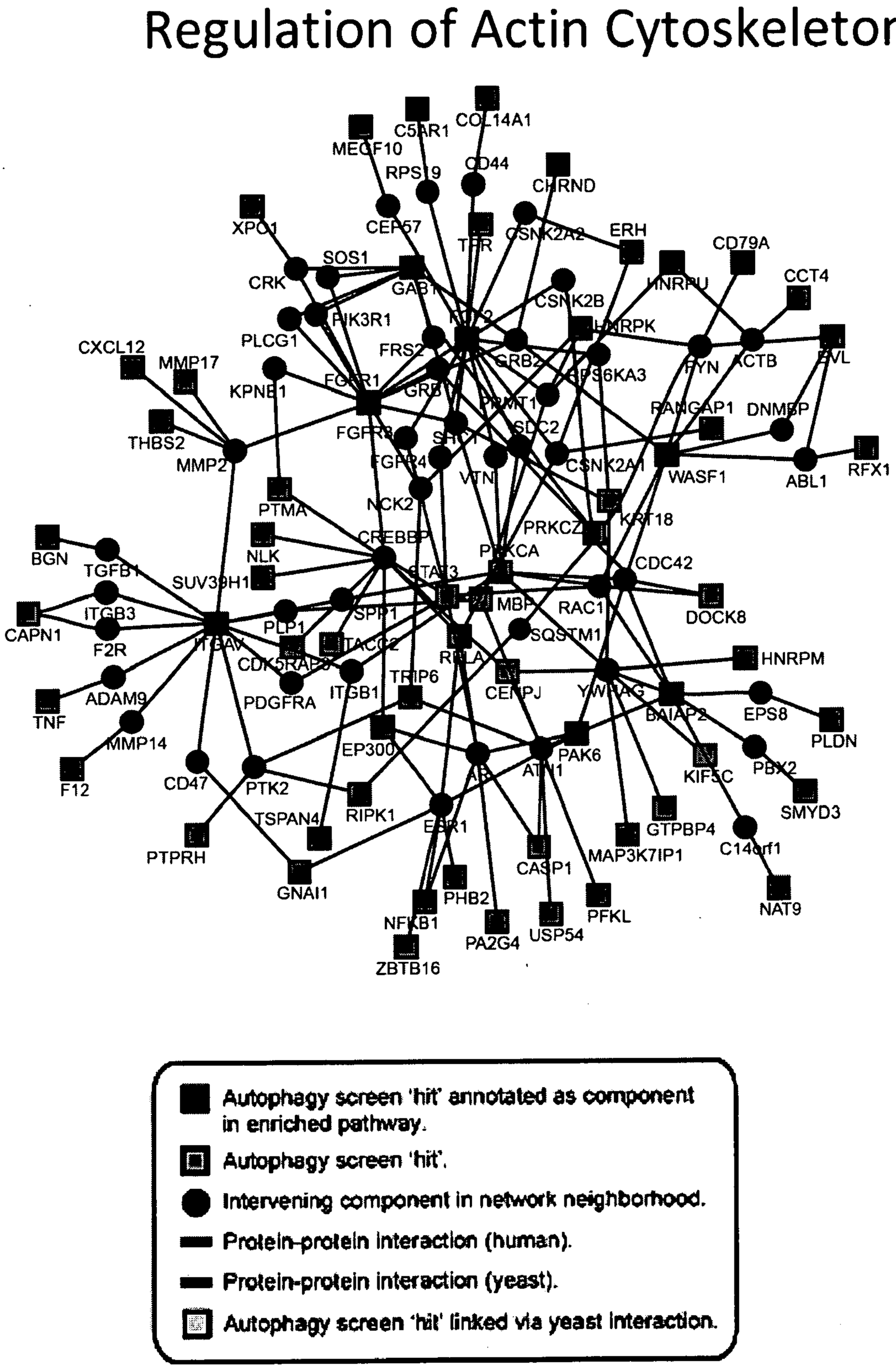
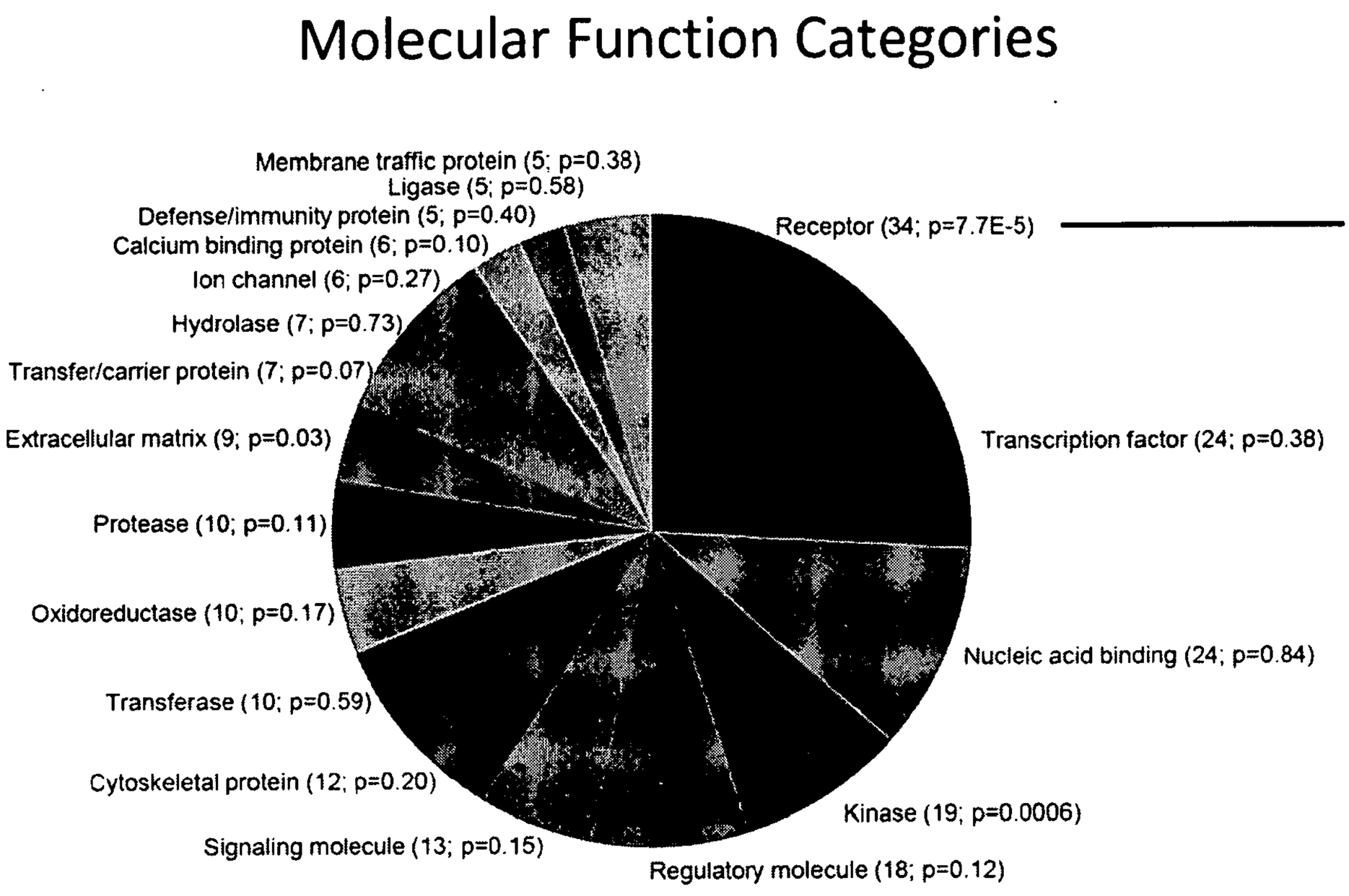

Regulation of Actin Cytoskeleton
C5AR1
COL14A1
MEGF10
RPS19
CD44
CHRND
XPO1
CEP57
TFR
CSNK2A2
ERH
CD79A
SOS1
CRK
GAB1
HNRPU
CCT4
CSNK2B
PIK3R1
HNRPK
PLCG1
FRS2
GRB2
CXCL12
MMP17
RPS6KA3
PYN
ACTB
EVL
KPNB1
FGFR1
PRMT1
RANGAP1
DNMBP
THBS2
FGFR3
SDC2
MMP2
FGFR4
CSNK2A1
WASF1
ABL1
RFX1
VTN
PTMA
NCK2
KRT18
PRKCZ
CREBBP
BGN
NLK
PRKCA
TGFB1
CDC42
SUV39H1
STAT3
MBP
RAC1
DOCK8
ITGB3
SPP1
CAPN1
PLP1
SQSTM1
F2R
ITGAV
TACC2
HNRPM
CDK5RAP3
RHOA
TRIP6
ADAM9
ITGB1
CEP1PJ
YWHAG
PDGFRA
BAIAP2
EPS8
TNF
PLDN
MMP14
EP300
PAK6
KIF5C
PBX2
F12
CD47
PTK2
ATM1
SMYD3
RIPK1
TSPAN4
GTPBP4
PTPRH
MAP3K7IP1
C14orf1
GNAI1
CASP1
PHB2
PFKL
NAT9
NFKB1
USP54
PA2G4
ZBTB16
Autophagy screen 'hit' annotated as component in enriched pathway.
Autophagy screen 'hit'.
Intervening component in network neighborhood.
Protein-protein interaction (human).
Protein-protein interaction (yeast).
Autophagy screen 'hit' linked via yeast interaction.

Figure 25

A.


Molecular Function Categories
Membrane traffic protein (5; p=0.38)
Ligase (5; p=0.58)
Defense/immunity protein (5; p=0.40)
Calcium binding protein (6; p=0.10)
Ion channel (6; p=0.27)
Hydrolase (7; p=0.73)
Transfer/carrier protein (7; p=0.07)
Extracellular matrix (9; p=0.03)
Protease (10; p=0.11)
Oxidoreductase (10; p=0.17)
Transferase (10; p=0.59)
Cytoskeletal protein (12; p=0.20)
Signaling molecule (13; p=0.15)
Receptor (34; p=7.7E-5)
Transcription factor (24; p=0.38)
Nucleic acid binding (24; p=0.84)
Kinase (19; p=0.0006)
Regulatory molecule (18; p=0.12)

B.

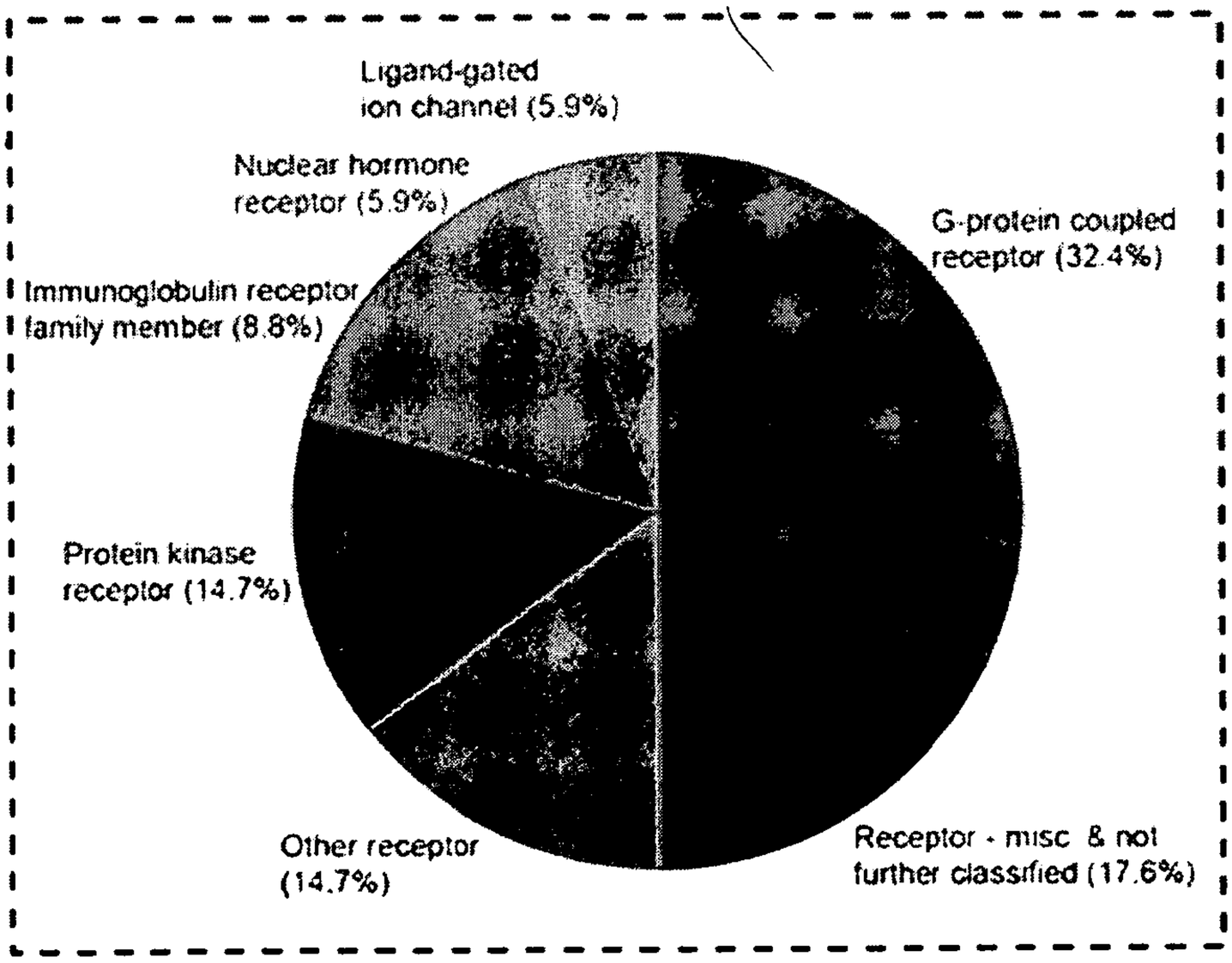

Ligand-gated
ion channel (5.9%)
Nuclear hormone
receptor (5.9%)
Immunoglobulin receptor
family member (8.8%)
Protein kinase
receptor (14.7%)
Other receptor
(14.7%)
G-protein coupled
receptor (32.4%)
Receptor - misc & not
further classified (17.6%)

Figure 26.

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Cell adhesion molecule	ITGAV	3685	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51);ITGAV
Cell adhesion molecule	MEGF10	84466	multiple EGF-like-domains 10;MEGF10
Cell adhesion molecule	TGFBI	7045	transforming growth factor, beta-induced, 68kDa;TGFBI
Cell adhesion molecule	TSPAN4	7106	tetraspanin 4;TSPAN4
Cell junction protein	GJA3	2700	gap junction protein, alpha 3, 46kDa (connexin 46);GJA3
Cell junction protein	TRIP6	7205	thyroid hormone receptor interactor 6;TRIP6
Cell junction protein	GJA4	2701	gap junction protein, alpha 4, 37kDa (connexin 37);GJA4
Chaperone	CCT4	10575	chaperonin containing TCP1, subunit 4 (delta);CCT4
Chaperone	PDCL	5082	phosducin-like;PDCL
Chaperone	PFDN2	5202	prefoldin subunit 2;PFDN2
Cytoskeletal protein	CKAP5	9793	cytoskeleton associated protein 5;CKAP5
Cytoskeletal protein	WASF1	8936	WAS protein family, member 1;WASF1
Cytoskeletal protein	SORBS3	10174	sorbin and SH3 domain containing 3;SORBS3
Cytoskeletal protein	TRIP6	7205	thyroid hormone receptor interactor 6;TRIP6
Cytoskeletal protein	KRT18	3875	keratin 18;KRT18
Cytoskeletal protein	TUBGCP6	85378	tubulin, gamma complex associated protein 6;TUBGCP6.
Cytoskeletal protein	LOC285643	285643	KIF4B;unassigned
Cytoskeletal protein	KIF11	3832	kinesin family member 11;KIF11
Cytoskeletal protein	CENPE	1062	centromere protein E, 312kDa;CENPE
Cytoskeletal protein	KIF5C	3800	kinesin family member 5C;KIF5C
Cytoskeletal protein	TUBB2A	7280	tubulin, beta 2A;TUBB2A
Cytoskeletal protein	MYL3	4634	myosin, light chain 3, alkali; ventricular, skeletal, slow;MYL3
Defense/Immunity protein	AGER	177	advanced glycosylation end product-specific receptor;AGER
Defense/Immunity protein	NPTX1	4884	neuronal pentraxin I;NPTX1
Defense/Immunity protein	HLA-DRB1	3123	major histocompatibility complex, class II, DR beta 1;HLA-DRB1
Defense/Immunity protein	FCER1A	2205	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide;FCER1A
Defense/Immunity protein	CD300C	10871	CD300c molecule;CD300C
Extracellular matrix	BGN	633	biglycan;BGN
Extracellular matrix	COL14A1	7373	collagen, type XIV, alpha 1 (undulin);COL14A1
Extracellular matrix	MEGF10	84466	multiple EGF-like-domains 10;MEGF10
Extracellular matrix	MMP10	4319	matrix metallopeptidase 10 (stromelysin 2);MMP10
Extracellular matrix	NTN2L	4917	netrin 2-like (chicken);NTN2L
Extracellular matrix	MATN3	4148	matrilin 3;MATN3

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Extracellular matrix	TLR3	7098	toll-like receptor 3;TLR3
Extracellular matrix	THBS2	7058	thrombospondin 2;THBS2
Extracellular matrix	MMP17	4326	matrix metallopeptidase 17 (membrane-inserted);MMP17
Hydrolase	CHID1	66005	chitinase domain containing 1;CHID1
Hydrolase	RNPEPL1	57140	arginyl aminopeptidase (aminopeptidase B)-like 1;RNPEPL1
Hydrolase	ARSE	415	arylsulfatase E (chondrodysplasia punctata 1);ARSE
Hydrolase	PLCH2	9651	phospholipase C, eta 2;PLCH2
Hydrolase	HMBS	3145	hydroxymethylbilane synthase;HMBS
Hydrolase	PNKD	25953	paroxysmal nonkinesiogenic dyskinesia;PNKD
Hydrolase	NUDT1	4521	nudix (nucleoside diphosphate linked moiety X)-type motif 1;NUDT1
Ion channel	TRPM3	80036	transient receptor potential cation channel, subfamily M, member 3;TRPM3
Ion channel	CHRND	1144	cholinergic receptor, nicotinic, delta;CHRND
Ion channel	CLCN1	1180	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant);CLCN1
Ion channel	P2RX1	5023	purinergic receptor P2X, ligand-gated ion channel, 1;P2RX1
Ion channel	TRPA1	8989	transient receptor potential cation channel, subfamily A, member 1;TRPA1
Ion channel	CATSPER4	378807	cation channel, sperm associated 4;CATSPER4
Isomerase	BPGM	669	2,3-bisphosphoglycerate mutase;BPGM
Kinase	IRAK3	11213	IRAK3
Kinase	FGFR1	2260	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1
Kinase	PAK6	56924	p21(CDKN1A)-activated kinase 6;PAK6
Kinase	CHKA	1119	choline kinase alpha;CHKA
Kinase	RAGE	5891	renal tumor antigen;RAGE
Kinase	IHPK3	117283	inositol hexaphosphate kinase 3;IHPK3
Kinase	TPR	7175	translocated promoter region (to activated MET oncogene);TPR
Kinase	CAMKV	79012	CaM kinase-like vesicle-associated;CAMKV
Kinase	PRKAA2	5563	protein kinase, AMP-activated, alpha 2 catalytic subunit;PRKAA2
Kinase	PRKCZ	5590	protein kinase C, zeta;PRKCZ
Kinase	PRKCA	5578	protein kinase C, alpha;PRKCA
Kinase	CARKL	23729	carbohydrate kinase-like;CARKL
Kinase	EPHA6	285220	EPH receptor A6;EPHA6
Kinase	PFKL	5211	phosphofructokinase, liver;PFKL
Kinase	GRK6	2870	G protein-coupled receptor kinase 6;GRK6
Kinase	NLK	51701	nemo-like kinase;NLK

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Kinase	AURKA	6790	aurora kinase A;AURKA
Kinase	RIPK1	8737	receptor (TNFRSF)-interacting serine-threonine kinase 1;RIPK1
Kinase	PLXNA2	5362	plexin A2;PLXNA2
Ligase	TRIM8	81603	tripartite motif-containing 8;TRIM8
Ligase	UBE1L2	55236	ubiquitin-activating enzyme E1-like 2;UBE1L2
Ligase	MCCC1	56922	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha);MCCC1
Ligase	TRIM69	140691	tripartite motif-containing 69;TRIM69
Ligase	UBE2D1	7321	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast);UBE2D1
Lyase	HMGCL	3155	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria);HMGCL
Membrane traffic protein	COPB2	9276	coatomer protein complex, subunit beta 2 (beta prime);COPB2
Membrane traffic protein	ARCN1	372	archain 1;ARCN1
Membrane traffic protein	CPNE6	9362	copine VI (neuronal);CPNE6
Membrane traffic protein	COPE	11316	coatomer protein complex, subunit epsilon;COPE
Membrane traffic protein	ATG5	9474	ATG5 autophagy related 5 homolog (S. cerevisiae);ATG5
Nucleic acid binding	HSFY2	159119	heat shock transcription factor, Y linked 2;HSFY2
Nucleic acid binding	CHAF1B	8208	chromatin assembly factor 1, subunit B (p60);CHAF1B
Nucleic acid binding	DDX24	57062	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24;DDX24
Nucleic acid binding	HNRPK	3190	heterogeneous nuclear ribonucleoprotein K;HNRPK
Nucleic acid binding	PPARD	5467	peroxisome proliferator-activated receptor delta;PPARD
Nucleic acid binding	SP140	11262	SP140 nuclear body protein;SP140
Nucleic acid binding	RELA	5970	v-rel reticuloendotheliosis viral oncogene homolog A, nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, p65 (avian);RELA
Nucleic acid binding	EP300	2033	E1A binding protein p300;EP300
Nucleic acid binding	SETDB1	9869	SET domain, bifurcated 1;SETDB1
Nucleic acid binding	GCM2	9247	glial cells missing homolog 2 (Drosophila);GCM2
Nucleic acid binding	HIVEP2	3097	human immunodeficiency virus type I enhancer binding protein 2;HIVEP2
Nucleic acid binding	HNRPU	3192	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A);HNRPU
Nucleic acid binding	HNRPM	4670	heterogeneous nuclear ribonucleoprotein M;HNRPM

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Nucleic acid binding	SUV39H1	6839	suppressor of variegation 3-9 homolog 1 (Drosophila);SUV39H1
Nucleic acid binding	PA2G4	5036	proliferation-associated 2G4, 38kDa;PA2G4
Nucleic acid binding	STAT3	6774	signal transducer and activator of transcription 3 (acute-phase response factor);STAT3
Nucleic acid binding	RORC	6097	RAR-related orphan receptor C;RORC
Nucleic acid binding	SMARCD1	6602	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1;SMARCD1
Nucleic acid binding	SF3A2	8175	SF3A2
Nucleic acid binding	HIST2H3C	126961	histone cluster 2, H3c;HIST2H3C
Nucleic acid binding	POLR3G	10622	polymerase (RNA) III (DNA directed) polypeptide G (32kD);POLR3G
Nucleic acid binding	DBX1	120237	developing brain homeobox 1;unassigned
Nucleic acid binding	HOXD11	3237	homeobox D11;HOXD11
Nucleic acid binding	ICT1	3396	ICT1
Oxidoreductase	CYP27A1	1593	cytochrome P450, family 27, subfamily A, polypeptide 1;CYP27A1
Oxidoreductase	SDHB	6390	succinate dehydrogenase complex, subunit B, iron sulfur (Ip);SDHB
Oxidoreductase	OGDH	4967	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide);OGDH
Oxidoreductase	QSCN6	5768	quiescin Q6;QSCN6
Oxidoreductase	EGLN2	112398	egl nine homolog 2 (C. elegans);EGLN2
Oxidoreductase	TH	7054	tyrosine hydroxylase;TH
Oxidoreductase	COX5A	9377	cytochrome c oxidase subunit Va;COX5A
Oxidoreductase	SOD1	6647	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult));SOD1
Oxidoreductase	GPX2	2877	glutathione peroxidase 2 (gastrointestinal);GPX2
Oxidoreductase	GFER	2671	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae);GFER
Phosphatase	PTPRH	5794	protein tyrosine phosphatase, receptor type, H;PTPRH
Phosphatase	PTPRU	10076	protein tyrosine phosphatase, receptor type, U;PTPRU
Protease	TMPRSS5	80975	transmembrane protease, serine 5 (spinesin);TMPRSS5
Protease	USP24	23358	ubiquitin specific peptidase 24;USP24
Protease	USP19	10869	ubiquitin specific peptidase 19;USP19
Protease	RNPEPL1	57140	arginyl aminopeptidase (aminopeptidase B)-like 1;RNPEPL1
Protease	MMP10	4319	matrix metallopeptidase 10 (stromelysin 2);MMP10
Protease	RELN	5649	reelin;RELN

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Protease	F12	2161	coagulation factor XII (Hageman factor);F12
Protease	CASP1	834	CASP1
Protease	MMP17	4326	matrix metallopeptidase 17 (membrane-inserted);MMP17
Protease	CAPN1	823	calpain 1, (mu/I) large subunit;CAPN1
Receptor	TRPM3	80036	transient receptor potential cation channel, subfamily M, member 3;TRPM3
Receptor	TMPRSS5	80975	transmembrane protease, serine 5 (spinesin);TMPRSS5
Receptor	IRAK3	11213	interleukin-1 receptor-associated kinase 3;IRAK3
Receptor	ADMR	11318	adrenomedullin receptor;ADMR
Receptor	FGFR1	2260	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome);FGFR1
Receptor	UNC13B	10497	unc-13 homolog B (C. elegans);UNC13B
Receptor	PTGER2	5732	prostaglandin E receptor 2 (subtype EP2), 53kDa;PTGER2
Receptor	AGER	177	advanced glycosylation end product-specific receptor;AGER
Receptor	BGN	633	biglycan;BGN
Receptor	GABBR2	9568	gamma-aminobutyric acid (GABA) B receptor, 2;GABBR2
Receptor	PPARD	5467	peroxisome proliferator-activated receptor delta;PPARD
Receptor	TPR	7175	translocated promoter region (to activated MET oncogene);TPR
Receptor	GHSR	2693	growth hormone secretagogue receptor;GHSR
Receptor	BAIAP2	10458	BAI1-associated protein 2;BAIAP2
Receptor	SORCS2	57537	sortilin-related VPS10 domain containing receptor 2;SORCS2
Receptor	PAQR6	79957	progestin and adipoQ receptor family member VI;PAQR6
Receptor	EPHA6	285220	EPH receptor A6;EPHA6
Receptor	TRHR	7201	thyrotropin-releasing hormone receptor;TRHR
Receptor	C5AR1	728	C5AR1
Receptor	GPR18	2841	G protein-coupled receptor 18;GPR18
Receptor	BAI3	577	brain-specific angiogenesis inhibitor 3;BAI3
Receptor	TLR3	7098	toll-like receptor 3;TLR3
Receptor	PTPRH	5794	protein tyrosine phosphatase, receptor type, H;PTPRH
Receptor	ADRA1A	148	adrenergic, alpha-1A-, receptor;ADRA1A
Receptor	UTS2R	2837	urotensin 2 receptor;UTS2R
Receptor	RORC	6097	RAR-related orphan receptor C;RORC
Receptor	CHRND	1144	cholinergic receptor, nicotinic, delta;CHRND

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Receptor	TACR2	6865	tachykinin receptor 2;TACR2
Receptor	P2RX1	5023	purinergic receptor P2X, ligand-gated ion channel, 1;P2RX1
Receptor	PLXNA2	5362	plexin A2;PLXNA2
Receptor	PTPRU	10076	protein tyrosine phosphatase, receptor type, U;PTPRU
Receptor	FCER1A	2205	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide;FCER1A
Receptor	CD300C	10871	CD300c molecule;CD300C
Receptor	TNFRSF19L	84957	tumor necrosis factor receptor superfamily, member 19-like;TNFRSF19L
Calcium binding protein	HRC	3270	histidine rich calcium binding protein;HRC
Calcium binding protein	PRKCZ	5590	protein kinase C, zeta;PRKCZ
Calcium binding protein	PRKCA	5578	protein kinase C, alpha;PRKCA
Calcium binding protein	F12	2161	coagulation factor XII (Hageman factor);F12
Calcium binding protein	PLCH2	9651	phospholipase C, eta 2;PLCH2
Calcium binding protein	MYL3	4634	myosin, light chain 3, alkali; ventricular, skeletal, slow;MYL3
Regulatory molecule	RANGAP1	5905	Ran GTPase activating protein 1;RANGAP1
Regulatory molecule	GTPBP4	23560	GTP binding protein 4;GTPBP4
Regulatory molecule	PDCL	5082	phosducin-like;PDCL
Regulatory molecule	TRIP6	7205	thyroid hormone receptor interactor 6;TRIP6
Regulatory molecule	CNKSR2	22866	connector enhancer of kinase suppressor of Ras 2;CNKSR2
Regulatory molecule	PSD	5662	pleckstrin and Sec7 domain containing;PSD
Regulatory molecule	MAP3K7IP1	10454	MAP3K7IP1
Regulatory molecule	DOCK8	81704	dedicator of cytokinesis 8;DOCK8
Regulatory molecule	PRKAG3	53632	protein kinase, AMP-activated, gamma 3 non-catalytic subunit;PRKAG3
Regulatory molecule	THBS2	7058	thrombospondin 2;THBS2
Regulatory molecule	GNAI1	2770	GNAI1
Regulatory molecule	FRAG1	27315	FGF receptor activating protein 1;unassigned
Regulatory molecule	RAB7A	7879	RAB7, member RAS oncogene family;RAB7
Regulatory molecule	CDKN2D	1032	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4);CDKN2D
Regulatory molecule	GNG5	2787	guanine nucleotide binding protein (G protein), gamma 5;GNG5
Regulatory molecule	GNG11	2791	guanine nucleotide binding protein (G protein), gamma 11;GNG11
Regulatory molecule	PDCD5	9141	programmed cell death 5;PDCD5
Regulatory molecule	WFDC2	10406	WAP four-disulfide core domain 2;WFDC2

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Signaling molecule	TNF	7124	tumor necrosis factor (TNF superfamily, member 2);TNF
Signaling molecule	TRAF1	7185	TNF receptor-associated factor 1;TRAF1
Signaling molecule	CLCF1	23529	cardiotrophin-like cytokine factor 1;CLCF1
Signaling molecule	SEMA4B	10509	sema domain, immunoglobulin domain (Ig), transmembrane domain (TM) and short cytoplasmic domain, (semaphorin) 4B;SEMA4B
Signaling molecule	THBS2	7058	thrombospondin 2;THBS2
Signaling molecule	TGFBI	7045	transforming growth factor, beta-induced, 68kDa;TGFBI
Signaling molecule	FGF2	2247	fibroblast growth factor 2 (basic);FGF2
Signaling molecule	LIF	3976	leukemia inhibitory factor (cholinergic differentiation factor);LIF
Signaling molecule	IGF1	3479	insulin-like growth factor 1 (somatomedin C);IGF1
Signaling molecule	TSPAN4	7106	tetraspanin 4;TSPAN4
Signaling molecule	ADM	133	adrenomedullin;ADM
Signaling molecule	GNRH2	2797	gonadotropin-releasing hormone 2;GNRH2
Signaling molecule	AMH	268	anti-Mullerian hormone;AMH
Synthase & synthetase	PROSC	11212	proline synthetase co-transcribed homolog (bacterial);PROSC
Transcription factor	ZFY	7544	zinc finger protein, Y-linked;ZFY
Transcription factor	RFX1	5989	regulatory factor X, 1 (influences HLA class II expression);RFX1
Transcription factor	HSFY2	159119	heat shock transcription factor, Y linked 2;HSFY2
Transcription factor	PPARD	5467	peroxisome proliferator-activated receptor delta;PPARD
Transcription factor	TCEB3	6924	transcription elongation factor B (SIII), polypeptide 3 (110kDa, elongin A);TCEB3
Transcription factor	NFIL3	4783	nuclear factor, interleukin 3 regulated;NFIL3
Transcription factor	PCGF1	84759	polycomb group ring finger 1;PCGF1
Transcription factor	SMYD3	64754	SET and MYND domain containing 3;SMYD3
Transcription factor	RELA	5970	RELA
Transcription factor	RBBP8	5932	retinoblastoma binding protein 8;RBBP8
Transcription factor	EP300	2033	E1A binding protein p300;EP300
Transcription factor	GCM2	9247	glial cells missing homolog 2 (Drosophila);GCM2
Transcription factor	GTF2IRD2	84163	GTF2I repeat domain containing 2;GTF2IRD2
Transcription factor	HIVEP2	3097	human immunodeficiency virus type I enhancer binding protein 2;HIVEP2
Transcription factor	TAF2	6873	TAF2 RNA polymerase II, TATA box binding protein (TBP)-associated factor, 150kDa;TAF2

Figure 26 (Continued)

Molecular Function Category	Gene Symbol	GeneID	Gene Name
Transcription factor	NFKB1	4790	nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 (p105);NFKB1
Transcription factor	PA2G4	5036	proliferation-associated 2G4, 38kDa;PA2G4
Transcription factor	STAT3	6774	signal transducer and activator of transcription 3 (acute-phase response factor);STAT3
Transcription factor	RORC	6097	RAR-related orphan receptor C;RORC
Transcription factor	PLAGL2	5326	pleiomorphic adenoma gene-like 2;PLAGL2
Transcription factor	DBX1	120237	developing brain homeobox 1;unassigned
Transcription factor	ZBTB16	7704	zinc finger and BTB domain containing 16;ZBTB16
Transcription factor	HOXD11	3237	homeobox D11;HOXD11
Transcription factor	ERH	2079	enhancer of rudimentary homolog (Drosophila);ERH
Transfer/carrier protein	SLC25A19	60386	solute carrier family 25 (mitochondrial deoxynucleotide carrier), member 19;SLC25A19
Transfer/carrier protein	SCAMP4	113178	secretory carrier membrane protein 4;SCAMP4
Transfer/carrier protein	KIAA0196	9897	KIAA0196;KIAA0196
Transfer/carrier protein	PRKCZ	5590	protein kinase C, zeta;PRKCZ
Transfer/carrier protein	PRKCA	5578	protein kinase C, alpha;PRKCA
Transfer/carrier protein	ATG7	10533	ATG7 autophagy related 7 homolog (S. cerevisiae);ATG7
Transfer/carrier protein	FABP1	2168	fatty acid binding protein 1, liver;FABP1
Transferase	PAK6	56924	p21(CDKN1A)-activated kinase 6;PAK6
Transferase	TRNT1	51095	tRNA nucleotidyl transferase, CCA-adding, 1;TRNT1
Transferase	EP300	2033	E1A binding protein p300;EP300
Transferase	SETDB1	9869	SET domain, bifurcated 1;SETDB1
Transferase	PGGT1B	5229	protein geranylgeranyltransferase type I, beta subunit;PGGT1B
Transferase	SUV39H1	6839	suppressor of variegation 3-9 homolog 1 (Drosophila);SUV39H1
Transferase	NNMT	4837	nicotinamide N-methyltransferase;NNMT
Transferase	NAT9	26151	N-acetyltransferase 9;NAT9
Transferase	ASMT	438	acetylserotonin O-methyltransferase;ASMT
Transferase	NUDT1	4521	nudix (nucleoside diphosphate linked moiety X)-type motif 1;NUDT1
Transporter	TOM1	10043	target of myb1 (chicken);TOM1
Transporter	CHRND	1144	cholinergic receptor, nicotinic, delta;CHRND
Transporter	PRAF2	11230	PRA1 domain family, member 2;PRAF2
Transporter	FXYD2	486	FXYD domain containing ion transport regulator 2;FXYD2

Figure 27.

A.

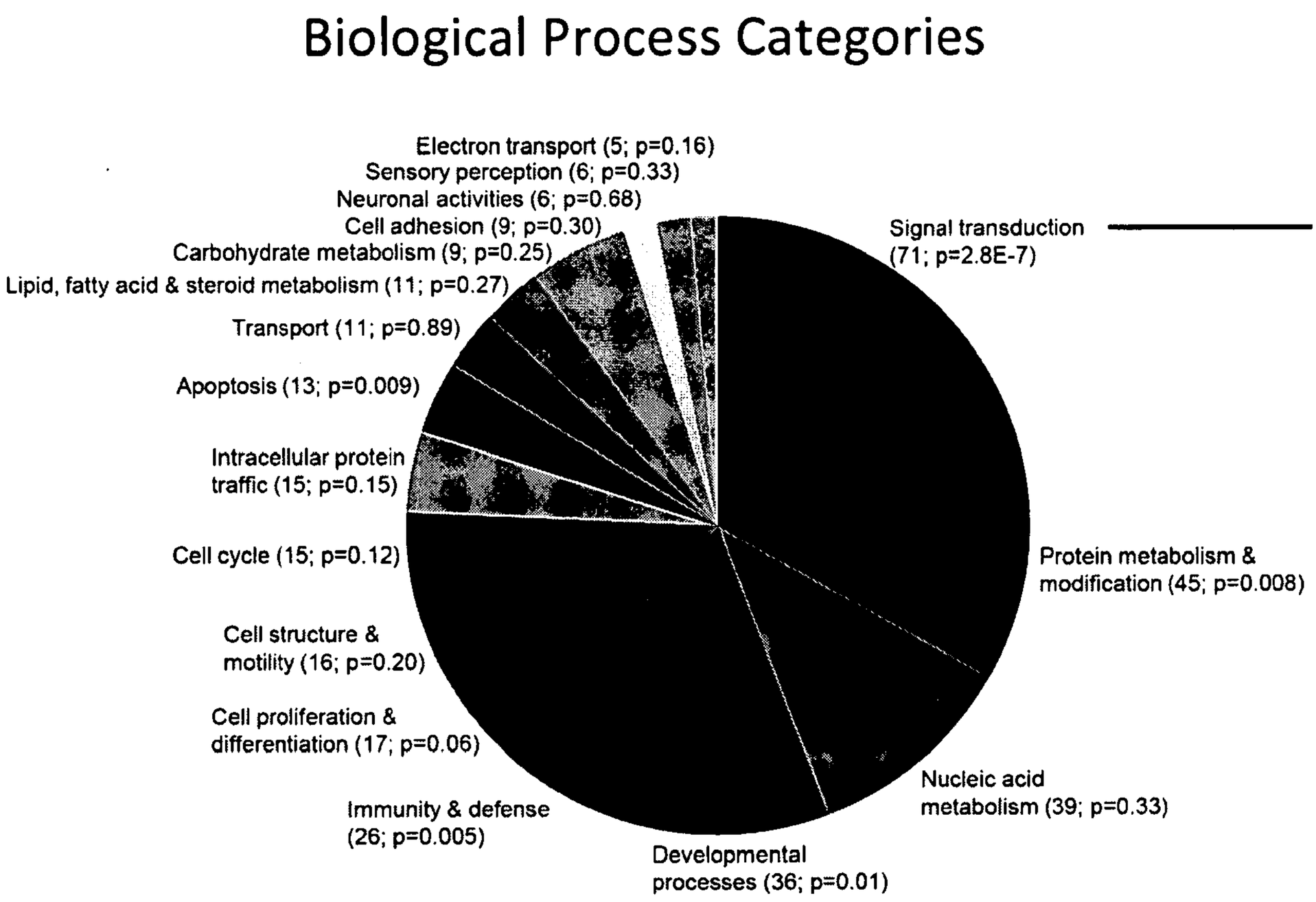

Biological Process Categories
Electron transport (5; p=0.16)
Sensory perception (6; p=0.33)
Neuronal activities (6; p=0.68)
Cell adhesion (9; p=0.30)
Carbohydrate metabolism (9; p=0.25)
Lipid, fatty acid & steroid metabolism (11; p=0.27)
Transport (11; p=0.89)
Apoptosis (13; p=0.009)
Intracellular protein traffic (15; p=0.15)
Cell cycle (15; p=0.12)
Cell structure & motility (16; p=0.20)
Cell proliferation & differentiation (17; p=0.06)
Immunity & defense (26; p=0.005)
Developmental processes (36; p=0.01)
Nucleic acid metabolism (39; p=0.33)
Protein metabolism & modification (45; p=0.008)
Signal transduction (71; p=2.8E-7)

Figure 27 (Continued)

B.

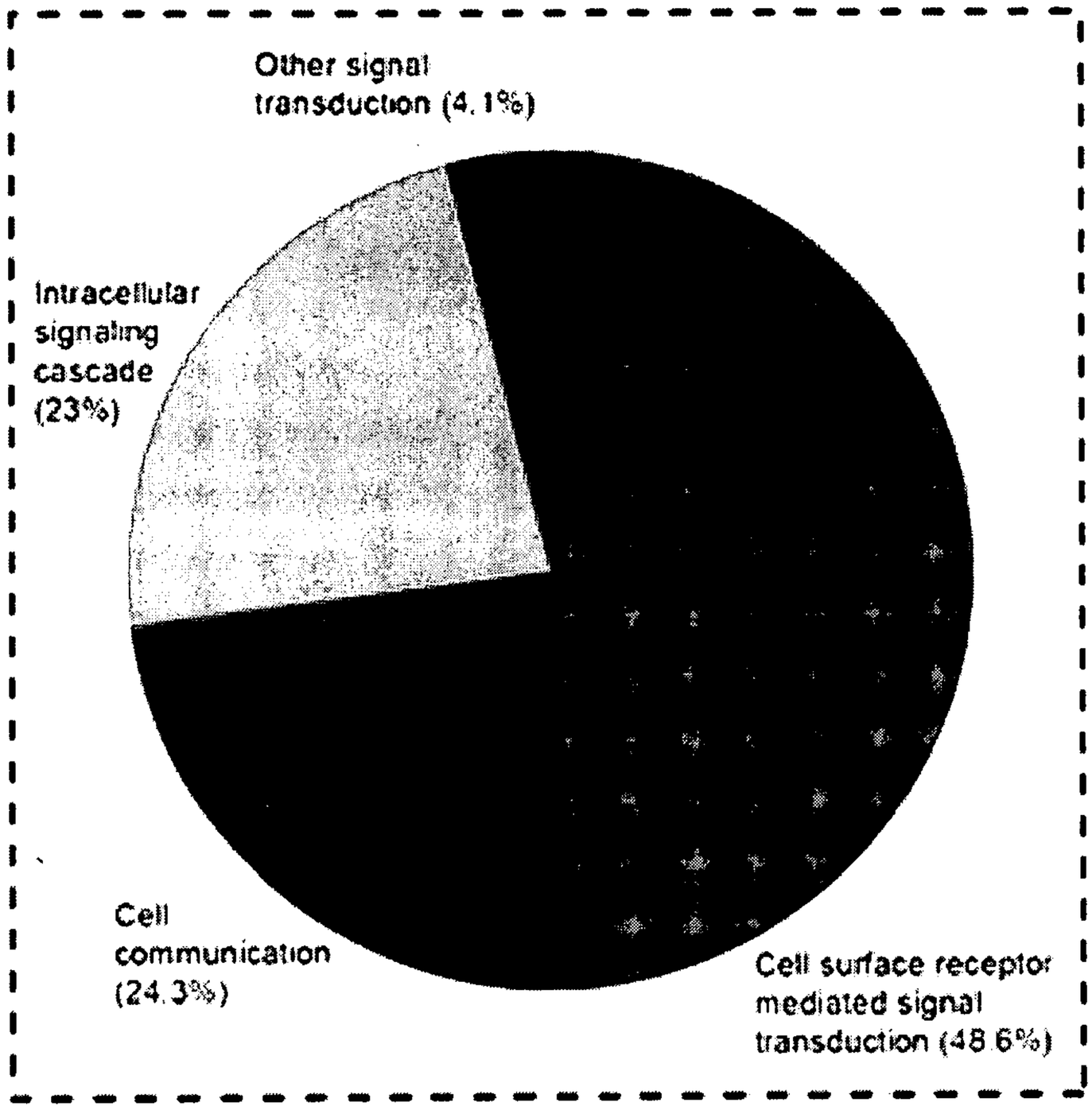


Figure 28.

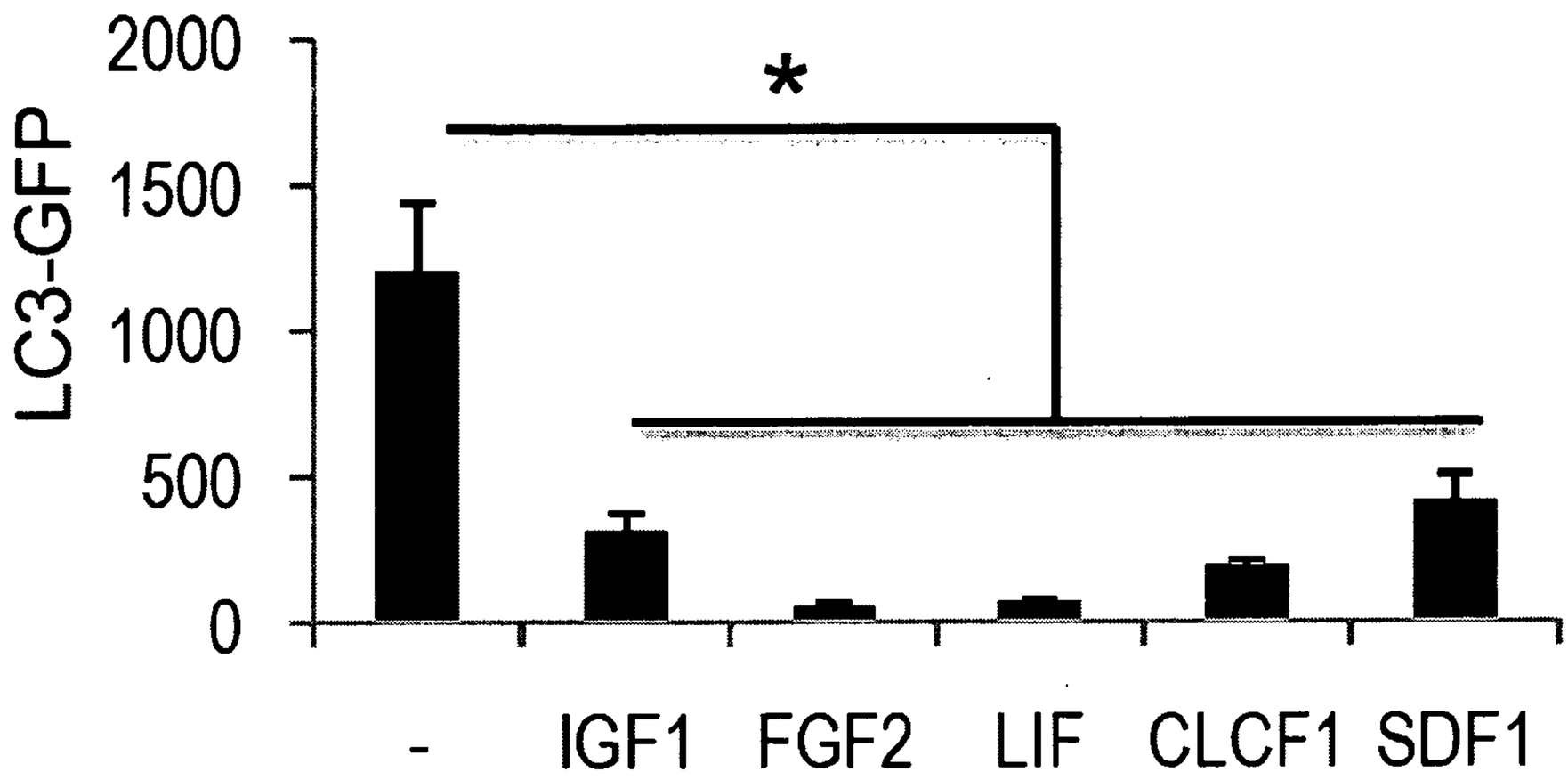


Figure 29.

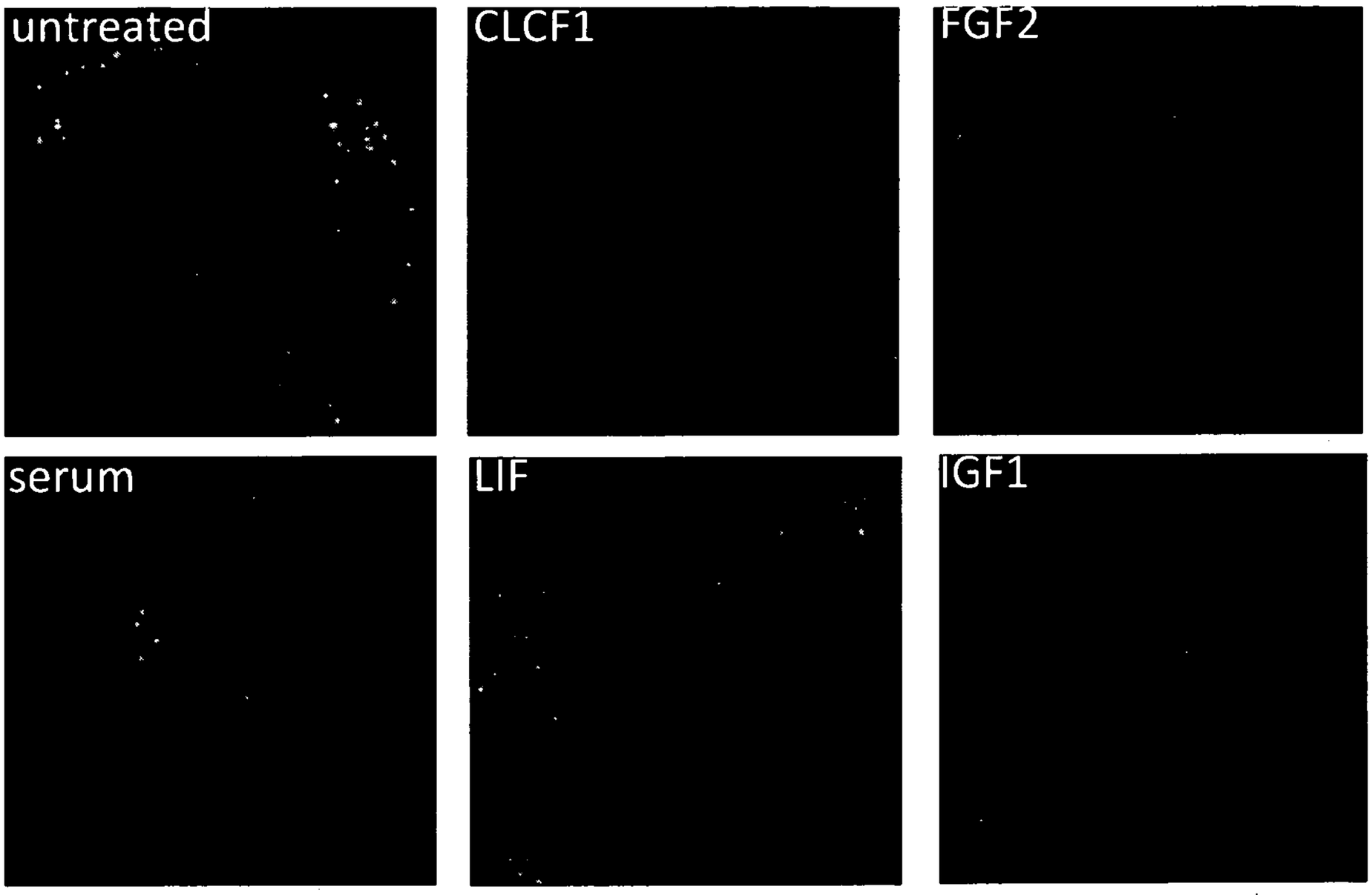
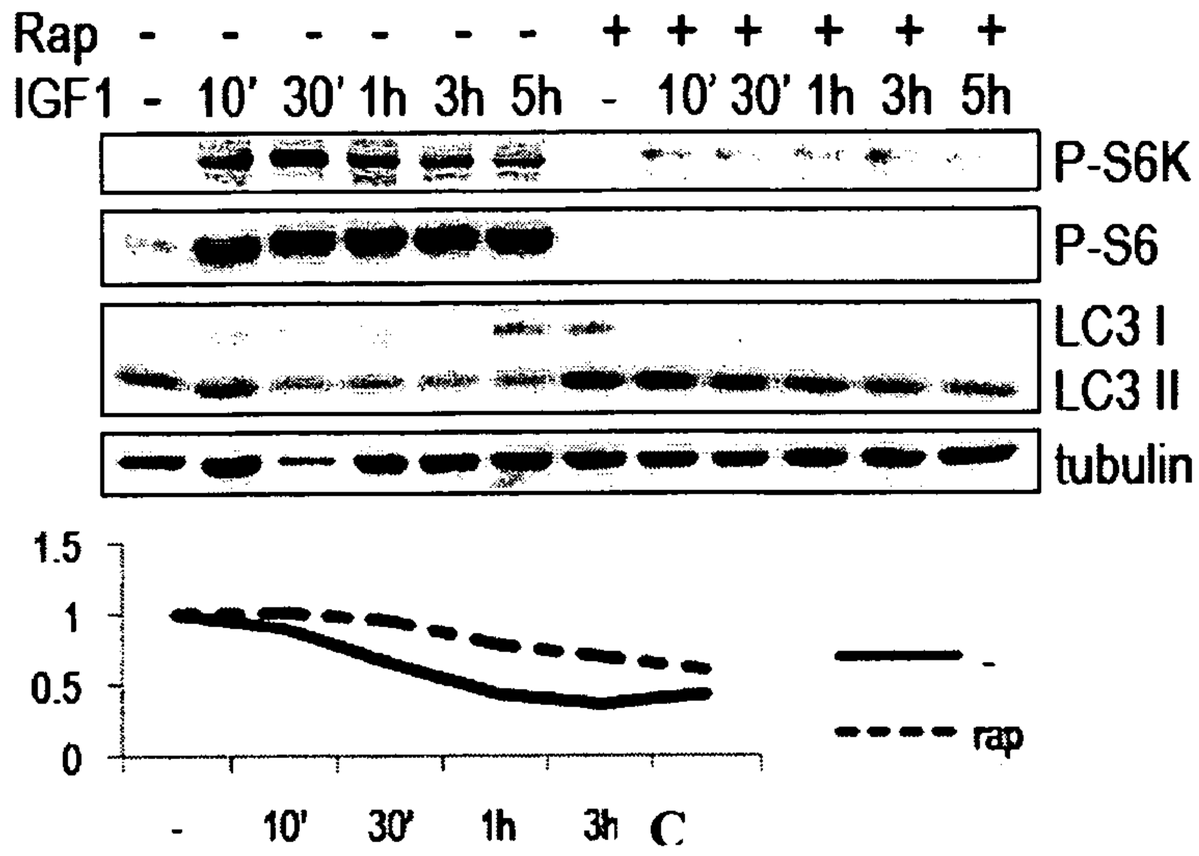
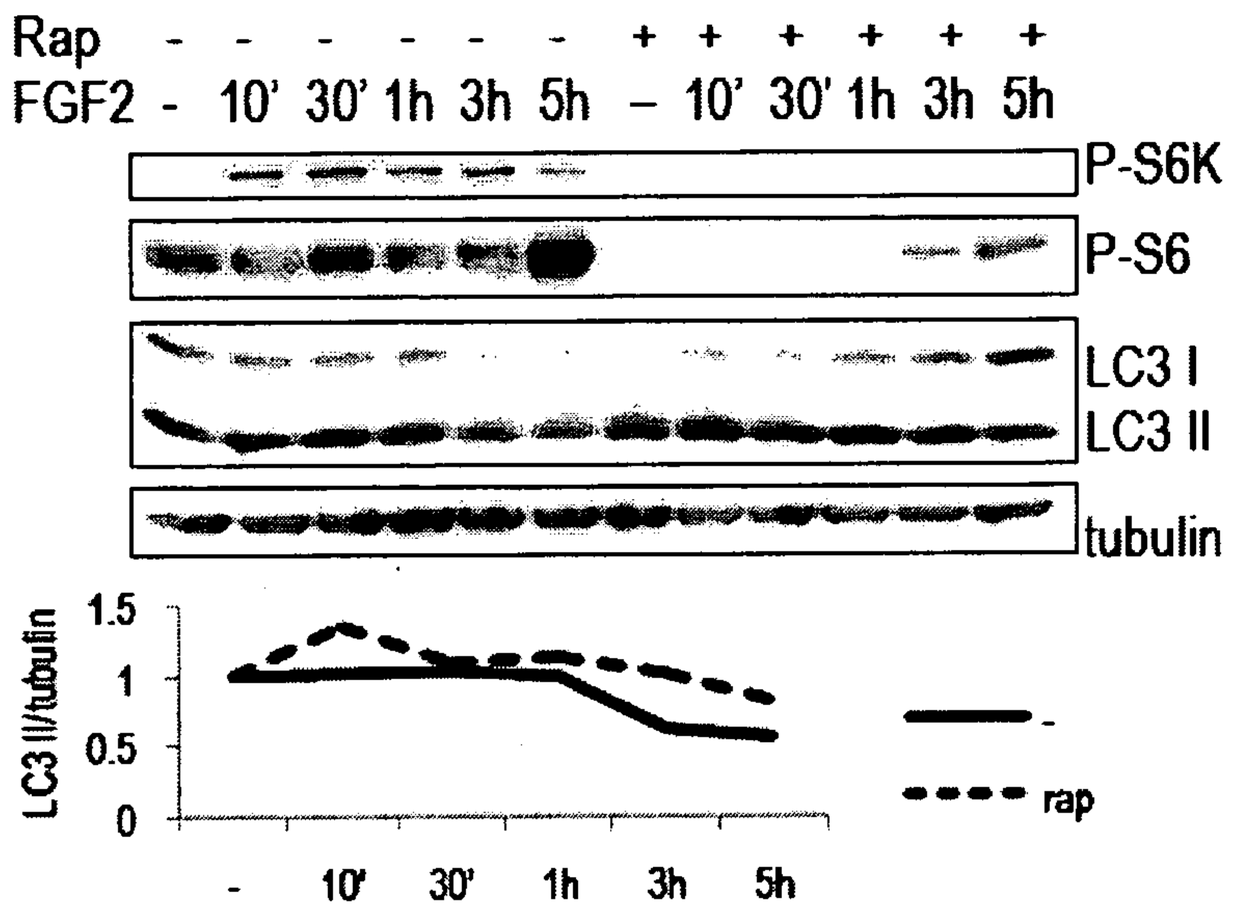


Figure 30.

A



B



C

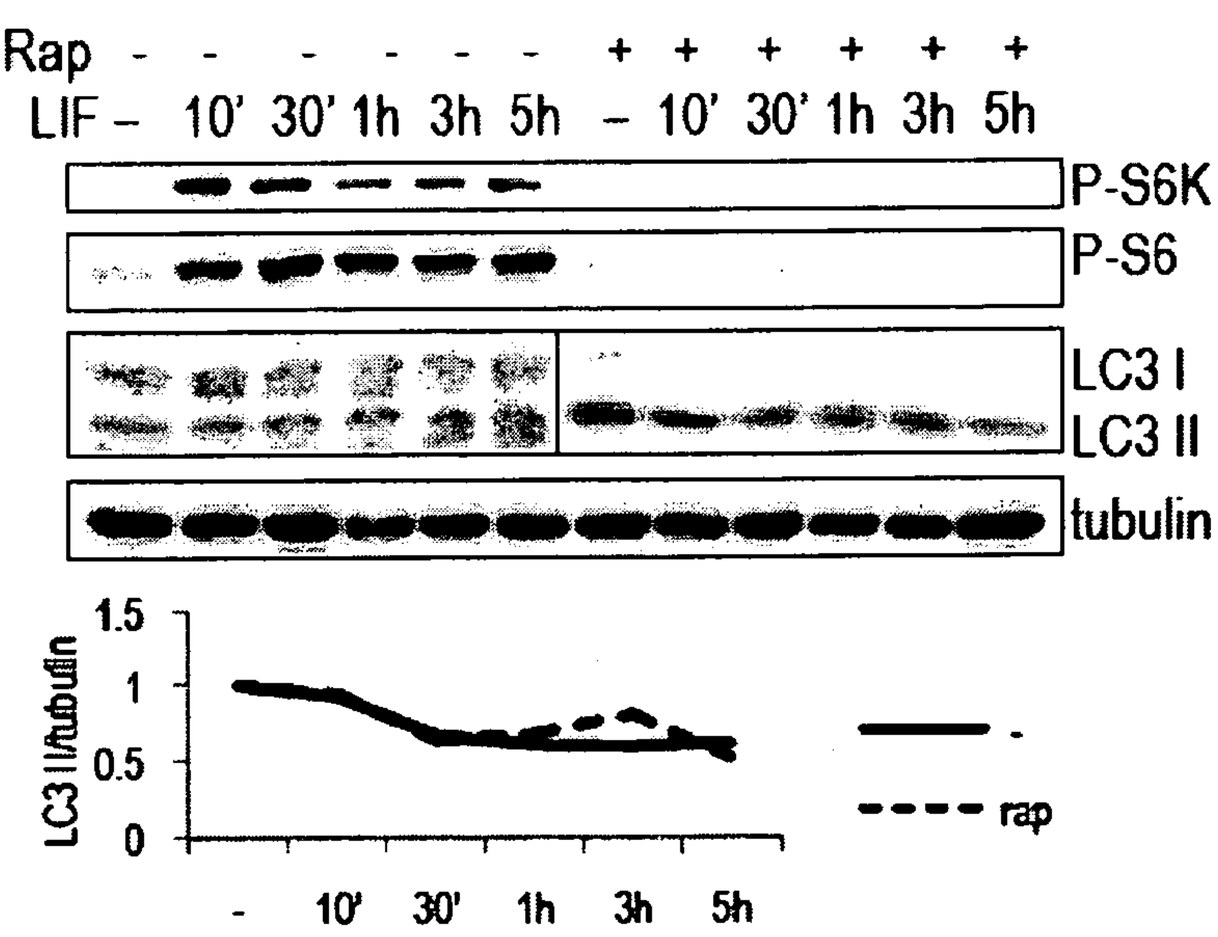


Figure 30 (cont.)

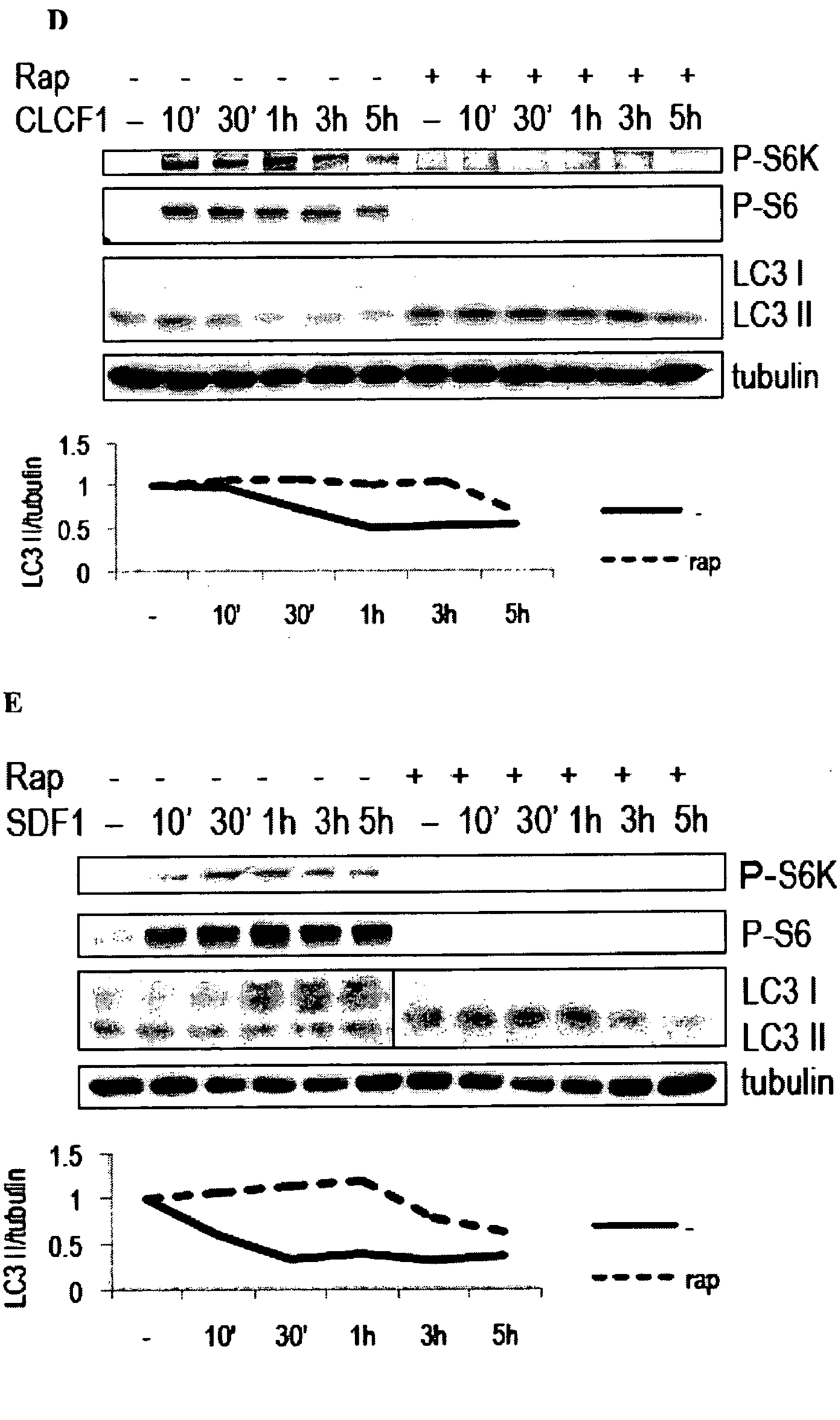
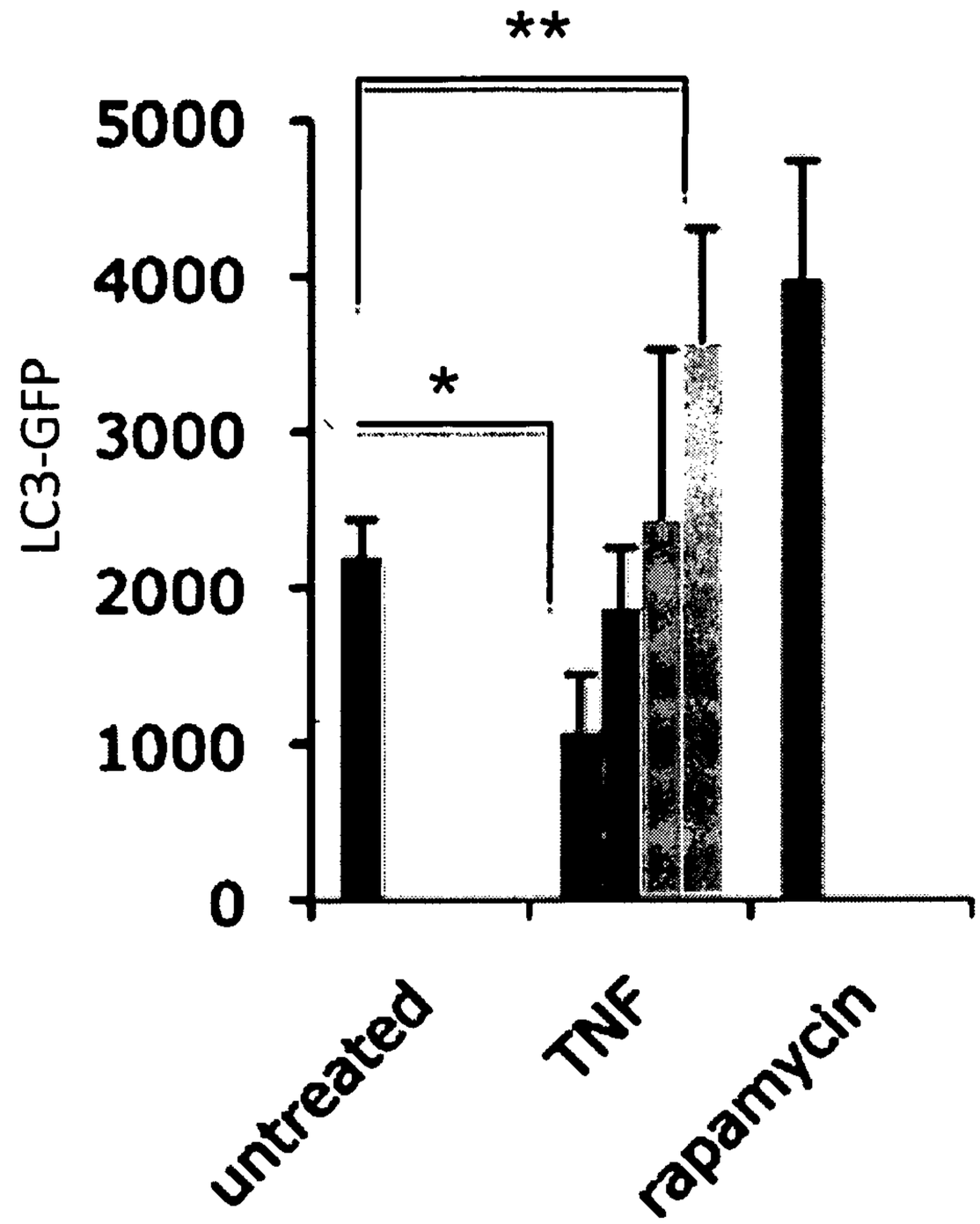


Figure 31.

A.


**
5000
4000
3000
2000
1000
0
LC3-GFP
*
untreated
TNF
rapamycin

B.

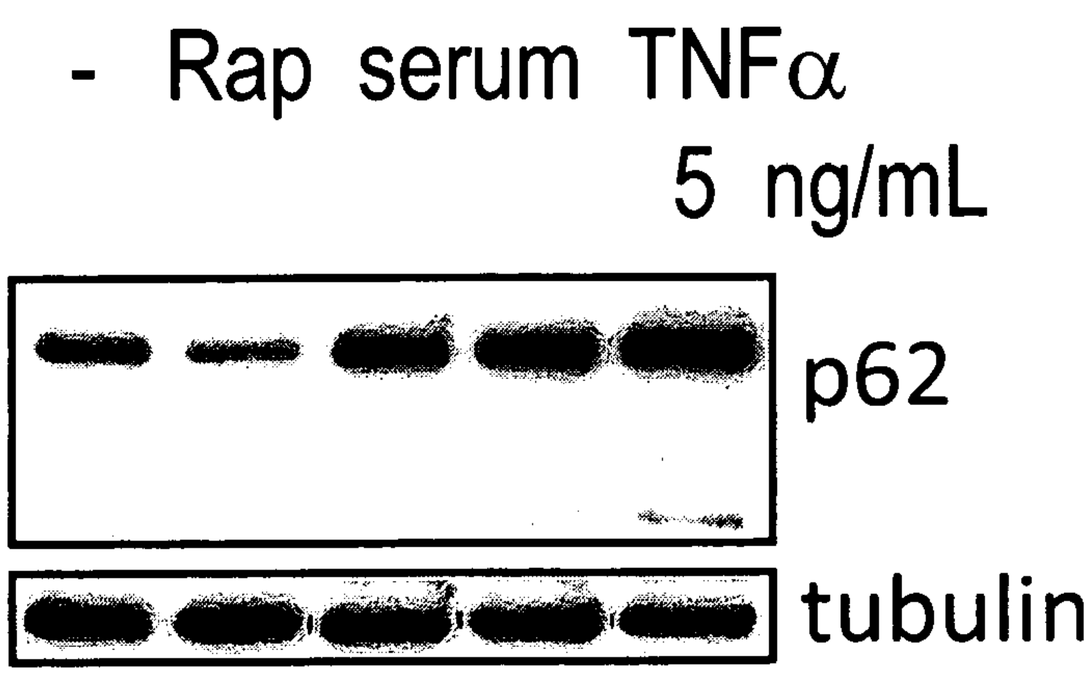

- Rap serum TNFα
5 ng/mL
p62
tubulin

Figure 32.

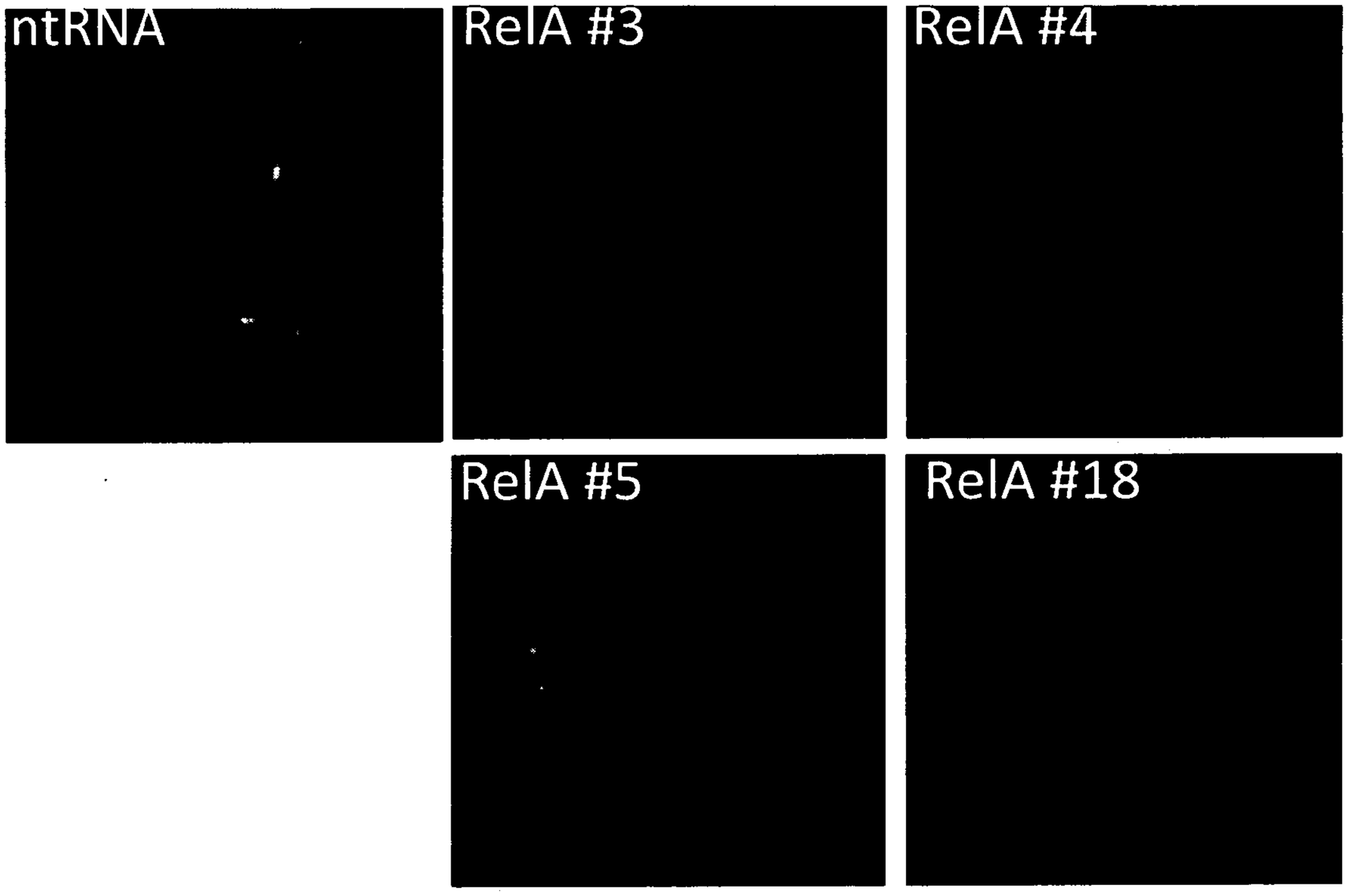


Figure 33.

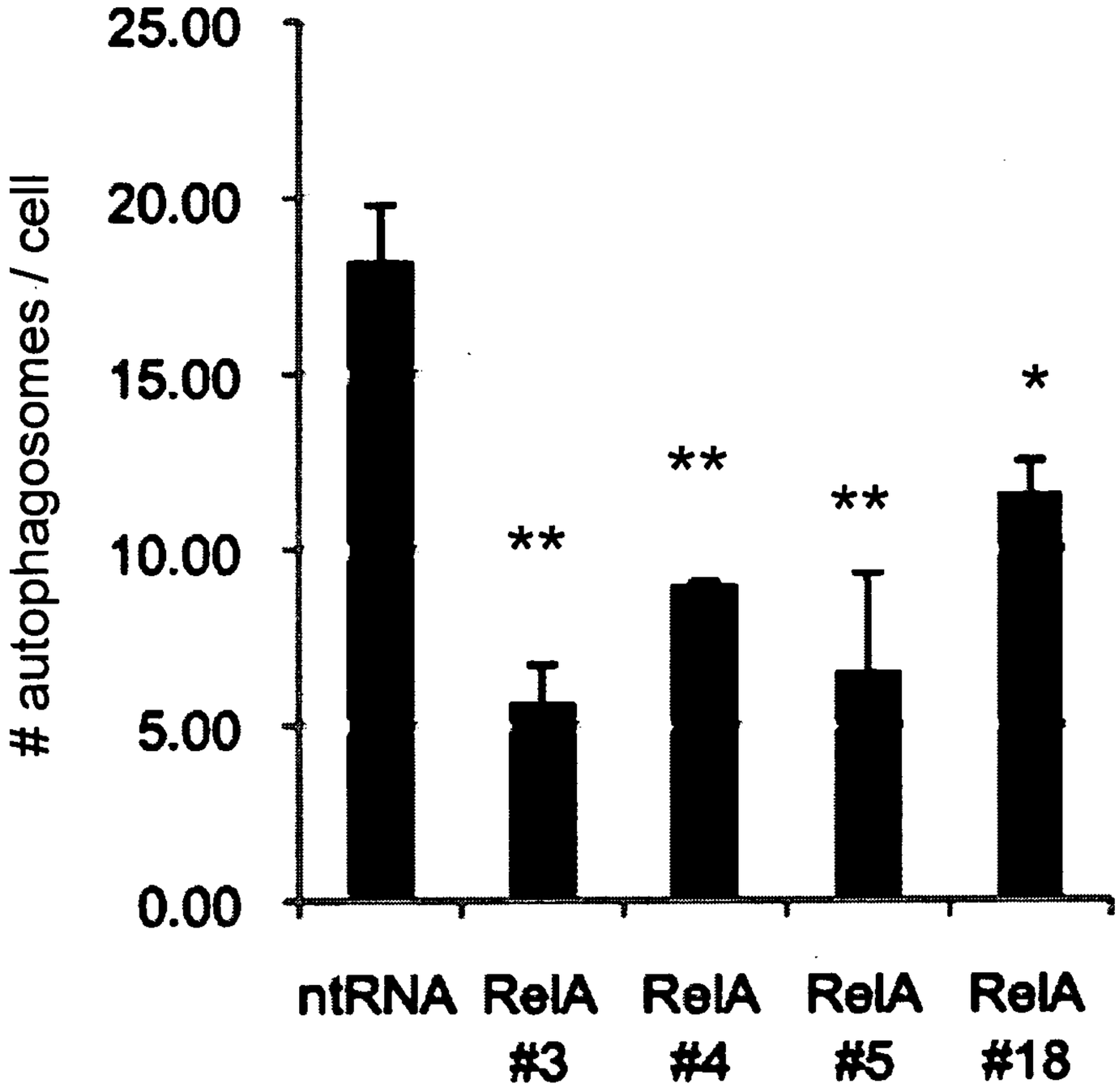
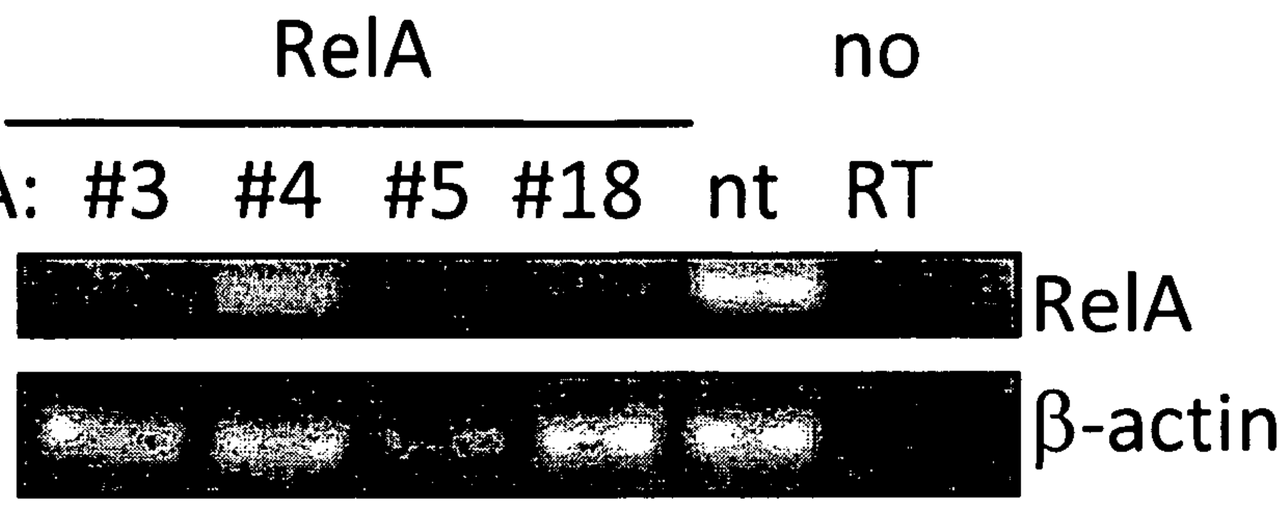

25.00
20.00
15.00
10.00
5.00
0.00
autophagosomes / cell
**
**
**
*
ntRNA
RelA #3
RelA #4
RelA #5
RelA #18

Figure 34.

A.


RelA
no
siRNA: #3 #4 #5 #18 nt RT
RelA
β-actin

B.

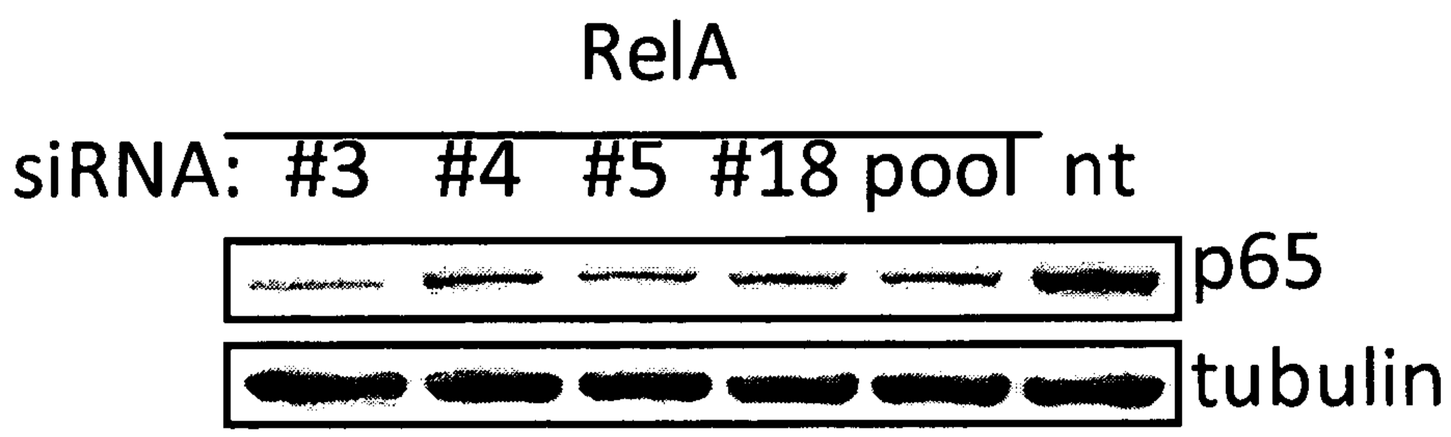
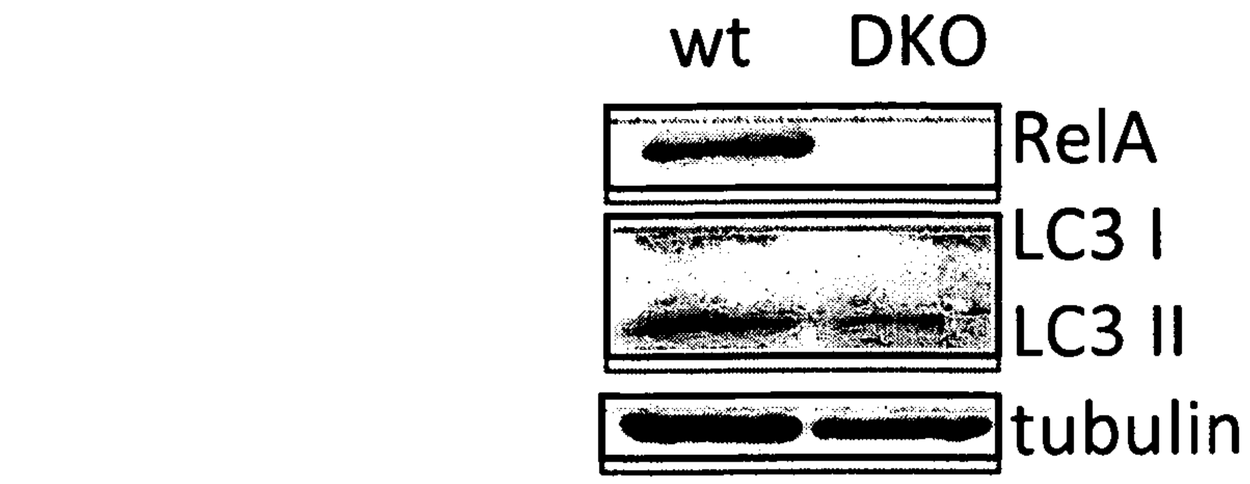

RelA
siRNA: #3 #4 #5 #18 pool nt
p65
tubulin

Figure 35.

A.


wt
DKO
RelA
LC3 I
LC3 II
tubulin
LC3II/tubulin: 1.0 0.6

B.

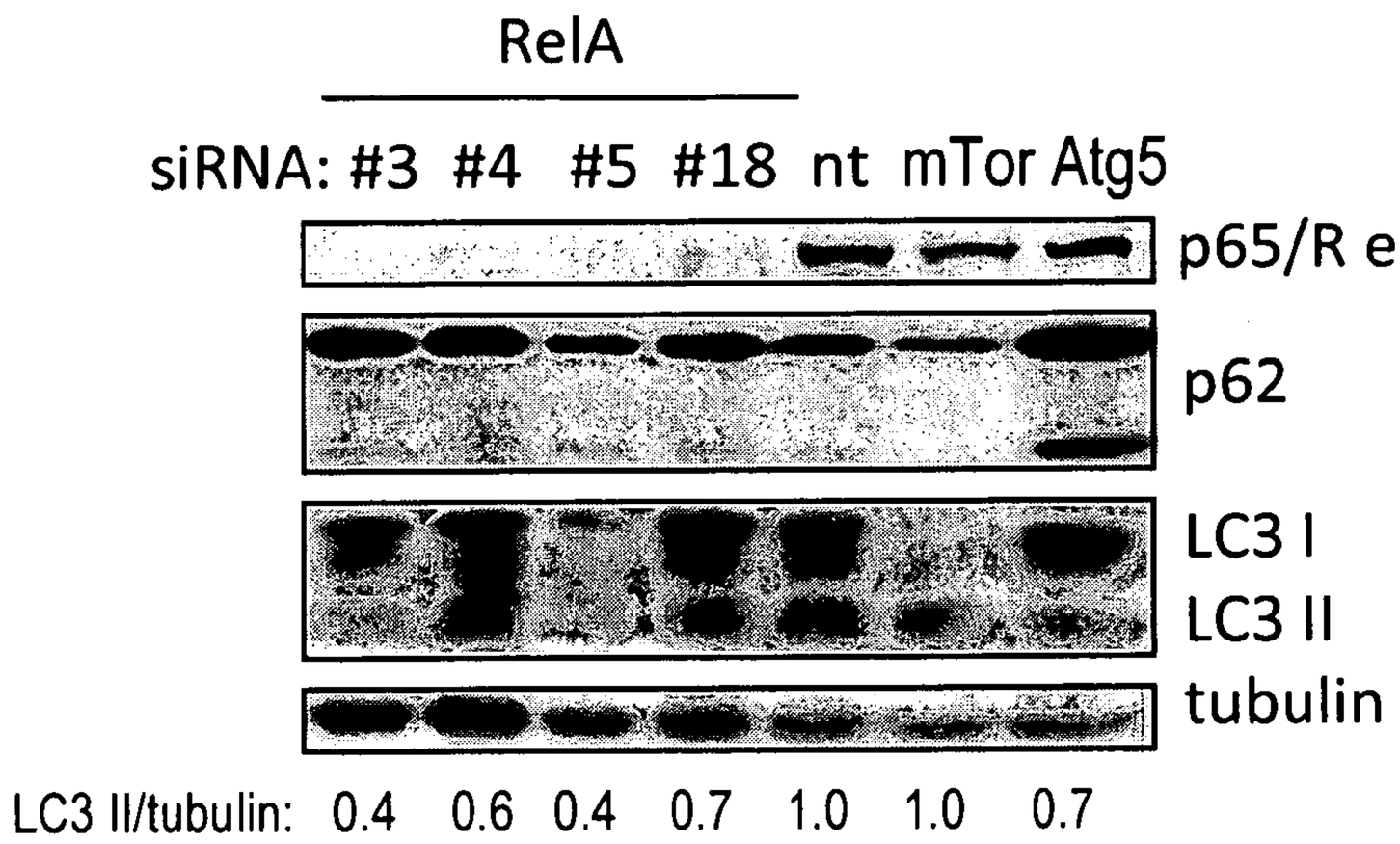

RelA
siRNA: #3 #4 #5 #18 nt mTor Atg5
p65/R elA
p62
LC3 I
LC3 II
tubulin
LC3 II/tubulin: 0.4 0.6 0.4 0.7 1.0 1.0 0.7

Figure 36.

A.

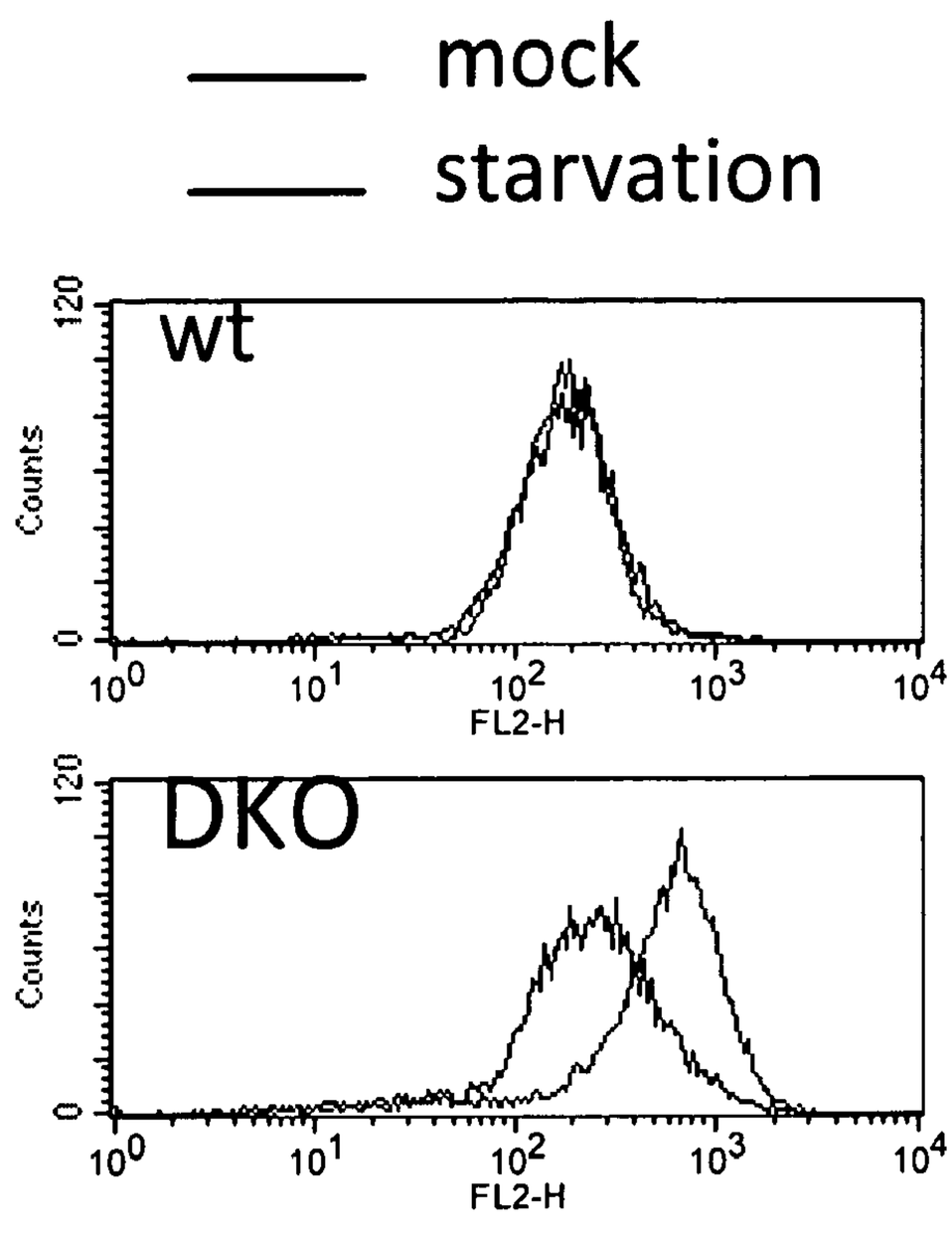
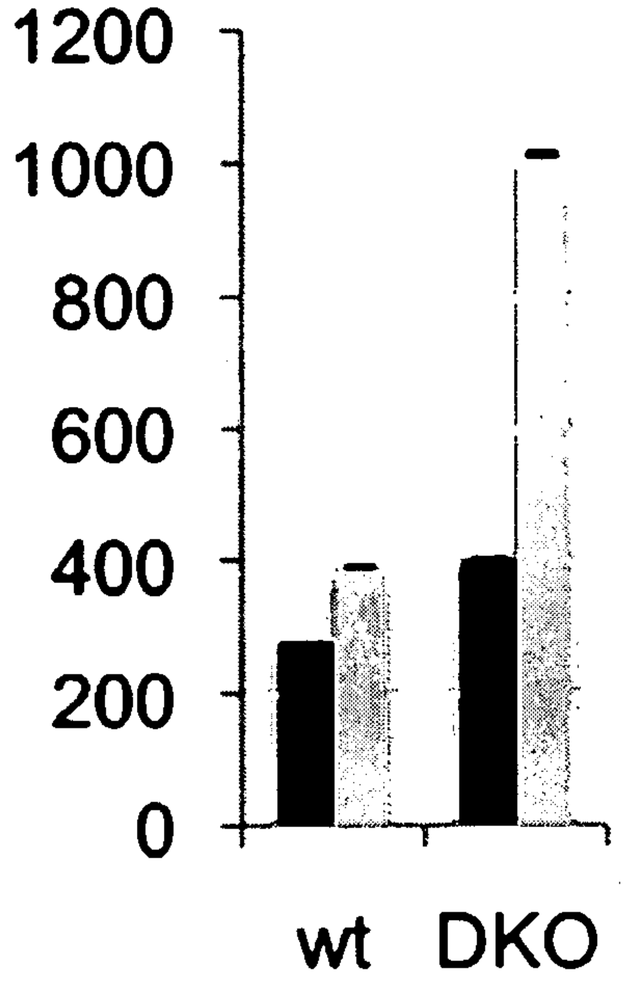


Figure 36 (Continued)

B.



C.

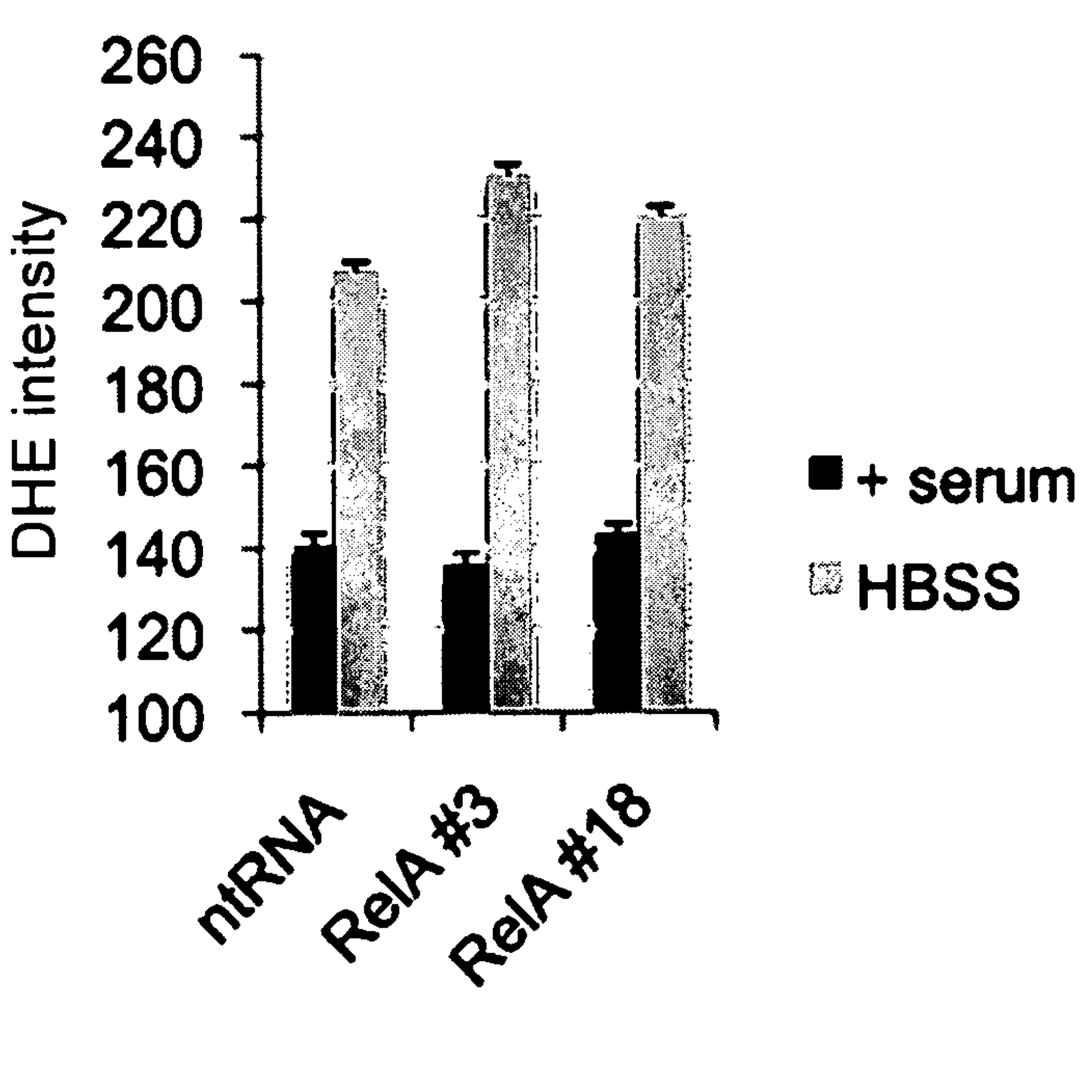


Figure 37.

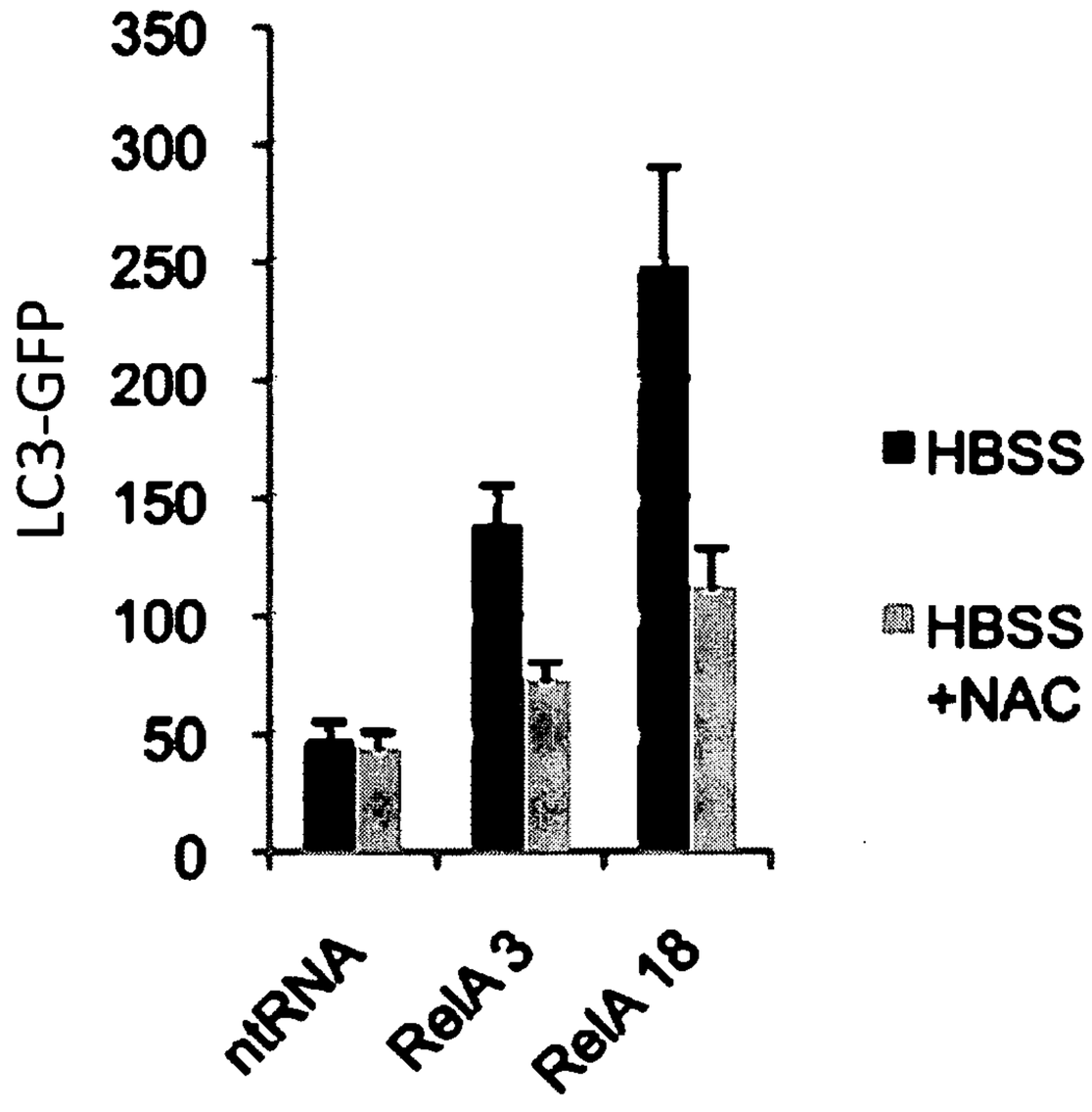

350
300
250
200
150
100
50
0
LC3-GFP
HBSS
HBSS +NAC
ntRNA
RelA 3
RelA 18

Figure 38.

Autophagy genes with mitochondrial localization.

		Motochondrial localization based on:	
Gene symbol	**GeneID**	**Prediction**	**Association/Function**
CYP27A1	1593	Maestro	GO: MF, BP or CC
HMGCL	3155	Maestro	GO: MF, BP or CC
SOD1	6647	Maestro	GO: MF, BP or CC
COX5A	9377	Maestro	GO: MF, BP or CC
TRNT1	51095	Maestro	GO: MF, BP or CC
MCCC1	56922	Maestro	GO: MF, BP or CC
SLC25A19	60386	Maestro	GO: MF, BP or CC
PIGY	84992	#N/A	GO: MF, BP or CC
GFER	2671	Maestro	GO: MF, BP or CC
ICT1	3396	Maestro	#N/A
	8936	#N/A	GO: MF, BP or CC
PNKD	25953	Maestro	GO: MF, BP or CC
CCT4	10575	Maestro	#N/A
NUDT1	4521	Maestro	#N/A
OGDH	4967	Maestro	GO: MF, BP or CC
PRKCA	5578	#N/A	GO: MF, BP or CC
SDHB	6390	Maestro	GO: MF, BP or CC
PHB2	11331	#N/A	GO: MF, BP or CC

Figure 39.

Hit genes with known connection to oxidative damage and/or reactive oxygen species (ROS)

GeneID	Gene symbol	Gene Name
124056	NOXO1	NOXO1 P41NOX P41NOXA P41NOXB P41NOXC MGC20258 NADPH oxidase organizer 1 isoform c
2877	GPX2	GPX2 GSHPX-GI gastrointestinal glutathione peroxidase 2
4521	NUDT1	NUDT1 MTH1 nudix-type motif 1 isoform p22
23560	GTPBP4	GTPBP4 NGB CRFG FLJ10686 FLJ10690 G protein-binding protein CRFG
4790	NFKB1	NFKB1 KBF1 EBP-1 MGC54151 NFKB-p50 NFKB-p105 NF-kappa-B nuclear factor kappa-B subunit 1
2079	ERH	ERH DROER enhancer of rudimentary homolog
8737	RIPK1	RIPK1 receptor (TNFRSF)-interacting serine-threonine kinase 1
6647	SOD1	SOD1 ALS ALS1 IPOA superoxide dismutase 1 soluble
5563	PRKAA2	PRKAA2 AMPK AMPK2 PRKAA AMP-activated protein kinase alpha 2 catalytic subunit
5970	RELA	RELA NFKB3 v-rel reticuloendotheliosis viral oncogene homolog A nuclear factor of kappa light polypeptide gene enhancer in B-cells 3 p65
10076	PTPRU	PTPRU FMI PTP PCP-2 PTP-J PTPRO PTPU2 GLEPP1 PTP-PI PTPPSI hPTP-J R-PTP-PSI protein tyrosine phosphatase receptor type U isoform 3 precursor
5229	PGGT1B	PGGT1B BGGI GGTI protein geranylgeranyltransferase type I beta subunit
2205	FCER1A	FCER1A FCE1A FcERI Fc fragment of IgE high affinity I receptor for; alpha polypeptide precursor
5590	PRKCZ	PRKCZ PKC2 protein kinase C zeta
4967	OGDH	OGDH E1k oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)
27315	FRAG1	FRAG1 FGF receptor activating protein 1
5768	QSOX1	QSCN6 quiescin Q6
10509	SEMA4B	SEMA4B SemC SEMAC KIAA1745 semaphorin 4B precursor
10043	TOM1	TOM1 target of myb1
5467	PPARD	PPARD FAAR NUC1 NUCI NR1C2 NUCII PPARB MGC3931 PPAR-beta peroxisome proliferative activated receptor delta isoform 2
7124	TNF	TNF DIF TNFA TNFSF2 CACHECTIN tumor necrosis factor alpha
9377	COX5A	COX5A COX-VA cytochrome c oxidase subunit Va precursor
5578	PRKCA	PRKCA PKCA PRKACA PKC-alpha protein kinase C alpha
6390	SDHB	SDHB SDH SDH1 SDHIP succinate dehydrogenase complex subunit B iron sulfur (Ip)
834	CASP1	CASP1 P45 IL1BC caspase 1 isoform gamma precursor
11331	PHB2	REA BAP repressor of estrogen receptor activity
6774	STAT3	STAT3 APRF FLJ20882 MGC16063 signal transducer and activator of transcription 3 isoform 3
728	C5AR1	C5R1 C5A C5AR CD88 complement component 5 receptor 1 (C5a ligand)

Figure 39 (Continued)

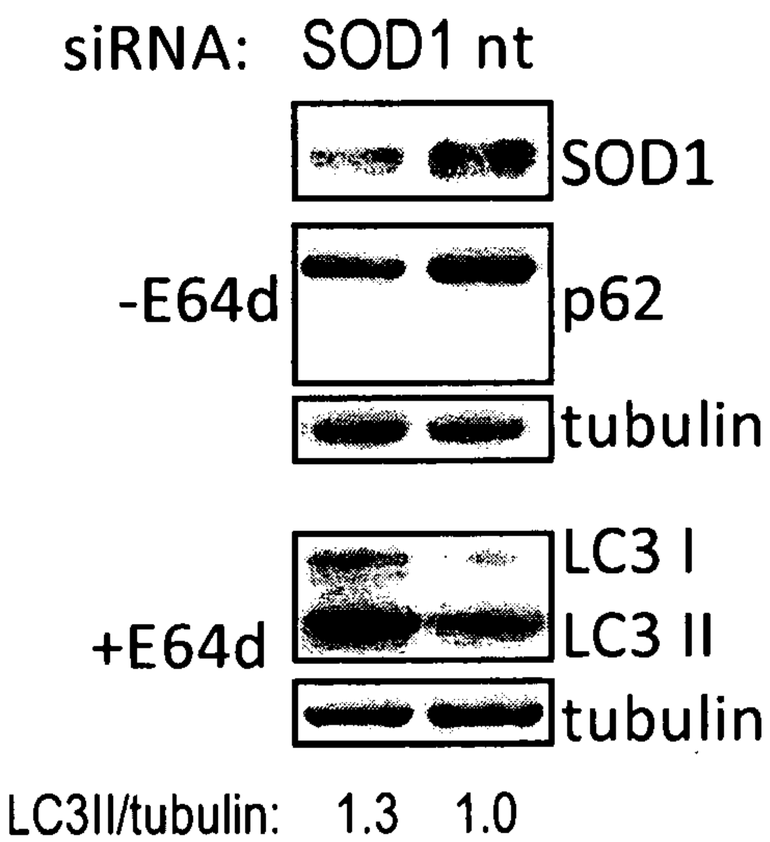
GeneID	Gene symbol	Gene Name
8989	TRPA1	TRPA1 ANKTM1 ankyrin-like protein 1
2700	GJA3	GJA3 CAE2 CAE3 CX46 CZP2 CZP3 gap junction protein alpha 3 46kDa (connexin 46)
10454	MAP3K7IP1	MAP3K7IP1 TAB1 MGC57664 mitogen-activated protein kinase kinase kinase 7 interacting protein 1 isoform beta
8936	WASF1	WASF1 SCAR1 WAVE1 KIAA0269 WAS protein family member 1
2176	FANCC	FANCC FA3 FAC FACC Fanconi anemia complementation group C
7058	THBS2	THBS2 TSP2 thrombospondin 2 precursor
1119	CHKA	CHKA CHK CKI choline kinase alpha isoform b
10406	WFDC2	WFDC2 HE4 WAP5 dJ461P17.6 WAP four-disulfide core domain 2 isoform 5
126961	HIST2H3C	HIST2H3C H3.2 H3/M H3F2 H3FM MGC9629 H3 histone
4319	MMP10	MMP10 SL-2 STMY2 matrix metalloproteinase 10 preproprotein
2168	FABP1	FABP1 FABPL L-FABP fatty acid binding protein 1 liver
140691	TRIM69	RNF36 Trif ring finger protein 36 isoform b
633	BGN	BGN PGI DSPG1 PG-S1 SLRR1A biglycan preproprotein
133	ADM	ADM adrenomedullin
25953	PNKD	MR-1 BRP17 FKSG19 TAHCCP2 KIAA1184 MGC31943 DKFZp564N1362 myofibrillogenesis regulator 1
3396	ICT1	ICT1 DS-1 immature colon carcinoma transcript 1
10575	CCT4	CCT4 SRB Cctd chaperonin containing TCP1 subunit 4 (delta)
10458	BAIAP2	BAIAP2 BAP2 IRSP53 BAI1-associated protein 2 isoform 3
3237	HOXD11	HOXD11 HOX4 HOX4F homeo box D11
2671	GFER	GFER ALR HSS ERV1 HPO1 HPO2 HERV1 erv1-like growth factor
823	CAPN1	CAPN1 CANP muCL CANPL1 muCANP calpain 1 large subunit
9474	ATG5	APG5L ASP APG5 hAPG5 APG5 autophagy 5-like
5023	P2RX1	P2RX1 P2X1 purinergic receptor P2X1
3190	HNRPK	HNRPK CSBP TUNP HNRNPK heterogeneous nuclear ribonucleoprotein K isoform b
372	ARCN1	ARCN1 COPD archain
7098	TLR3	TLR3 toll-like receptor 3
11318	GPR182	ADMR AMR 7TMR hrhAMR MGC34399 adrenomedullin receptor
7185	TRAF1	TRAF1 EBI6 MGC:10353 TNF receptor-associated factor 1
1032	CDKN2D	CDKN2D p19 INK4D p19-INK4D cyclin-dependent kinase inhibitor 2D
2247	FGF2	FGF2 BFGF FGFB HBGH-2 fibroblast growth factor 2
4148	MATN3	MATN3 HOA EDM5 matrilin 3 precursor
3875	KRT18	KRT18 K18 CYK18 keratin 18
5732	PTGER2	PTGER2 EP2 prostaglandin E receptor 2 (subtype EP2) 53kDa
1593	CYP27A1	CYP27A1 CTX CP27 CYP27 cytochrome P450 family 27 subfamily A polypeptide 1 precursor

Figure 39 (Continued)

GeneID	Gene symbol	Gene Name
4155	MBP	MBP myelin basic protein
7201	TRHR	TRHR thyrotropin-releasing hormone receptor
60386	SLC25A19	SLC25A19 DNC MUP1 MCPHA solute carrier family 25 (mitochondrial deoxynucleotide carrier) member 19
10204	NUTF2	NUTF2 NTF2 PP15 nuclear transport factor 2
6790	AURKA	STK6 AIK ARK1 BTAK STK15 MGC34538 serine/threonine protein kinase 6
2161	F12	F12 HAF coagulation factor XII precursor
3976	LIF	LIF CDF HILDA D-FACTOR leukemia inhibitory factor (cholinergic differentiation factor)
7045	TGFBI	TGFBI CSD CDB1 CDG2 CSD1 CSD2 CSD3 LCD1 BIGH3 CDGG1 transforming growth factor beta-induced 68kDa
7514	XPO1	XPO1 CRM1 exportin 1
23529	CLCF1	CLC BSF3 cardiotrophin-like cytokine
438	ASMT	ASMT ASMTY HIOMT HIOMTY acetylserotonin O-methyltransferase
6924	TCEB3	TCEB3 SIII TCEB3A elongin A
6387	CXCL12	CXCL12 PBSF SDF1 SDF1A SDF1B TPAR1 SCYB12 SDF-1a SDF-1b TLSF-a TLSF-b chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
268	AMH	AMH MIF MIS anti-Mullerian hormone
3145	HMBS	HMBS AIP UPS PBGD hydroxymethylbilane synthase
7704	ZBTB16	ZNF145 PLZF ZBTB16 zinc finger protein 145 (Kruppel-like expressed in promyelocytic leukemia)
5662	PSD	PSD TYL KIAA2011 pleckstrin and Sec7 domain containing
3479	IGF1	IGF1 IGFI insulin-like growth factor 1 (somatomedin C)
2837	UTS2R	GPR14 UTR UTR2 G protein-coupled receptor 14
2033	EP300	EP300 p300 E1A binding protein p300
5082	PDCL	PDCL PhLP DKFZp564M1863 phosducin-like
7879	RAB7A	RAB7 PSN CMT2B PRO2706 RAB7 member RAS oncogene family
55835	CENPJ	CENPJ CPAP LIP1 BM032 centromere protein J
7175	TPR	TPR translocated promoter region (to activated MET oncogene)
4014	LOR	LOR loricrin
2260	FGFR1	FGFR1 CEK FLG FLT2 KAL2 BFGFR C-FGR N-SAM fibroblast growth factor receptor 1 isoform 9 precursor
973	CD79A	CD79A IGA MB-1 CD79A antigen isoform 2 precursor
3123	HLA-DRB1	HLA-DRB1 HLA-DR1B major histocompatibility complex class II DR beta 1 precursor
7373	COL14A1	COL14A1 UND

Figure 40.

A


siRNA: SOD1 nt
SOD1
-E64d
p62
tubulin
LC3 I
+E64d
LC3 II
tubulin
LC3II/tubulin: 1.3 1.0

B.

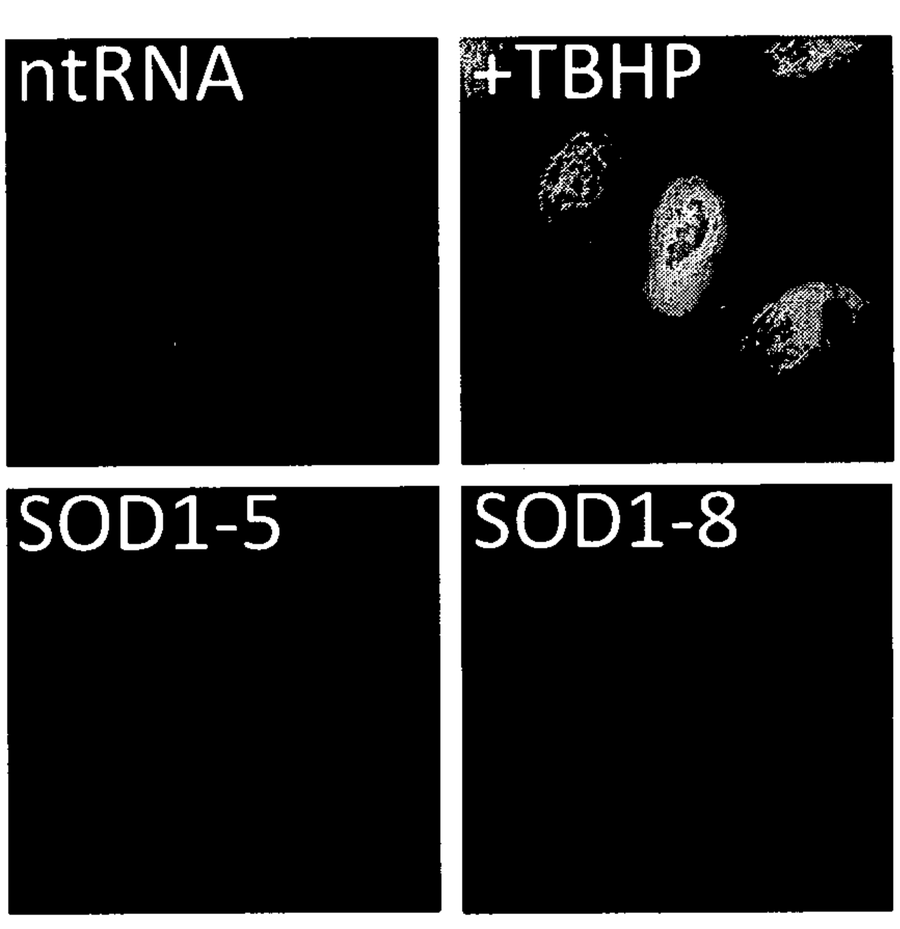

ntRNA
+TBHP
SOD1-5
SOD1-8

Figure 40 (Continued)

C.

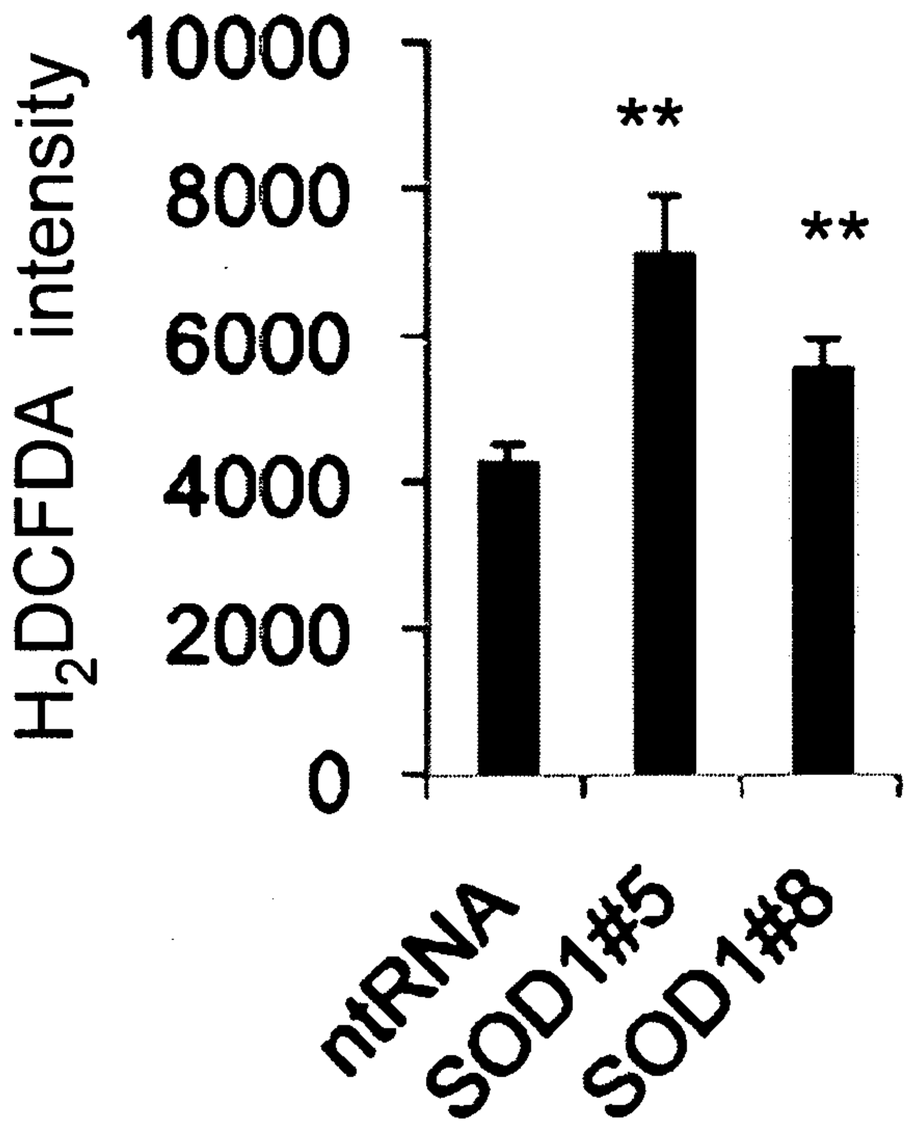
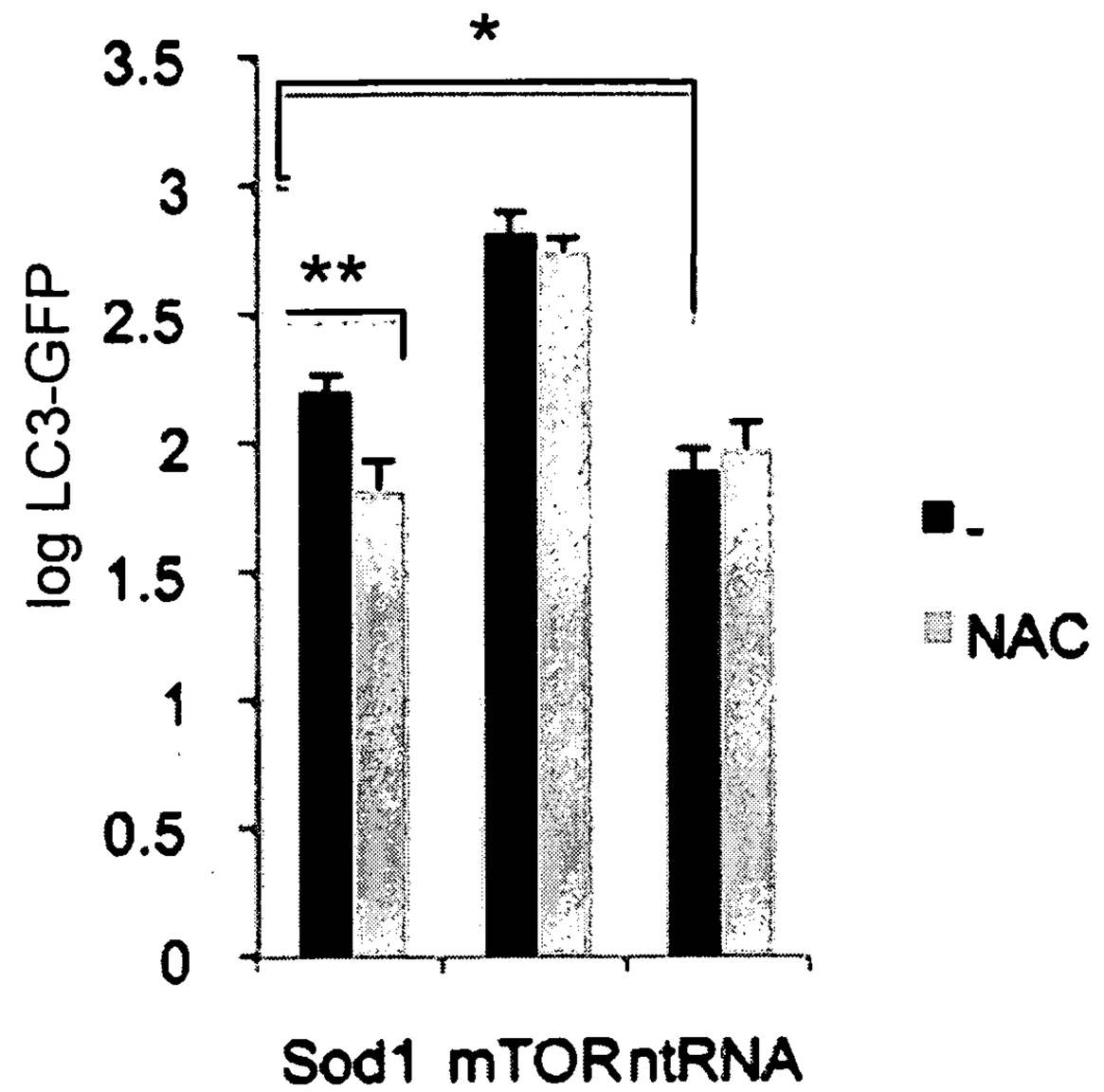


Figure 41.



z-score	Sod1	mTOR	ntRNA
-NAC:	1.5	4.5	
+NAC:	-0.4	4.1	0.3

Figure 42

Genes whose knock-down induces autophagy in the absence but not the presence of antioxidant.

Gene Symbol	Gene ID	Gene Name
CHID1	66005	chitinase domain containing 1
IHPK3	117283	inositol hexaphosphate kinase 3
NLK	51701	nemo-like kinase
PIGY	84992	phosphatidylinositol glycan anchor biosynthesis, class Y
STAT3	6774	signal transducer and activator of transcription 3 (acute-phase response factor)
TRNT1	51095	tRNA nucleotidyl transferase, CCA-adding, 1
SCOTIN	51246	scotin
NNMT	4837	nicotinamide N-methyltransferase
SOD1	6647	superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult))
TAF2	6873	TAF2 RNA polymerase II, TATA box binding protein (TBP)-associated factor, 150kDa
DBX1	120237	developing brain homeobox 1
USP24	23358	ubiquitin specific peptidase 24
CYP27A1	1593	cytochrome P450, family 27, subfamily A, polypeptide 1
FANCC	2176	Fanconi anemia, complementation group C
MUC3A	4584	mucin 3A, cell surface associated
SMARCD1	6602	SWI/SNF related, matrix associated, actin dependent regulator of chromatin, subfamily d, member 1
NPTN	27020	neuroplastin
TNFRSF17	608	tumor necrosis factor receptor superfamily, member 17
COX5A	9377	cytochrome c oxidase subunit Va
NUDT1	4521	nudix (nucleoside diphosphate linked moiety X)-type motif 1
IGF1	3479	insulin-like growth factor 1 (somatomedin C)
ADRA1A	148	adrenergic, alpha-1A-, receptor
CD79A	973	CD79a molecule, immunoglobulin-associated alpha
WASF1	8936	WAS protein family, member 1
OA48-18	10414	acid-inducible phosphoprotein
LIF	3976	leukemia inhibitory factor (cholinergic differentiation factor)
HMGCL	3155	3-hydroxymethyl-3-methylglutaryl-Coenzyme A lyase (hydroxymethylglutaricaciduria)
MMACHC	25974	methylmalonic aciduria (cobalamin deficiency) cblC type, with homocystinuria
FRAG1	27315	FGF receptor activating protein 1
MCCC1	56922	methylcrotonoyl-Coenzyme A carboxylase 1 (alpha)
AMH	268	anti-Mullerian hormone
PLXNA2	5362	plexin A2

Figure 42 (Continued)

Gene Symbol	Gene ID	Gene Name
ASMT	438	acetylserotonin O-methyltransferase
CASP1	834	caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase)
MMP17	4326	matrix metallopeptidase 17 (membrane-inserted)
RBBP8	5932	retinoblastoma binding protein 8
TRPA1	8989	transient receptor potential cation channel, subfamily A, member 1
ADM	133	adrenomedullin
PAK6	56924	p21(CDKN1A)-activated kinase 6
POLR3G	10622	polymerase (RNA) III (DNA directed) polypeptide G (32kD)
AGER	177	advanced glycosylation end product-specific receptor
CPNE6	9362	copine VI (neuronal)
MAP3K7IP1	10454	mitogen-activated protein kinase kinase kinase 7 interacting protein 1
USP54	159195	ubiquitin specific peptidase 54
GPX2	2877	glutathione peroxidase 2 (gastrointestinal)
NUPR1	26471	nuclear protein 1
EVL	51466	Enah/Vasp-like
PA2G4	5036	proliferation-associated 2G4, 38kDa
GTPBP4	23560	GTP binding protein 4
PRKAA2	5563	protein kinase, AMP-activated, alpha 2 catalytic subunit
EGLN2	112398	egl nine homolog 2 (C. elegans)
PSD	5662	pleckstrin and Sec7 domain containing
ZBTB16	7704	zinc finger and BTB domain containing 16
PLAGL2	5326	pleiomorphic adenoma gene-like 2
CEND1	51286	cell cycle exit and neuronal differentiation 1
RNPEPL1	57140	arginyl aminopeptidase (aminopeptidase B)-like 1
CHAF1B	8208	chromatin assembly factor 1, subunit B (p60)
PTMA	5757	prothymosin, alpha (gene sequence 28)
GNAI1	2770	guanine nucleotide binding protein (G protein), alpha inhibiting activity polypeptide 1
GNG5	2787	guanine nucleotide binding protein (G protein), gamma 5
CHKA	1119	choline kinase alpha
RFX1	5989	regulatory factor X, 1 (influences HLA class II expression)
HLA-DRB1	3123	major histocompatibility complex, class II, DR beta 1
DOCK8	81704	dedicator of cytokinesis 8
WFDC2	10406	WAP four-disulfide core domain 2
NOXO1	124056	NADPH oxidase organizer 1
KIAA0133	9816	KIAA0133
NUTF2	10204	nuclear transport factor 2

Figure 42 (Continued)

Gene Symbol	Gene ID	Gene Name
SEMA4B	10509	sema domain, immunoglobulin domain (Ig), transmembrane domain (TM) and short cytoplasmic domain, (semaphorin) 4B
PRKCZ	5590	protein kinase C, zeta
SP140	11262	SP140 nuclear body protein
CARKL	23729	carbohydrate kinase-like
HNRPM	4670	heterogeneous nuclear ribonucleoprotein M
F12	2161	coagulation factor XII (Hageman factor)
TNF	7124	tumor necrosis factor (TNF superfamily, member 2)
TRIP6	7205	thyroid hormone receptor interactor 6
SF3A2	8175	splicing factor 3a, subunit 2, 66kDa
SIX2	10736	sine oculis homeobox homolog 2 (Drosophila)
FABP1	2168	fatty acid binding protein 1, liver
QSCN6	5768	quiescin Q6
COPE	11316	coatomer protein complex, subunit epsilon
ATG16L2	89849	ATG16 autophagy related 16-like 2 (S. cerevisiae)
PRAF2	11230	PRA1 domain family, member 2
GFER	2671	growth factor, augmenter of liver regeneration (ERV1 homolog, S. cerevisiae)
HIST2H3C	126961	histone cluster 2, H3c
TSPAN4	7106	tetraspanin 4
FGF2	2247	fibroblast growth factor 2 (basic)
CENPJ	55835	centromere protein J
HRC	3270	histidine rich calcium binding protein
TRIM69	140691	tripartite motif-containing 69
UNC13B	10497	unc-13 homolog B (C. elegans)
PROSC	11212	proline synthetase co-transcribed homolog (bacterial)
NFIL3	4783	nuclear factor, interleukin 3 regulated
TACC2	10579	transforming, acidic coiled-coil containing protein 2
C2orf13	200558	chromosome 2 open reading frame 13
FGFR1	2260	fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome)
PAQR6	79957	progestin and adipoQ receptor family member VI
LOC285643	285643	KIF4B
BPGM	669	2,3-bisphosphoglycerate mutase
PLDN	26258	pallidin homolog (mouse)
KIAA0196	9897	KIAA0196
BAI3	577	brain-specific angiogenesis inhibitor 3

Figure 42 (Continued)

Gene Symbol	Gene ID	Gene Name
SCAMP4	113178	secretory carrier membrane protein 4
C18orf8	29919	chromosome 18 open reading frame 8
MKLN1	4289	muskelin 1, intracellular mediator containing kelch motifs
SLC25A19	60386	solute carrier family 25 (mitochondrial thiamine pyrophosphate carrier), member 19
UBE1L2	55236	ubiquitin-activating enzyme E1-like 2
ARSE	415	arylsulfatase E (chondrodysplasia punctata 1)
UBE2D1	7321	ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast)
TRIM8	81603	tripartite motif-containing 8
ICT1	3396	immature colon carcinoma transcript 1
GNG11	2791	guanine nucleotide binding protein (G protein), gamma 11
TUBGCP6	85378	tubulin, gamma complex associated protein 6
SIDT1	54847	SID1 transmembrane family, member 1
MATN3	4148	matrilin 3
LOR	4014	loricrin
SORBS3	10174	sorbin and SH3 domain containing 3

Figure 43

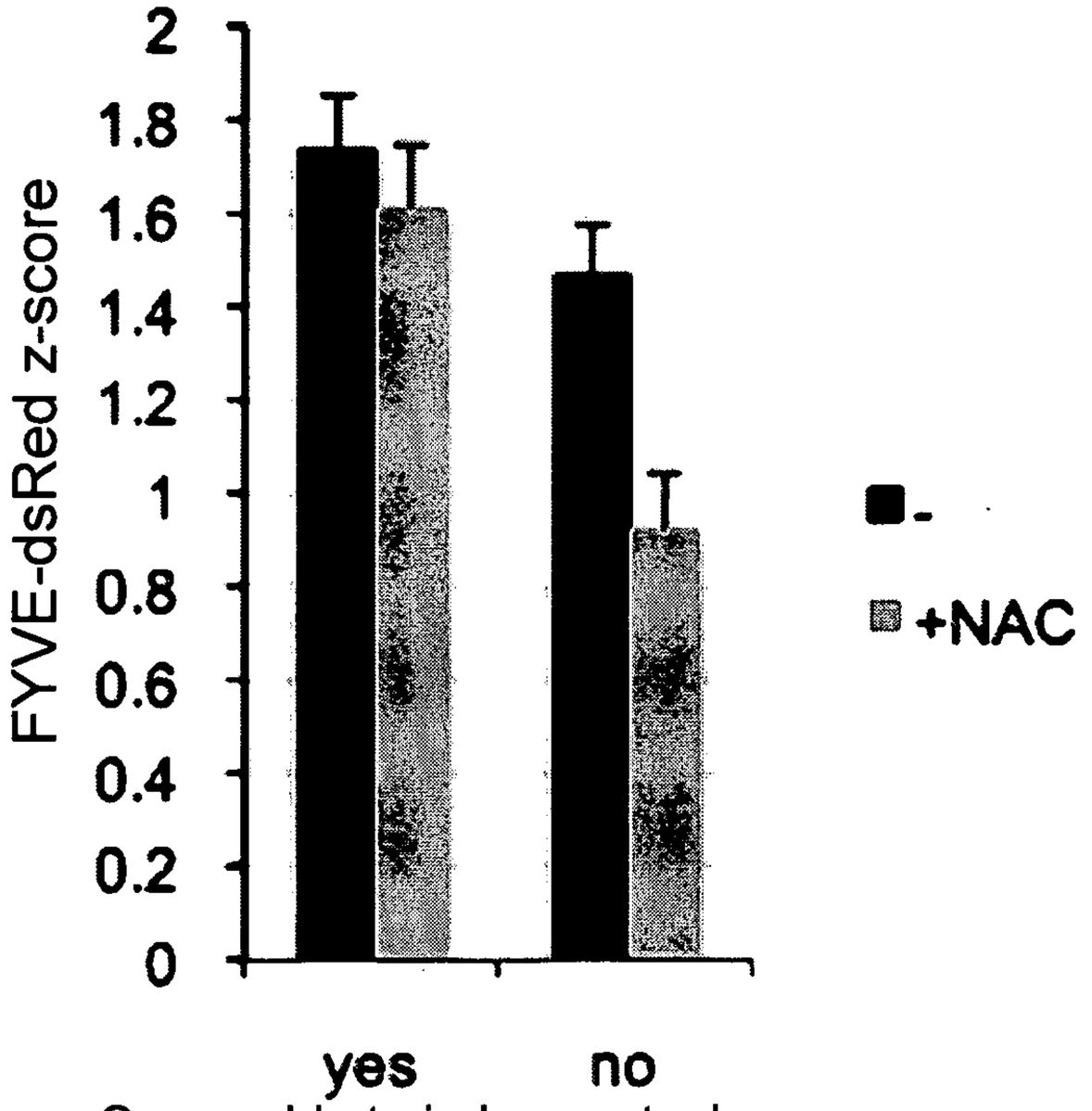

FYVE-dsRed z-score
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8
-
+NAC
yes
no
Genes able to induce autophagy
in presence of NAC

Figure 44

Genes whose knock-down induces autophagy in the presence of antioxidant.

Gene Symbol	Gene ID	Gene Name
CKAP5	9793	cytoskeleton associated protein 5
DDX24	57062	DEAD (Asp-Glu-Ala-Asp) box polypeptide 24
XPO1	7514	exportin 1 (CRM1 homolog, yeast)
RAB7A	7879	RAB7A, member RAS oncogene family
PPARD	5467	peroxisome proliferator-activated receptor delta
CENPE	1062	centromere protein E, 312kDa
HNRPK	3190	heterogeneous nuclear ribonucleoprotein K
ITGAV	3685	integrin, alpha V (vitronectin receptor, alpha polypeptide, antigen CD51)
TRPM3	80036	transient receptor potential cation channel, subfamily M, member 3
PNKD	25953	paroxysmal nonkinesiogenic dyskinesia
CAPN1	823	calpain 1, (mu/I) large subunit
PTCRA	171558	pre T-cell antigen receptor alpha
TLR3	7098	toll-like receptor 3
ZFY	7544	zinc finger protein, Y-linked
KIF11	3832	kinesin family member 11
EP300	2033	E1A binding protein p300
SORCS2	57537	sortilin-related VPS10 domain containing receptor 2
C5AR1	728	complement component 5a receptor 1
HOXD11	3237	homeobox D11
PRKCA	5578	protein kinase C, alpha
GHSR	2693	growth hormone secretagogue receptor
NCR3	259197	natural cytotoxicity triggering receptor 3
TMPRSS5	80975	transmembrane protease, serine 5 (spinesin)
IRAK3	11213	interleukin-1 receptor-associated kinase 3
CAMKV	79012	CaM kinase-like vesicle-associated
PTGER2	5732	prostaglandin E receptor 2 (subtype EP2), 53kDa
CDKN2D	1032	cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4)
NTN2L	4917	netrin 2-like (chicken)
FCER1A	2205	Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide
SSPN	8082	sarcospan (Kras oncogene-associated gene)
PPFIA4	8497	protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4
OGDH	4967	oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide)

Figure 44 (Continued)

Gene Symbol	Gene ID	Gene Name
TRHR	7201	thyrotropin-releasing hormone receptor
CCT4	10575	chaperonin containing TCP1, subunit 4 (delta)
HIVEP2	3097	human immunodeficiency virus type I enhancer binding protein 2
GRK6	2870	G protein-coupled receptor kinase 6
RELN	5649	reelin
SUV39H1	6839	suppressor of variegation 3-9 homolog 1 (Drosophila)
TINP1	10412	TGF beta-inducible nuclear protein 1
CHRND	1144	cholinergic receptor, nicotinic, delta
ERH	2079	enhancer of rudimentary homolog (Drosophila)
TUBB2A	7280	tubulin, beta 2A
BAIAP2	10458	BAI1-associated protein 2
FXYD2	486	FXYD domain containing ion transport regulator 2
RANGAP1	5905	Ran GTPase activating protein 1
ARCN1	372	archain 1
COPB2	9276	coatomer protein complex, subunit beta 2 (beta prime)
SDHB	6390	succinate dehydrogenase complex, subunit B, iron sulfur (Ip)
PFDN2	5202	prefoldin subunit 2
TNFRSF19L	84957	tumor necrosis factor receptor superfamily, member 19-like
UTS2R	2837	urotensin 2 receptor
NAGK	55577	N-acetylglucosamine kinase
CDK5RAP3	80279	CDK5 regulatory subunit associated protein 3
GJA4	2701	gap junction protein, alpha 4, 37kDa
PHB2	11331	prohibitin 2
GJA3	2700	gap junction protein, alpha 3, 46kDa
MYL3	4634	myosin, light chain 3, alkali; ventricular, skeletal, slow
CXCL12	6387	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)
PFKL	5211	phosphofructokinase, liver
TRAF1	7185	TNF receptor-associated factor 1
NPTX1	4884	neuronal pentraxin I
C11orf68	83638	chromosome 11 open reading frame 68
PCGF1	84759	polycomb group ring finger 1
TOM1	10043	target of myb1 (chicken)
RORC	6097	RAR-related orphan receptor C
KRT18	3875	keratin 18
AURKA	6790	aurora kinase A

Figure 44 (Continued)

Gene Symbol	Gene ID	Gene Name
GNRH2	2797	gonadotropin-releasing hormone 2
HMBS	3145	hydroxymethylbilane synthase
INTS4	92105	integrator complex subunit 4
TACR2	6865	tachykinin receptor 2
PTPRH	5794	protein tyrosine phosphatase, receptor type, H
GABBR2	9568	gamma-aminobutyric acid (GABA) B receptor, 2
FBXL20	84961	F-box and leucine-rich repeat protein 20
RAGE	5891	renal tumor antigen
CLCF1	23529	cardiotrophin-like cytokine factor 1
GTF2IRD2	84163	GTF2I repeat domain containing 2
MMP10	4319	matrix metallopeptidase 10 (stromelysin 2)
PLCH2	9651	phospholipase C, eta 2
BGN	633	biglycan
GCM2	9247	glial cells missing homolog 2 (Drosophila)
CD300C	10871	CD300c molecule
RIPK1	8737	receptor (TNFRSF)-interacting serine-threonine kinase 1
CDCA8	55143	cell division cycle associated 8
TH	7054	tyrosine hydroxylase
COL14A1	7373	collagen, type XIV, alpha 1 (undulin)
ADMR	11318	adrenomedullin receptor
USP19	10869	ubiquitin specific peptidase 19
PTPRU	10076	protein tyrosine phosphatase, receptor type, U
EPHA6	203806	EPH receptor A6
PRKAG3	53632	protein kinase, AMP-activated, gamma 3 non-catalytic subunit
HNRPU	3192	heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A)
THBS2	7058	thrombospondin 2
HSFY2	159119	heat shock transcription factor, Y linked 2
CLCN1	1180	chloride channel 1, skeletal muscle (Thomsen disease, autosomal dominant)
SETDB1	9869	SET domain, bifurcated 1
LOC388959	388959	hypothetical LOC388959
NAT9	26151	N-acetyltransferase 9

Figure 45

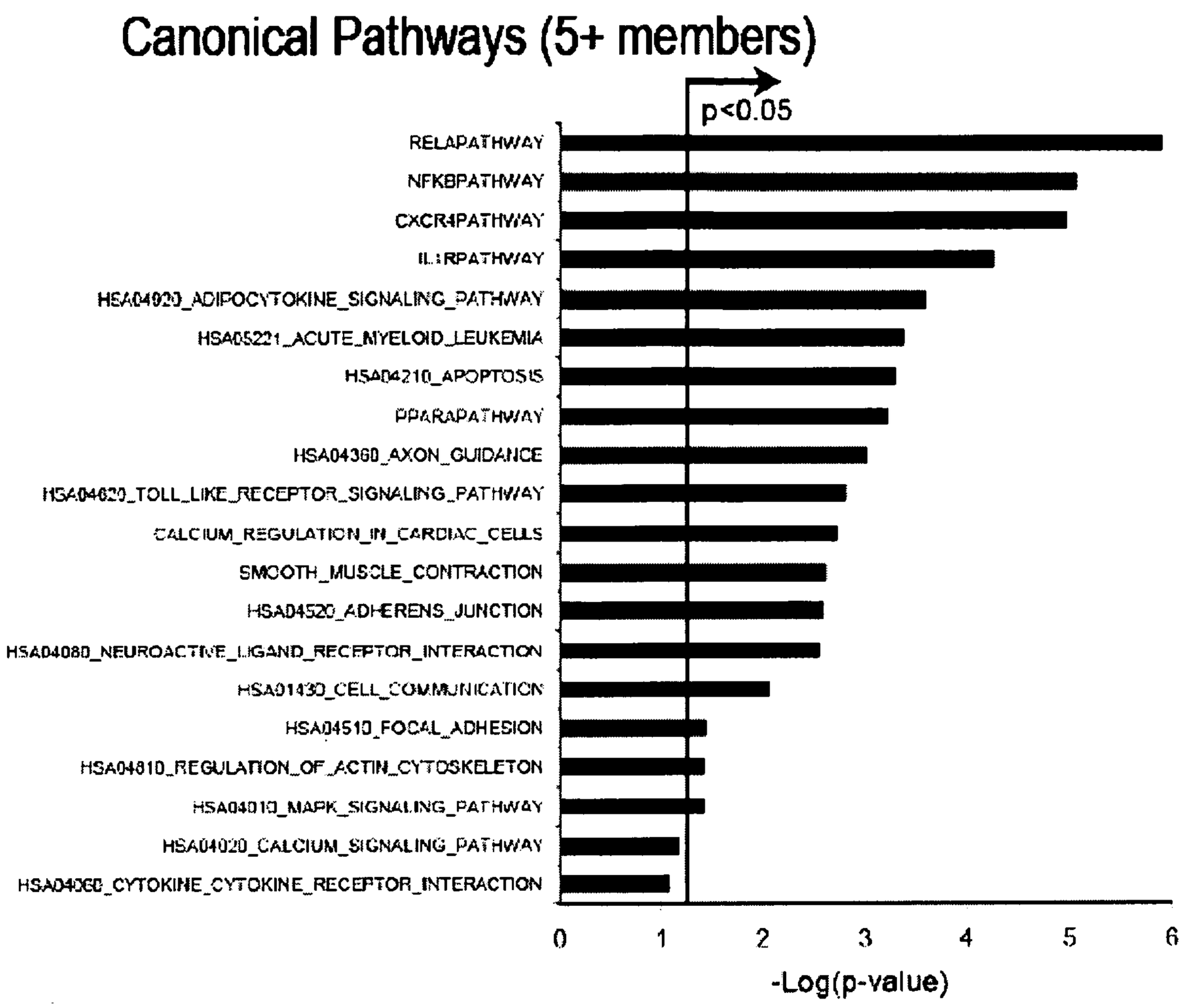

Canonical Pathways (5+ members)
p<0.05
RELAPATHWAY
NFKBPATHWAY
CXCR4PATHWAY
IL1RPATHWAY
HSA04920_ADIPOCYTOKINE_SIGNALING_PATHWAY
HSA05221_ACUTE_MYELOID_LEUKEMIA
HSA04210_APOPTOSIS
PPARAPATHWAY
HSA04360_AXON_GUIDANCE
HSA04620_TOLL_LIKE_RECEPTOR_SIGNALING_PATHWAY
CALCIUM_REGULATION_IN_CARDIAC_CELLS
SMOOTH_MUSCLE_CONTRACTION
HSA04520_ADHERENS_JUNCTION
HSA04080_NEUROACTIVE_LIGAND_RECEPTOR_INTERACTION
HSA01430_CELL_COMMUNICATION
HSA04510_FOCAL_ADHESION
HSA04810_REGULATION_OF_ACTIN_CYTOSKELETON
HSA04010_MAPK_SIGNALING_PATHWAY
HSA04020_CALCIUM_SIGNALING_PATHWAY
HSA04060_CYTOKINE_CYTOKINE_RECEPTOR_INTERACTION
0
1
2
3
4
5
6
-Log(p-value)

Figure 46

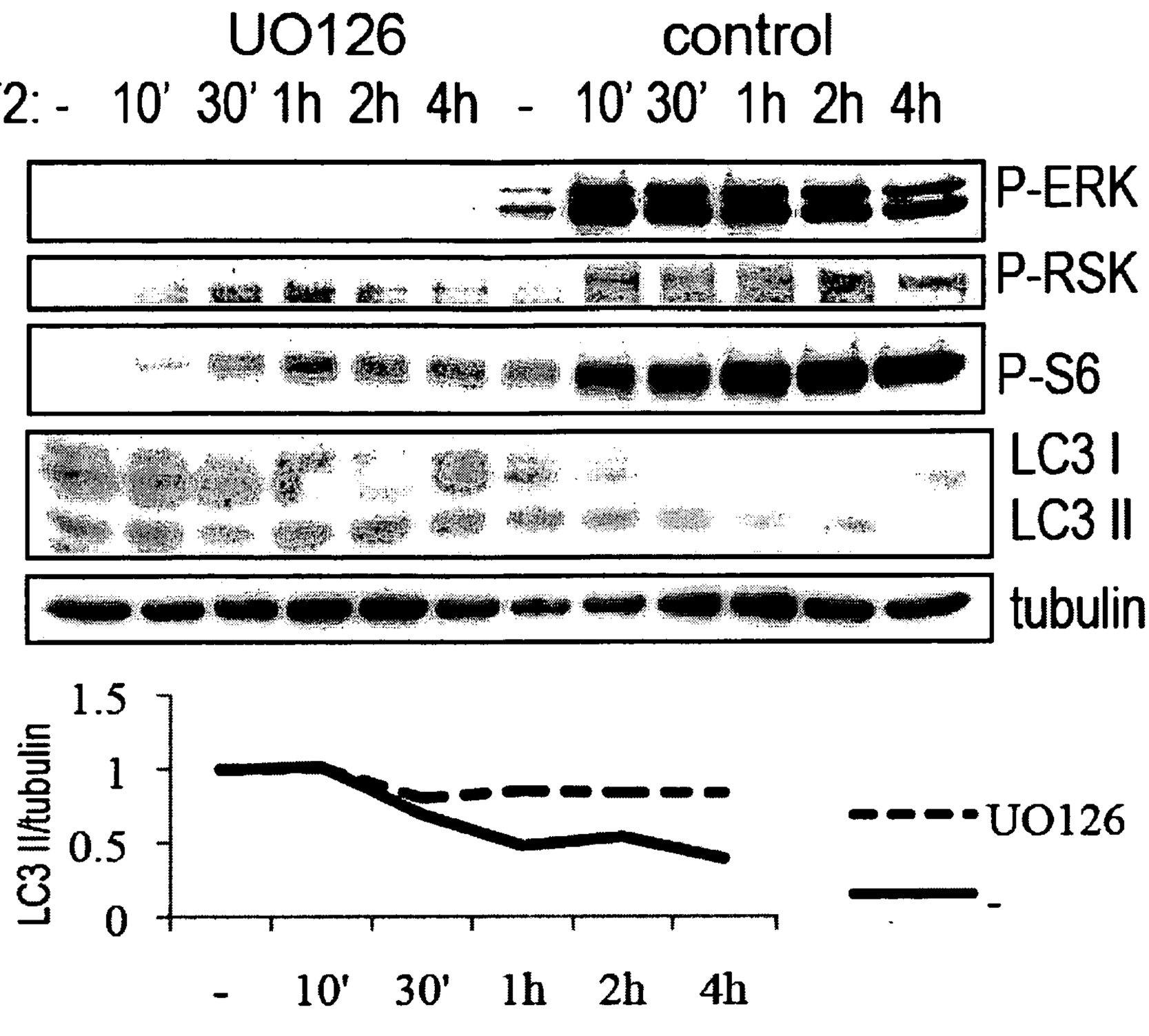

UO126
control
FGF2: - 10' 30' 1h 2h 4h - 10' 30' 1h 2h 4h
P-ERK
P-RSK
P-S6
LC3 I
LC3 II
tubulin
LC3 II/tubulin
1.5
1
0.5
0
UO126
-
- 10' 30' 1h 2h 4h

Figure 47

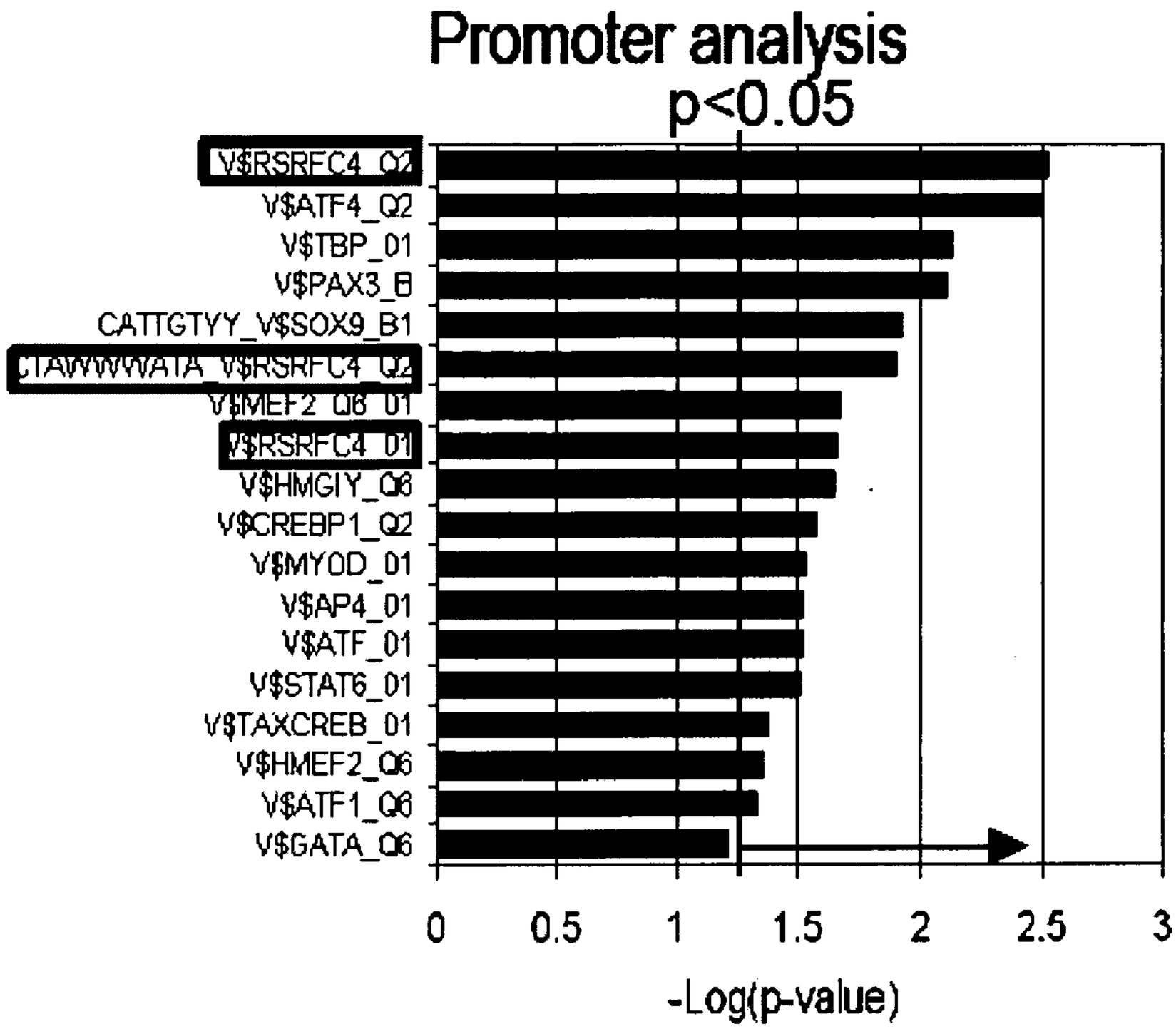

Promoter analysis
p<0.05
V$RSRFC4_Q2
V$ATF4_Q2
V$TBP_01
V$PAX3_B
CATTGTYY_V$SOX9_B1
CTAWWWATA_V$RSRFC4_Q2
V$MEF2_Q6_01
V$RSRFC4_01
V$HMGIY_Q6
V$CREBP1_Q2
V$MYOD_01
V$AP4_01
V$ATF_01
V$STAT6_01
V$TAXCREB_01
V$HMEF2_Q6
V$ATF1_Q6
V$GATA_Q6
0
0.5
1
1.5
2
2.5
3
-Log(p-value)

Figure 48

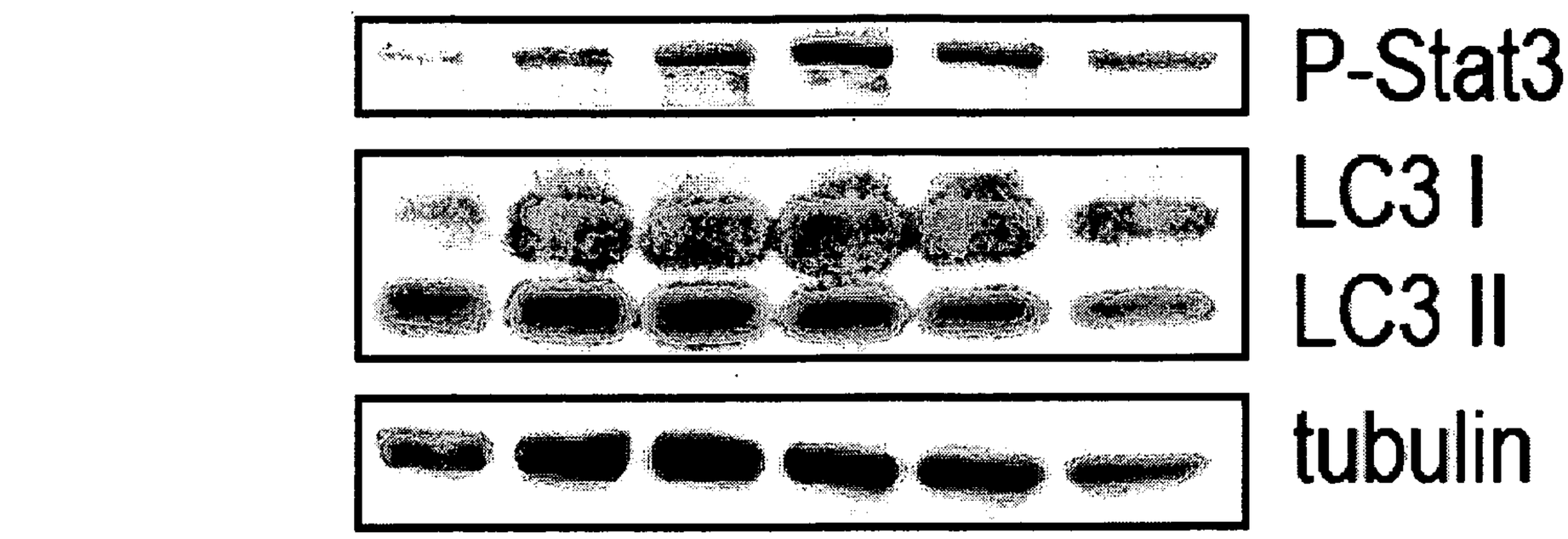


Figure 49

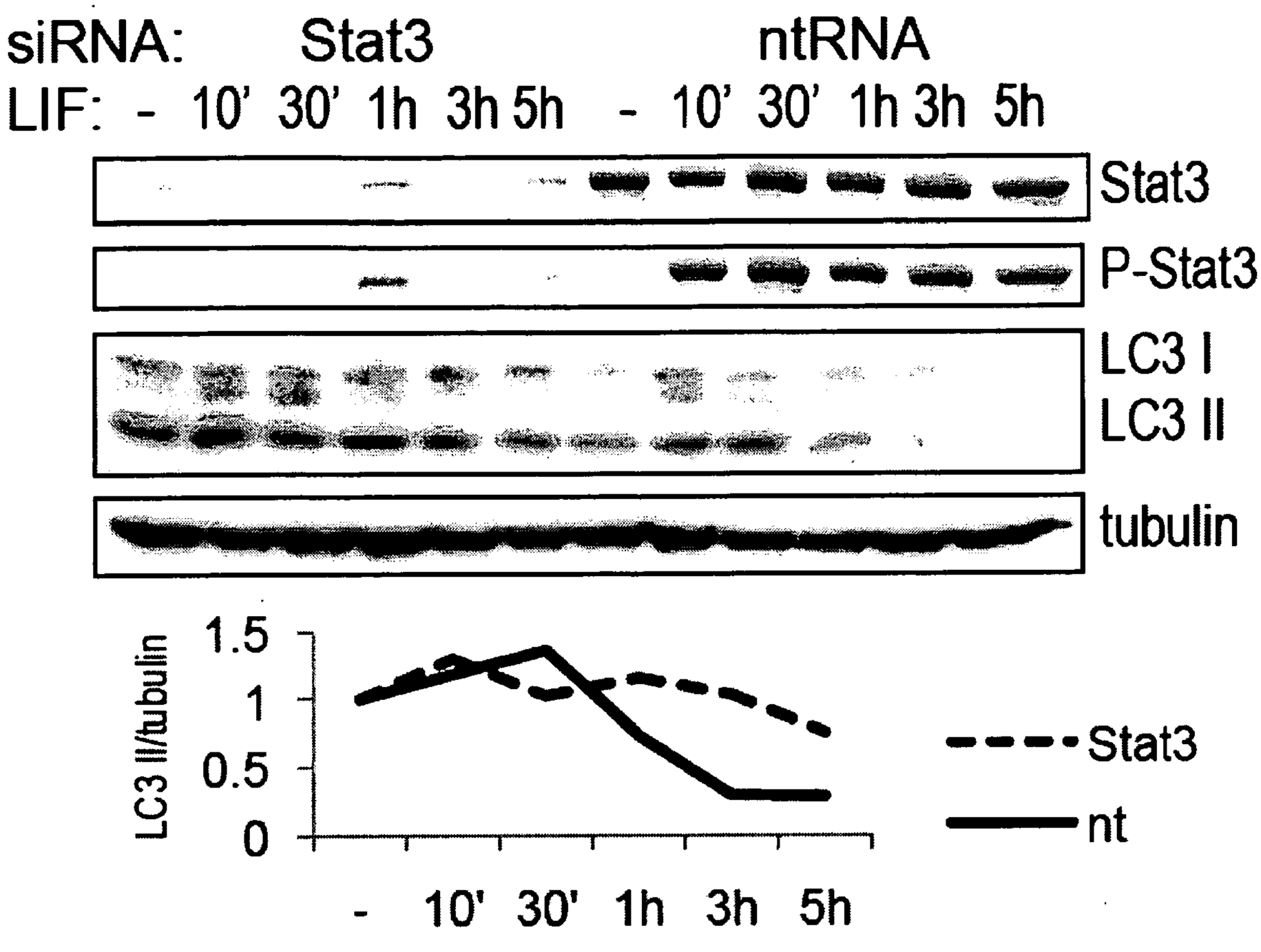

siRNA: Stat3 ntRNA
LIF: - 10' 30' 1h 3h 5h - 10' 30' 1h 3h 5h
Stat3
P-Stat3
LC3 I
LC3 II
tubulin
LC3 II/tubulin
1.5
1
0.5
0
- 10' 30' 1h 3h 5h
Stat3
nt

Figure 50

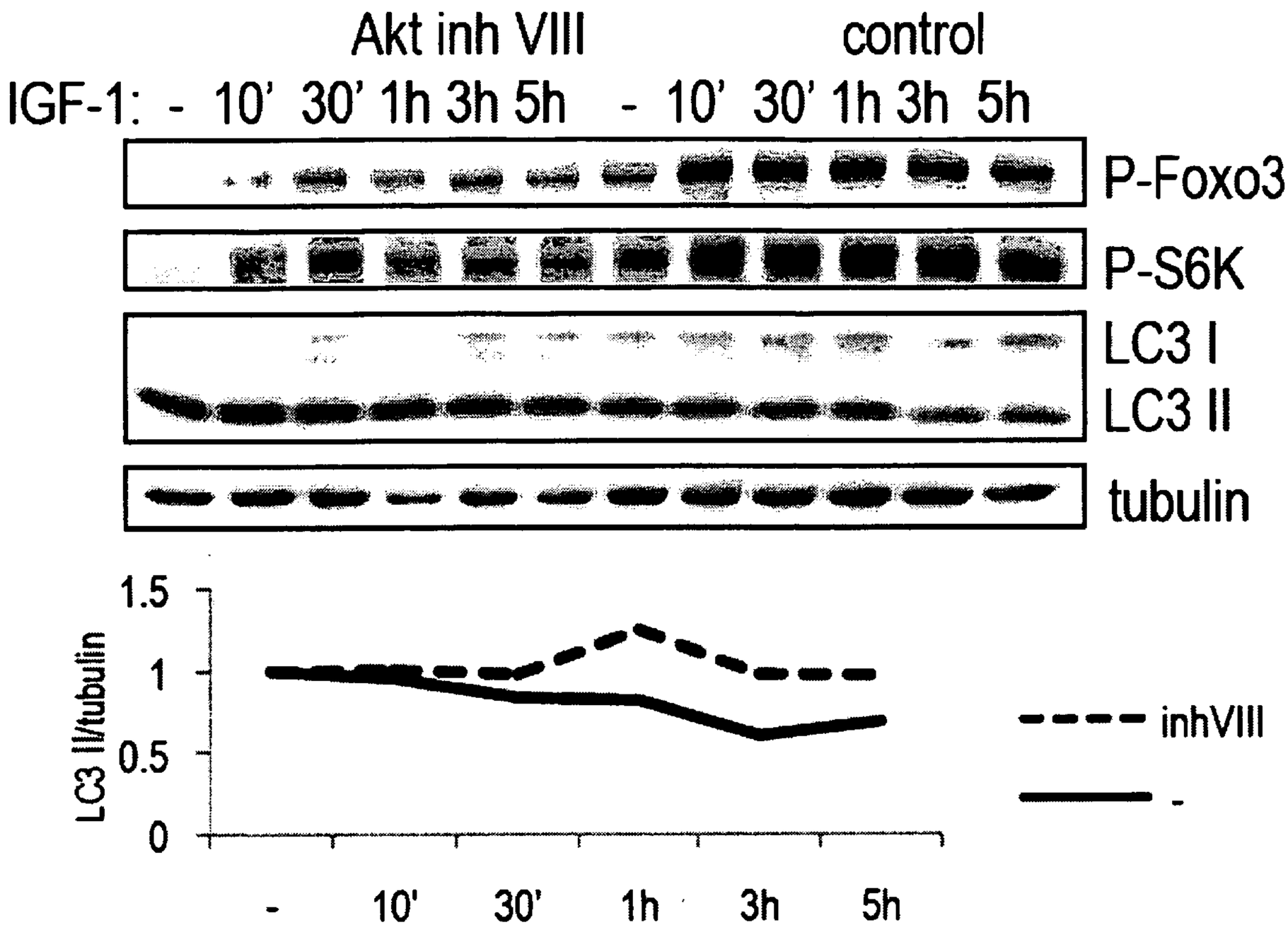

Akt inh VIII
control
IGF-1: - 10' 30' 1h 3h 5h - 10' 30' 1h 3h 5h
P-Foxo3
P-S6K
LC3 I
LC3 II
tubulin
LC3 II/tubulin
1.5
1
0.5
0
- 10' 30' 1h 3h 5h
inhVIII
-

Figure 51

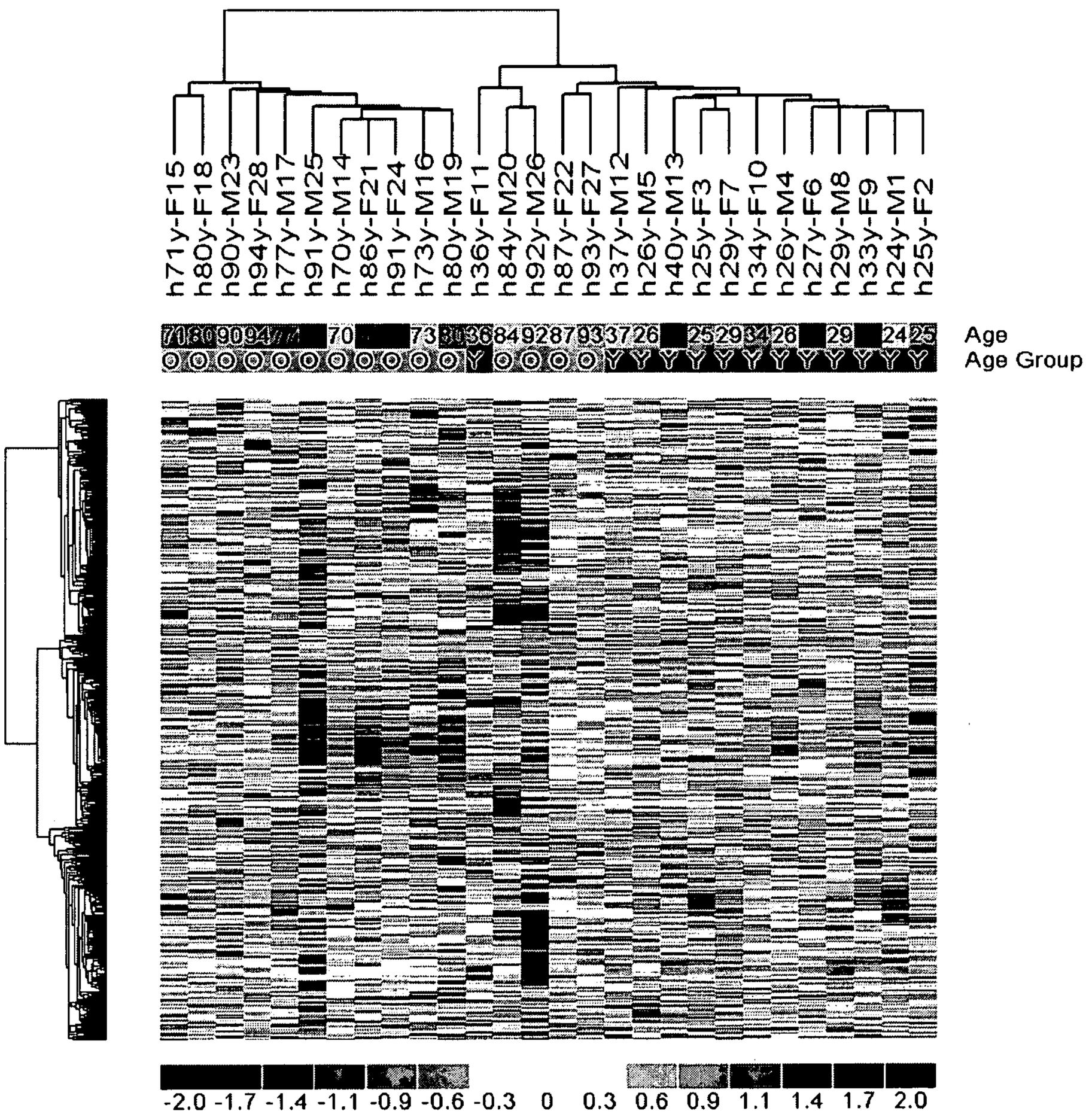

F15
F18
M23
F28
M17
M25
M14
F21
F24
M16
M19
F11
M20
M26
F22
F27
M12
M5
M13
F3
F7
F10
M4
F6
M8
F9
M1
F2
h71y-
h80y-
h90y-
h94y-
h77y-
h91y-
h70y-
h86y-
h91y-
h73y-
h80y-
h36y-
h84y-
h92y-
h87y-
h93y-
h37y-
h26y-
h40y-
h25y-
h29y-
h34y-
h26y-
h27y-
h29y-
h33y-
h24y-
h25y-
71 80 90 94 77 70 73 36 84 92 87 93 37 26 25 29 34 26 29 24 25
Age
Age Group
-2.0 -1.7 -1.4 -1.1 -0.9 -0.6 -0.3 0 0.3 0.6 0.9 1.1 1.4 1.7 2.0

Figure 52

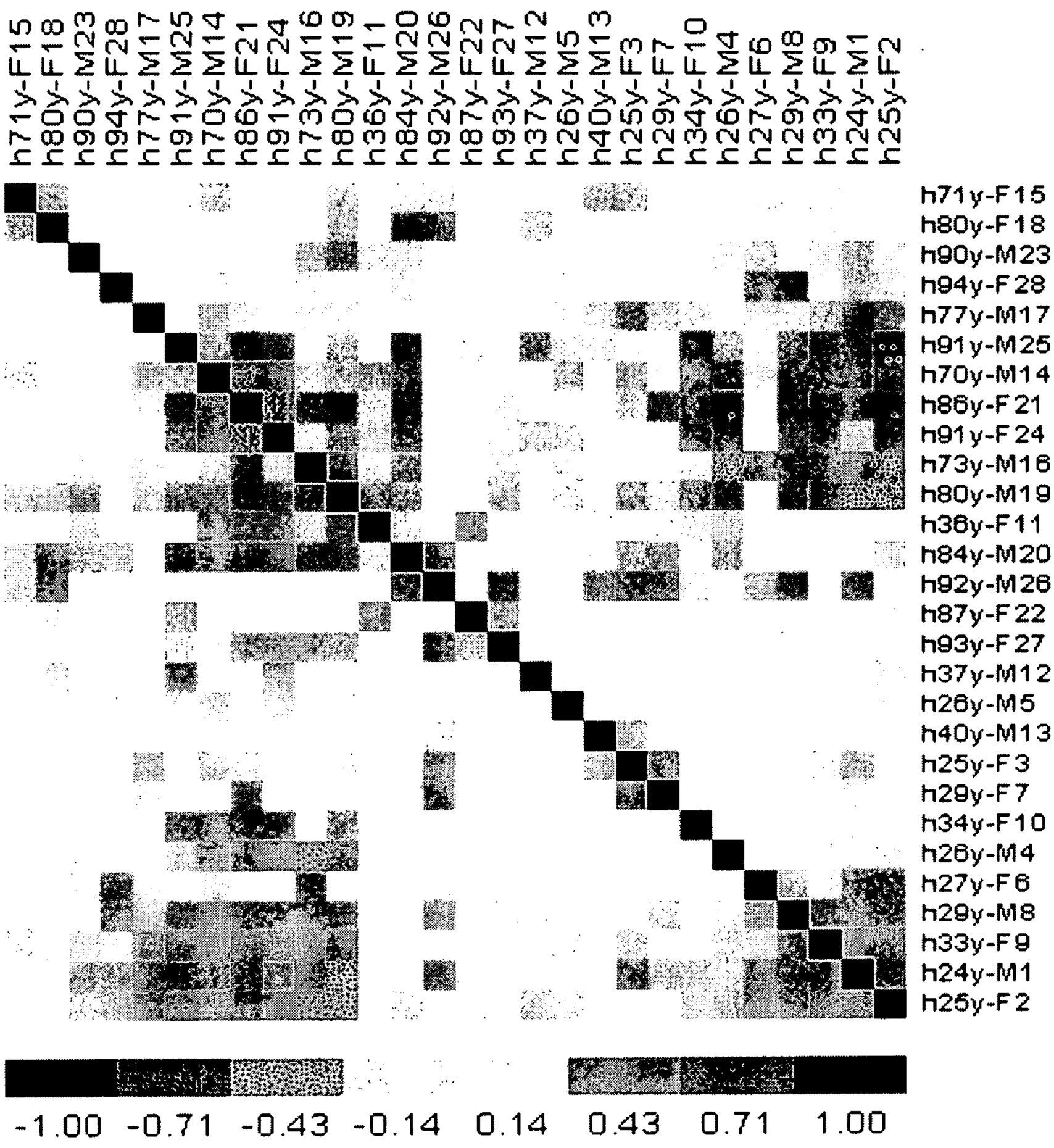

h71y-F15
h80y-F18
h90y-M23
h94y-F28
h77y-M17
h91y-M25
h70y-M14
h86y-F21
h91y-F24
h73y-M16
h80y-M19
h36y-F11
h84y-M20
h92y-M26
h87y-F22
h93y-F27
h37y-M12
h26y-M5
h40y-M13
h25y-F3
h29y-F7
h34y-F10
h26y-M4
h27y-F6
h29y-M8
h33y-F9
h24y-M1
h25y-F2
-1.00 -0.71 -0.43 -0.14 0.14 0.43 0.71 1.00

Figure 53

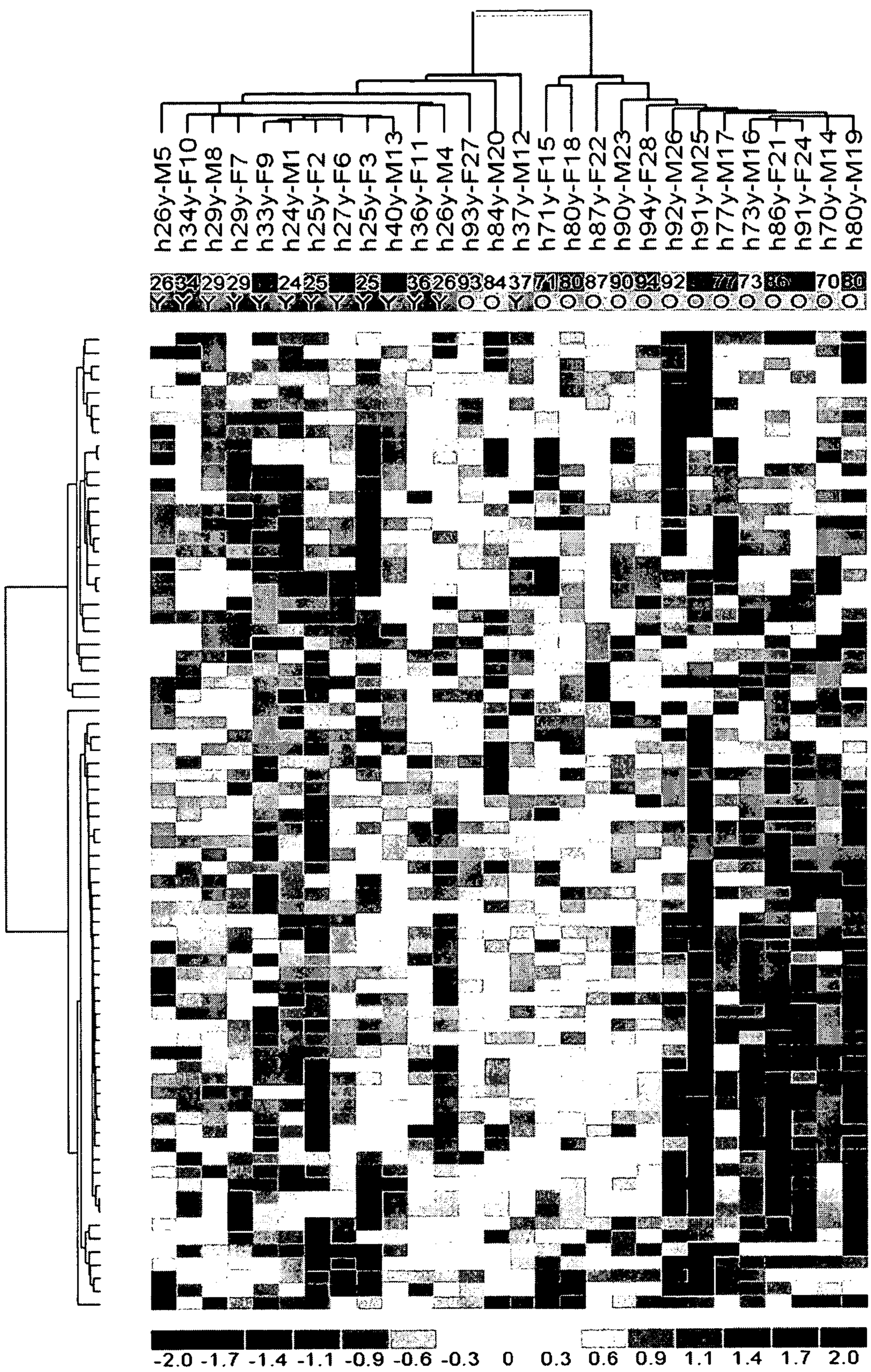

h26y-M5
h34y-F10
h29y-M8
h29y-F7
h33y-F9
h24y-M1
h25y-F2
h27y-F6
h25y-F3
h40y-M13
h36y-F11
h26y-M4
h93y-F27
h84y-M20
h37y-M12
h71y-F15
h80y-F18
h87y-F22
h90y-M23
h94y-F28
h92y-M26
h91y-M25
h77y-M17
h73y-M16
h86y-F21
h91y-F24
h70y-M14
h80y-M19
-2.0 -1.7 -1.4 -1.1 -0.9 -0.6 -0.3 0 0.3 0.6 0.9 1.1 1.4 1.7 2.0

Figure 54

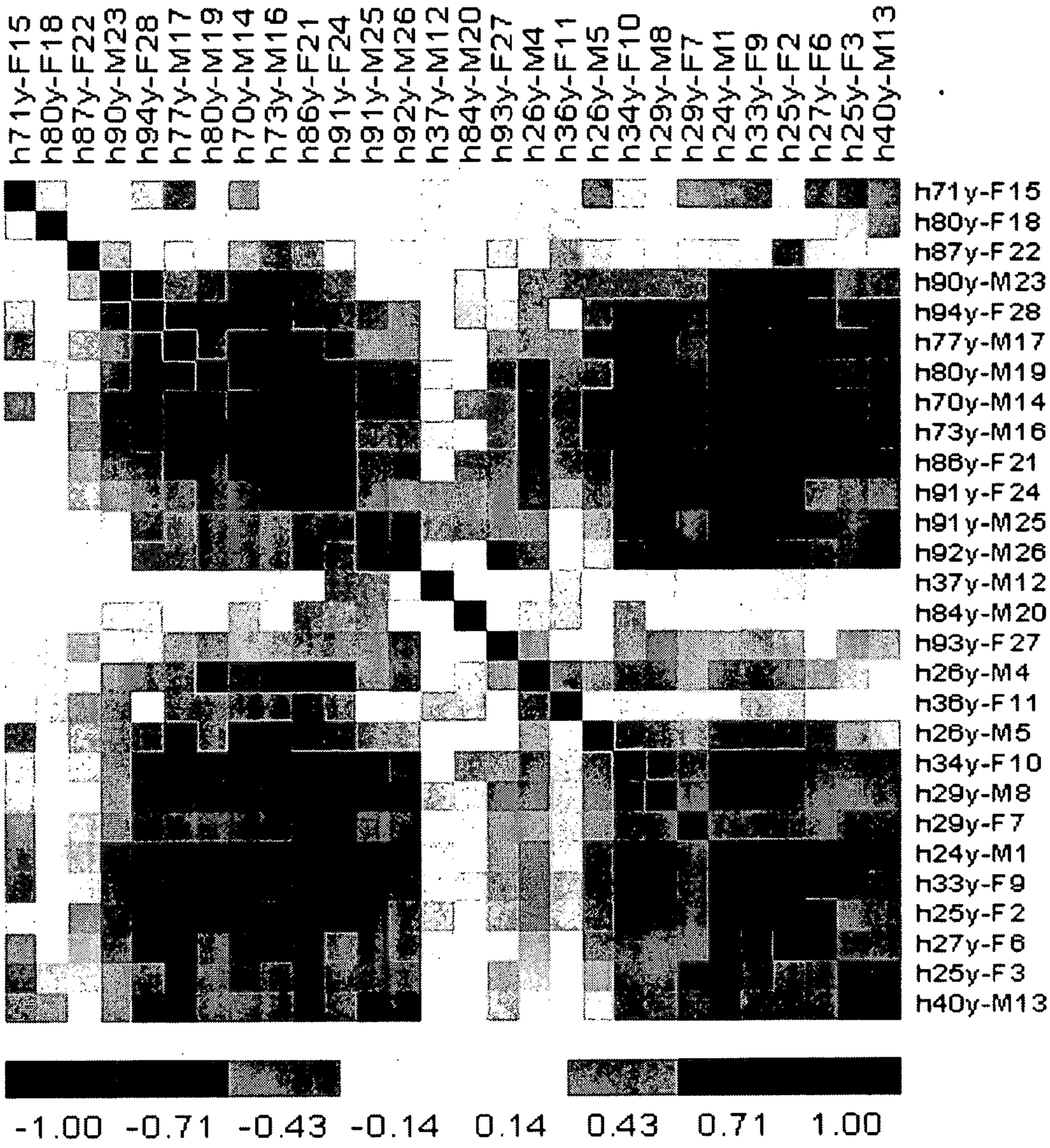

h71y-F15
h80y-F18
h87y-F22
h90y-M23
h94y-F28
h77y-M17
h80y-M19
h70y-M14
h73y-M16
h86y-F21
h91y-F24
h91y-M25
h92y-M26
h37y-M12
h84y-M20
h93y-F27
h26y-M4
h36y-F11
h26y-M5
h34y-F10
h29y-M8
h29y-F7
h24y-M1
h33y-F9
h25y-F2
h27y-F6
h25y-F3
h40y-M13
-1.00 -0.71 -0.43 -0.14 0.14 0.43 0.71 1.00

Figure 55.

Autophagy hit genes differentially regulated in human brain during aging.

Gene Symbol	Gene ID	fold change	P value
PLDN	26258	-1.87	0
NLK	51701	-1.74	0.000001
COX5A	9377	-1.56	0.000001
CAMKV	79012	-1.55	0.000001
HIVEP2	3097	-1.96	0.000002
SDHB	6390	-1.57	0.000004
GABBR2	9568	-1.55	0.000007
CNKSR2	22866	-1.7	0.000008
GABBR2	9568	-1.82	0.000009
HIVEP2	3097	-1.48	0.00001
GABBR2	9568	-1.78	0.000014
NLK	51701	-1.54	0.00004
TUBB2A	7280	-1.37	0.000056
NLK	51701	-1.95	0.000056
PLDN	26258	-1.65	0.000075
ATG5	9474	-1.39	0.000083
CNKSR2	22866	-1.74	0.000095
UBE2D1	7321	-1.62	0.000097
SMYD3	64754	-1.38	0.000108
PRKCZ	5590	-1.35	0.000115
ATG5	9474	-1.43	0.000115
CCT4	10575	-1.42	0.000133
BAI3	577	-1.36	0.000148
IGF1	3479	-2.2	0.000193
RANGAP1	5905	-1.47	0.00065
ARCN1	372	-1.22	0.000786
BAIAP2	10458	-1.45	0.001107
GABBR2	9568	-1.28	0.001125
PLCH2	9651	-1.68	0.001224
TACC2	10579	-1.49	0.001403
KIF5C	3800	-1.43	0.001787
CDKN2D	1032	-1.41	0.001867
PA2G4	5036	-1.3	0.002141
ATG5	9474	-1.42	0.002334
KIF5C	3800	-1.21	0.00252
RELN	5649	-1.69	0.002788
GTPBP4	23560	-1.25	0.003265

Figure 55 (Continued)

Gene Symbol	Gene ID	fold change	P value
GTPBP4	23560	-1.36	0.003307
PDCD5	9141	-1.28	0.003321
CPNE6	9362	-1.63	0.00385
TRNT1	51095	-1.49	0.004337
GNAI1	2770	-1.32	0.005034
PAK6	56924	-1.33	0.007166
ATG7	10533	-1.32	0.012585
PAK6	56924	-1.38	0.013333
ATG16L2	89849	-1.39	0.020407
MEGF10	84466	1.86	0.000054
USP54	159195	1.53	0.000059
ZBTB16	7704	1.38	0.00039
CROP	51747	1.43	0.000489
GAB1	2549	1.35	0.001026
PTMA	5757 /// 643287	1.26	0.001133
SSPN	8082	1.6	0.001447
NUPR1	26471	2.22	0.002563
SORBS3	10174	1.31	0.002784
MBP	4155	1.78	0.00307
FGF2	2247	1.63	0.00338
ITGAV	3685	1.31	0.003674
FGFR1	2260	1.31	0.003808
GRK6	2870	1.52	0.003929
SDHB	6390	2.01	0.006541
IHPK3	117283	1.49	0.007656
PTMA	5757	1.35	0.008051
HMBS	3145	1.34	0.00915
BGN	10194	1.73	0.011653
PTMA	5757	1.3	0.014135
GNG5	2787	1.31	0.015485
PAQR6	79957	1.44	0.015659
TRIP6	7205	1.36	0.019916
MBP	4155	1.38	0.02253
TRAF1	7185	1.35	0.022563
BGN	633	1.71	0.023652
RORC	6097	1.39	0.025701
C5AR1	728	2.77	0.033053

Figure 56.

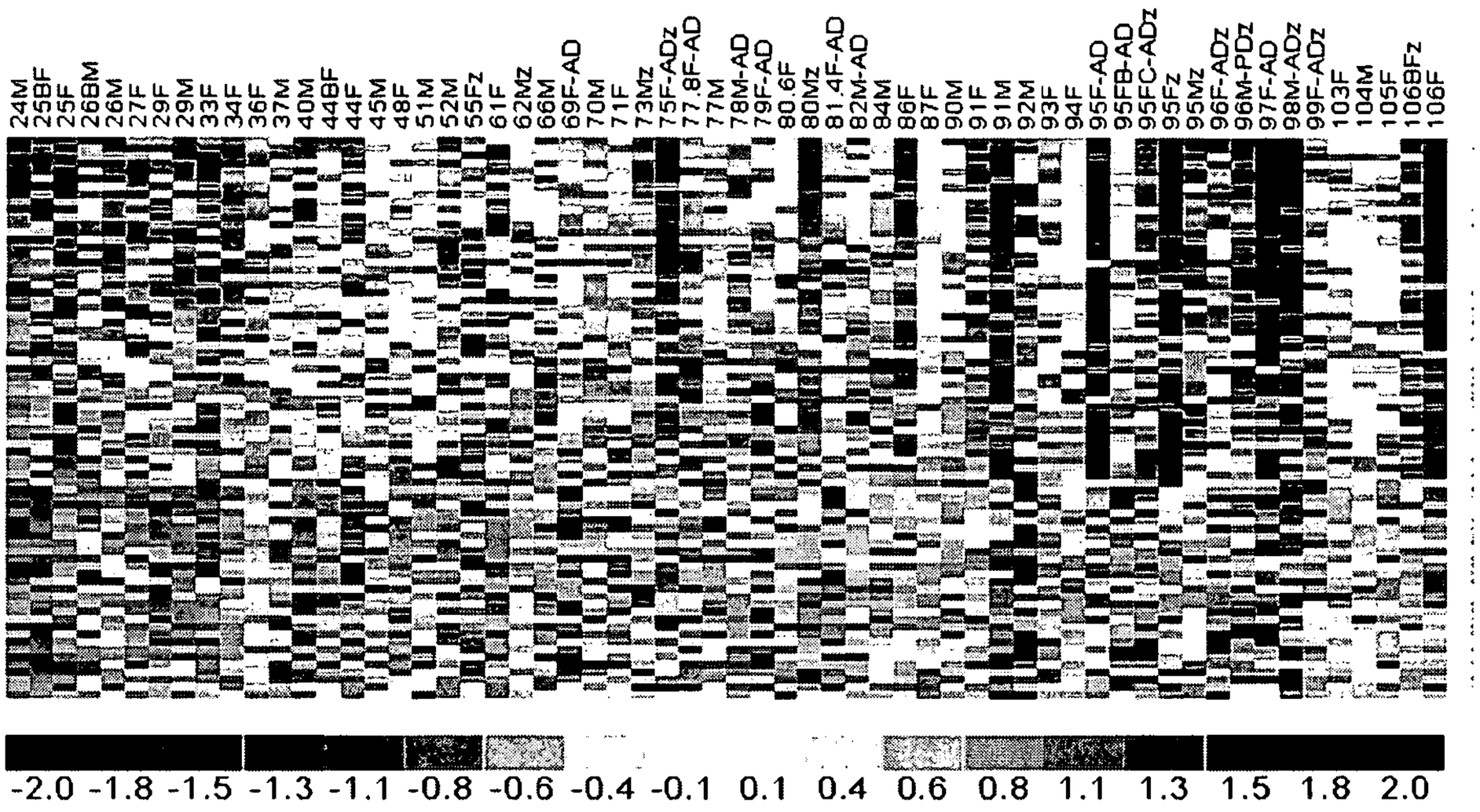

24M
25BF
25F
26BM
26M
27F
29F
29M
33F
34F
36F
37M
40M
44BF
44F
45M
48F
51M
52M
55Fz
61F
62Mz
66M
69F-AD
70M
71F
73Mz
75F-ADz
77.8F-AD
77M
78M-AD
79F-AD
80.6F
80Mz
81.4F-AD
82M-AD
84M
86F
87F
90M
91F
91M
92M
93F
94F
95F-AD
95FB-AD
95FC-ADz
95Fz
95Mz
96F-ADz
96M-PDz
97F-AD
98M-ADz
99F-ADz
103F
104M
105F
106BFz
106F
-2.0 -1.8 -1.5 -1.3 -1.1 -0.8 -0.6 -0.4 -0.1 0.1 0.4 0.6 0.8 1.1 1.3 1.5 1.8 2.0

Figure 57

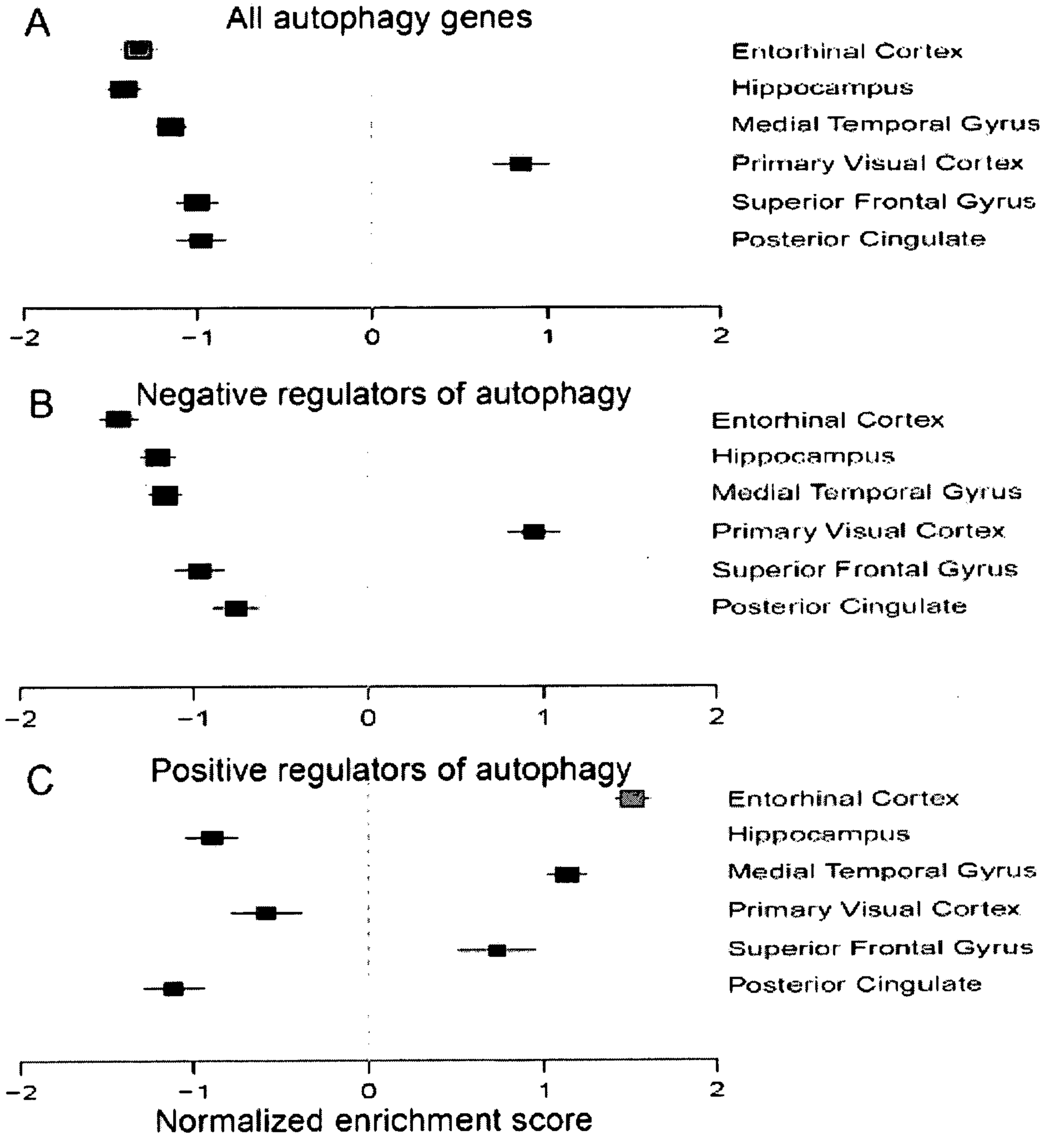

A
All autophagy genes
Entorhinal Cortex
Hippocampus
Medial Temporal Gyrus
Primary Visual Cortex
Superior Frontal Gyrus
Posterior Cingulate
−2
−1
0
1
2
B
Negative regulators of autophagy
Entorhinal Cortex
Hippocampus
Medial Temporal Gyrus
Primary Visual Cortex
Superior Frontal Gyrus
Posterior Cingulate
−2
−1
0
1
2
C
Positive regulators of autophagy
Entorhinal Cortex
Hippocampus
Medial Temporal Gyrus
Primary Visual Cortex
Superior Frontal Gyrus
Posterior Cingulate
−2
−1
0
1
2
Normalized enrichment score

Figure 58

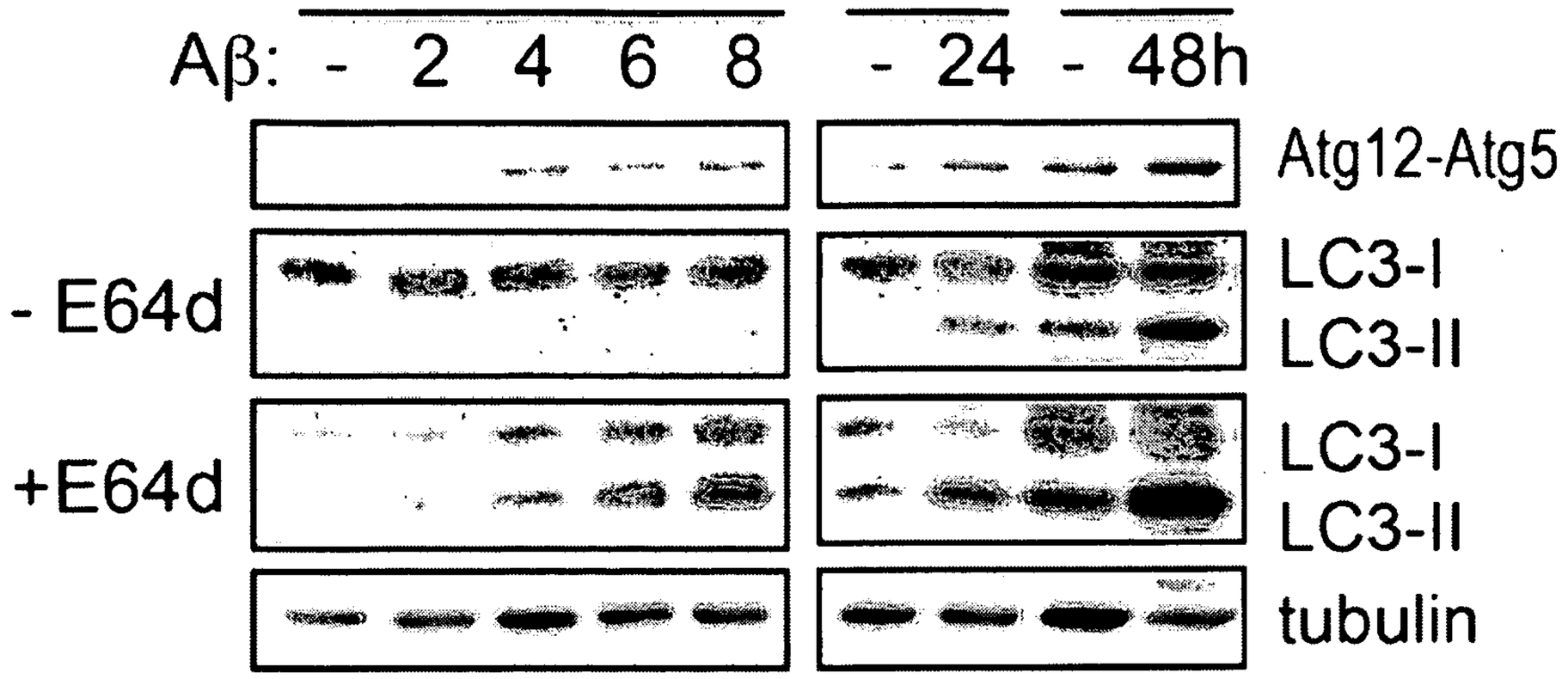

Aβ: - 2 4 6 8 - 24 - 48h
- E64d
+E64d
Atg12-Atg5
LC3-I
LC3-II
LC3-I
LC3-II
tubulin

Figure 59

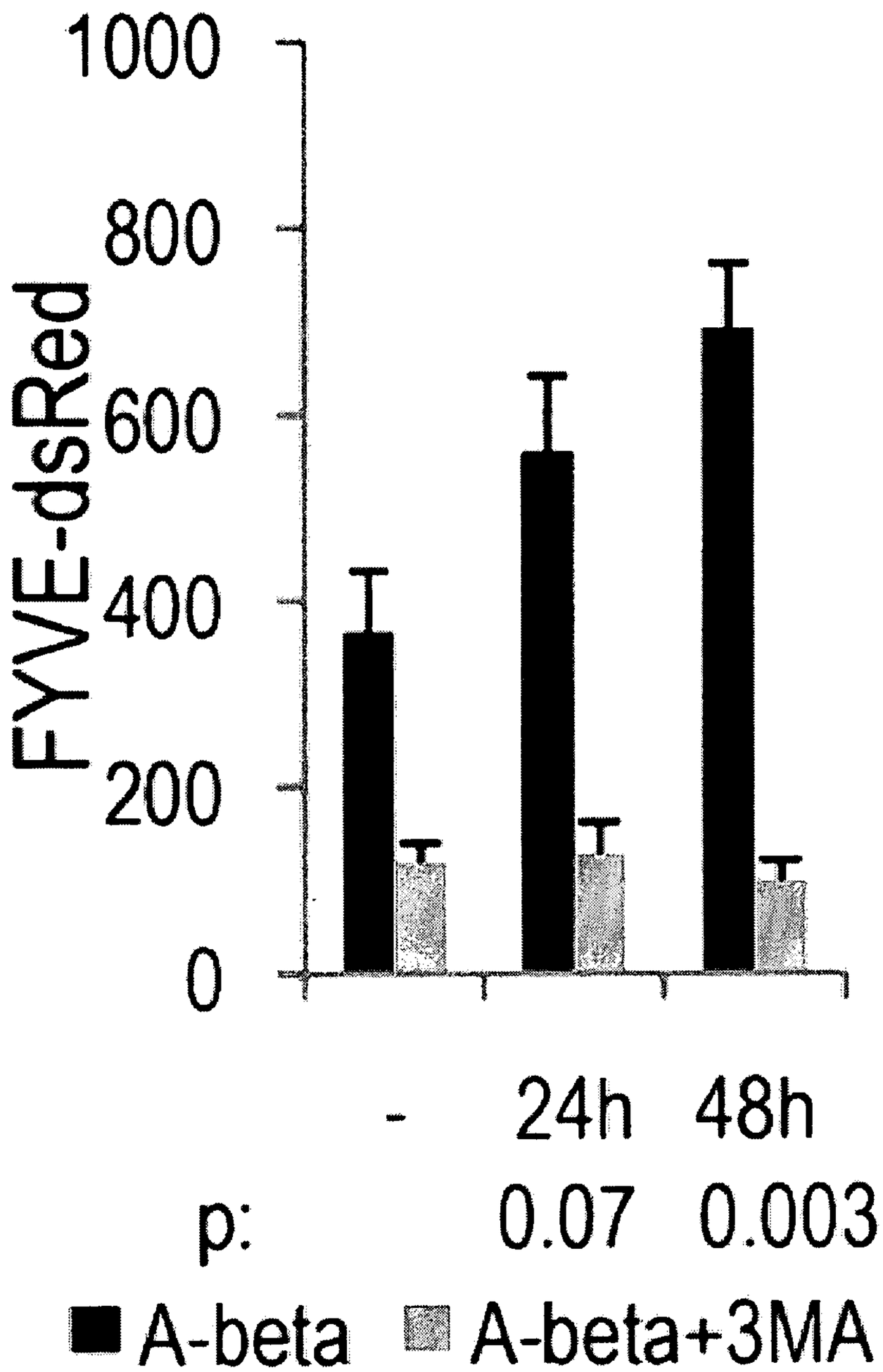

1000
800
600
400
200
0
FYVE-dsRed
-
24h
48h
p:
0.07
0.003
A-beta
A-beta+3MA

Figure 60

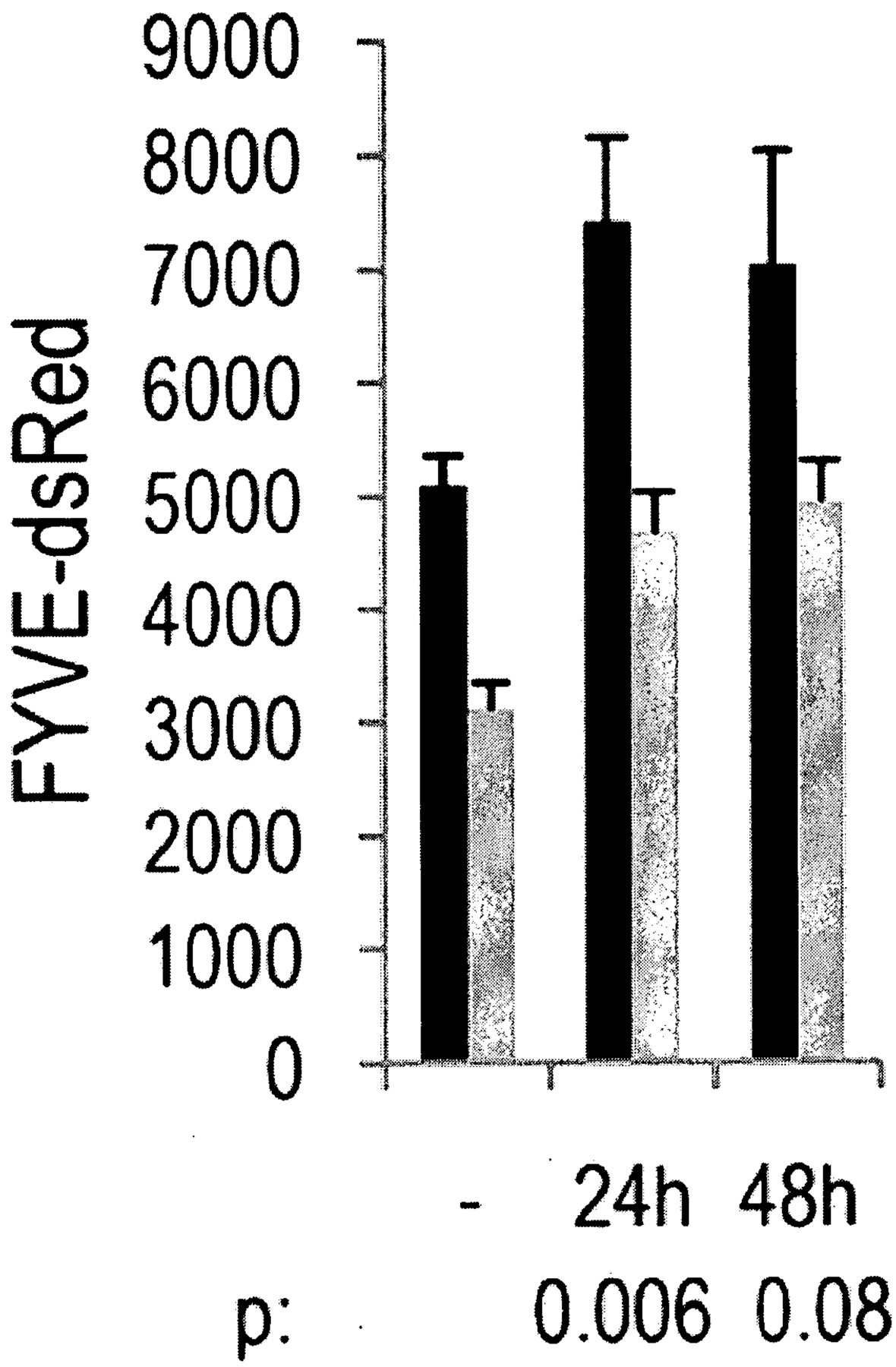

9000
8000
7000
6000
5000
4000
3000
2000
1000
0
FYVE-dsRed
-
24h
48h
p:
0.006
0.08

Figure 61

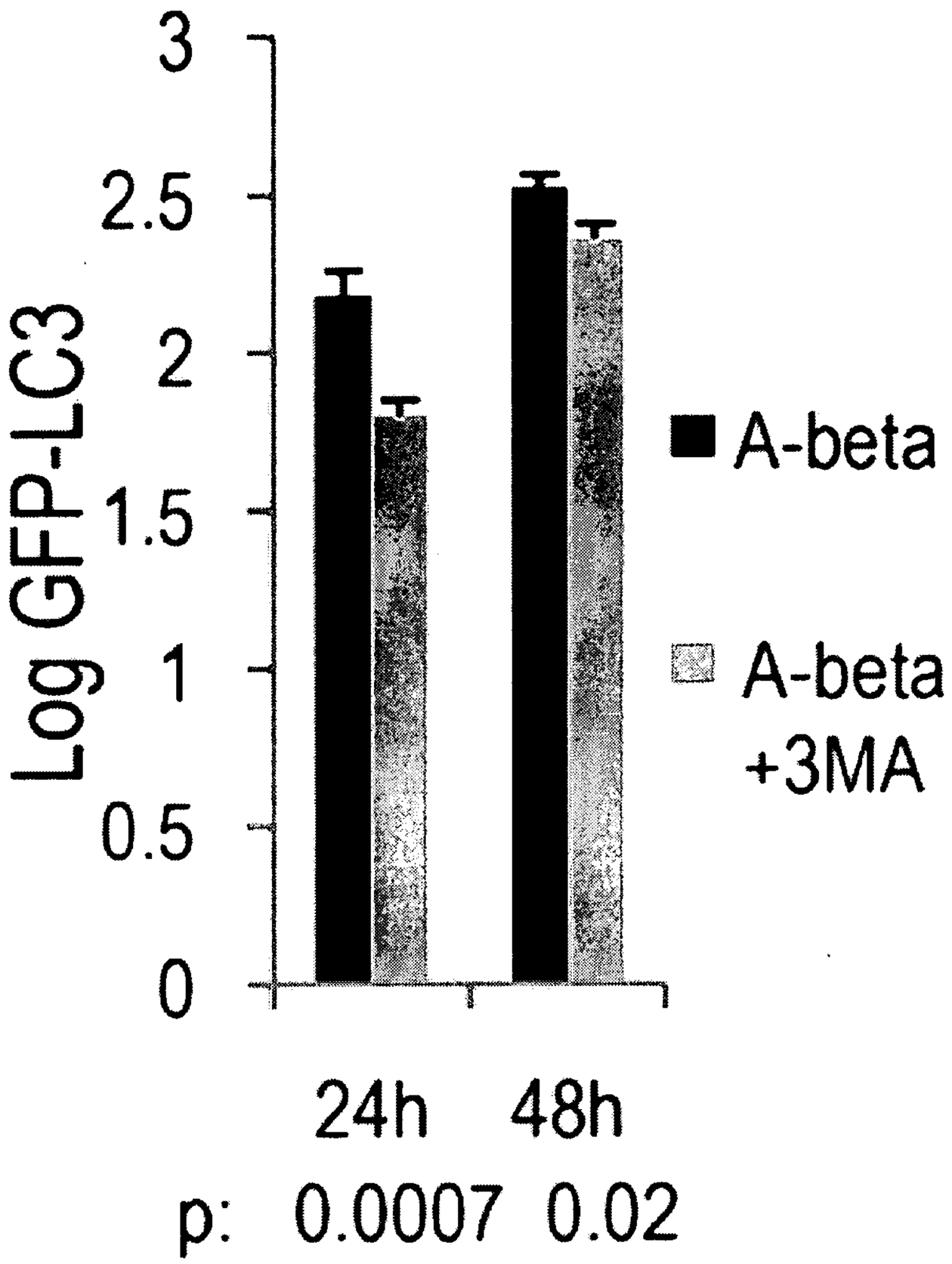


Figure 62

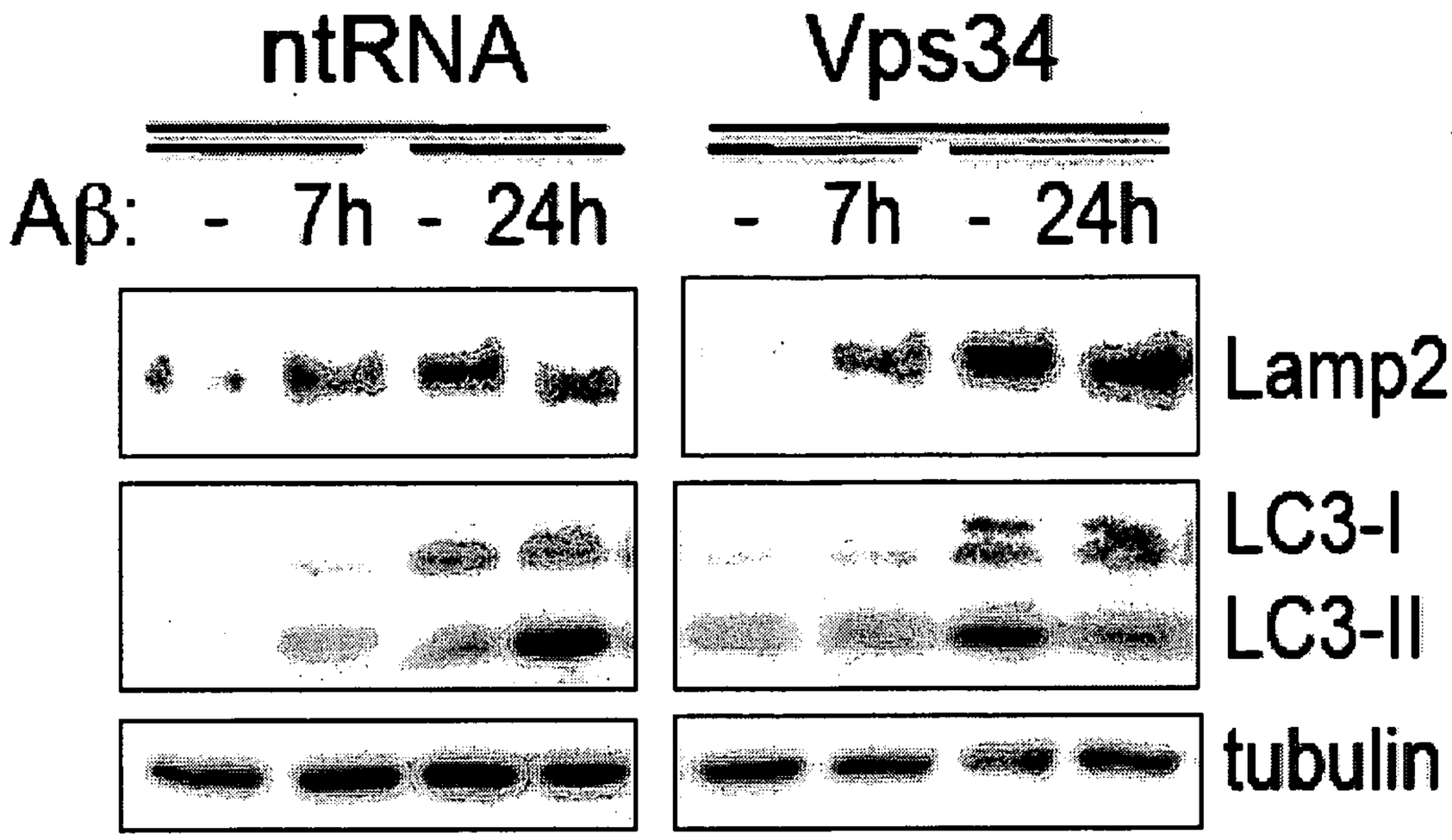


Figure 63.

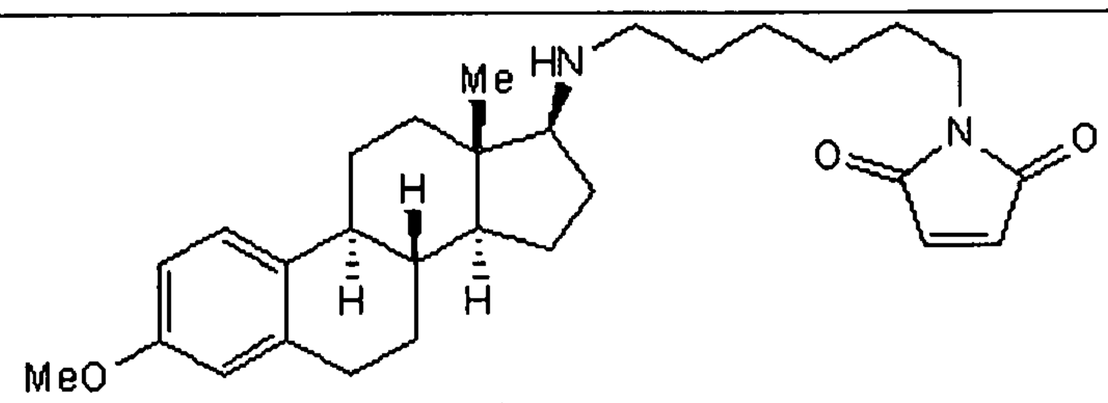
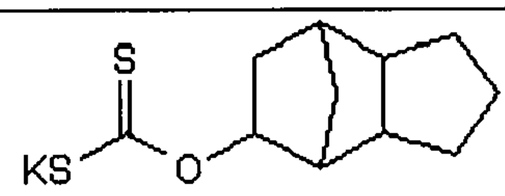
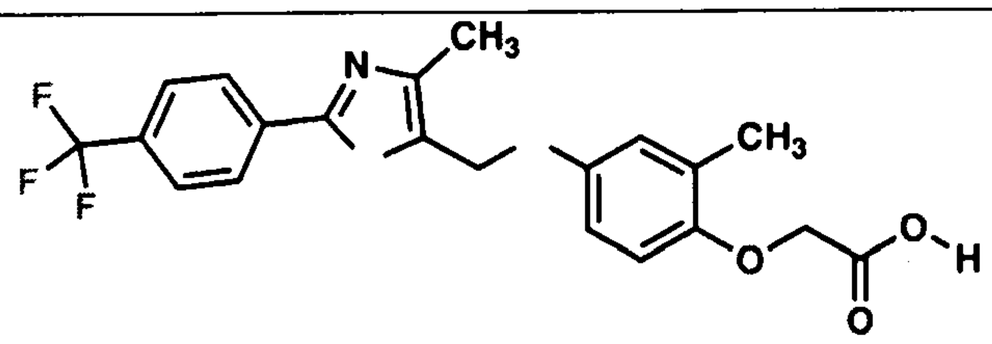
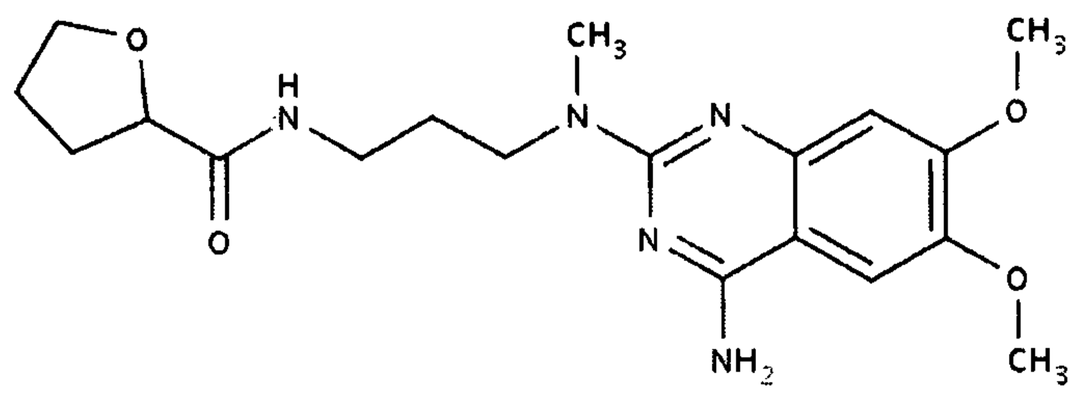
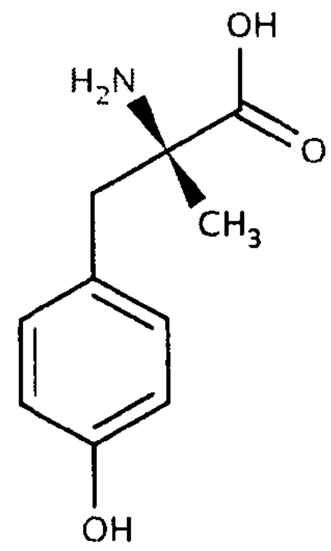
U73122	
D609	
GW501516	
alfuzosin	
Alpha-methyl-para-tyrosine	

Figure 63 (Continued)

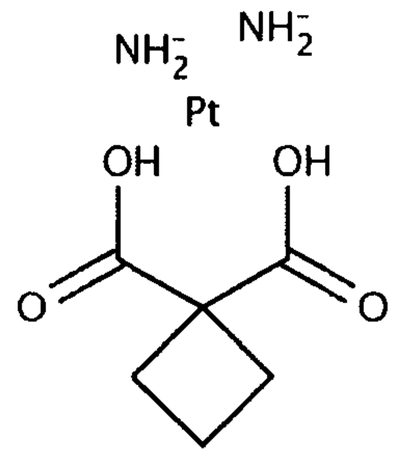
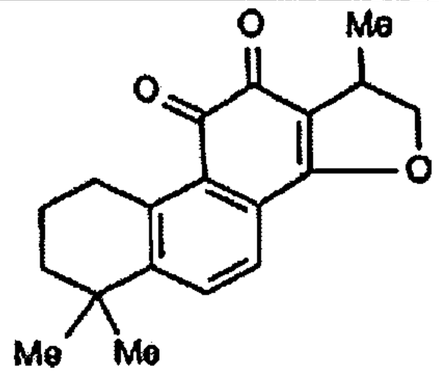
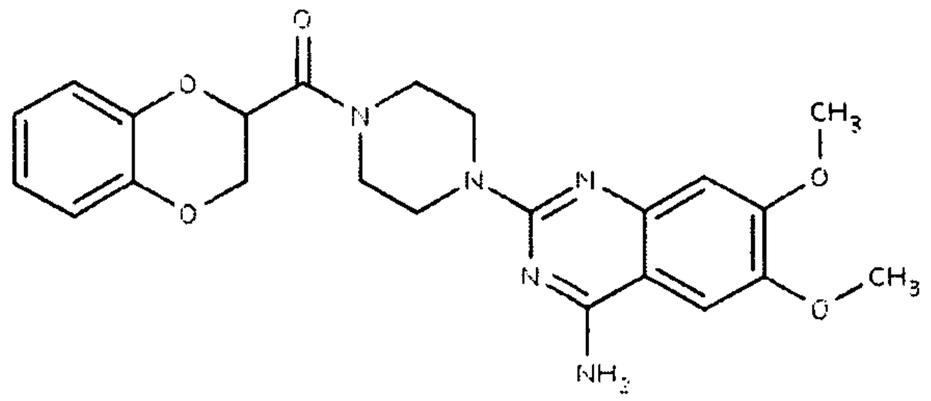
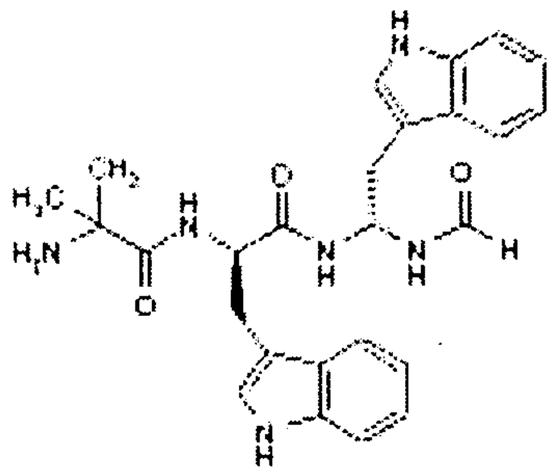
carboplatin	
cryptotanshinone	 Cryptotanshinone (*110-5*)
doxazosine	
EP1572	

Figure 63 (Continued)

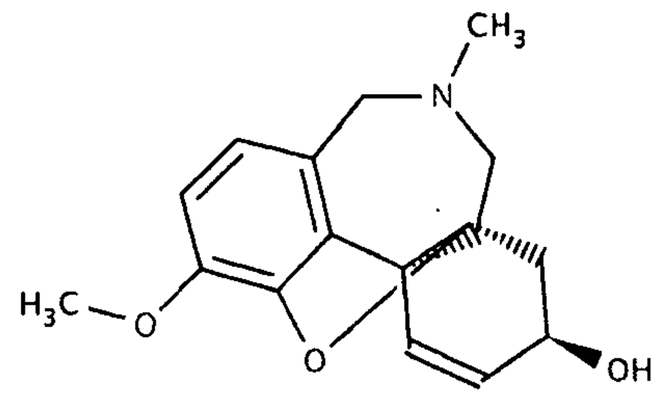
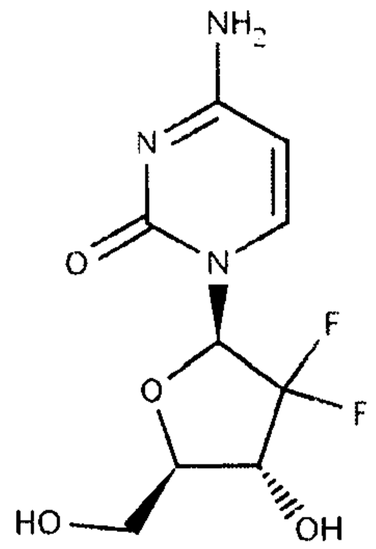
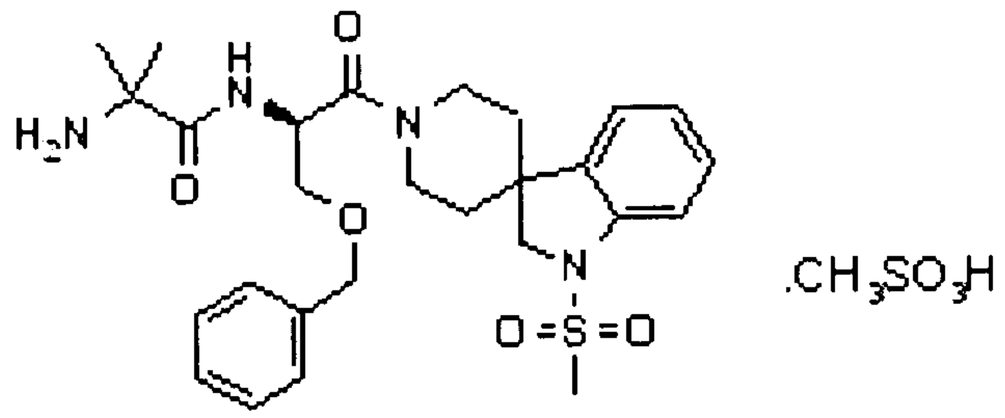
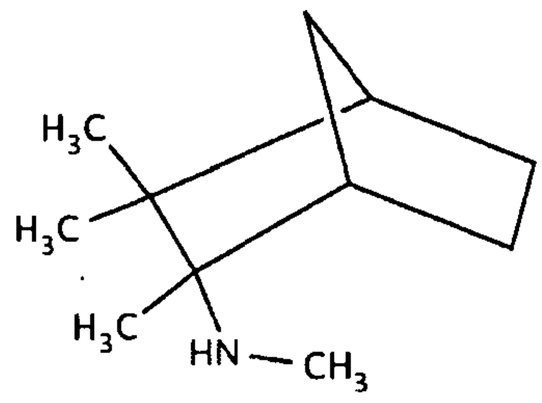
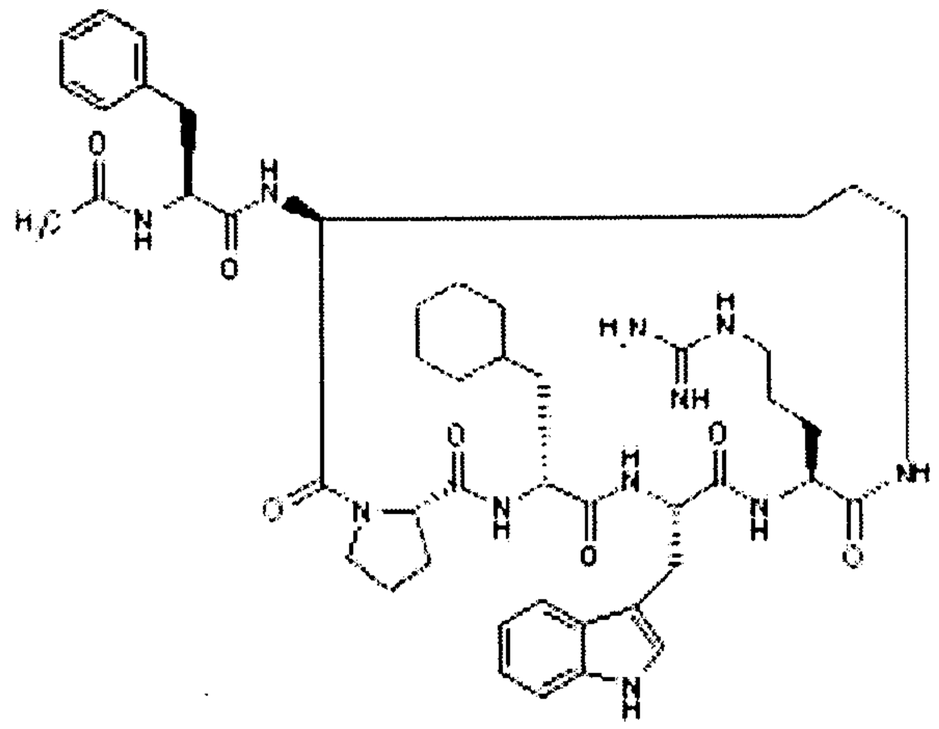
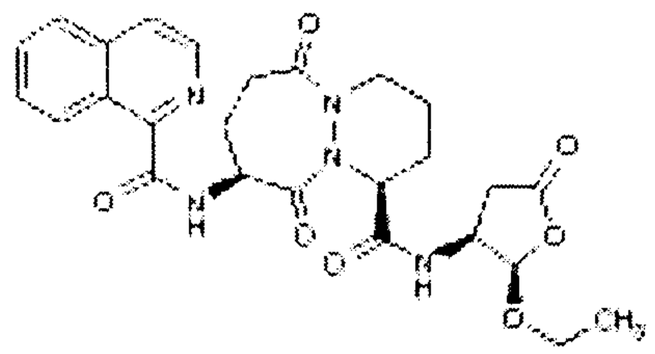
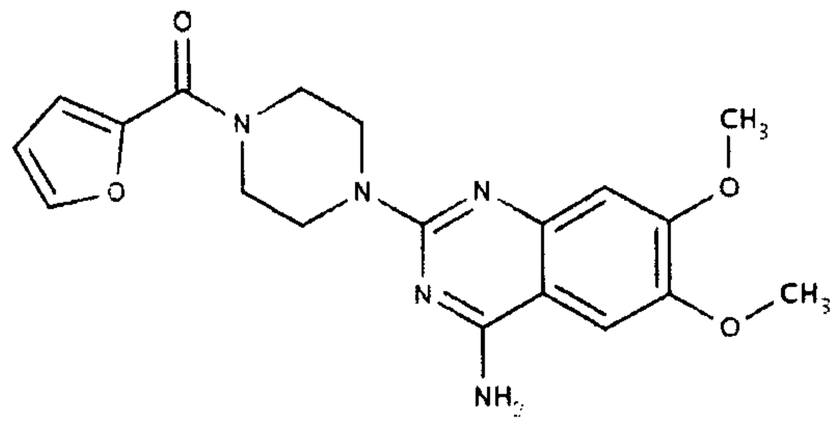
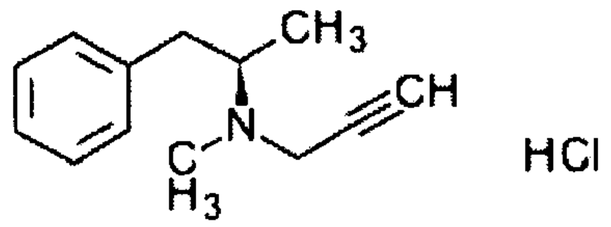
galantamine	
gemcitabine	
Ibutamoren Mesylate	
mecamylamine	

Figure 63 (Continued)

PMX53	
pralnacasan	
prazosin	
S-0373	 HCl

D00785

Figure 63 (Continued)

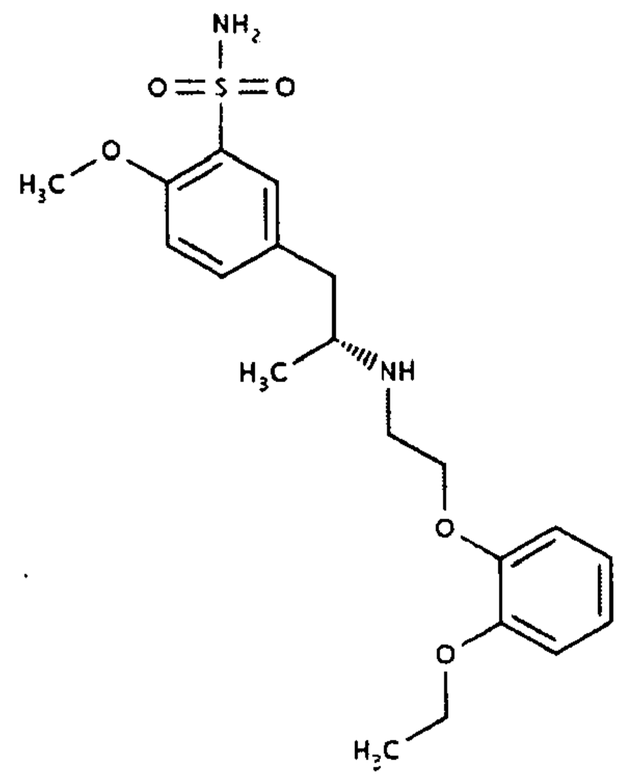
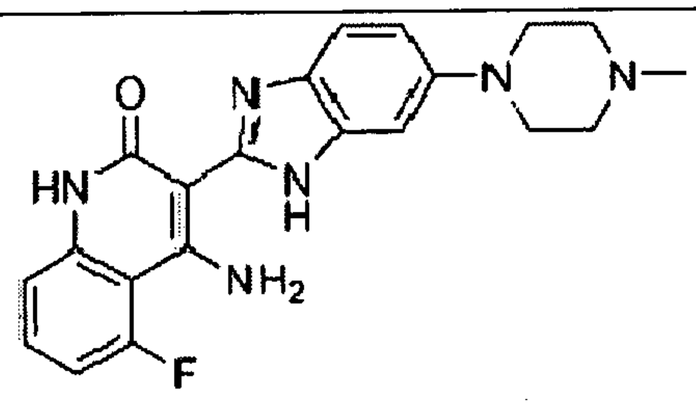
tamsulosin	
TK1258	

Figure 64

NM_001124.1| Homo sapiens adrenomedullin (ADM), mRNA
CTGGATAGAACAGCTCAAGCCTTGCCACTTCGGGCTTCTCACTGCAGCTGGGCTTGGACTTCGGAGTTTT
GCCATTGCCAGTGGGACGTCTGAGACTTTCTCCTTCAAGTACTTGGCAGATCACTCTCTTAGCAGGGTCT
GCGCTTCGCAGCCGGGATGAAGCTGGTTTCCGTCGCCCTGATGTACCTGGGTTCGCTCGCCTTCCTAGGC
GCTGACACCGCTCGGTTGGATGTCGCGTCGGAGTTTCGAAAGAAGTGGAATAAGTGGGCTCTGAGTCGTG
GGAAGAGGGAACTGCGGATGTCCAGCAGCTACCCCACCGGGCTCGCTGACGTGAAGGCCGGGCCTGCCCA
GACCCTTATTCGGCCCCAGGACATGAAGGGTGCCTCTCGAAGCCCCGAAGACAGCAGTCCGGATGCCGCC
CGCATCCGAGTCAAGCGCTACCGCCAGAGCATGAACAACTTCCAGGGCCTCCGGAGCTTTGGCTGCCGCT
TCGGGACGTGCACGGTGCAGAAGCTGGCACACCAGATCTACCAGTTCACAGATAAGGACAAGGACAACGT
CGCCCCCAGGAGCAAGATCAGCCCCCAGGGCTACGGCCGCCGGCGCCGGCGCTCCCTGCCCGAGGCCGGC
CCGGGTCGGACTCTGGTGTCTTCTAAGCCACAAGCACACGGGGCTCCAGCCCCCCCGAGTGGAAGTGCTC
CCCACTTTCTTTAGGATTTAGGCGCCCATGGTACAAGGAATAGTCGCGCAAGCATCCCGCTGGTGCCTCC
CGGGACGAAGGACTTCCCGAGCGGTGTGGGGACCGGGCTCTGACAGCCCTGCGGAGACCCTGAGTCCGGG
AGGCACCGTCCGGCGGCGAGCTCTGGCTTTGCAAGGGCCCCTCCTTCTGGGGGCTTCGCTTCCTTAGCCT
TGCTCAGGTGCAAGTGCCCCAGGGGGCGGGGTGCAGAAGAATCCGAGTGTTTGCCAGGCTTAAGGAGAGG
AGAAACTGAGAAATGAATGCTGAGACCCCCGGAGCAGGGGTCTGAGCCACAGCCGTGCTCGCCCACAAAC
TGATTTCTCACGGCGTGTCACCCCACCAGGGCGCAAGCCTCACTATTACTTGAACTTTCCAAAACCTAAA
GAGGAAAAGTGCAATGCGTGTTGTACATACAGAGGTAACTATCAATATTTAAGTTTGTTGCTGTCAAGAT
TTTTTTTGTAACTTCAAATATAGAGATATTTTTGTACGTTATATATTGTATTAAGGGCATTTTAAAAGCA
ATTATATTGTCCTCCCCTATTTTAAGACGTGAATGTCTCAGCGAGGTGTAAAGTTGTTCGCCGCGTGGAA
TGTGAGTGTGTTTGTGTGCATGAAAGAGAAAGACTGATTACCTCCTGTGTGGAAGAAGGAAACACCGAGT
CTCTGTATAATCTATTTACATAAAATGGGTGATATGCGAACAGCAAACC

NM_001501.1| Homo sapiens gonadotropin-releasing hormone 2 (GNRH2), transcript variant 1, mRNA
CTGCAGCTGCCTGAAGGAGCCATCTCATCCACAGCTCTTCCTTGAGCAGCCATGGCCAGCTCCAGGCGAG
GCCTCCTGCTCCTGCTGCTGCTGACTGCCCACCTTGGACCCTCAGAGGCTCAGCACTGGTCCCATGGCTG
GTACCCTGGAGGAAAGCGAGCCCTCAGCTCAGCCCAGGATCCCCAGAATGCCCTTAGGCCCCCAGGAAGG
GCCCTGGACACTGCAGCAGGCAGCCCAGTCCAGACTGCCCATGGCCTCCCAAGTGATGCCCTGGCTCCCC
TGGACGACAGCATGCCCTGGGAGGGCAGGACCACGGCCCAGTGGTCCCTTCACAGGAAGCGACACCTGGC
ACGGACACTGCTGACCGCAGCCCGAGAGCCCCGCCCCGCCCCGCCATCCTCCAATAAAGTGTGAGGTTCT
CCG

NM_002425.1| Homo sapiens matrix metallopeptidase 10 (stromelysin 2) (MMP10), mRNA
AAAGAAGGTAAGGGCAGTGAGAATGATGCATCTTGCATTCCTTGTGCTGTTGTGTCTGCCAGTCTGCTCT
GCCTATCCTCTGAGTGGGGCAGCAAAAGAGGAGGACTCCAACAAGGATCTTGCCCAGCAATACCTAGAAA
AGTACTACAACCTCGAAAAGGATGTGAAACAGTTTAGAAGAAAGGACAGTAATCTCATTGTTAAAAAAAT
CCAAGGAATGCAGAAGTTCCTTGGGTTGGAGGTGACAGGGAAGCTAGACACTGACACTCTGGAGGTGATG
CGCAAGCCCAGGTGTGGAGTTCCTGACGTTGGTCACTTCAGCTCCTTTCCTGGCATGCCGAAGTGGAGGA
AAACCCACCTTACATACAGGATTGTGAATTATACACCAGATTTGCCAAGAGATGCTGTTGATTCTGCCAT
TGAGAAAGCTCTGAAAGTCTGGGAAGAGGTGACTCCACTCACATTCTCCAGGCTGTATGAAGGAGAGGCT
GATATAATGATCTCTTTCGCAGTTAAAGAACATGGAGACTTTTACTCTTTTGATGGCCCAGGACACAGTT
TGGCTCATGCCTACCCACCTGGACCTGGGCTTTATGGAGATATTCACTTTGATGATGATGAAAAATGGAC
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CACTCAGCCAACACTGAAGCTTTGATGTACCCACTCTACAACTCATTCACAGAGCTCGCCCAGTTCCGCC
TTTCGCAAGATGATGTGAATGGCATTCAGTCTCTCTACGGACCTCCCCCTGCCTCTACTGAGGAACCCCT
GGTGCCCACAAAATCTGTTCCTTCGGGATCTGAGATGCCAGCCAAGTGTGATCCTGCTTTGTCCTTCGAT
GCCATCAGCACTCTGAGGGGAGAATATCTGTTCTTTAAAGACAGATATTTTTGGCGAAGATCCCACTGGA
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GTACAAGCAGGTTATCCAAGAGGCATCCATACCCTGGGTTTTCCTCCAACCATAAGGAAAATTGATGCAG
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TAGCCAGTCCATGGAGCAAGGCTTCCCTAGACTAATAGCTGATGACTTTCCAGGAGTTGAGCCTAAGGTT

GATGCTGTATTACAGGCATTTGGATTTTTCTACTTCTTCAGTGGATCATCACAGTTTGAGTTTGACCCCA
ATGCCAGGATGGTGACACACATATTAAAGAGTAACAGCTGGTTACATTGCTAGGCGAGATAGGGGGAAGA
CAGATATGGGTGTTTTTAATAAATCTAATAATTATTCATCTAATGTATTATGAGCCAAAATGGTTAATTT
TTCCTGCATGTTCTGTGACTGAAGAAGATGAGCCTTGCAGATATCTGCATGTGTCATGAAGAATGTTTCT
GGAATTCTTCACTTGCTTTTGAATTGCACTGAACAGAATTAAGAAATACTCATGTGCAATAGGTGAGAGA
ATGTATTTTCATAGATGTGTTATTACTTCCTCAATAAAAAGTTTTATTTTGGGCCTGTTCCTT

NM_000751.1| Homo sapiens cholinergic receptor, nicotinic, delta (CHRND), mRNA
TGGGATGGAGGGGCCAGTGCTGACACTGGGGCTGCTGGCTGCCCTGGCGGTGTGTGGCAGCTGGGGGCTG
AACGAGGAGGAGCGGCTGATCCGGCACCTGTTTCAAGAGAAGGGCTACAACAAGGAGCTCCGGCCCGTGG
CACACAAAGAGGAGAGTGTGGACGTTGCCCTGGCCCTCACACTCTCCAACCTCATCTCCCTGAAAGAAGT
TGAGGAGACCCTCACTACCAATGTGTGGATAGAGCACGGCTGGACAGACAACCGGCTGAAGTGGAATGCT
GAAGAATTTGGAAACATCAGTGTCCTGCGCCTCCCCCCGGACATGGTGTGGCTCCCAGAGATTGTGCTGG
AGAACAACAATGACGGCTCCTTCCAGATCTCCTACTCCTGCAACGTGCTTGTCTACCACTACGGCTTCGT
GTACTGGCTGCCACCTGCCATCTTCCGCTCCTCCTGCCCCATCTCTGTCACCTATTTCCCCTTCGACTGG
CAGAACTGCTCCCTCAAGTTCAGTTCCCTCAAGTATACGGCCAAAGAGATCACCCTGAGCCTGAAACAGG
ATGCCAAGGAGAACCGCACCTACCCCGTGGAGTGGATCATCATTGATCCTGAAGGCTTCACAGAGAACGG
GGAGTGGGAGATAGTCCACCGGCCGGCCAGGGTCAACGTGGACCCCAGAGCCCCTCTGGACAGCCCCAGC
CGCCAGGACATCACCTTCTACCTCATCATCCGCCGCAAGCCCCTCTTCTACATCATCAACATCCTGGTGC
CCTGCGTGCTCATCTCCTTCATGGTCAACCTGGTCTTCTACCTACCGGCTGACAGTGGTGAGAAGACATC
AGTGGCCATCTCGGTGCTCCTGGCTCAGTCTGTCTTCCTGCTGCTCATCTCCAAGCGTCTGCCTGCCACA
TCCATGGCCATCCCCCTTATCGGCAAGTTCCTGCTCTTCGGCATGGTGCTGGTCACCATGGTTGTGGTGA
TCTGTGTCATCGTGCTCAACATCCACTTCCGAACACCCAGCACCCATGTGCTGTCTGAGGGGGTCAAGAA
GCTCTTCCTGGAGACCCTGCCGGAGCTCCTGCACATGTCCCGCCCAGCAGAGGATGGACCCAGCCCTGGG
GCCCTGGTGCGGAGGAGCAGCTCCCTGGGATACATCTCCAAGGCCGAGGAGTACTTCCTGCTCAAGTCCC
GCAGTGACCTCATGTTCGAGAAGCAGTCAGAGCGGCATGGGCTGGCCAGGCGCCTCACCACTGCACGCCG
GCCCCCAGCAAGCTCTGAGCAGGCCCAGCAGGAACTCTTCAATGAGCTGAAGCCAGCTGTGGATGGGGCA
AACTTCATTGTTAACCACATGAGGGACCAGAACAATTACAATGAGGAGAAAGACAGCTGGAACCGAGTGG
CCCGCACAGTGGACCGCCTCTGCCTGTTTGTGGTGACGCCTGTCATGGTGGTGGGCACAGCCTGGATCTT
CCTGCAGGGCGTTTACAACCAGCCACCACCCCAGCCTTTTCCTGGGGACCCCTACTCCTACAACGTGCAG
GACAAGCGCTTCATCTAGGGTGGGCCTGTTGGGGAGCCAGGAGACAGCAGGGTCTGAGAGAGGAGCCACA
GTCCCTAATGACACCCACTCCTAGCCCTGAGGCTCGTGCCCCTCAGACTGGGGAAGAGTCCAAGGAAGGG
AGGGAGCAGCCACTCCTCAATGCTCAATGGCTCCCCTGAAATCAAGACAGGGGCCACCCGA

NM_001443.1| Homo sapiens fatty acid binding protein 1, liver (FABP1), mRNA
AGAGCCGCAGGTCAGTCGTGAAGAGGGAGCTCTATTGCCACCATGAGTTTCTCCGGCAAGTACCAACTGC
AGAGCCAGGAAAACTTTGAAGCCTTCATGAAGGCAATCGGTCTGCCGGAAGAGCTCATCCAGAAGGGGAA
GGATATCAAGGGGGTGTCGGAAATCGTGCAGAATGGGAAGCACTTCAAGTTCACCATCACCGCTGGGTCC
AAAGTGATCCAAAACGAATTCACGGTGGGGGAGGAATGTGAGCTGGAGACAATGACAGGGGAGAAAGTCA
AGACAGTGGTTCAGTTGGAAGGTGACAATAAACTGGTGACAACTTTCAAAAACATCAAGTCTGTGACCGA
ACTCAACGGCGACATAATCACCAATACCATGACATTGGGTGACATTGTCTTCAAGAGAATCAGCAAGAGA
ATTTAAACAAGTCTGCATTTCATATTATTTTAGTGTGTAAAATTAATGTAATAAAGTGAACTTTGTTTT

NM_001545.1| Homo sapiens immature colon carcinoma transcript 1 (ICT1), mRNA
GCATGGCGGCCACCAGGTGCCTGCGCTGGGGCCTGAGCCGAGCCGGAGTCTGGCTGCTCCCACCGCCCGC
ACGGTGCCCACGCCGGGCGCTGCACAAGCAGAAAGACGGCACTGAGTTCAAGAGCATCTACAGCCTGGAC
AAGCTCTACCCCGAATCTCAGGGCTCGGACACCGCCTGGAGGGTCCCGAATGGTGCAAAGCAAGCCGACA
GTGACATCCCTCTAGATCGCTTGACAATATCTTATTGTCGGAGTAGTGGTCCTGGGGGGCAGAATGTGAA
CAAAGTGAATTCCAAGGCAGAAGTCAGGTTCCATTTGGCAACTGCCGAGTGGATCGCGGAGCCCGTGCGG
CAGAAGATAGCCATCACGCATAAAAACAAGATCAACAGGTTAGGAGAGTTGATCCTTACCTCTGAGAGCA
GCCGCTATCAGTTCCGGAATCTGGCAGATTGCCTGCAGAAAATTCGAGACATGATCACTGAGGCCAGCCA
GACACCGAAGGAGCCAACAAAAGAAGATGTTAAACTTCATAGAATCAGGATAGAAAACATGAATCGGGAA
AGGCTGAGACAAAAGAGAATTCATTCTGCTGTAAAGACAAGCAGGAGGGTCGACATGGACTGAAATCACC
CTCTGCAGCTGGGAGGGCTCTTCTGGGCGTCCGGGCAGCTGCAGCTGAGAGGACTTTCACACCATAAGGA
GATTTCTGTTTTTCTTTTTGGCTGTTAATGCTTGTCTATAACATTGGAGCCATCACAAGAATGTTCATTT
GGAATGAAGGCTGCAGGCACTGGTTGCAGACGTCTTTATAGGCAGTCACCATGTTGTCAAACCTTAATAA
TGCACCTCATGTATTAGTCACAATAAAAATCAGAACTCAAAAAAAAAA

NM_004122.1| Homo sapiens growth hormone secretagogue receptor (GHSR), transcript variant 1b, mRNA
ATGTGGAACGCGACGCCCAGCGAAGAGCCGGGGTTCAACCTCACACTGGCCGACCTGGACTGGGATGCTT
CCCCCGGCAACGACTCGCTGGGCGACGAGCTGCTGCAGCTCTTCCCCGCGCCGCTGCTGGCGGGCGTCAC
AGCCACCTGCGTGGCACTCTTCGTGGTGGGTATCGCTGGCAACCTGCTCACCATGCTGGTGGTGTCGCGC
TTCCGCGAGCTGCGCACCACCACCAACCTCTACCTGTCCAGCATGGCCTTCTCCGATCTGCTCATCTTCC
TCTGCATGCCCCTGGACCTCGTTCGCCTCTGGCAGTACCGGCCCTGGAACTTCGGCGACCTCCTCTGCAA
ACTCTTCCAATTCGTCAGTGAGAGCTGCACCTACGCCACGGTGCTCACCATCACAGCGCTGAGCGTCGAG
CGCTACTTCGCCATCTGCTTCCCACTCCGGGCCAAGGTGGTGGTCACCAAGGGGCGGGTGAAGCTGGTCA
TCTTCGTCATCTGGGCCGTGGCCTTCTGCAGCGCCGGGCCCATCTTCGTGCTAGTCGGGGTGGAGCACGA
GAACGGCACCGACCCTTGGGACACCAACGAGTGCCGCCCCACCGAGTTTGCGGTGCGCTCTGGACTGCTC
ACGGTCATGGTGTGGGTGTCCAGCATCTTCTTCTTCCTTCCTGTCTTCTGTCTCACGGTCCTCTACAGTC
TCATCGGCAGGAAGCTGTGGCGGAGGAGGCGCGGCGATGCTGTCGTGGGTGCCTCGCTCAGGGACCAGAA
CCACAAGCAAACCGTGAAAATGCTGGGTGGGTCTCAGCGCGCGCTCAGGCTTTCTCTCGCGGGTCCTATC
CTCTCCCTGTGCCTTCTCCCTTCTCTCTGA

NM_005796.1| Homo sapiens nuclear transport factor 2 (NUTF2), mRNA
GGAAGGGACAGTCGGCCGCAGACCGCGCTGGGTTGCCGCTGCCGCTGCCGCCATCGTGCCAGCCCCTCGG
GTCTCCGTGAGGCCGGGTGACGCTCCAGAATGGGAGACAAGCCAATTTGGGAGCAGATTGGATCCAGCTT
CATTCAACATTACTACCAGTTATTTGATAATGATAGAACCCAACTAGGCGCAATTTACATTGACGCGTCA
TGCCTTACGTGGGAAGGACAACAGTTCCAGGGGAAAGCTGCCATTGTGGAGAAGTTGTCTAGCCTTCCGT
TCCAGAAAATTCAGCACAGCATCACCGCGCAGGACCATCAGCCCACTCCAGATAGCTGCATCATCAGCAT
GGTTGTGGGCCAGCTTAAGGCGGATGAAGACCCCATCATGGGGTTCCACCAGATGTTCCTATTAAAGAAC
ATCAACGATGCTTGGGTTTGCACCAATGACATGTTCAGGCTCGCCCTGCACAACTTTGGCTGACCTCCTC
TCAGCTAGGCACTCACGCTGTTTCCTCCTCCCTCCTCTTCCCAATACTATTCCCACTCCTCCAGATGCTC
CAAATATCATGCACAAATGAGCAGGGCCGCGGTGGGAGTGGGCGCAGTGCGCTGCTGCCACTGAGGTGTT
GTGCATGATGTTTGGATGCTAGACTAGTTGCATCTGACGGGAGAAGTTTGTGTTGTACCAGCGCATGCCT
TGGAAAGACTTAAGTAATGCAAAAGGTTGTCCTTTTTTTTTTTTTTTTTTTTTTAATCTACTGACAAGTT
GCTCTAGTAACCCAAAGAAGTGAAGGAGAAAGCAGCTGCCTCACCGCCCAGACATTGATTTGTTCAGATG
TTTCAATGCCTCATGATACAATAAAACCACAAAAATTTTCTTAACAAAAAAAAA

NM_006181.1| Homo sapiens netrin 3 (NTN3), mRNA
GAGGACGCGCCAACATCCCCGCTGCTGTGCTGGGCCCGGGGCGTGCCCGCCGCTGCTCCCACCTCTGGGC
CGGGCTGGGGCCGCCCGGGGGCCCTGTTCCTCGGCATTGCGGGCCTGGTGGGCAGAACCGCGGAGAGGGC
TTCTTTTCCCCAAGGGCAGCGTCTTGGGGCCCGGCCACTGGCTGACCCGCAGCGGCTCCGGCCATGCCTG
GCTGGCCCTGGGGGCTGCTGCTGACGGCAGGCACGCTCTTCGCCGCCCTGAGTCCTGGGCCGCCGGCGCC
CGCCGACCCCTGCCACGATGAGGGGGGTGCGCCCCGCGGCTGCGTGCCAGGACTGGTGAACGCCGCCCTG
GGCCGCGAGGTGCTGGCTTCCAGCACGTGCGGGCGGCCGGCCACTCGGGCCTGCGACGCCTCCGACCCGC
GACGGGCACACTCCCCCGCCCTCCTTACTTCCCCAGGGGGCACGGCCAGCCCTCTGTGCTGGCGCTCGGA
GTCCCTGCCTCGGGCGCCCCTCAACGTGACTCTCACGGTGCCCCTGGGCAAGGCTTTTGAGCTGGTCTTC
GTGAGCCTGCGCTTCTGCTCAGCTCCCCCAGCCTCCGTGGCCCTGCTCAAGTCTCAGGACCATGGCCGCA
GCTGGGCCCCGCTGGGCTTCTTCTCCTCCCACTGTGACCTGGACTATGGCCGTCTGCCTGCCCCTGCCAA
TGGCCCAGCTGGCCCAGGGCCTGAGGCCCTGTGCTTCCCCGCACCCCTGGCCCAGCCTGATGGCAGCGGC
CTTCTGGCCTTCAGCATGCAGGACAGCAGCCCCCCAGGCCTGGACCTGGACAGCAGCCCAGTGCTCCAAG
ACTGGGTGACCGCCACCGACGTCCGTGTAGTGCTCACAAGGCCTAGCACGGCAGGTGACCCCAGGGACAT
GGAGGCCGTCGTCCCTTACTCCTACGCAGCCACCGACCTCCAGGTGGGCGGGCGCTGCAAGTGCAATGGA
CATGCCTCACGGTGCCTGCTGGACACACAGGGCCACCTGATCTGCGACTGTCGGCATGGCACCGAGGGCC
CTGACTGCGGCCGCTGCAAGCCCTTCTACTGCGACAGGCCATGGCAGCGGGCCACTGCCCGGGAATCCCA
CGCCTGCCTCGCTTGCTCCTGCAACGGCCATGCCCGCCGCTGCCGCTTCAACATGGAGCTGTACCGACTG
TCCGGCCGCCGCAGCGGGGGTGTCTGTCTCAACTGCCGGCACAACACCGCCGGCCGCCACTGCCACTACT
GCCGGGAGGGCTTCTATCGAGACCCTGGCCGTGCCCTGAGTGACCGTCGGGCTTGCAGGGCCTGCGACTG
TCACCCGGTTGGTGCTGCTGGCAAGACCTGCAACCAGACCACAGGCCAGTGTCCCTGCAAGGATGGCGTC
ACTGGCCTCACCTGCAACCGCTGCGCGCCTGGCTTCCAGCAAAGCCGCTCCCCAGTGGCGCCCTGTGTTA
AGACCCCTATCCCTGGACCCACTGAGGACAGCAGCCCTGTGCAGCCCCAGGACTGTGACTCGCACTGCAA
ACCTGCCCGTGGCAGCTACCGCATCAGCCTAAAGAAGTTCTGCAAGAAGGACTATGCGGTGCAGGTGGCG
GTGGGTGCGCGCGGCGAGGCGCGCGGCGCGTGGACACGCTTCCCGGTGGCGGTGCTCGCCGTGTTCCGGA
GCGGAGAGGAGCGCGCGCGGCGCGGGAGTAGCGCGCTGTGGGTGCCCGCCGGGGATGCGGCCTGCGGCTG
CCCGCGCCTGCTCCCCGGCCGCCGCTACCTCCTGCTGGGGGGCGGGCCTGGAGCCGCGGCTGGGGGCGCG
GGGGGCCGGGGGCCCGGGCTCATCGCCGCCCGCGGAAGCCTCGTGCTACCCTGGAGGGACGCGTGGACGC
GGCGCCTGCGGAGGCTGCAGCGACGCGAACGGCGGGGGCGCTGCAGCGCCGCCTGAGCCCGCCGGCTGGG
CAGGGCGGCCGCTGCTCCCACATCTA

NM_007213.1| Homo sapiens PRA1 domain family, member 2 (PRAF2), mRNA
CACATTAACCGGCAGGATGTCGGAGGTGCGGCTGCCACCGCTACGCGCCCTGGACGACTTTGTTCTGGGG
TCGGCGCGTCTGGCGGCTCCGGATCCATGCGACCCGCAGCGATGGTGCCACCGCGTCATCAACAACCTCC
TCTACTACCAAACCAACTACCTTCTCTGCTTCGGCATCGGCCTCGCTCTCGCCGGGTACGTGCGGCCACT
TCATACGCTCCTGAGCGCGCTGGTAGTGGCGGTGGCCCTCGGCGTGCTGGTGTGGGCAGCTGAGACCCGC
GCAGCTGTGCGCCGCTGCCGCCGCAGCCACCCTGCAGCCTGCCTGGCCGCAGTGCTTGCCGTCGGCCTCC
TGGTGCTCTGGGTCGCGGGCGGCGCTTGCACCTTCCTGTTCAGCATCGCCGGGCCGGTGCTTCTGATCCT
GGTGCACGCCTCGTTGCGCCTGCGCAACCTTAAGAACAAGATTGAGAACAAGATCGAGAGCATTGGTCTC
AAGCGGACGCCAATGGGCCTGCTACTAGAGGCACTGGGACAAGAGCAGGAGGCTGGATCCTAGGCCCCTG
GGATCTGTACCCAGGACCTGGAGAATACCACCCCACCCCCAGCCCATAATTGGGACCCAGAGCCCTTTCC
CAGCACTTAAAACAGGAGCCTAGAGCCCCCTGCCCAAACAAAACAGGACATCTGTGACCGCCCTACCCCC
ACGCCAGCCCCAAACTAAGATATCCCTCACACCCAGCCCCCATTACCTAGGGACAAGAGTCTTCCCCAGC
CTTGAACCTAGGACCAAGAGCCACCTACATCCAGCCCCAAAACTGGGGCTTCAGGCCAGAGCATCCATGG
CCAATTTCAAATTGTGAACCCAGAGACACTCCCATCCACCCTTCTCCATGCTCATCCCCAAACTGGGGCC
TGGAGCAAGGCACTCTCAAATCTTGAACCCTGGACCAAAGCTTTTCCAGACCCCACCCTACCTTCCAACC
CAGGTCAAGACATTGCCAAATCTTGAACTCAGAACCCAAGTGTTCCATGCCCCTGTGTGGATGGAGTCGG
GTATCCTGACTGTTGGACCCCTGGTCCAGGTGATCCCGACCCTCACCAGTCCCATTTGCCTCCCTCCAGC
TCTGCTTAGGCATTTTGCCCCTCACCCCAATGTTCCACACCATCGACAACCAAGGGGTGAGGTGGGGACA
GGCCTCAGCAGGGAATGGGGCGTATATGTTAGTGTTGCTGCAACAATAAAGCCTGTTGCATCTCTCATGC
CAA

NM_014226.1| Homo sapiens renal tumor antigen (RAGE), mRNA
CGCATGCGCAGGCGACAGCTCATGGCGTTCAGGGCCTGACGGTTGCTAGGGTGACAGGGACACAACATGG
CGGCGGGATCTCTAACGCTCTCCTTCGAGGGACCACCACGGAGATCCTAGTGCGGGACCCCGCCTCAGGG
AAGTGGAAAGCAGGGGGACAACCTTCCTGCTTCCTTCTTTTCCGTCCAGTGTCGGCAAGGGGTTGTCACC
GGCTTCCGCATCCAAGATGAAGAACTATAAAGCAATTGGCAAAATAGGAGAGGGAACGTTTTCTGAAGTT
ATGAAGATGCAAAGCCTGAGAGATGGAAACTACTATGCATGTAAACAAATGAAGCAGCGCTTTGAAAGTA
TTGAGCAAGTCAACAACCTACGAGAGATCCAAGCACTGAGGCGCCTGAATCCGCACCCAAACATTCTTAT
GTTGCATGAAGTGGTTTTTGACAGAAAATCTGGTTCTCTTGCACTAATATGTGAACTTATGGACATGAAT
ATTTATGAGCTAATACGAGGGAGAAGATACCCATTATCAGAAAAAAAAATTATGCACTATATGTACCAGT
TATGTAAGTCCCTGGATCATATTCACAGAAATGGAATATTTCACAGAGATGTAAAACCAGAAAATATACT
AATAAAGCAGGATGTCCTGAAATTAGGGGACTTTGGCTCCTGCCGGAGTGTCTATTCCAAGCAGCCGTAC
ACGGAATACATCTCCACCCGCTGGTACCGGGCCCCGGAGTGTCTCCTCACTGATGGGTTCTACACGTACA
AGATGGACCTGTGGAGCGCCGGCTGTGTGTTCTACGAGATCGCCAGTCTGCAGCCCCTCTTTCCTGGAGT
AAATGAACTGGACCAAATCTCAAAAATCCACGATGTCATCGGCACACCCGCTCAGAAGATCCTCACCAAG
TTCAAACAGTCGAGAGCTATGAATTTTGATTTTCCTTTTAAAAAGGGATCAGGAATACCTCTACTAACAA
CCAATTTGTCCCCACAATGCCTCTCCCTCCTGCACGCAATGGTGGCCTATGATCCCGATGAGAGAATCGC
CGCCCACCAGGCCCTGCAGCACCCCTACTTCCAAGAACAGAGGAAAACAGAGAAGCGGGCTCTGGGCAGC
CACAGAAAAGCTGGCTTTCCGGAGCACCCTGTGGCACCGGAACCACTCAGTAACAGCTGCCAGATTTCCA
AGGAGGGCAGAAAGCAGAAACAGTCCCTAAAGCAAGAGGAGGACCGTCCCAAGAGACGAGGACCGGCCTA
TGTCATGGAACTGCCCAAACTAAAGCTTTCGGGAGTGGTCAGACTGTCGTCTTACTCCAGCCCCACGCTG
CAGTCCGTGCTTGGATCTGGAACAAATGGAAGAGTGCCGGTGCTGAGACCCTTGAAGTGCATCCCTGCGA
GCAAGAAGACAGATCCGCAGAAGGACCTTAAGCCTGCCCCGCAGCAGTGTCGCCTGCCCACCATAGTGCG
GAAAGGCGGAAGATAACTGAGCAGCACCGTCGTCTCGACTTCGGAGGCAACACCAAGCCCGACCGGGCCA
GGCCTGGGTGATCTGCTGCTGAGACGCCACGGAGGGCTGGGGATGCGCCTGCGTCCGTTTCGCGCTGGCC
GGGGCTCTGGGTGCTGCCCTGCGCCCTGCCGCACCCGCGGCCCGCGCAGCTGCCTAGGATGTTCTGGGCT
AATATACTTGTAAAACCACCGCATTCTAGGGTTTTCTTTCATTTTCGTTAAGAATTTGGGGCAGGAAATA
CTTTGTAACTTTGTATATGAATCAAAACAAACGAGCAGGCATTTCTGTGATGTGTTGGGCGTGGTTGGAA
GGTGGGTTCTGCGTGTCCCTTCCCAGCGCTGCTGGTCAGTCGTGGAGCGCCATCATGTCTTACCAGTGAC
GCTGCTGACACCCCTGACTTTTATTAAAGAATAAGCTGTCGTTAAAAAAAAAAAAAAAAAAAAA

NM_018949.1| Homo sapiens urotensin 2 receptor (UTS2R), mRNA
ATGGCGCTGACCCCCGAGTCCCCGAGCAGCTTCCCTGGGCTGGCCGCCACCGGCAGCTCTGTGCCGGAGC
CGCCTGGCGGCCCCAACGCAACCCTCAACAGCTCCTGGGCCAGCCCGACCGAGCCCAGCTCCCTGGAGGA
CCTGGTGGCCACGGGCACCATTGGGACTCTGCTGTCGGCCATGGGCGTGGTGGGCGTGGTGGGCAACGCC
TACACGCTGGTGGTCACCTGCCGCTCCCTGCGTGCGGTGGCCTCCATGTACGTCTACGTGGTCAACCTGG
CGCTGGCCGACCTGCTGTACCTGCTCAGCATCCCCTTCATCGTGGCCACCTACGTCACCAAGGAGTGGCA
CTTCGGGGACGTGGGCTGCCGCGTGCTCTTCGGCCTGGACTTCCTGACCATGCACGCCAGCATCTTCACG
CTGACCGTCATGAGCAGCGAGCGCTACGCTGCGGTGCTGCGGCCGCTGGACACCGTGCAGCGCCCCAAGG
GCTACCGCAAGCTGCTGGCGCTGGGCACCTGGCTGCTGGCGCTGCTGCTGACGCTGCCCGTGATGCTGGC
CATGCGGCTGGTGCGCCGGGGTCCCAAGAGCCTGTGCCTGCCCGCCTGGGGCCCGCGCGCCCACCGCGCC

TACCTGACGCTGCTCTTCGCCACCAGCATCGCGGGGCCCGGGCTGCTCATCGGGCTGCTCTACGCGCGCC
TGGCCCGCGCCTACCGCCGCTCGCAGCGCGCCTCCTTCAAGCGGGCCCGGCGGCCGGGGGCGCGCGCGCT
GCGCCTGGTGCTGGGCATCGTGCTGCTCTTCTGGGCCTGCTTCCTGCCCTTCTGGCTGTGGCAGCTGCTC
GCCCAGTACCACCAGGCCCCGCTGGCGCCGCGGACGGCGCGCATCGTCAACTACCTGACCACCTGCCTCA
CCTACGGCAACAGCTGCGCCAACCCCTTCCTCTACACGCTGCTCACCAGGAACTACCGCGACCACCTGCG
CGGCCGCGTGCGGGGCCCGGGCAGCGGGGGAGGCCGGGGGCCCGTTCCCTCCCTGCAGCCCCGCGCCCGC
TTCCAGCGCTGTTCGGGCCGCTCCCTGTCTTCCTGCAGCCCACAGCCCACTGACAGCCTCGTGCTGGCCC
CAGCGGCCCCGGCCCGACCTGCGCCCGAGGGTCCCAGGGCCCCGGCGTGA

NM_022743.1| Homo sapiens SET and MYND domain containing 3 (SMYD3), mRNA
GTGGATTTTAGAGATACCTCCCCTCCTTCTGCTCAGCTGCCTTGCAGTAATTAAACTCTTTCTCTGCTGC
AACACCCCTACTGTTCTCCGTGTATTGGCTTTTCTGGGCAGCAGGAAGGAAAAGCTGATGCGATGCTCTC
AGTGCCGCGTCGCCAAATACTGTAGTGCTAAGTGTCAGAAAAAAGCTTGGCCAGACCACAAGCGGGAATG
CAAATGCCTTAAAAGCTGCAAACCCAGATATCCTCCAGACTCCGTTCGACTTCTTGGCAGAGTTGTCTTC
AAACTTATGGATGGAGCACCTTCAGAATCAGAGAAGCTTTACTCATTTTATGATCTGGAGTCAAATATTA
ACAAACTGACTGAAGATAAGAAAGAGGGCCTCAGGCAACTCGTAATGACATTTCAACATTTCATGAGAGA
AGAAATACAGGATGCCTCTCAGCTGCCACCTGCCTTTGACCTTTTTGAAGCCTTTGCAAAAGTGATCTGC
AACTCTTTCACCATCTGTAATGCGGAGATGCAGGAAGTTGGTGTTGGCCTATATCCCAGTATCTCTTTGC
TCAATCACAGCTGTGACCCCAACTGTTCGATTGTGTTCAATGGGCCCCACCTCTTACTGCGAGCAGTCCG
AGACATCGAGGTGGGAGAGGAGCTCACCATCTGCTACCTGGATATGCTGATGACCAGTGAGGAGCGCCGG
AAGCAGCTGAGGGACCAGTACTGCTTTGAATGTGACTGTTTCCGTTGCCAAACCCAGGACAAGGATGCTG
ATATGCTAACTGGTGATGAGCAAGTATGGAAGGAAGTTCAAGAATCCCTGAAAAAAATTGAAGAACTGAA
GGCACACTGGAAGTGGGAGCAGGTTCTGGCCATGTGCCAGGCGATCATAAGCAGCAATTCTGAACGGCTT
CCCGATATCAACATCTACCAGCTGAAGGTGCTCGACTGCGCCATGGATGCCTGCATCAACCTCGGCCTGT
TGGAGGAAGCCTTGTTCTATGGTACTCGGACCATGGAGCCATACAGGATTTTTTTCCCAGGAAGCCATCC
CGTCAGAGGGGTTCAAGTGATGAAAGTTGGCAAACTGCAGCTACATCAAGGCATGTTTCCCCAAGCAATG
AAGAATCTGAGACTGGCTTTTGATATTATGAGAGTGACACATGGCAGAGAACACAGCCTGATTGAAGATT
TGATTCTACTTTTAGAAGAATGCGACGCCAACATCAGAGCATCCTAAGGGAACGCAGTCAGAGGGAAATA
CGGCGTGTGTCTTTGTTGAATGCCTTATTGAGGTCACACACTCTATGCTTTGTTAGCTGTGTGAACCTCT
CTTATTGGAAATTCTGTTCCGTGTTTGTGTAGGTAAATAAAGGCAGACATGGTTTGCAAACCACAAGAAT
CATTAGTTGTAGAGAAGCACGATTATAATAAATTCAAAACATTTGGTTGAGGATGCCAAAAAAAAAAAAA
AAAAAAA

NM_005186.2| Homo sapiens calpain 1, (mu/I) large subunit (CAPN1), mRNA
AAGGAGAGAGGGAGGGCGGAGGGCGGAGGGGCGGCGGGAGGAGGGCGGGGAGGAGCGCTCTTCCTGGTTG
GGCCCTGCCCTGAGCTGCCACCGGGAAGCCAGCCTCAGGGACTGCAGCGACCCCCAAACACCCCTCCCCC
AGGATGTCGGAGGAGATCATCACGCCGGTGTACTGCACTGGGGTGTCAGCCCAAGTGCAGAAGCAGCGGG
CCAGGGAGCTGGGCCTGGGCCGCCATGAGAATGCCATCAAGTACCTGGGCCAGGATTATGAGCAGCTGCG
GGTGCGATGCCTGCAGAGTGGGACCCTCTTCCGTGATGAGGCCTTCCCCCCGGTACCCCAGAGCCTGGGT
TACAAGGACCTGGGTCCCAATTCCTCCAAGACCTATGGCATCAAGTGGAAGCGTCCCACGGAACTGCTGT
CAAACCCCCAGTTCATTGTGGATGGAGCTACCCGCACAGACATCTGCCAGGGAGCACTGGGGGACTGCTG
GCTCTTGGCGGCCATTGCCTCCCTCACTCTCAACGACACCCTCCTGCACCGAGTGGTTCCGCACGGCCAG
AGCTTCCAGAATGGCTATGCCGGCATCTTCCATTTCCAGCTGTGGCAATTTGGGGAGTGGGTGGACGTGG
TCGTGGATGACCTGCTGCCCATCAAGGACGGGAAGCTAGTGTTCGTGCACTCTGCCGAAGGCAACGAGTT
CTGGAGCGCCCTGCTTGAGAAGGCCTATGCCAAGGTAAATGGCAGCTACGAGGCCCTGTCAGGGGGCAGC
ACCTCAGAGGGCTTTGAGGACTTCACAGGCGGGGTTACCGAGTGGTACGAGTTGCGCAAGGCTCCCAGTG
ACCTCTACCAGATCATCCTCAAGGCGCTGGAGCGGGGCTCCCTGCTGGGCTGCTCCATAGACATCTCCAG
CGTTCTAGACATGGAGGCCATCACTTTCAAGAAGTTGGTGAAGGGCCATGCCTACTCTGTGACCGGGGCC
AAGCAGGTGAACTACCGAGGCCAGGTGGTGAGCCTGATCCGGATGCGGAACCCCTGGGGCGAGGTGGAGT
GGACGGGAGCCTGGAGCGACAGCTCCTCAGAGTGGAACAACGTGGACCCATATGAACGGGACCAGCTCCG
GGTCAAGATGGAGGACGGGGAGTTCTGGATGTCATTCCGAGACTTCATGCGGGAGTTCACCCGCCTGGAG
ATCTGCAACCTCACACCCGACGCCCTCAAGAGCCGGACCATCCGCAAATGGAACACCACACTCTACGAAG
GCACCTGGCGGCGGGGGAGCACCGCGGGGGGCTGCCGAAACTACCCAGCCACCTTCTGGGTGAACCCTCA
GTTCAAGATCCGGCTGGATGAGACGGATGACCCGGACGACTACGGGGACCGCGAGTCAGGCTGCAGCTTC
GTGCTCGCCCTTATGCAGAAGCACCGTCGCCGCGAGCGCCGCTTCGGCCGCGACATGGAGACTATTGGCT
TCGCGGTCTACGAGGTCCCTCCGGAGCTGGTGGGCCAGCCGGCCGTACACTTGAAGCGTGACTTCTTCCT
GGCCAATGCGTCTCGGGCGCGCTCAGAGCAGTTCATCAACCTGCGAGAGGTCAGCACCCGCTTCCGCCTG
CCACCCGGGGAGTATGTGGTGGTGCCCTCCACCTTCGAGCCCAACAAGGAGGGCGACTTCGTGCTGCGCT
TCTTCTCAGAGAAGAGTGCTGGGACTGTGGAGCTGGATGACCAGATCCAGGCCAATCTCCCCGATGAGCA
AGTGCTCTCAGAAGAGGAGATTGACGAGAACTTCAAGGCCCTCTTCAGGCAGCTGGCAGGGGAGGACATG
GAGATCAGCGTGAAGGAGTTGCGGACAATCCTCAATAGGATCATCAGCAAACACAAAGACCTGCGGACCA

AGGGCTTCAGCCTAGAGTCGTGCCGCAGCATGGTGAACCTCATGGATCGTGATGGCAATGGGAAGCTGGG
CCTGGTGGAGTTCAACATCCTGTGGAACCGCATCCGGAATTACCTGTCCATCTTCCGGAAGTTTGACCTG
GACAAGTCGGGCAGCATGAGTGCCTACGAGATGCGGATGGCCATTGAGTCGGCAGGCTTCAAGCTCAACA
AGAAGCTGTACGAGCTCATCATCACCCGCTACTCGGAGCCCGACCTGGCGGTCGACTTTGACAATTTCGT
TTGCTGCCTGGTGCGGCTAGAGACCATGTTCCGATTTTTCAAAACTCTGGACACAGATCTGGATGGAGTT
GTGACCTTTGACTTGTTTAAGTGGTTGCAGCTGACCATGTTTGCATGAGGCAGGGACTCGGTCCCCCTTG
CCGTGCTCCCCTCCCTCCTCGTCTGCCAAGCCTCGCCTCCTACCACACCACACCAGGCCACCCCAGCTGC
AAGTGCCTTCCTTGGAGCAGAGAGGCAGCCTCGTCCTCCTGTCCCCTCTCCTCCCAGCCACCATCGTTCA
TCTGCTCCGGGCAGAACTGTGTGGCCCCTGCCTGTGCCAGCCATGGGCTCGGGATGGACTCCCTGGGCCC
CACCCATTGCCAAGCCAGGAAGGCAGCTTTCGCTTGTTCCTGCCTCGGGACAGCCCCGGGTTTCCCCAGC
ATCCTGATGTGTCCCCTCTCCCCACTTCAGAGGCCACCCACTCAGCACCACCGGCCTGGCCTTGCCTGCA
GACTATAAACTATAACCACTAGCTCGACACAGTCTGCAGTCCAGGCGTGTGGAGCCGCCTCCCGGCTCGG
GGAGGCCCCGGGGCTGGGAACGCCTGTGCCTTCCTGCGCCGAAGCCAACGCCCCCTCTGTCCTTCCCTGG
CCCTGCTGCCGACCAGGAGCTGCCCAGCCTGTGGGCGGTCGGCCTTCCCTCCTTCGCTCCTTTTTTATAT
TAGTGATTTTAAAGGGGACTCTTCAGGGACTTGTGTACTGGTTATGGGGGTGCCAGAGGCACTAGGCTTG
GGGTGGGGAGGTCCCGTGTTCCATATAGAGGAACCCCAAATAATAAAAGGCCCCACATCTGTCTGTG

NM_006032.2| Homo sapiens copine VI (neuronal) (CPNE6), mRNA
GCCGGAGCTGGAGCCGGCGCCAGCGGCTGGAGCAGCAAGGGAGTCAGGGCCAGGGCCAGAGAGCCGGAGA
GAGGAGCCCCCGACCGAGAGCCCAGTGACATGTCGGACCCTGAGATGGGATGGGTGCCTGAGCCCCCAAC
CATGACGCTGGGGGCCTCTCGGGTGGAGCTGCGGGTGTCCTGCCATGGCCTCCTGGACCGGGACACACTC
ACCAAACCCCACCCCTGCGTGCTGCTCAAGCTCTACTCTGATGAGCAGTGGGTGGAGGTAGAGCGCACAG
AGGTGCTTCGCTCCTGTTCCAGCCCTGTCTTCTCCCGGGTGCTGGCCCTTGAGTATTTTTTTGAGGAGAA
GCAGCCCCTGCAGTTCCACGTGTTCGATGCCGAGGACGGAGCCACCAGCCCCCGAAATGATACCTTCCTC
GGCTCTACGGAGTGCACCTTGGGCCAGATTGTGTCACAAACCAAGGTCACTAAGCCATTATTGCTGAAGA
ATGGGAAGACTGCGGGCAAGTCCACCATCACGATCGTGGCCGAGGAGGTATCAGGCACAAACGACTATGT
GCAACTCACCTTCAGAGCCTACAAGCTGGACAACAAGGATCTGTTCAGCAAGTCTGACCCTTTCATGGAA
ATCTATAAGACCAACGAGGACCAAAGTGATCAGCTGGTCTGGAGAACTGAGGTGGTGAAGAACAACCTGA
ACCCCAGCTGGGAGCCGTTCCGCCTGTCCCTGCATTCCCTATGCAGCTGTGATGTTCACCGACCTCTCAA
GTTCCTGGTGTATGACTATGACTCCAGTGGGAAGCATGACTTCATCGGCGAGTTCACCAGCACTTTCCAG
GAGATGCAGGAAGGGACGGCAAACCCTGGGCAGGAGATGCAGTGGGACTGTATCAACCCCAAGTATCGGG
ACAAGAAGAAGAATTACAAGAGCTCAGGGACGGTAGTGCTGGCCCAGTGCACGGTGGAGAAGGTGCACAC
CTTCCTGGATTACATCATGGGTGGCTGCCAGATCAGCTTCACGGTGGCCATTGACTTCACTGCCTCCAAT
GGGGACCCGAGGAGCAGCCAGTCCCTGCACTGCCTCAGTCCCCGACAGCCCAACCACTACCTGCAGGCCC
TGCGTGCAGTGGGAGGCATCTGCCAGGACTATGACAGTGATAAGCGGTTCCCAGCTTTTGGCTTTGGGGC
TCGAATCCCCCCCAACTTCGAGGTGTCCCATGACTTTGCTATCAACTTTGACCCGGAAAATCCTGAATGT
GAAGAGATCTCAGGGGTCATCGCCTCCTACCGTCGTTGCCTGCCCCAGATCCAGCTCTACGGCCCCACCA
ATGTGGCCCCCATCATCAACCGTGTGGCTGAGCCGGCCCAGCGGGAGCAGAGCACCGGCCAAGCCACGAA
GTACTCGGTGCTGCTGGTGCTCACTGACGGTGTGGTGAGCGACATGGCTGAGACTCGCACTGCTATCGTG
CGTGCCTCCCGCCTGCCCATGTCCATCATCATCGTAGGCGTGGGCAATGCTGACTTCTCTGACATGCGGC
TGCTGGATGGCGACGACGGCCCCTTGCGCTGCCCCCGAGGGGTGCCTGCAGCCCGAGACATTGTCCAGTT
CGTGCCCTTCCGAGACTTCAAGGATGCTGCCCCCTCTGCACTCGCCAAGTGTGTCCTGGCTGAGGTGCCA
CGGCAGGTGGTGGAGTACTACGCCAGCCAGGGCATCAGCCCTGGGGCTCCCAGGCCCTGCACACTGGCTA
CGACTCCCAGCCCTAGCCCGTGACTGCCTCCCTCCGGACCGACACTCCCTCAGCCTCTCAGTGCCTGTCC
TGACCCTCGTGACTCCAGTGACCAATGCCTCCACCTCTTGGACCAGGTGTGCCCCCTGGGTTCTGGACGT
GAGTGGTGGGTCCTGCTCCTATCTCTCCAAACCCCATACCCTTCAATGCTGTGGCCCCTCAGTGACTTCC
TTGGGTGATCCTGACTTTCTAGCCATTAATAAAGAGAACTGCTCCTAGCAAAAAAAAA

NM_000604.2| Homo sapiens fibroblast growth factor receptor 1 (fms-related tyrosine kinase 2, Pfeiffer syndrome) (FGFR1), transcript variant 1, mRNA
CCTCTTGCGGCCACAGGCGCGGCGTCCTCGGCGGCGGGCGGCAGCTAGCGGGAGCCGGGACGCCGGTGCA
GCCGCAGCGCGCGGAGGAACCCGGGTGTGCCGGGAGCTGGGCGGCCACGTCCGGACGGGACCGAGACCCC
TCGTAGCGCATTGCGGCGACCTCGCCTTCCCCGGCCGCGAGCGCGCCGCTGCTTGAAAAGCCGCGGAACC
CAAGGACTTTTCTCCGGTCCGAGCTCGGGGCGCCCCGCAGGCGCACGGTACCCGTGCTGCAGTCGGGCAC
GCCGCGGCGCCGGGGGCCTCCGCAGGGCGATGGAGCCGGTCTGCAAGGAAAGTGAGGCGCCGCCGCTGCG
TTCTGGAGGAGGGGGGCACAAGGTCTGGAGACCCCGGGTGGCGGACGGGAGCCCTCCCCCCGCCCCGCCT
CCGGGGCACCAGCTCCGGCTCCATTGTTCCCGCCCGGGCTGGAGGCGCCGAGCACCGAGCGCCGCCGGGA
GTCGAGCGCCGGCCGCGGAGCTCTTGCGACCCCGCCAGGACCCGAACAGAGCCCGGGGGCGGCGGGCCGG
AGCCGGGGACGCGGGCACACGCCCGCTCGCACAAGCCACGGCGGACTCTCCCGAGGCGGAACCTCCACGC
CGAGCGAGGGTCAGTTTGAAAAGGAGGATCGAGCTCACTGTGGAGTATCCATGGAGATGTGGAGCCTTGT

CACCAACCTCTAACTGCAGAACTGGGATGTGGAGCTGGAAGTGCCTCCTCTTCTGGGCTGTGCTGGTCAC
AGCCACACTCTGCACCGCTAGGCCGTCCCCGACCTTGCCTGAACAAGCCCAGCCCTGGGGAGCCCCTGTG
GAAGTGGAGTCCTTCCTGGTCCACCCCGGTGACCTGCTGCAGCTTCGCTGTCGGCTGCGGGACGATGTGC
AGAGCATCAACTGGCTGCGGGACGGGGTGCAGCTGGCGGAAAGCAACCGCACCCGCATCACAGGGGAGGA
GGTGGAGGTGCAGGACTCCGTGCCCGCAGACTCCGGCCTCTATGCTTGCGTAACCAGCAGCCCCTCGGGC
AGTGACACCACCTACTTCTCCGTCAATGTTTCAGATGCTCTCCCCTCCTCGGAGGATGATGATGATGATG
ATGACTCCTCTTCAGAGGAGAAAGAAACAGATAACACCAAACCAAACCGTATGCCCGTAGCTCCATATTG
GACATCCCCAGAAAAGATGGAAAAGAAATTGCATGCAGTGCCGGCTGCCAAGACAGTGAAGTTCAAATGC
CCTTCCAGTGGGACCCCAAACCCCACACTGCGCTGGTTGAAAAATGGCAAAGAATTCAAACCTGACCACA
GAATTGGAGGCTACAAGGTCCGTTATGCCACCTGGAGCATCATAATGGACTCTGTGGTGCCCTCTGACAA
GGGCAACTACACCTGCATTGTGGAGAATGAGTACGGCAGCATCAACCACACATACCAGCTGGATGTCGTG
GAGCGGTCCCCTCACCGGCCCATCCTGCAAGCAGGGTTGCCCGCCAACAAAACAGTGGCCCTGGGTAGCA
ACGTGGAGTTCATGTGTAAGGTGTACAGTGACCCGCAGCCGCACATCCAGTGGCTAAAGCACATCGAGGT
GAATGGGAGCAAGATTGGCCCAGACAACCTGCCTTATGTCCAGATCTTGAAGACTGCTGGAGTTAATACC
ACCGACAAAGAGATGGAGGTGCTTCACTTAAGAAATGTCTCCTTTGAGGACGCAGGGGAGTATACGTGCT
TGGCGGGTAACTCTATCGGACTCTCCCATCACTCTGCATGGTTGACCGTTCTGGAAGCCCTGGAAGAGAG
GCCGGCAGTGATGACCTCGCCCCTGTACCTGGAGATCATCATCTATTGCACAGGGGCCTTCCTCATCTCC
TGCATGGTGGGGTCGGTCATCGTCTACAAGATGAAGAGTGGTACCAAGAAGAGTGACTTCCACAGCCAGA
TGGCTGTGCACAAGCTGGCCAAGAGCATCCCTCTGCGCAGACAGGTAACAGTGTCTGCTGACTCCAGTGC
ATCCATGAACTCTGGGGTTCTTCTGGTTCGGCCATCACGGCTCTCCTCCAGTGGGACTCCCATGCTAGCA
GGGGTCTCTGAGTATGAGCTTCCCGAAGACCCTCGCTGGGAGCTGCCTCGGGACAGACTGGTCTTAGGCA
AACCCCTGGGAGAGGGCTGCTTTGGGCAGGTGGTGTTGGCAGAGGCTATCGGGCTGGACAAGGACAAACC
CAACCGTGTGACCAAAGTGGCTGTGAAGATGTTGAAGTCGGACGCAACAGAGAAAGACTTGTCAGACCTG
ATCTCAGAAATGGAGATGATGAAGATGATCGGGAAGCATAAGAATATCATCAACCTGCTGGGGGCCTGCA
CGCAGGATGGTCCCTTGTATGTCATCGTGGAGTATGCCTCCAAGGGCAACCTGCGGGAGTACCTGCAGGC
CCGGAGGCCCCCAGGGCTGGAATACTGCTACAACCCCAGCCACAACCCAGAGGAGCAGCTCTCCTCCAAG
GACCTGGTGTCCTGCGCCTACCAGGTGGCCCGAGGCATGGAGTATCTGGCCTCCAAGAAGTGCATACACC
GAGACCTGGCAGCCAGGAATGTCCTGGTGACAGAGGACAATGTGATGAAGATAGCAGACTTTGGCCTCGC
ACGGGACATTCACCACATCGACTACTATAAAAAGACAACCAACGGCCGACTGCCTGTGAAGTGGATGGCA
CCCGAGGCATTATTTGACCGGATCTACACCCACCAGAGTGATGTGTGGTCTTTCGGGGTGCTCCTGTGGG
AGATCTTCACTCTGGGCGGCTCCCCATACCCCGGTGTGCCTGTGGAGGAACTTTTCAAGCTGCTGAAGGA
GGGTCACCGCATGGACAAGCCCAGTAACTGCACCAACGAGCTGTACATGATGATGCGGGACTGCTGGCAT
GCAGTGCCCTCACAGAGACCCACCTTCAAGCAGCTGGTGGAAGACCTGGACCGCATCGTGGCCTTGACCT
CCAACCAGGAGTACCTGGACCTGTCCATGCCCCTGGACCAGTACTCCCCCAGCTTTCCCGACACCCGGAG
CTCTACGTGCTCCTCAGGGGAGGATTCCGTCTTCTCTCATGAGCCGCTGCCCGAGGAGCCCTGCCTGCCC
CGACACCCAGCCCAGCTTGCCAATGGCGGACTCAAACGCCGCTGACTGCCACCCACACGCCCTCCCCAGA
CTCCACCGTCAGCTGTAACCCTCACCCACAGCCCCTGCTGGGCCCACCACCTGTCCGTCCCTGTCCCCTT
TCCTGCTGGCAGGAGCCGGCTGCCTACCAGGGGCCTTCCTGTGTGGCCTGCCTTCACCCCACTCAGCTCA
CCTCTCCCTCCACCTCCTCTCCACCTGCTGGTGAGAGGTGCAAAGAGGCAGATCTTTGCTGCCAGCCACT
TCATCCCCTCCCAGATGTTGGACCAACACCCCTCCCTGCCACCAGGCACTGCCTGGAGGGCAGGGAGTGG
GAGCCAATGAACAGGCATGCAAGTGAGAGCTTCCTGAGCTTTCTCCTGTCGGTTTGGTCTGTTTTGCCTT
CACCCATAAGCCCCTCGCACTCTGGTGGCAGGTGCCTTGTCCTCAGGGCTACAGCAGTAGGGAGGTCAGT
GCTTCGTGCCTCGATTGAAGGTGACCTCTGCCCCAGATAGGTGGTGCCAGTGGCTTATTAATTCCGATAC
TAGTTTGCTTTGCTGACCAAATGCCTGGTACCAGAGGATGGTGAGGCGAAGGCCAGGTTGGGGGCAGTGT
TGTGGCCCTGGGGCCCAGCCCCAAACTGGGGGCTCTGTATATAGCTATGAAGAAAACACAAAGTGTATAA
ATCTGAGTATATATTTACATGTCTTTTTAAAAGGGTCGTTACCAGAGATTTACCCATCGGGTAAGATGCT
CCTGGTGGCTGGGAGGCATCAGTTGCTATATATTAAAAACAAAAAAGAAAAAAAAGGAAAATGTTTTTAA
AAAGGTCATATATTTTTTGCTACTTTTGCTGTTTTATTTTTTTAAATTATGTTCTAAAC

NM_032875.1| Homo sapiens F-box and leucine-rich repeat protein 20 (FBXL20), mRNA

GAGAGCCGAGCTAGCGACGAGCAGTCGTTGCGGCCGCCGGCGCCGCGGGAGGTGGTGGAGGCCTAGCCGG
AGCCGAGAGGTCTCTTGTTCCCGTCCCACGGTCCCGGCGTCACCCCTCCGGCGCCCAGTCCCCGTCCCGG
AACTCCCGGGCCTGTCCTGGGCCCCCGGTCTGTGCACTCCGCTCGCCGCAGCGCCCGGCCCGGGCCGCAC
CCGCCGGCCCCATGAGGAGGGACGTGAACGGAGTGACCAAGAGCAGGTTTGAGATGTTCTCAAATAGTGA
TGAAGCTGTAATCAATAAAAAACTTCCCAAAGAACTCCTGTTACGGATATTTTCTTTTCTAGATGTTGTT
ACCCTGTGCCGCTGTGCTCAGGTCTCCAGGGCCTGGAATGTTCTGGCTCTGGATGGCAGTAACTGGCAGC
GAATTGACCTATTTGATTTCCAGAGGGATATTGAGGGCCGAGTAGTGGAGAATATTTCAAAACGATGTGG
GGGCTTTTTACGAAAGTTAAGTCTTCGTGGATGTCTTGGAGTGGGAGACAATGCATTAAGAACCTTTGCA
CAAAACTGCAGGAACATTGAAGTACTGAATCTAAATGGGTGTACAAAGACAACAGACGCTACATGTACTA
GCCTTAGCAAGTTCTGTTCCAAACTCAGGCACCTTGACTTGGCTTCCTGTACATCAATAACAAACATGTC

Figure 64 (Continued)

TCTAAAAGCTCTGAGTGAGGGATGTCCACTGTTGGAGCAGTTGAACATTTCCTGGTGTGACCAAGTAACC
AAGGATGGCATTCAAGCACTAGTGAGGGGCTGTGGGGGTCTCAAGGCCTTATTCTTAAAAGGCTGCACGC
AGCTAGAAGATGAAGCTCTCAAGTACATAGGTGCACACTGCCCTGAACTGGTGACTTTGAACTTGCAGAC
TTGCTTGCAAATCACAGATGAAGGTCTCATTACTATATGCAGAGGGTGCCATAAGTTACAATCCCTTTGT
GCCTCTGGCTGCTCCAACATCACAGATGCCATCCTGAATGCTCTAGGTCAGAACTGCCCACGGCTTAGAA
TATTGGAAGTGGCAAGATGTTCTCAATTAACAGATGTGGGCTTTACCACTCTAGCCAGGAATTGCCATGA
ACTTGAAAAGATGGACCTGGAAGAGTGTGTTCAGATAACAGATAGCACATTAATCCAACTTTCTATACAC
TGTCCTCGACTTCAAGTATTGAGTCTGTCTCACTGTGAGCTGATCACAGATGATGGAATTCGTCACCTGG
GGAATGGGGCCTGCGCCCATGACCAGCTGGAGGTGATTGAGCTGGACAACTGCCCACTAATCACAGATGC
ATCCCTGGAGCACTTGAAGAGCTGTCATAGCCTTGAGCGGATAGAACTCTATGACTGCCAGCAAATCACA
CGGGCTGGAATCAAGAGACTCAGGACCCATTTACCCAATATTAAAGTCCACGCCTACTTCGCACCTGTCA
CTCCACCCCCATCAGTAGGGGGCAGCAGACAGCGCTTCTGCAGATGCTGCATCATCCTATGACAATGGAG
GTGGTCAACCTTGGCGAACTGAGTATTTAATGACACTTCTAGAGCTACCGTGGAGTCTCTCCAGTGGAAG
CAACCCCAGTGTTCTGAGCAAGGGTTACAAAGTGAGGGAGGGCAGTGTCCAGATCCCCAGAGCCACACAT
ACATACACATACACACCCTTACCCCCATCCACTCTAGCTTTGTGACCATGGGACTGAAGTTTGTGATGGC
TTTTTTATCAAGTAGATTGGTAAAATTTAACCATTCCTGTTGAGGTGCCCATAAGAAAATCATAGGCCAA
GATAGGGAGGGGCATTCCAGCAAACCCCGTGTTAATGCTACTGTGGTTTTTAAATTTTTGTCTAGGGGTT
TCTTTGGGGATTTTAGAACAGCATCTGCTGTCCTCCGGGGTCAAGAAAAGCATGGAAAGACAATATATGA
TGTACCCAGGGACCAGAAAGAAAATTTCTTTGCATCTTAGAAATGGTAGACATTCATTGTGACTAAAGAG
CTTCTATGCTTCCTTGTTTCCATGCCAACATGCTGAGCATGCTCACAAAGAAGGCTCGTCCATTCCTCCT
GTGTTTTAGTATTTGGCCCAGAGGTTTCCTAAATGGTTGCCTTGAAATCACTGTGGTCCAAATGTAATTC
TTACACACTCAAATTATCACTGTCTGTAGCACACTTGTGCACCTGTCTTACATTCTCTGTTGCTCCCCCC
CACACTCTTGCTCAGTCTGTCACCTGTTCAGTCTGCTTACTCACTCAATTGTTACCCTTTTGCTGTTGTC
GTGTTTACAGTTTGCATTTTGAATGATTAGTTGGGATTACCAAACATTTTTTAAAAAGATATTATCAATA
AATATTTTTTTAATTCTAAATTTTAAAAAAAAAAAAAAAAAA

NM_020414.3| Homo sapiens DEAD (Asp-Glu-Ala-Asp) box polypeptide 24 (DDX24), mRNA
CCGGAACTGCAGTTGCTGCTGCAGCTGAGGTACAGCGGCGGTTTCTGAGGTTCTTCACTCGCGACTGACG
GAGCTGCGGTGGCGTCTCCACACGCAACCATGAAGTTGAAGGACACAAAATCAAGGCCAAAGCAGTCAAG
CTGTGGCAAATTTCAGACAAAGGGAATCAAAGTTGTGGGAAAATGGAAGGAAGTGAAGATTGACCCAAAT
ATGTTTGCAGATGGACAGATGGATGACTTGGTGTGCTTTGAGGAATTGACAGATTACCAGTTGGTCTCCC
CTGCCAAGAATCCCTCCAGTCTCTTCTCAAAGGAAGCACCCAAGAGAAAGGCACAAGCTGTTTCAGAAGA
AGAGGAGGAGGAGGAGGGAAAGTCTAGCTCACCAAAGAAAAAGATCAAGTTGAAGAAAAGTAAAAATGTA
GCAACTGAAGGAACCAGTACCCAGAAAGAATTTGAAGTGAAAGATCCTGAGCTGGAGGCCCAGGGAGATG
ACATGGTTTGTGATGATCCGGAGGCTGGGGAGATGACATCAGAAAACCTGGTCCAAACTGCTCCAAAAAA
GAAGAAAAATAAAGGGAAAAAAGGGTTGGAGCCTTCTCAGAGCACTGCTGCCAAGGTGCCCAAAAAAGCG
AAGACATGGATTCCTGAAGTTCATGATCAGAAAGCAGATGTGTCAGCTTGGAAGGACCTGTTTGTTCCCA
GGCCGGTTCTCCGAGCACTCAGCTTTCTAGGCTTCTCTGCACCCACACCAATCCAAGCCCTGACCTTGGC
ACCTGCCATCCGTGACAAACTGGACATCCTTGGGGCTGCTGAGACAGGAAGTGGGAAAACTCTTGCCTTT
GCCATCCCAATGATTCATGCGGTGTTGCAGTGGCAGAAGAGGAATGCTGCCCCTCCTCCAAGTAACACCG
AAGCACCACCTGGAGAGACCAGAACTGAGGCCGGAGCTGAGACTAGATCACCAGGCAAGGCTGAAGCTGA
GTCTGATGCATTGCCTGACGATACTGTAATTGAGAGTGAAGCACTGCCCAGTGATATTGCAGCCGAGGCC
AGAGCCAAGACTGGAGGCACTGTCTCAGACCAGGCGTTGCTCTTTGGTGACGATGATGCTGGTGAAGGGC
CTTCTTCCCTGATCAGGGAGAAACCTGTTCCCAAACAGAATGAGAATGAGGAGGAAAATCTTGATAAAGA
GCAGACTGGAAATCTAAAACAGGAGTTGGATGACAAAAGCGCCACCTGTAAGGCATATCCAAAGCGTCCT
CTGCTTGGACTGGTTCTGACTCCCACTCGAGAGCTGGCCGTCCAGGTCAAACAGCACATTGATGCTGTGG
CCAGGTTTACAGGAATTAAAACTGCTATTTTGGTTGGTGGAATGTCCACGCAGAAACAGCAGAGGATGCT
GAACCGTCGTCCTGAGATTGTGGTTGCTACTCCAGGCCGGCTGTGGGAATTAATTAAAGAAAAGCATTAT
CATTTGAGGAACCTTCGGCAGCTCAGGTGCCTGGTAGTGGATGAGGCTGACCGGATGGTTGAGAAAGGCC
ATTTTGCTGAGCTCTCACAGCTGCTAGAGATGCTCAATGACTCCCAATACAACCCAAAGAGACAAACGCT
TGTTTTTTCTGCCACACTCACCCTGGTGCATCAGGCTCCTGCTCGAATCCTTCATAAGAAGCACACCAAG
AAAATGGATAAAACAGCCAAACTTGACCTCCTTATGCAGAAAATTGGCATGAGGGGCAAGCCCAAGGTCA
TTGACCTCACAAGGAATGAGGCCACGGTGGAGACGCTAACAGAGACCAAGATCCATTGTGAGACTGATGA
GAAAGACTTCTACTTGTACTACTTCCTGATGCAGTATCCAGGCCGCAGCTTAGTGTTTGCCAACAGTATC
TCCTGCATCAAACGCCTCTCTGGGCTCCTCAAAGTCCTTGATATCATGCCCTTGACCCTGCATGCCTGTA
TGCACCAGAAGCAGAGGCTCAGAAACCTGGAGCAGTTTGCCCGTCTGGAAGACTGTGTTCTCTTGGCAAC
AGATGTGGCAGCTCGGGGTCTGGATATTCCTAAAGTCCAGCATGTCATCCATTACCAGGTCCCACGTACC
TCGGAGATTTATGTCCACCGAAGTGGTCGAACTGCTCGAGCTACCAATGAAGGCCTCAGTCTGATGCTCA
TTGGGCCTGAGGATGTGATCAACTTTAAGAAGATTTACAAAACGCTCAAGAAAGATGAGGATATCCCACT
GTTCCCCGTGCAGACAAAATACATGGATGTGGTCAAGGAGCGAATCCGTTTAGCTCGACAGATTGAGAAA

TCTGAGTATCGGAACTTCCAGGCTTGCCTGCACAACTCTTGGATTGAGCAGGCAGCAGCTGCCCTGGAGA
TTGAGCTGGAAGAAGACATGTATAAGGGAGGAAAAGCTGACCAGCAAGAAGAACGTCGGAGACAAAAGCA
GATGAAGGTTCTGAAGAAGGAGCTGCGCCACCTGCTGTCCCAGCCACTGTTTACGGAGAGCCAGAAAACC
AAGTATCCCACTCAGTCTGGCAAGCCGCCCCTGCTTGTGTCTGCCCCAAGTAAGAGCGAGTCTGCTTTGA
GCTGTCTCTCCAAGCAGAAGAAGAAGACAAAGAAGCCGAAGGAGCCACAGCCGGAACAGCCACAGCC
AAGTACAAGTGCAAATTAACTGGTCAAGTGTGTCAGTGACTGCACATTGGTTTCTGTTCTCTGGCTATTT
GCAAAACCTCTCCCACCCTTGTGTTTCACTCCACCACCAACCCCAGGTAAAAAAGTCTCCCTCTCTTCCA
CTCACACCCATAGCGGGAGAGACCTCATGCAGATTTGCATTGTTTTGGAGTAAGAATTCAATGCAGCAGC
TTAATTTTTCTGTATTGCAGTGTTTATAGGCTTCTTGTGTGTTAAACTTGATTTCATAAATTAAAAACAA
TGGTCAGAAAAAAAAAAAAAAAAAAA

NM_053046.2| Homo sapiens egl nine homolog 2 (C. elegans) (EGLN2),
transcript variant 1, mRNA
GCTTTCCCCTGCCTGCCTGTCTCTAGTTTCTCTCACATCCCTTTTTTTTTTTCCTTTCTCTAGCCACCCT
GAAGGGTCCCTTCCCAAGCCCTTAGGGACCGCAGAGGACTTGGGGACCAGCAAGCAACCCCCAGGGCACG
AGAAGAGCTCTTGCTGTCTGCCCTGCCTCACCCTGCCCCACGCCAGGCCCGGTGGCCCCCAGCTGCATCA
AGTGGAGGCGGAGGAGGAGGCGGAGGAGGGTGGCACCATGGGCCCGGGCGGTGCCCTCCATGCCCGGGGG
ATGAAGACACTGCTGCCATGGACAGCCCGTGCCAGCCGCAGCCCCTAAGTCAGGCTCTCCCTCAGTTACC
AGGGTCTTCGTCAGAGCCCTTGGAGCCTGAGCCTGGCCGGGCCAGGATGGGAGTGGAGAGTTACCTGCCC
TGTCCCCTGCTCCCCTCCTACCACTGTCCAGGAGTGCCTAGTGAGGCCTCGGCAGGGAGTGGGACCCCCA
GAGCCACAGCCACCTCTACCACTGCCAGCCCTCTTCGGGACGGTTTTGGCGGGCAGGATGGTGGTGAGCT
GCGGCCGCTGCAGAGTGAAGGCGCTGCAGCGCTGGTCACCAAGGGGTGCCAGCGATTGGCAGCCCAGGGC
GCACGGCCTGAGGCCCCCAAACGGAAATGGGCCGAGGATGGTGGGGATGCCCCTTCACCCAGCAAACGGC
CCTGGGCCAGGCAAGAGAACCAGGAGGCAGAGCGGGAGGGTGGCATGAGCTGCAGCTGCAGCAGTGGCAG
TGGTGAGGCCAGTGCTGGGCTGATGGAGGAGGCGCTGCCCTCTGCGCCCGAGCGCCTGGCCCTGGACTAT
ATCGTGCCCTGCATGCGGTACTACGGCATCTGCGTCAAGGACAGCTTCCTGGGGGCAGCACTGGGCGGTC
GCGTGCTGGCCGAGGTGGAGGCCCTCAAACGGGGTGGGCGCCTGCGAGACGGGCAGCTAGTGAGCCAGAG
GGCGATCCCGCCGCGCAGCATCCGTGGGGACCAGATTGCCTGGGTGGAAGGCCATGAACCAGGCTGTCGA
AGCATTGGTGCCCTCATGGCCCATGTGGACGCCGTCATCCGCCACTGCGCAGGGCGGCTGGGCAGCTATG
TCATCAACGGGCGCACCAAGGCCATGGTGGCGTGTTACCCAGGCAACGGGCTCGGGTACGTAAGGCACGT
TGACAATCCCCACGGCGATGGGCGCTGCATCACCTGTATCTATTACCTGAATCAGAACTGGGACGTTAAG
GTGCATGGCGGCCTGCTGCAGATCTTCCCTGAGGGCCGGCCCGTGGTAGCCAACATCGAGCCACTCTTTG
ACCGGTTGCTCATTTTCTGGTCTGACCGGCGGAACCCCCACGAGGTGAAGCCAGCCTATGCCACCAGGTA
CGCCATCACTGTCTGGTATTTTGATGCCAAGGAGCGGGCAGCAGCCAAAGACAAGTATCAGCTAGCATCA
GGACAGAAAGGTGTCCAAGTACCTGTATCACAGCCGCCTACGCCCACCTAGTGGCCAGTCCCAGAGCCGC
ATGGCAGACAGCTTAAATGACTTCAGGAGAGCCCTGGGCCTGTGCTGGCTGCTCCTTCCCTGCCACCGCT
GCTGCTTCTGACTTTGCCTCTGTCCTGCCTGGTGTGGAGGGCTCTGTCTGTTGCTGAGGACCAAGGAGGA
GAAGAGACCTTTGCTGCCCCATCATGGGGGCTGGGGTTGTCACCTGGACAGGGGGCAGCCGTGGAGGCCA
CCGTTACCAACTGAAGCTGGGGGCCTGGGTCCTACCCTGTCTGGTCATGACCCCATTAGGTATGGAGAGC
TGGGAGGAGGCATTGTCACTTCCCACCAGGATGCAGGACTTGGGGTTGAGGTGAGTCATGGCCTCTTGCT
GGCAATGGGGTGGGAGGAGTACCCCCAAGTCCTCTCACTCCTCCAGCCTGGAATGTGAAGTGACTCCCCA
ACCCCTTTGGCCATGGCAGGCACCTTTTGGACTGGGCTGCCACTGCTTGGGCAGAGTAAAAGGTGCCAGG
AGGAGCATGGGTGTGGAAGTCCTGTCAGCCAAGAAATAAAAGTTTACCTCAGAGCTGCAAAAAAAAAAAA
AAAAAAAAAAA

NM_003265.2| Homo sapiens toll-like receptor 3 (TLR3), mRNA
CACTTTCGAGAGTGCCGTCTATTTGCCACACACTTCCCTGATGAAATGTCTGGATTTGGACTAAAGAAAA
AAGGAAAGGCTAGCAGTCATCCAACAGAATCATGAGACAGACTTTGCCTTGTATCTACTTTTGGGGGGGC
CTTTTGCCCTTTGGGATGCTGTGTGCATCCTCCACCACCAAGTGCACTGTTAGCCATGAAGTTGCTGACT
GCAGCCACCTGAAGTTGACTCAGGTACCCGATGATCTACCCACAAACATAACAGTGTTGAACCTTACCCA
TAATCAACTCAGAAGATTACCAGCCGCCAACTTCACAAGGTATAGCCAGCTAACTAGCTTGGATGTAGGA
TTTAACACCATCTCAAAACTGGAGCCAGAATTGTGCCAGAAACTTCCCATGTTAAAAGTTTTGAACCTCC
AGCACAATGAGCTATCTCAACTTTCTGATAAAACCTTTGCCTTCTGCACGAATTTGACTGAACTCCATCT
CATGTCCAACTCAATCCAGAAAATTAAAAATAATCCCTTTGTCAAGCAGAAGAATTTAATCACATTAGAT
CTGTCTCATAATGGCTTGTCATCTACAAAATTAGGAACTCAGGTTCAGCTGGAAAATCTCCAAGAGCTTC
TATTATCAAACAATAAAATTCAAGCGCTAAAAAGTGAAGAACTGGATATCTTTGCCAATTCATCTTTAAA
AAAATTAGAGTTGTCATCGAATCAAATTAAAGAGTTTTCTCCAGGGTGTTTTCACGCAATTGGAAGATTA
TTTGGCCTCTTTCTGAACAATGTCCAGCTGGGTCCCAGCCTTACAGAGAAGCTATGTTTGGAATTAGCAA
ACACAAGCATTCGGAATCTGTCTCTGAGTAACAGCCAGCTGTCCACCACCAGCAATACAACTTTCTTGGG
ACTAAAGTGGACAAATCTCACTATGCTCGATCTTTCCTACAACAACTTAAATGTGGTTGGTAACGATTCC
TTTGCTTGGCTTCCACAACTAGAATATTTCTTCCTAGAGTATAATAATATACAGCATTTGTTTTCTCACT

CTTTGCACGGGCTTTTCAATGTGAGGTACCTGAATTTGAAACGGTCTTTTACTAAACAAAGTATTTCCCT
TGCCTCACTCCCCAAGATTGATGATTTTTCTTTTCAGTGGCTAAAATGTTTGGAGCACCTTAACATGGAA
GATAATGATATTCCAGGCATAAAAAGCAATATGTTCACAGGATTGATAAACCTGAAATACTTAAGTCTAT
CCAACTCCTTTACAAGTTTGCGAACTTTGACAAATGAAACATTTGTATCACTTGCTCATTCTCCCTTACA
CATACTCAACCTAACCAAGAATAAAATCTCAAAAATAGAGAGTGATGCTTTCTCTTGGTTGGGCCACCTA
GAAGTACTTGACCTGGGCCTTAATGAAATTGGGCAAGAACTCACAGGCCAGGAATGGAGAGGTCTAGAAA
ATATTTTCGAAATCTATCTTTCCTACAACAAGTACCTGCAGCTGACTAGGAACTCCTTTGCCTTGGTCCC
AAGCCTTCAACGACTGATGCTCCGAAGGGTGGCCCTTAAAAATGTGGATAGCTCTCCTTCACCATTCCAG
CCTCTTCGTAACTTGACCATTCTGGATCTAAGCAACAACAACATAGCCAACATAAATGATGACATGTTGG
AGGGTCTTGAGAAACTAGAAATTCTCGATTTGCAGCATAACAACTTAGCACGGCTCTGGAAACACGCAAA
CCCTGGTGGTCCCATTTATTTCCTAAAGGGTCTGTCTCACCTCCACATCCTTAACTTGGAGTCCAACGGC
TTTGACGAGATCCCAGTTGAGGTCTTCAAGGATTTATTTGAACTAAAGATCATCGATTTAGGATTGAATA
ATTTAAACACACTTCCAGCATCTGTCTTTAATAATCAGGTGTCTCTAAAGTCATTGAACCTTCAGAAGAA
TCTCATAACATCCGTTGAGAAGAAGGTTTTCGGGCCAGCTTTCAGGAACCTGACTGAGTTAGATATGCGC
TTTAATCCCTTTGATTGCACGTGTGAAAGTATTGCCTGGTTTGTTAATTGGATTAACGAGACCCATACCA
ACATCCCTGAGCTGTCAAGCCACTACCTTTGCAACACTCCACCTCACTATCATGGGTTCCCAGTGAGACT
TTTTGATACATCATCTTGCAAAGACAGTGCCCCCTTTGAACTCTTTTTCATGATCAATACCAGTATCCTG
TTGATTTTTATCTTTATTGTACTTCTCATCCACTTTGAGGGCTGGAGGATATCTTTTTATTGGAATGTTT
CAGTACATCGAGTTCTTGGTTTCAAAGAAATAGACAGACAGACAGAACAGTTTGAATATGCAGCATATAT
AATTCATGCCTATAAAGATAAGGATTGGGTCTGGGAACATTTCTCTTCAATGGAAAAGGAAGACCAATCT
CTCAAATTTTGTCTGGAAGAAAGGGACTTTGAGGCGGGTGTTTTTGAACTAGAAGCAATTGTTAACAGCA
TCAAAAGAAGCAGAAAAATTATTTTTGTTATAACACACCATCTATTAAAAGACCCATTATGCAAAAGATT
CAAGGTACATCATGCAGTTCAACAAGCTATTGAACAAAATCTGGATTCCATTATATTGGTTTTCCTTGAG
GAGATTCCAGATTATAAACTGAACCATGCACTCTGTTTGCGAAGAGGAATGTTTAAATCTCACTGCATCT
TGAACTGGCCAGTTCAGAAAGAACGGATAGGTGCCTTTCGTCATAAATTGCAAGTAGCACTTGGATCCAA
AAACTCTGTACATTAAATTTATTTAAATATTCAATTAGCAAAGGAGAAACTTTCTCAATTTAAAAAGTTC
TATGGCAAATTTAAGTTTTCCATAAAGGTGTTATAATTTGTTTATTCATATTTGTAAATGATTATATTCT
ATCACAATTACATCTCTTCTAGGAAAATGTGTCTCCTTATTTCAGGCCTATTTTTGACAATTGACTTAAT
TTTACCCAAAATAAAACATATAAGCACGTAAAAAAAAAAAAAAAAAA

NM_016479.3| Homo sapiens shisa homolog 5 (Xenopus laevis) (SHISA5), mRNA

CGGACAGAGGTTCCGGGAACCAGCCGGGCCGGGGCGGGGCGGGGCGAGGGAGAGGGGCGGCCGCGCGGAT
CACTGAGGCTGTGGCGGCACTGCGCCCGGCGCTCGCGTCCGTCCGCCCGTCCGCCCGCCCAGCCATGACT
GCGCCGGTCCCCGCGCCGCGGATCCTGTTGCCGTTGCTGTTGCTGCTGCTGCTAACGCCGCCTCCGGGTG
CACGTGGTGAGGTGTGTATGGCTTCCCGTGGACTCAGCCTCTTCCCCGAGTCCTGTCCAGATTTCTGCTG
TGGTACCTGTGATGACCAATACTGCTGCTCTGACGTGCTGAAGAAATTTGTGTGGAGCGAGGAAAGGTGT
GCTGTGCCTGAGGCCAGCGTGCCTGCCAGTGTAGAGCCGGTGGAGCAGCTGGGCTCGGCGCTGAGGTTTC
GCCCTGGCTACAACGACCCCATGTCAGGGTTCGGAGCGACCTTGGCCGTTGGCCTGACCATCTTTGTGCT
GTCTGTCGTCACTATCATCATCTGCTTCACCTGCTCCTGCTGCTGCCTTTACAAGACGTGCCGCCGACCA
CGTCCGGTTGTCACCACCACCACATCCACCACTGTGGTGCATGCCCCTTATCCTCAGCCTCCAAGTGTGC
CGCCCAGCTACCCTGGACCAAGCTACCAGGGCTACCACACCATGCCGCCTCAGCCAGGGATGCCAGCAGC
ACCCTACCCAATGCAGTACCCACCACCTTACCCAGCCCAGCCCATGGGCCCACCGGCCTACCACGAGACC
CTGGCTGGAGGAGCAGCCGCGCCCTACCCCGCCAGCCAGCCTCCTTACAACCCGGCCTACATGGATGCCC
CGAAGGCGGCCCTCTGAGCATTCCCTGGCCTCTCTGGCTGCCACTTGGTTATGTTGTGTGTGTGCGTGAG
TGGTGTGCAGGCGCGGTTCCTTACGCCCCATGTGTGCTGTGTGTGTCCAGGCACGGTTCCTTACGCCCCA
TGTGTGCTGTGTGTGTCCTGCCTGTATATGTGGCTTCCTCTGATGCTGACAAGGTGGGGAACAATCCTTG
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AACTCAAAGAATGGGGTGGTGGGGGGCACCCTGTGAGGTGGCCCCTGAGAGGTGGGGGCCTCTCCAGGGC
ACATCTGGAGTTCTTCTCCAGCTTACCCTAGGGTGACCAAGTAGGGCCTGTCACACCAGGGTGGCGCAGC
TTTCTGTGTGATGCAGATGTGTCCTGGTTTCGGCAGCGTAGCCAGCTGCTGCTTGAGGCCATGGCTCGTC
CCCGGAGTTGGGGGTACCCGTTGCAGAGCCAGGGACATGATGCAGGCGAAGCTTGGGATCTGGCCAAGTT
GGACTTTGATCCTTTGGGCAGATGTCCCATTGCTCCCTGGAGCCTGTCATGCCTGTTGGGGATCAGGCAG
CCTCCTGATGCCAGAACACCTCAGGCAGAGCCCTACTCAGCTGTACCTGTCTGCCTGGACTGTCCCCTGT
CCCCGCATCTCCCCTGGGACCAGCTGGAGGGCCACATGCACACACAGCCTAGCTGCCCCCAGGGAGCTCT
GCTGCCCTTGCTGGCCCTGCCCTTCCCACAGGTGAGCAGGGCTCCTGTCCACCAGCACACTCAGTTCTCT
TCCCTGCAGTGTTTTCATTTTATTTTAGCCAAACATTTTGCCTGTTTTCTGTTTCAAACATGATAGTTGA
TATGAGACTGAAACCCCTGGGTTGTGGAGGGAAATTGGCTCAGAGATGGACAACCTGGCAACTGTGAGTC
CCTGCTTCCCGACACCAGCCTCATGGAATATGCAACAACTCCTGTACCCCAGTCCACGGTGTTCTGGCAG
CAGGGACACCTGGGCCAATGGGCCATCTGGACCAAAGGTGGGGTGTGGGGCCCTGGATGGCAGCTCTGGC
CCAGACATGAATACCTCGTGTTCCTCCTCCCTCTATTACTGTTTCACCAGAGCTGTCTTAGCTCAAATCT
GTTGTGTTTCTGAGTCTAGGGTCTGTACACTTGTTTATAATAAATGCAATCGTTTGGAGCTGCTGCCCCC

TTTCTTCCTGGCCTCGGCTGCTGGAATTGGAATCAGGCTGTACTCTTTCCATCCATTTGGGCTTCT

NM_004708.2| Homo sapiens programmed cell death 5 (PDCD5), mRNA
AGGCCGCGCTCGCGCCGAGGGGCTGCGAGAGTGACCGCGGCTGCTCCAGCGCTGACGCCGAGCCATGGCG
GACGAGGAGCTTGAGGCGCTGAGGAGACAGAGGCTGGCCGAGCTGCAGGCCAAACACGGGGATCCTGGTG
ATGCGGCCCAACAGGAAGCAAAGCACAGGGAAGCAGAAATGAGAAACAGTATCTTAGCCCAAGTTCTGGA
TCAGTCGGCCCGGGCCAGGTTAAGTAACTTAGCACTTGTAAAGCCTGAAAAAACTAAAGCAGTAGAGAAT
TACCTTATACAGATGGCAAGATATGGACAACTAAGTGAGAAGGTATCAGAACAAGGTTTAATAGAAATCC
TTAAAAAAGTAAGCCAACAAACAGAAAAGACAACAACAGTGAAATTCAACAGAAGAAAAGTAATGGACTC
TGATGAAGATGACGATTATTGAACTACAAGTGCTCACAGACTAGAACTTAACGGAACAAGTCTAGGACAG
AAGTTAAGATCTGATTATTTACTTTGTTTATTGTCTATATGCCTTTTAAAAAAATAAACTTGTTATGCAA
AATAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_001192.2| Homo sapiens tumor necrosis factor receptor superfamily, member 17 (TNFRSF17), mRNA
AAGACTCAAACTTAGAAACTTGAATTAGATGTGGTATTCAAATCCTTAGCTGCCGCGAAGACACAGACAG
CCCCCGTAAGAACCCACGAAGCAGGCGAAGTTCATTGTTCTCAACATTCTAGCTGCTCTTGCTGCATTTG
CTCTGGAATTCTTGTAGAGATATTACTTGTCCTTCCAGGCTGTTCTTTCTGTAGCTCCCTTGTTTTCTTT
TTGTGATCATGTTGCAGATGGCTGGGCAGTGCTCCCAAAATGAATATTTTGACAGTTTGTTGCATGCTTG
CATACCTTGTCAACTTCGATGTTCTTCTAATACTCCTCCTCTAACATGTCAGCGTTATTGTAATGCAAGT
GTGACCAATTCAGTGAAAGGAACGAATGCGATTCTCTGGACCTGTTTGGGACTGAGCTTAATAATTTCTT
TGGCAGTTTTCGTGCTAATGTTTTTGCTAAGGAAGATAAACTCTGAACCATTAAAGGACGAGTTTAAAAA
CACAGGATCAGGTCTCCTGGGCATGGCTAACATTGACCTGGAAAAGAGCAGGACTGGTGATGAAATTATT
CTTCCGAGAGGCCTCGAGTACACGGTGGAAGAATGCACCTGTGAAGACTGCATCAAGAGCAAACCGAAGG
TCGACTCTGACCATTGCTTTCCACTCCCAGCTATGGAGGAAGGCGCAACCATTCTTGTCACCACGAAAAC
GAATGACTATTGCAAGAGCCTGCCAGCTGCTTTGAGTGCTACGGAGATAGAGAAATCAATTTCTGCTAGG
TAATTAACCATTTCGACTCGAGCAGTGCCACTTTAAAAATCTTTTGTCAGAATAGATGATGTGTCAGATC
TCTTTAGGATGACTGTATTTTTCAGTTGCCGATACAGCTTTTTGTCCTCTAACTGTGGAAACTCTTTATG
TTAGATATATTTCTCTAGGTTACTGTTGGGAGCTTAATGGTAGAAACTTCCTTGGTTTCATGATTAAACT
CTTTTTTTTCCTGA

NM_032871.3| Homo sapiens RELT tumor necrosis factor receptor (RELT), transcript variant 1, mRNA
CGCGTCCTGTCCCGGCGCCTTCCGCGCACCGGCCACCGCCAGTCTCCGAGCCCCGTGACCTGCAGGTCCT
CCGGCCGCGACTCCGGGCCGGCCCTGCGTGGTGGCTGTCGGGGGGCGCGCGGGGAAGCAGCGGAACTTGC
GGTGTGAGGGGCCGGCGGGGCCCGGAGCGGTCCCCGGCCCGGGAAACTTGGACCGAGACCAGGCCGGCGA
CCACCAGGGGCCTGAGGATGAAGCCAAGTCTGCTGTGCCGGCCCCTGTCCTGCTTCCTTATGCTGCTGCC
CTGGCCTCTCGCCACCCTGACATCAACAACCCTTTGGCAGTGCCCACCTGGGGAGGAGCCCGACCTGGAC
CCAGGGCAGGGCACATTATGCAGGCCCTGCCCCCCAGGCACCTTCTCAGCTGCATGGGGCTCCAGCCCAT
GCCAGCCCCATGCCCGTTGCAGCCTTTGGAGGAGGCTGGAGGCCCAGGTGGGCATGGCAACTCGAGATAC
ACTCTGTGGAGACTGCTGGCCTGGGTGGTTTGGGCCTTGGGGGGTTCCCCGCGTTCCATGTCAACCATGT
TCCTGGGCACCTCTGGGTACTCATGGCTGTGATGAGTGGGGGCGGCGGGCCCGACGTGGCGTGGAGGTGG
CAGCAGGGGCCAGCAGCGGTGGTGAGACACGGCAGCCTGGGAACGGCACCCGGGCAGGTGGCCCAGAGGA
GACAGCCGCCCAGTACGCGGTCATCGCCATCGTCCCTGTCTTCTGCCTCATGGGGCTGTTGGGCATCCTG
GTGTGCAACCTCCTCAAGCGGAAGGGCTACCACTGCACGGCGCACAAGGAGGTCGGGCCCGGCCCTGGAG
GTGGAGGCAGTGGAATCAACCCTGCCTACCGGACTGAGGATGCCAATGAGGACACCATTGGGGTCCTGGT
GCGCTTGATCACAGAGAAGAAAGAGAATGCTGCGGCCCTGGAGGAGCTGCTGAAAGAGTACCACAGCAAA
CAGCTGGTGCAGACGAGCCACAGGCCTGTGTCCAAGCTGCCGCCAGCGCCCCCGAACGTGCCACACATCT
GCCCGCACCGCCACCATCTCCACACCGTGCAGGGCCTGGCCTCGCTCTCTGGCCCCTGCTGCTCCCGCTG
TAGCCAGAAGAAGTGGCCCGAGGTGCTGCTGTCCCCTGAGGCTGTAGCCGCCACTACTCCTGTTCCCAGC
CTTCTGCCTAACCCGACCAGGGTTCCCAAGGCCGGGGCCAAGGCAGGGCGTCAGGGCGAGATCACCATCT
TGTCTGTGGGCAGGTTCCGCGTGGCTCGAATTCCTGAGCAGCGGACAAGTTCAATGGTGTCTGAGGTGAA
GACCATCACGGAGGCTGGGCCCTCGTGGGGTGATCTCCCTGACTCCCCACAGCCTGGCCTCCCCCCTGAG
CAGCAGGCCCTGCTAGGAAGTGGCGGAAGCCGTACAAAGTGGCTGAAGCCCCCAGCAGAGAACAAGGCCG
AGGAGAACCGCTATGTGGTCCGGCTAAGTGAGAGCAACCTGGTCATCTGAGGGGCGGTCTAGTCTAAGGA
CACTGCGGCCCTGCCCTGGGAGGTTCCGAAGGCTTCCTGGAGGAGGTGGAGCTGCAGCTGGGACTGTGAG
GACCGAGAAGCAATGGCCCAGCAGACGAGACAGCAAAGACCAAGGCCTGGAGGTGGGAGCGTCTGCCCCA
GTGAGGAGGCAGGTGGCCGGCGGGCACTGTGTACAGGAGCAGGCTGAGCCCCGCCCCTGGCCCTGCTGCC
ATGTTGCTCCCCTGAAGGATGCCCCGACCCCCGTGCCTGCCCTGGCTGGATCCTAGGAGCCCACGGGATT
CTCTGTATCATCAGAGGCTGGGCTTGGCAGAGGGGAGGGGCCTGTGCCCGTCACCCCTGGCCCCATTCCT
TGGTAATTAGCCACACCCTTGCCTCTGTACAGGGCCCTAGAGCAGATGTGCGTCCCCCTCCTCTTCCAGC

AGGTCTATAAAGGGAAGGGGTAGCAGAAAGTCCTGGGCTAGGAGAGTGAGTCCCTGGGTTCTAATCTTGG
GCACATCTGTGGCCATCGCTGGGTCCATTTTTCTGACTGTGAAGTAAGGAGAGACGTCTCAGTACCCAGG
GCCTCTTCAGCTCTTTGTAGGTTCTGGGCTGGGTTGTGGGGGACTGGGGAGCTGGGCTCTACCATCCCTC
CCATTAGTAGCTTTATCCAGCCCCGTTTTTGCTGCTTCCAGGGCCTCTGCCTTCAAGGCCCCCATGGGGC
TGTCCATCCATGGCTCTGCCTACGGAAGGGGCTTAATGCATGTGCCTGCCCCTCCCCCAGCTGTTTTTAA
TGAAACTGAAAAAATAGACTTGATCCCGGCAGGACTGTGATACAGAGCCCTAGCCTGCCCAGCCAGCCCC
AAGATCTCAGGAGCTTTAGGGAGAAGACTTGGTGGGGCTGGAGCACACCTTGGGCCTCAGTGGTTTCTGT
GTCCCTGTGGTGCCAGTGCTTCTGGGCAGTGCAGGCGGCTGCCAGGCCCAGCCCTGACTTCCACTCTGGC
TCAGCAACCTGGTTATTTATGTGGGGCCGTGCAGGCATGGGCCCACTGCCTGTCCATCCTGTTTCTCTTA
TTTATTGAAACTCACCATTGCCCTATCCTTGTGTCTCCACCCCCTTCCATGTGTTGAATAATAAAAGGTG
GGAAAGTGCTGTCACGAGGAGCTCCTTTTCTCTGTGCCCTTCCCAGCTTATCCGCCTGTTCCACTTGTAC
CTGTCCAGCCCTGCCCTGTGTTGGGCACCGTGACAGGCTGCCCTCCCCTCACAGTTCCTGCACCTCAGCA
GCCAGGGAAGCAGGGCCCAGAGGAGACCCATCAACTCATCTGGTTCTCACCAAAGCCCCTAGCTAGAGGC
AGGGCTGTCCCACAGAAATAGTGTGAGCCACATATGTGACTTTAAATTTTCTGACAGTCACATTTCAAAA
AGTAAAAAGAAGCAGGTGAACTTAATTTTTTTTTAACCCAGTCTATCCAAAATATTTTCATTTCAATATA
AAATCAGTATAAAAAGTGTGAATGAGCTGTTTTTGCTTTTGCTTTGAAATCTGGAGTGTATCTTACACAT
CTTGATTGGAACTCAAATATTTCAAGGGCTCCATAGCCACGTGGCCAGCAGCTACCACACTGGTCAGCGC
AGCACCTGAGGAAATGCACTGTGTGGAGCCCGGGAGGTAATTGGGGGTGAGGAAGATGTGCGGGCCAGGG
AATGTGTTCCACAGAGAGGGACAGCATGTGCAAAGGCTGAGCACCAACACATAGTGGTCACCTGGGCCTT
ATGCCCAGGGAGCCTCCAAAGCATTCAGCTCCTGCTTGGCCAGTGTGGAGAGGAAAGGAGCCACAGGAGC
GGGTGCTACACCCGGCAAGACAGAGCGTCTCCATCTAAAATTCCTAGGGCAAGCTTCCAACCCGGCCCAA
GGGCCGCATGTGGCCCAGGATATGGCTTTGAATGGGGCCTAACACAAATTCATAAACTTTCTTAAAACAT
TGAAAAAAAAAAAAAAAAAA

NM_021192.2| Homo sapiens homeobox D11 (HOXD11), mRNA
ATGAACGACTTTGACGAGTGCGGCCAGAGCGCAGCCAGCATGTACCTGCCGGGCTGCGCCTACTATGTGG
CCCCGTCTGACTTCGCTAGCAAGCCTTCGTTCCTTTCCCAACCGTCGTCCTGCCAGATGACTTTCCCCTA
CTCTTCCAACCTGGCTCCGCACGTCCAGCCCGTGCGCGAAGTGGCCTTCCGCGACTACGGCCTGGAGCGC
GCCAAGTGGCCGTACCGCGGCGGCGGCGGCGGCGGCAGCGCGGGGGGCGGCAGCAGCGGGGGCGGCCCCG
GCGGGGGCGGCGGCGGCGCGGGGGGCTACGCTCCCTACTACGCGGCGGCGGCGGCGGCGGCTGCGGCGGC
CGCGGCGGCCGAGGAGGCGGCCATGCAACGCGAGCTTCTCCCGCCCGCGGGCCGCCGGCCGGACGTGCTC
TTCAAGGCGCCTGAGCCGGTGTGCGCTGCGCCGGGGCCGCCGCACGGCCCCGCGGGCGCCGCCTCCAACT
TCTACAGCGCGGTGGGCCGCAATGGCATCTTGCCACAGGGCTTCGACCAGTTCTACGAGGCAGCGCCCGG
GCCCCCGTTCGCCGGGCCGCAGCCCCCGCCGCCACCCGCGCCGCCACAGCCCGAGGGCGCAGCCGACAAG
GGCGACCCCAGGACCGGGGCTGGTGGCGGCGGGGGCAGTCCCTGCACCAAGGCGACCCCTGGCTCGGAGC
CCAAGGGGGCAGCAGAAGGCAGCGGTGGCGACGGCGAGGGCCCCCCGGGAGAGGCGGGGGCCGAGAAGAG
CAGCAGCGCAGTTGCCCCCCAGCGGTCCCGGAAAAAGCGCTGTCCCTATACCAAGTACCAGATCCGCGAA
CTGGAACGCGAGTTTTTCTTTAACGTGTACATAAACAAAGAGAAAAGACTTCAACTCTCTCGGATGCTCA
ACCTCACTGACCGGCAAGTCAAAATCTGGTTCCAGAATCGCAGGATGAAAGAAAAGAAACTGAACAGAGA
CCGTCTGCAGTATTTCACTGGAAACCCCTTATTTTGAGAGCTCCAGGAAGCGCCCTCACCCCAGCCCCAC
TCACCCACCCTCCTTCCCACCAGCCTGCTCTCCGCAGGCCCACTGTCCTTGGGTTTAATGACGTCTCTTC
TCTGTGGAACTTCACGATTCCTTCCCACGGTCAACTCGGGACCTCCCAGCGACCACTGCAGCCTGCGGAC
GAGGCCGGGACTTGGCCGAGCGGATCCTAATAAGGGGAAAATGGTAAATGCAAACGTCCCGTTACAATTT
TACCGCCAGTGTGCTGTCGTTCCCCCTCCCCCTCTCCGAGTCCTCGTGGGGACACGGCGGGGTCTGTAGG
AAGTTGGGCCGGGTTGGGGGTTGCTAGAAGGCGCTGGTGTTTTGCTCTGAGTTTTAAGAGATCCCTTCCT
TCCTCTTCGGTGAATGCAGGTTATTTAAACTTTGGGAAATGTACTTTTAGTCTGTCATATCAA

NM_024897.2| Homo sapiens progestin and adipoQ receptor family member VI (PAQR6), transcript variant 1, mRNA
TTCCTCCATCAGCCTGGCTGGCAGCAGCCTTGGACTCCGCCCGTGGAGCCCTGGGCCTGTTGACCCACCA
GCTTAGGAGCACCCACCAAGCTCTGGGTAAGGAAGCTCACCTTCTGGGGCTCTTCTGGGAAAATAGAGGT
CAACGTGGAGGTACCAGGCCACCATGCTCAGTCTCAAGCTGCCCCAACTTCTTCAAGTCCACCAGGTCCC
CCGGGTGTTCTGGGAAGATGGCATCATGTCTGGCTACCGCCGCCCCACCAGCTCGGCTTTGGACTGTGTC
CTCAGCTCCTTCCAGATGACCAACGAGACGGTCAACATCTGGACTCACTTCCTGCCCACCTGGTGAGGGG
AGGCTCTGCCCCAGGCCGCGGCCTTGAGCTCAGAGGGGGTACCCAGGCGGGCAGGGACCGTCCAGGCCCA
CGGGCTGCAGCGGCAGTCGCGGGGGTCCGCGGCGGCCTGAGCACGCGCCCGCCGCAGGTACTTCCTGTGG
CGGCTCCTGGCGCTGGCGGGCGGCCCCGGCTTCCGTGCGGAGCCGTACCACTGGCCGCTGCTGGTCTTCC
TGCTGCCCGCCTGCCTCTACCCCTTCGCGTCGTGCTGCGCGCACACCTTCAGCTCCATGTCGCCCCGCAT
GCGCCACATCTGCTACTTCCTCGACTACGGCGCGCTCAGCCTCTACAGTCTGGGCTGCGCCTTCCCCTAT
GCCGCCTACTCCATGCCGGCCTCCTGGCTGCACGGCCACCTGCACCAGTTCTTTGTGCCTGCCGCCGCAC
TCAACTCCTTCCTGTGCACCGGCCTCTCCTGCTACTCCCGTTTCCTGGAGCTGGAAAGCCCTGGGCTCAG

Figure 64 (Continued)

TAAGGTCCTCCGCACAGGAGCCTTCGCCTATCCATTCCTGTTCGACAACCTCCCACTCTTTTATCGGCTC
GGGCTGTGCTGGGGCAGGGGCCACGGCTGTGGGCAGGAGGCCCTGAGCACCAGCCATGGCTACCATCTCT
TCTGCGCGCTGCTCACTGGCTTCCTCTTCGCCTCCCACCTGCCTGAAAGGCTGGCACCAGGACGCTTTGA
TTACATCGGTGAGGGCACGCCTGGCCCGGCCCGGGAAGAGGCAGGGGCAGATGCCTTCCCAGAGCACAGA
ATGAACTGGGCCACAGCCACCAGTTATTCCACATCTGTGCAGTGCTGGGCACCCACTTCCAGCTGGAGGC
AGTGCTGGCTGATATGGGATCACGCAGAGCCTGGCTGGCCACACAGGAACCTGCCCTGGGCCTGGCAGGC
ACAGTGGCCACACTGGTCTTGGCTGCAGCTGGGAACCTACTCATTATTGCTGCTTTCACAGCCACCCTGC
TTCGGGCCCCCAGTACATGCCCTCTGCTGCAGGGTGGCCCACTGGAGGGGGGTACCCAGGCCAAACAACA
GTGAGGCCCCATCCCTGACCCTGTCCTGGAGGGGGCAGAGGCCAGGCCCCAGTGCTGACGAGGAGCCCAG
ATTTGGGCCTAATCAGGTGGGGACGCATCTCAGCCTGGAACCAACAGGGGCTGAGGAGAGAGGGCACAGG
AGAGAGGGCAGAGAAGAGGAGGGGTGTCTAGGGGGACTGGCAGAGTGTGAGAGGGACCGTGAGGGGGCTC
TTGATGGGAGTGGAAGAAGTGCTGAGGGTCTGAGAGGGGAGATGCATGCGTGTCCAGGCTGAAGATGCCC
CTATATTCTGTCAAAGGTTGGCGGGGGGAGGTGTTGGGGTCCTTTCATCTGGCTCCGTTTCTGGTGCTTC
TGGAAGTCTCTGCTCAGCACAGGGAAGAACTAACACGACTAACCTAGGCCTACCCTGAATGCTTCTTGCT
AACCAGGCCGAGAGGCCACACACTTGCCCCCCCCATCCCCACAAACCAGGTAATGCCAGTTTGCCAGCAGC
TATTTGCCTATAGAGATGAGTCTGTCCTGGTCATAACTGTGTGCTCAAGGTGTCCAGGCTTTTGGGGGTG
GGCCTATCTGGGTGCATTATGGATGGTTTGGTGGATTGAGGTGTGGGGAGGAGGGTCCTAGGCTAGAGGG
GGTATCCCTAGTTAGACTTTGGGAAGCCACCTTCAATGTTTTCTGGAACAAGGCAGGTACAAATAAAAAA
ATAAAACTTTGGAAAGC

NM_000594.2| Homo sapiens tumor necrosis factor (TNF superfamily, member 2) (TNF), mRNA
CTCCCTCAGCAAGGACAGCAGAGGACCAGCTAAGAGGGAGAGAAGCAACTACAGACCCCCCCTGAAAACA
ACCCTCAGACGCCACATCCCCTGACAAGCTGCCAGGCAGGTTCTCTTCCTCTCACATACTGACCCACGGC
TCCACCCTCTCTCCCCTGGAAAGGACACCATGAGCACTGAAAGCATGATCCGGGACGTGGAGCTGGCCGA
GGAGGCGCTCCCCAAGAAGACAGGGGGGCCCCAGGGCTCCAGGCGGTGCTTGTTCCTCAGCCTCTTCTCC
TTCCTGATCGTGGCAGGCGCCACCACGCTCTTCTGCCTGCTGCACTTTGGAGTGATCGGCCCCCAGAGGG
AAGAGTTCCCCAGGGACCTCTCTCTAATCAGCCCTCTGGCCCAGGCAGTCAGATCATCTTCTCGAACCCC
GAGTGACAAGCCTGTAGCCCATGTTGTAGCAAACCCTCAAGCTGAGGGGCAGCTCCAGTGGCTGAACCGC
CGGGCCAATGCCCTCCTGGCCAATGGCGTGGAGCTGAGAGATAACCAGCTGGTGGTGCCATCAGAGGGCC
TGTACCTCATCTACTCCCAGGTCCTCTTCAAGGGCCAAGGCTGCCCCTCCACCCATGTGCTCCTCACCCA
CACCATCAGCCGCATCGCCGTCTCCTACCAGACCAAGGTCAACCTCCTCTCTGCCATCAAGAGCCCCTGC
CAGAGGGAGACCCCAGAGGGGGCTGAGGCCAAGCCCTGGTATGAGCCCATCTATCTGGGAGGGGTCTTCC
AGCTGGAGAAGGGTGACCGACTCAGCGCTGAGATCAATCGGCCCGACTATCTCGACTTTGCCGAGTCTGG
GCAGGTCTACTTTGGGATCATTGCCCTGTGAGGAGGACGAACATCCAACCTTCCCAAACGCCTCCCCTGC
CCCAATCCCTTTATTACCCCCTCCTTCAGACACCCTCAACCTCTTCTGGCTCAAAAAGAGAATTGGGGGC
TTAGGGTCGGAACCCAAGCTTAGAACTTTAAGCAACAAGACCACCACTTCGAAACCTGGGATTCAGGAAT
GTGTGGCCTGCACAGTGAAGTGCTGGCAACCACTAAGAATTCAAACTGGGGCCTCCAGAACTCACTGGGG
CCTACAGCTTTGATCCCTGACATCTGGAATCTGGAGACCAGGGAGCCTTTGGTTCTGGCCAGAATGCTGC
AGGACTTGAGAAGACCTCACCTAGAAATTGACACAAGTGGACCTTAGGCCTTCCTCTCTCCAGATGTTTC
CAGACTTCCTTGAGACACGGAGCCCAGCCCTCCCCATGGAGCCAGCTCCCTCTATTTATGTTTGCACTTG
TGATTATTTATTATTTATTTATTATTTATTTATTTACAGATGAATGTATTTATTTGGGAGACCGGGGTAT
CCTGGGGGACCCAATGTAGGAGCTGCCTTGGCTCAGACATGTTTTCCGTGAAAACGGAGCTGAACAATAG
GCTGTTCCCATGTAGCCCCCTGGCCTCTGTGCCTTCTTTTGATTATGTTTTTTAAAATATTTATCTGATT
AAGTTGTCTAAACAATGCTGATTTGGTGACCAACTGTCACTCATTGCTGAGCCTCTGCTCCCCAGGGGAG
TTGTGTCTGTAATCGCCCTACTATTCAGTGGCGAGAAATAAAGTTTGCTTAGAAAAGAA

NM_001136.3| Homo sapiens advanced glycosylation end product-specific receptor (AGER), transcript variant 1, mRNA
GCCAGGACCCTGGAAGGAAGCAGGATGGCAGCCGGAACAGCAGTTGGAGCCTGGGTGCTGGTCCTCAGTC
TGTGGGGGGCAGTAGTAGGTGCTCAAAACATCACAGCCCGGATTGGCGAGCCACTGGTGCTGAAGTGTAA
GGGGGCCCCCAAGAAACCACCCCAGCGGCTGGAATGGAAACTGAACACAGGCCGGACAGAAGCTTGGAAG
GTCCTGTCTCCCCAGGGAGGAGGCCCCTGGGACAGTGTGGCTCGTGTCCTTCCCAACGGCTCCCTCTTCC
TTCCGGCTGTCGGGATCCAGGATGAGGGGATTTTCCGGTGCCAGGCAATGAACAGGAATGGAAAGGAGAC
CAAGTCCAACTACCGAGTCCGTGTCTACCAGATTCCTGGGAAGCCAGAAATTGTAGATTCTGCCTCTGAA
CTCACGGCTGGTGTTCCCAATAAGGTGGGGACATGTGTGTCAGAGGGAAGCTACCCTGCAGGGACTCTTA
GCTGGCACTTGGATGGGAAGCCCCTGGTGCCTAATGAGAAGGGAGTATCTGTGAAGGAACAGACCAGGAG
ACACCCTGAGACAGGGCTCTTCACACTGCAGTCGGAGCTAATGGTGACCCCAGCCCGGGGAGGAGATCCC
CGTCCCACCTTCTCCTGTAGCTTCAGCCCAGGCCTTCCCCGACACCGGGCCTTGCGCACAGCCCCCATCC
AGCCCCGTGTCTGGGAGCCTGTGCCTCTGGAGGAGGTCCAATTGGTGGTGGAGCCAGAAGGTGGAGCAGT
AGCTCCTGGTGGAACCGTAACCCTGACCTGTGAAGTCCCTGCCCAGCCCTCTCCTCAAATCCACTGGATG

AAGGATGGTGTGCCCTTGCCCCTTCCCCCCAGCCCTGTGCTGATCCTCCCTGAGATAGGGCCTCAGGACC
AGGGAACCTACAGCTGTGTGGCCACCCATTCCAGCCACGGGCCCCAGGAAAGCCGTGCTGTCAGCATCAG
CATCATCGAACCAGGCGAGGAGGGGCCAACTGCAGGCTCTGTGGGAGGATCAGGGCTGGGAACTCTAGCC
CTGGCCCTGGGGATCCTGGGAGGCCTGGGGACAGCCGCCCTGCTCATTGGGGTCATCTTGTGGCAAAGGC
GGCAACGCCGAGGAGAGGAGAGGAAGGCCCCAGAAAACCAGGAGGAAGAGGAGGAGCGTGCAGAACTGAA
TCAGTCGGAGGAACCTGAGGCAGGCGAGAGTAGTACTGGAGGGCCTTGAGGGGCCCACAGACAGATCCCA
TCCATCAGCTCCCTTTTCTTTTTCCCTTGAACTGTTCTGGCCTCAGACCAACTCTCTCCTGTATAATCTC
TCTCCTGTATAACCCCACCTTGCCAAGCTTTCTTCTACAACCAGAGCCCCCCACAATGATGATTAAACAC
CTGACACATCTTGA

NM_002558.2| Homo sapiens purinergic receptor P2X, ligand-gated ion channel, 1 (P2RX1), mRNA
ACTTGAGGAGTCGACTGAGAGCTTTATCTTTGCCCCTGTGACGGGTGGCCTGTGGTTTCCTGCCAACAAC
TCCTGTTTCCGGAGGCCCAACAAGGCCCACGCAGAGCCAGGAGGGGCAGTGGGGCTGGGCCTGGGTGGCC
CCACCAGCCCCGCCCCATCTATCTTTGGGAATTTATTTGTCCATGGGCGAGGCTGGCCTGCAGTCTGTTG
CCTTCCAGGGGCCAAGAGCTGCTCTGATCACCCAGGGATTCTCTCTCCAACCCAAGTGCCTCCAGCTGAC
CTCTGGCTCCTGTCCTCTGGCTCCACCTGCACCGCCCTGCTCTTCCTAAGGGGCCAGGAAGCCCCCAGAA
GCTCTACCATCGACGTGGGTGGTGGCACCCGGCTCACCCTGAGAGCAGAGGCCGTGCAGGGGGCTCAGTT
CTGAGCCCCAGCCGGCCCACCATGGCACGGCGGTTCCAGGAGGAGCTGGCCGCCTTCCTCTTCGAGTATG
ACACCCCCCGCATGGTGCTGGTGCGTAATAAGAAGGTGGGCGTTATCTTCCGACTGATCCAGCTGGTGGT
CCTGGTCTACGTCATCGGGTGGGTGTTTCTCTATGAGAAGGGCTACCAGACCTCGAGCGGCCTCATCAGC
AGTGTCTCTGTGAAACTCAAGGGCCTGGCCGTGACCCAGCTCCCTGGCCTCGGCCCCCAGGTCTGGGATG
TGGCTGACTACGTCTTCCCAGCCCAGGGGGACAACTCCTTCGTGGTCATGACCAATTTCATCGTGACCCC
GAAGCAGACTCAAGGCTACTGCGCAGAGCACCCAGAAGGGGGCATATGCAAGGAAGACAGTGGCTGTACC
CCTGGGAAGGCCAAGAGGAAGGCCCAAGGCATCCGCACGGGCAAGTGTGTGGCCTTCAACGACACTGTGA
AGACGTGTGAGATCTTTGGCTGGTGCCCCGTGGAGGTGGATGACGACATCCCGCGCCCTGCCCTTCTCCG
AGAGGCCGAGAACTTCACTCTTTTCATCAAGAACAGCATCAGCTTTCCACGCTTCAAGGTCAACAGGCGC
AACCTGGTGGAGGAGGTGAATGCTGCCCACATGAAGACCTGCCTCTTTCACAAGACCCTGCACCCCCTGT
GCCCAGTCTTCCAGCTTGGCTACGTGGTGCAAGAGTCAGGCCAGAACTTCAGCACCCTGGCTGAGAAGGG
TGGAGTGGTTGGCATCACCATCGACTGGCACTGTGACCTGGACTGGCACGTACGGCACTGCAGACCCATC
TATGAGTTCCATGGGCTGTACGAAGAGAAAAATCTCTCCCCAGGCTTCAACTTCAGGTTTGCCAGGCACT
TTGTGGAGAACGGGACCAACTACCGTCACCTCTTCAAGGTGTTTGGGATTCGCTTTGACATCCTGGTGGA
CGGCAAGGCCGGGAAGTTTGACATCATCCCTACAATGACCACCATCGGCTCTGGAATTGGCATCTTTGGG
GTGGCCACAGTTCTCTGTGACCTGCTGCTGCTTCACATCCTGCCTAAGAGGCACTACTACAAGCAGAAGA
AGTTCAAATACGCTGAGGACATGGGGCCAGGGGCGGCTGAGCGTGACCTCGCAGCTACCAGCTCCACCCT
GGGCCTGCAGGAGAACATGAGGACATCCTGATGCTCGGGCCCCAACTCCTGACTGGGTGCAGCGTGAGGC
TTCAGCCTGGAGCCCTGGTGGGTCCCAGCCAGGGCAGAGGGGCCTCCCCAGGAAGTCTCCTACCCTCTCA
GCCAGGCAGAGAGCAGTTTGCCAGAAGCTCAGGGTGCATAGTAGGAGAGACCTGTGCAAATCTGAGCTCC
GGCTCCGACCCCACACACCCTGAGGGAGCCCTACCCTAGCCTCAGCCGCTCCTGGTGGGGAATGGCTGGG
GCTGGGCAGGACCCTCCCACACACCTGCACCCTAGCTTCGTGCTTCTCTCTCCGGACTCTCATTATCCAA
CCCGCTGCCTCCATTTCTCTAGATCTGTGCTCTCCGATGTGGCAGTCAGTAACCATAGGTGACTAAATTA
AACTAAAATAAAATAGAATGAAACACAAAATTCAATTCCTCGGCTGAACTAGCCACATTTCAACTGCTCA
GTAGATACGTGTGGTTAGTGGCTGCCATACTGGACAGCTCGGGGCATTTTCACTGTCAAAGAAAGTTCTA
TTAGACAGCCCTGCTTGAGCCCTGTTTCTTCCTGGCCTTCGGTTTCCCTGGGGAACTTATCGACAATGCA
AGCTCCTGGGCCCACCCCCAGACCTCCTGAACCAAAAGCTCCAGGGCTGGCCGTATGATCTGTGTGGATG
GCAAACTCCCCAGGCCATTCTGGGACCTAAGTTTAAGAAGTGCCGTCCTCGAACTTTCTGACTCTAAGCT
CCTGAGCGGGAGTCAGACTTAGCCCTGAGCCTGCACTTCCTGTTCAGGTGCAGACACTGAACAGGGTCTC
AAACACCTTCAGCATGTGTGTTGTGTGCTCACGTGCCACACAGTGTCTCATGCACACAACCCAGTGTACA
CACCACCTACGTGCACACAGCATCCTTCCACACTGTGTATGTGAACAGCTTGGGCCCTGCAAACACAACC
ATCTACACACATCTACACCCCCAAGCACACACACATGGTCCGTGCCATGTCACCTCCATAGGGAAAGGCT
TCTCTCCAAGTGTGCCAGGCCAGGACAGCCCTCCCAGCCATGAATCCTTACTCAGCTACCTCGGGTTGGG
GTGGGAGCCCCAGCCAAATCCTGGGCTCCCTGCCTGTGGCTCAGCCCCAGCTCCCAAGGCCTGCCTGGCT
CTGTCTGAACAGAAGGTCTGGGGGAAGCGACGGGTGGAGTACAATAAAGGGAATGAGGACAAACAAAAAA
AAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_025197.2| Homo sapiens CDK5 regulatory subunit associated protein 3 (CDK5RAP3), transcript variant 2, mRNA
CGGAGGCTCGGCCACAACGCCACTGGATTGGTGGTAGGGCGGGGCGGGCCACAGTCTCCAGCCTGAAGCG
GAAGTGGAGGAAAGATGGAGGACCATCAGCACGTGCCCATCGACATCCAGACCAGCAAGCTGCTCGATTG
GCTGGTGGACAGAAGGCACTGCAGCCTGAAATGGCAGAGTCTGGTGCTGACGATCCGCGAGAAGATCAAT

GCTGCCATCCAGGACATGCCAGAGAGCGAAGAGATCGCCCAGCTGCTGTCTGGGTCCTACATTCACTACT
TTCACTGCCTAAGAATCCTGGACCTTCTCAAAGGCACAGAGGCCTCCACGAAGAATATTTTTGGCCGATA
CTCTTCACAGCGGATGAAGGATTGGCAGGAGATTATAGCTCTGTATGAGAAGGACAACACCTACTTAGGT
AAAGTGGCCCGGCCTGGGAGCCCTGGTATCCATGGGGAAGCCCACTCTCAGAGTTCTGAGATACCAGGCT
TATAGGAGGCACAGTCTGTGAGTGGGAAGAGACTGGAGTGTAGATGTTGCCCATTTGTAGGTGGTAAAAT
CAATTGTTTTTGATGGAATTGATTTTCCCTGAGTGGAGTGCTGGGGGAAGGAGGAGGTCCAGGCCGGTAG
TGGCCATTCGCCGTGCCTCAGCGAGCAGGTGTGTGTGGGTCCTCCACCACTCACCTCTTGGTTAGCGGGA
GTGTGCTGCCCCCACCCCCACCCCCGCACCCCCATTCTACACAAGGCAGAAGAGGCACGGGTTTTCCTGG
GAGCGAATATCAAGTGCCTGAGAGCAACTACAGGACTAACTGTGTTTGGGTTGGGTGTAGTATAAATAAT
AATAATGGCTAATATTTCCTGAGCATCTACTAAATGCAAGGAATTGTGCTTGGTGTGTCATGTGGATTCT
CTCTTGCATCTTCATGATAAATGTTATTGTCGCTGTTTTACCGATGAGGGTTGGATTAGAGGGGTTAAAC
AACTTGTCTTAGGCTCCACAGCTGGGAACAAGTGGGGCTGGGAAGCTGACTTCGTGCTCTTCACCACCAC
AAAGGATGTGTGTGCATCCTGGGGCATGCCTGCCTCATGTGGGGGTGTCCTGGGCTGAATTTCCTGGGCA
CTTCTCAGTGGAACTCTCTAGCCTCCTGGTTCGGAATGTCAACTATGAGATCCCCTCACTGAAGAAGCAG
ATTGCCAAGTGCCAGCAGCTGCAGCAAGAATACAGCCGCAAGGAGGAGGAGTGCCAGGCAGGGGCTGCCG
AGATGCGGGAGCAGTTCTACCACTCCTGCAAGCAGTATGGCATCACGGGCGAAAATGTCCGAGGAGAACT
GCTGGCCCTGGTGAAGGACCTGCCGAGTCAGCTGGCTGAGATTGGGGCAGCGGCTCAGCAGTCCCTGGGG
GAAGCCATTGACGTGTACCAGGCGTCTGTGGGGTTTGTGTGTGAGAGCCCCACAGAGCAGGTGTTGCCAA
TGCTGCGGTTCGTGCAGAAGCGGGGAAACTCAACGGTGTACGAGTGGAGGACAGGGACAGAGCCCTCTGT
GGTGGAACGACCCCACCTCGAGGAGCTTCCTGAGCAGGTGGCAGAAGATGCGATTGACTGGGGCGACTTT
GGGGTAGAGGCAGTGTCTGAGGGGACTGACTCTGGCATCTCTGCCGAGGCTGCTGGAATCGACTGGGGCA
TCTTCCCGGAATCAGATTCAAAGGATCCTGGAGGTGATGGGATAGACTGGGGAGACGATGCTGTTGCTTT
GCAGATCACAGTGCTGGAAGCAGGAACCCAGGCTCCAGAAGGTGTTGCCAGGGGCCCAGATGCCCTGACA
CTGCTTGAATACACTGAGACCCGGAATCAGTTCCTTGATGAGCTCATGGAGCTTGAGATCTTCTTAGCCC
AGAGAGCAGTGGAGTTGAGTGAGGAGGCAGATGTCCTGTCTGTGAGCCAGTTCCAGCTGGCTCCAGCCAT
CCTGCAGGGCCAGACCAAAGAGAAGATGGTTACCATGGTGTCAGTGCTGGAGGATCTGATTGGCAAGCTT
ACCAGTCTTCAGCTGCAACACCTGTTTATGATCCTGGCCTCACCAAGGTATGTGGACCGAGTGACTGAAT
TCCTCCAGCAAAAGCTGAAGCAGTCCCAGCTGCTGGCTTTGAAGAAAGAGCTGATGGTGCAGAAGCAGCA
GGAGGCACTTGAGGAGCAGGCGGCTCTGGAGCCTAAGCTGGACCTGCTACTGGAGAAGACCAAGGAGCTG
CAGAAGCTGATTGAAGCTGACATCTCCAAGAGGTACAGCGGGCGCCCTGTGAACCTGATGGGAACCTCTC
TGTGACACCCTCCGTGTTCTTGCCTGCCCATCTTCTCCGCTTTTGGGATGAAGATGATAGCCAGGGCTGT
TGTTTTGGGGCCCTTCAAGGCAAAAGACCAGGCTGACTGGAAGATGGAAAGCCACAGGAAGGAAGCGGCA
CCTGATGGTGATCTTGGCACTCTCCATGTTCTCTACAAGAAGCTGTGGTGATTGGCCCTGTGGTCTACCA
GGCGAAAACCACAGATTCTCCTTCTAGTTAGTATAGCGGACTTAATAAAAGAGGAAAAAACTCTTGCTTC
AGTA

NM_002001.2| Homo sapiens Fc fragment of IgE, high affinity I, receptor for; alpha polypeptide (FCER1A), mRNA
AGTCTCCAGCATCCTCCACCTGTCTACCACCGAGCATGGGCCTATATTTGAAGCCTTAGATCTCTCCAGC
ACAGTAAGCACCAGGAGTCCATGAAGAAGATGGCTCCTGCCATGGAATCCCCTACTCTACTGTGTGTAGC
CTTACTGTTCTTCGCTCCAGATGGCGTGTTAGCAGTCCCTCAGAAACCTAAGGTCTCCTTGAACCCTCCA
TGGAATAGAATATTTAAAGGAGAGAATGTGACTCTTACATGTAATGGGAACAATTTCTTTGAAGTCAGTT
CCACCAAATGGTTCCACAATGGCAGCCTTTCAGAAGAGACAAATTCAAGTTTGAATATTGTGAATGCCAA
ATTTGAAGACAGTGGAGAATACAAATGTCAGCACCAACAAGTTAATGAGAGTGAACCTGTGTACCTGGAA
GTCTTCAGTGACTGGCTGCTCCTTCAGGCCTCTGCTGAGGTGGTGATGGAGGGCCAGCCCCTCTTCCTCA
GGTGCCATGGTTGGAGGAACTGGGATGTGTACAAGGTGATCTATTATAAGGATGGTGAAGCTCTCAAGTA
CTGGTATGAGAACCACAACATCTCCATTACAAATGCCACAGTTGAAGACAGTGGAACCTACTACTGTACG
GGCAAAGTGTGGCAGCTGGACTATGAGTCTGAGCCCCTCAACATTACTGTAATAAAAGCTCCGCGTGAGA
AGTACTGGCTACAATTTTTTATCCCATTGTTGGTGGTGATTCTGTTTGCTGTGGACACAGGATTATTTAT
CTCAACTCAGCAGCAGGTCACATTTCTCTTGAAGATTAAGAGAACCAGGAAAGGCTTCAGACTTCTGAAC
CCACATCCTAAGCCAAACCCCAAAAACAACTGATATAATTACTCAAGAAATATTTGCAACATTAGTTTTT
TTCCAGCATCAGCAATTGCTACTCAATTGTCAAACACAGCTTGCAATATACATAGAAACGTCTGTGCTCA
AGGATTTATAGAAATGCTTCATTAAACTGAGTGAAACTGGTTAAGTGGCATGTAATAGTAAGTGCTCAAT
TAACATTGGTTGAATAAATGAGAGAATGAATAGATTCATTTATTAGCATTTGTAAAAGAGATGTTCAATT
TCAATAAAATAAATATAAAACCATGTAACAGAATGCTTCTGAGTAAAAAAAAAAAAAAAAAAAAAAAAA
A

NM_177229.2| Mus musculus RIKEN cDNA 5730405M06 gene (5730405M06Rik), mRNA
GGAGGGGGAGGGAGTGGGGTTGAGGCAAGATGGCGGCTAAGGTGGCGAAGCTGCGGCGGCGGCGGCTGGA
GTGAGCGCCCAGCTCGCCGCGCTGCCGAGCGAGGGCCGGGCGGAGGGCCGCGCGCCGGGGCCGCGCCCCG

GTCGTCCGGCCGTGGGGCGCGCCGGCTCTCACTCCGCCAGCTCCCGGTTGCTTCCTAGAAACATGGTTGT
TTGTTGGACTTGATCTCACAGCCCAGTGAGGACGTCTTTACTGATAATGTCAAGTTCAGGTTACCCTCCC
AACCAAGGAGCTTTCAGCACAGAGCAAAGTCGTTATCCTTCACATTCTGTCCAGTATACCTTTCCCAGTA
CCCGACACCAGCAGGAATTTGCAGTTCCTGACTACCGCTCTTCTCATATTGAAGTTAGCCAGGCATCACA
GCTTTTACAGCAGCAGCAGCAGCAGCAGCAGCTTCGAAGACGACCTTCCTTGCTTTCAGAATTTCACCCG
GGTTCTGACAGGCCTCAAGAAAGGAGAACTGGATATGAACAGTTTCACTCAGGACCCTCACCGGTGGATC
ATGATTCCTTGGAGTCCAAGCGCCCACGCCTGGAGCCAGTTTCTGATGCCCATTTCCAGCGTGTTAGTGC
TGCGGTTTTACCTTTAGTTCACTCGCTGCCAGAAGGCTTGAGGTCGTCTGCAGATGCTAAGAAGGATTCA
GCATTTGGAAGCAAACATGAAGCTCCATCCTCTCCTTTGGCTGGGCAACCATGTGGAGATGACCAAAATG
CTTCACCTTCAAAGCTTTCAAAGGAGGAGTTAATACAGAGTATGGACCGGGTAGACCGAGAGATTGCAAA
AGTAGAGCAGCAGATCCTTAAATTGAAAAAGAAACAACAACAGCTAGAAGAAGAAGCAGCTAAGCCCCCT
GAACCAGAGAAGCCTGTGTCCCCTCCTCCTGTGGAACAAAAACACCGTAGTATTGTCCAAATTATTTATG
ATGAGAATCGGAAAAAAGCAGAAGAAGCTCATAAAATATTTGAAGGTCTTGGCCCAAAAGTTGAACTGCC
ACTCTATAACCAGCCATCAGATACCAAGGTGTACCATGAGAACATCAAGACTGGAGTACCTGCAAGGCGC
ATGATGAAGTAAGATAATGAAGCTTTTTCATTTAAGACTAGTGATGACACCGTCCCCCACCAAGCCACAT
CTGCCATTGCAAAGTAGAAGTGCCACGGATCATTTTATCATTATTCAAAACCCTATTCTAGGGAAATCCC
TTCCATGACTCTTGCTAATGACGGTATTTGTAGACCCTTCTGCCATTTAATGTCCTGGTGCCTTGGAAGG
CTCAGTCCCCTCATGTGGGTGAGGAGGCTGTTCCAGAATGTAGTTTGCTTTGCCTTGCTCATCCTAGTAC
CAGGCCTTTTATGGATGCAGATCTTGGAATAAGAAAATAAGTGAAAAATGAACTTAATTTCTTGATCTGA
TTGAAGTAAGAGAAGCTAGATAGGCTTTTTTTTAAAAAAAAATTGTTGTTTAATTATCATACTTTTAGAG
AATTTTAGGTGAAGTATTAACAGTTGGTGACAAAAATGTGATATTTCTATTTATTTGCATTTAAAAGTAT
ATCAGAACCTAAAATGATTTACTATTACAGTTACTAATGAGCACATTATTTATATTTTTCCTTTTACGTT
CTGAGAATGTGTACAATAATTGACTACTTTGCAGAACTGGTGGCAAATATATGTGCATGGTAGTATGCAT
GACTATTACAATTTAGTCATTTTAATTTCTGTTAAGTATGTAGTTCATAATTAAAGTCATCTATTC

NM_007273.3| Homo sapiens prohibitin 2 (PHB2), transcript variant 2, mRNA
AAGTTCGGGTCCGTAGTGGGCTAAGGGGGAGGGTTTCAAAGGGAGCGCACTTCCGCTGCCCTTTCTTTCG
CCAGCCTTACGGGCCCGAACCCTCGTGTGAAGGGTGCAGTACCTAAGCCGGAGCGGGGTAGAGGCGGGCC
GGCACCCCCTTCTGACCTCCAGTGCCGCCGGCCTCAAGATCAGACATGGCCCAGAACTTGAAGGACTTGG
CGGGACGGCTGCCCGCCGGGCCCCGGGGCATGGGCACGGCCCTGAAGCTGTTGCTGGGGGCCGGCGCCGT
GGCCTACGGTGTGCGCGAATCTGTGTTCACCGTGGAAGGCGGGCACAGAGCCATCTTCTTCAATCGGATC
GGTGGAGTGCAGCAGGACACTATCCTGGCCGAGGGCCTTCACTTCAGGATCCCTTGGTTCCAGTACCCCA
TTATCTATGACATTCGGGCCAGACCTCGAAAAATCTCCTCCCCTACAGGCTCCAAAGACCTACAGATGGT
GAATATCTCCCTGCGAGTGTTGTCTCGACCCAATGCTCAGGAGCTTCCTAGCATGTACCAGCGCCTAGGG
CTGGACTACGAGGAACGAGTGTTGCCGTCCATTGTCAACGAGGTGCTCAAGAGTGTGGTGGCCAAGTTCA
ATGCCTCACAGCTGATCACCCAGCGGGCCCAGGTATCCCTGTTGATCCGCCGGGAGCTGACAGAGAGGGC
CAAGGACTTCAGCCTCATCCTGGATGATGTGGCCATCACAGAGCTGAGCTTTAGCCGAGAGTACACAGCT
GCTGTAGAAGCCAAACAAGTGGCCCAGCAGGAGGCCCAGCGGGCCCAATTCTTGGTAGAAAAAGCAAAGC
AGGAACAGCGGCAGAAAATTGTGCAGGCCGAGGGTGAGGCCGAGGCTGCCAAGATGCTTGGAGAAGCACT
GAGCAAGAACCCTGGCTACATCAAACTTCGCAAGATTCGAGCAGCCCAGAATATCTCCAAGACGATCGCC
ACATCACAGAATCGTATCTATCTCACAGCTGACAACCTTGTGCTGAACCTACAGGATGAAAGTTTCACCA
GGGGAAGTGACAGCCTCATCAAGGGTAAGAAATGAGCCTAGTCACCAAGAACTCCACCCCCAGAGGAAGT
GGATCTGCTTCTCCAGTTTTTGAGGAGCCAGCCAGGGGTCCAGCACAGCCCTACCCCGCCCCAGTATCAT
GCGATGGTCCCCCACACCGGTTCCCTGAACCCCTCTTGGATTAAGGAAGACTGAAGACTAGCCCCTTTTC
TGGGAAATTACTTTCCTCCTCCCTGTGTTAACTGGGGCTGTTGGGGACAGTGCGTGATTTCTCAGTGATT
TCCTACAGTGTTGTTCCCTCCCTCAAGGCTGGGAGGAGATAAACACCAACCCAGGAATTCTCAATAAATT
TTTATTACTTAACCTG

NM_021059.2| Homo sapiens histone cluster 2, H3c (HIST2H3C), mRNA
CGCCGCTGCGCTGGTAAGCCTGTGTTTTGGTTCGCTATGGCCCGTACTAAGCAGACTGCTCGCAAGTCGA
CCGGCGGCAAGGCCCCGAGGAAGCAGCTGGCCACCAAGGCGGCCCGCAAGAGCGCGCCGGCCACGGGCGG
GGTGAAGAAGCCGCACCGCTACCGGCCCGGCACCGTAGCCCTGCGGGAGATCCGGCGCTACCAGAAGTCC
ACGGAGCTGCTGATCCGCAAGCTGCCCTTCCAGCGGCTGGTACGCGAGATCGCGCAGGACTTTAAGACGG
ACCTGCGCTTCCAGAGCTCGGCCGTGATGGCGCTGCAGGAGGCCAGCGAGGCCTACCTGGTGGGGCTGTT
CGAAGACACGAACCTGTGCGCCATCCACGCCAAGCGCGTGACCATTATGCCCAAGGACATCCAGCTGGCC
CGCCGCATCCGTGGAGAGCGGGCTTAAGAAGTGGCGGTTCGGCCGGAGGTTCCATCGTATCCAAAAGGCT
CTTTTCAGAGCCACCCA

NM_153716.1| Homo sapiens heat shock transcription factor, Y linked 2
(HSFY2), transcript variant 1, mRNA
AACCATTGTGATGGTCTAGATAAGTGTACATGCTTAGGCCTTCTGAAGCAGCATTTGAAGCTGCAGTCCT

GAAAACCATGCAGGCCGGAAGAGTAGATAAAGAAATATTTATTTGAGATGGCACATGTTTCTTCAGAAAC
TCAAGATGTTTCCCCCAAAGATGAATTAACTGCTTCAGAAGCCTCCACTAGGTCTCCATTGTGTGAACAC
ACCTTCCCTGGGGACTCAGACTTACGGTCAATGATTGAAGAACATGCTTTTCAGGTTTTGTCACAAGGAT
CCTTGTTAGAAAGTCCAAGTTACACAGTTTGTGTCTCTGAGCCAGATAAAGATGATGATTTTCTTTCTCT
GAACTTTCCCAGGAAACTTTGGAAAATAGTGGAAAGTGACCAATTCAAGTCTATTTCATGGGATGAGAAT
GGAACTTGCATAGTGATTAATGAAGAACTCTTCAAGAAAGAAATTTTGGAAACAAAGGCTCCTTACAGAA
TATTTCAAACTGATGCTATCAAAAGTTTTGTTCGACAGCTCAACCTTTATGGATTTAGTAAAATTCAACA
GAATTTTCAAAGATCTGCCTTTCTAGCCACCTTTCTGTCAGAAGAGAAAGAATCGTCTGTCTTAAGCAAG
TTAAAGTTCTATTATAATCCAAATTTCAAGCGTGGCTATCCCCAACTTTTAGTAAGAGTGAAGAGAAGAA
TTGGTGTTAAAAATGCTTCACCTATATCTACTTTATTCAACGAAGATTTCAACAAGAAGCATTTTAGAGC
AGGGGCTAACATGGAGAATCATAATTCTGCCTTAGCTGCTGAAGCTAGTGAAGAAAGTTTATTTTCAGCC
TCTAAAAATTTAAATATGCCTCTAACAAGGGAATCTTCTGTCAGACAGATAATTGCAAATTCATCTGTCC
CCATTAGAAGTGGTTTCCCTCCTCCTTCACCTTCAACCTCAGTTGGACCATCAGAACAAATTGCAACAGA
TCAACATGCTATTTTAAATCAGTTGACCACTATTCATATGCACTCTCATAGTACCTACATGCAAGCAAGG
GGCCACATTGTGAATTTTATTACAACCACAACTTCTCAATACCACATCATATCTCCCTTACAAAATGGTT
ATTTTGGGCTGACAGTGGAACCATCTGCTGTTCCCACACGATATCCTCTGGTATCAGTCAATGAGGCTCC
ATATCGTAACATGCTACCAGCAGGCAACCCGTGGTTGCAAATGCCTACGATCGCTGATAGATCAGCTGCC
CCTCATTCCAGGCTAGCTCTTCAACCATCACCACTGGACAAATATCACCCTAATTACAACTGATCTGCCA
TTAAAAGAGGACCAGATTATGAATGACAACAGAGACTAACATTTACATTGACAAAAAACCCTAAAAATTT
CTGCAATTATCTTATTGAACAATAAAATTGCATGTTTACTTCT

NM_002083.2| Homo sapiens glutathione peroxidase 2 (gastrointestinal) (GPX2), mRNA
CTTCCTGGCTCCTCCTTCCTCCCCACCCCTCTAATAGGCTCATAAGTGGGCTCAGGCCTCTCTGCGGGGC
TCACTCTGCGCTTCACCATGGCTTTCATTGCCAAGTCCTTCTATGACCTCAGTGCCATCAGCCTGGATGG
GGAGAAGGTAGATTTCAATACGTTCCGGGGCAGGGCCGTGCTGATTGAGAATGTGGCTTCGCTCTGAGGC
ACAACCACCCGGGACTTCACCCAGCTCAACGAGCTGCAATGCCGCTTTCCCAGGCGCCTGGTGGTCCTTG
GCTTCCCTTGCAACCAATTTGGACATCAGGAGAACTGTCAGAATGAGGAGATCCTGAACAGTCTCAAGTA
TGTCCGTCCTGGGGGTGGATACCAGCCCACCTTCACCCTTGTCCAAAAATGTGAGGTGAATGGGCAGAAC
GAGCATCCTGTCTTCGCCTACCTGAAGGACAAGCTCCCCTACCCTTATGATGACCCATTTTCCCTCATGA
CCGATCCCAAGCTCATCATTTGGAGCCCTGTGCGCCGCTCAGATGTGGCCTGGAACTTTGAGAAGTTCCT
CATAGGGCCGGAGGGAGAGCCCTTCCGACGCTACAGCCGCACCTTCCCAACCATCAACATTGAGCCTGAC
ATCAAGCGCCTCCTTAAAGTTGCCATATAGATGTGAACTGCTCAACACACAGATCTCCTACTCCATCCAG
TCCTGAGGAGCCTTAGGATGCAGCATGCCTTCAGGAGACACTGCTGGACCTCAGCATTCCCTTGATATCA
GTCCCCTTCACTGCAGAGCCTTGCCTTTCCCCTCTGCCTGTTTCCTTTTCCTCTCCCAACCCTCTGGTTG
GTGATTCAACTTGGGCTCCAAGACTTGGGTAAGCTCTGGGCCTTCACAGAATGATGGCACCTTCCTAAAC
CCTCATGGGTGGTGTCTGAGAGGCGTGAAGGGCCTGGAGCCACTCTGCTAGAAGAGACCAATAAAGGGCA
GGTGTGGAAACGGCAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_003338.3| Homo sapiens ubiquitin-conjugating enzyme E2D 1 (UBC4/5 homolog, yeast) (UBE2D1), mRNA
AAAAGAGTCTCGCCGGCGTCCCCGCCCGCACACTCGCGCACACTCGCGCTCGGGCGCACACGGAGCAGGG
ACCGGCGCCCGGAGCGAGCCAGGGAGCGGCTAACCGGGGACCCACCGCGCGGAGCCAGCCTAGCTGCCAG
CGAGCCCAACCCGCGACGACCCACGCCCCTGAGCCCCGCAGCCGACCCCTGCCGGCCGGTGTCCCCACCG
CCATCCCTGACCCATGGCGCTGAAGAGGATTCAGAAAGAATTGAGTGATCTACAGCGCGATCCACCTGCT
CACTGTTCAGCTGGACCTGTGGGAGATGACTTGTTCCACTGGCAAGCCACTATTATGGGGCCTCCTGATA
GCGCATATCAAGGTGGAGTCTTCTTTCTCACTGTACATTTTCCGACAGATTATCCTTTTAAACCACCAAA
GATTGCTTTCACAACAAAAATTTACCATCCAAACATAAACAGTAATGGAAGTATTTGTCTCGATATTCTG
AGGTCACAATGGTCACCAGCTCTGACTGTATCAAAAGTTTTATTGTCCATATGTTCTCTACTTTGTGATC
CTAATCCAGATGACCCCTTAGTACCAGATATTGCACAAATCTATAAATCAGACAAAGAAAAATACAACAG
ACATGCAAGAGAATGGACTCAGAAATATGCAATGTAAAAATCAAAAACATTTTCATATATACCAGAGTAC
TGTAAAATCTAGGTTTTTTTCAACATTAGCAGTAAATTGAGCACTGTTTACTGTTTCATTGTACCATGAA
ACCATTTGATTTTTACCCATTTTAAATGTGTTTCTGAAGCAAGACAAAACAAACTTCCAAAAATACCCTT
AAGACTGTGATGAGAGCATTTATCATTTTGTATGCATTGAGAAAGACATTTATTATGGTTTTTAAGATAC
TTGGACATCTGCATCTTCAGCTTACAAGATCTACAATGCAGCTGAAAAGCAACCAAATTATTTTTTGCTG
AAACTAGATGTTTTTACATGAGAAATACTGTATGTGTTGTCTAAGATGTCAGTTTTATAAATCTGTATTC
AGATTTCATTCTTTGTTAGCTCACTTTATAATTTGTATTTTTTTACTGTATAGACTAAATATATTCTATT
TACATGTATGTCAACTCATTACTTTTTTCCTGTGAACAGTATTGAAAAACCCCAACGGCTGATAATTAAG
TGAATTAACTGTGTCTCCCTTGTCTTAGGATATTCTGTAGATTGATTGCAGATTTCTTAAATCTGAAATG
ATCTTTACACTGTAATTCTCAGCATACTGATTATGGAGAAACACTTGTTTTGATTTTGTTATACTTGACT
TAACTTTATTGCAATGTGAATTAATTGCACTGCTAAGTAGGAAGATGTGTAACTTTTATTTGTTGCTATT

CACATTTGAATTTTTTCCTGTATAGGCAATATTATATTGACACCTTTTACAGATCTTACTGTAGCTTTTT
CCATATAAATAAAATGCTTTTTCTACTATTTGTCTTGATTACTTAAAAAAATAAAAATATAAGTAAGGAT
CAAAACTCTAAAATTTTGCATGAAAATTACATCCAAATTGTGAAAATCAGATCTATTTTGTTTGCCATTA
GTCACCATTAGTTATATAAATTTTATTGTTTTAGGTTAGTATCTCTTTACTAAATTGTCAGTCTATAAGA
TAATATATGTTGATCCCTTGCTGTAGAGGAGAATTTAGAGTAATTTGGGGTTTGTCTTGGATTATATCTA
AATGGATTATTTGTTAAAAGTACTGAAATGAGTATAAGGCAGTATCACCCATCCAAAAGAAAGGTCTTTA
TAGACCTGCACAGTCACTAGATTAATTCATTAAAATGCCCCCACCCTGATGTAATTGACATTACATTTCT
TAACATTTTAAAATCTAGAATTTCTAAAATGGAATTTAATGCCATCACAATTTGAAAAACTTTTTTTTTT
TTTTTACTATAGAAGTTACAAAGGAAGTTCTAAAATTATGCCTCCCTCTGTTTTTATAAGTTGCCATCGA
AAAGTGATTTAAATAAGCAGGTTATCTTTATAGATTTTAAAGAAAACTAGAAAGTTTTAATGTTTTAACT
TGGGGAAAAATACATCTCTTTAATGTTTAGCATGCTTGTCAACCTTGAGTGAGTGTCATTTTTAAGAACA
GTTGTAGCCCTTCTGATTATTGCAGTAGCTGTAGAAGTATGTAAGAATATGTGATGGGTGTAGTCATTAG
CAAAGCATTTAAATCACTTGAGTATTTTGTCATGGTTCATTATTATTAAAGCACAAAATAACCTATTGTT
AGAAAATATGTGTTTTTATAAATGAATGTAAAATAATTAAATGAATTGTGAAATGGATGTTTAAGAAAAT
ATAGGCTTAAAAAGTAAATCTATAAAATGATGTCTTAAAACAGCCATATCATGAAAAATTCTACTTAGCT
ATATTATTATAAGCTACATTTGCCCTGAATTTGAACACTCAACATCACTAGATTTAAATATTTAGTATAT
TTTGATAGTAAAGGGTTTTGTTTCTTGAATATCTTCACTTTAAACAAAAAAAAAAAAACAACTTTCATTTG
TGTGGCATTTATTTTTGGAAGTGTCTTCTTTTTTTTCTTTATTAAAGTTTTTGAAACTTGCAAAAAAAAA
AAAAAAAAA

NM_144603.2| Homo sapiens NADPH oxidase organizer 1 (NOXO1), transcript variant a, mRNA
CATTCTTCTTCTTCCTCCCAGCAGCTCTGCCTGGAAGTCCCCCAATCCCTGTGCTCCTGCCCCCAGATCG
TGGGCTTGGGGGACAGCAAGAAAGCTGCAAAGGACTCAGCTGGGGCGGCTCCACAAGGACCCCACAGGCT
TGAGGTGTTGCCAGGATTCTATCCCAGGGGGTTTTCTGGGTCCCCCAGAGCCCTCTCAAGCACGGGGAGG
CAGAAGGGTCCTCGCCCATTTCAGGAATCTGCAGCCTGGAAGCCACAGCCATGGCAGGCCCCCGATACCC
AGTTTCAGTGCAAGGGGCAGCCCTGGTGCAGATCAAGAGGCTCCAAACGTTTGCCTTCTCTGTGCGCTGG
TCAGACGGCAGCGACACCTTCGTGCGCAGGAGTTGGGACGAATTCAGGCAGCTCAAGACCCTCAAGGAGA
CCTTCCCGGTGGAGGCGGGCCTGCTGCGGAGATCTGACCGCGTTCTCCCAAAGCTTCTCGATGCACCACT
GTTGGGACGCGTGGGGCGCACGAGCCGCGGCCTGGCGCGCCTGCAGCTGTTGGAAACCTATTCTCGGAGG
CTGCTGGCGACTGCAGAGCGCGTGGCACGGAGCCCGACGATCACTGGCTTCTTCGCACCGCAACCCCTGG
ACCTGGAGCCCGCGCTGCCACCCGGCAGCCGGGTGATCCTGCCCACCCCAGAGGAGCAGCCTCTTTCTCG
CGCTGCGGGCCGCCTCTCCATCCACAGTCTGGAGGCTCAGAGCCTGCGCTGCCTGCAGCCCTTCTGTACC
CAGGACACGCGGGATAGGCCTTTTCAGGCGCAGGCCCAGGAGAGCCTGGACGTGCTGCTGCGGCACCCCT
CAGGCTGGTGGCTGGTGGAGAACGAAGACCGGCAGACCGCCTGGTTTCCAGCGCCCTACCTGGAGGAGGC
GGCCCCGGGCCAAGGCCGGGAGGGAGGCCCGTCCCTAGGGAGCAGCGGTCCCCAGTTCTGTGCTTCCCGC
GCCTACGAGAGCAGCCGCGCAGATGAGCTGTCCGTGCCCGCGGGGGCGCGCGTGCGCGTGTTGGAAACGT
CAGACCGCGGCTGGTGGCTATGCAGGTACGGCGACCGGGCGGGCCTACTCCCCGCGGTGCTGCTGCGGCC
GGAAGGGCTGGGCGCTCTCCTGAGCGGGACGGGGTTCCGTGGAGGAGACGACCCGGCGGGTGAGGCCCGG
GGCTTCCCTGAACCCTCCCAGGCCACCGCCCCTCCCCCCACCGTGCCCACCCGACCTTCGCCGGGCGCCA
TCCAGAGCCGCTGCTGCACCGTCACACGCAGGGCCCTGGAGCGGCGCCCACGGCGCCAGGGCCGCCCTCG
AGGGTGCGTGGACTCTGTGCCGCACCCCACGACGGAGCAGTGAGCGCGAGGATCCCGATGAGGGGATGGA
CCGCGCCATCCCAGGGCTGGGGAGGAGCGGGGAGGGCGGATCTCCCTGGCCAGCAGGGATGGGAGGGGGC
CATTTGCTGTTCCTCTGAGTAAAGCTTGTTCTGCATGAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
AAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_015654.3| Homo sapiens N-acetyltransferase 9 (GCN5-related, putative) (NAT9), mRNA
GGGAACGTGGCTGGTTGGAGGAGGTAGATCACCCTTTCTGCGGGGGACGATTTCGTCGGTGGTAGGCTGC
TACCATGAGGTTGAATCAGAACACCTTGCTGCTGGGGAAGAAGGTGGTCCTTGTACCCTACACCTCGGAG
CATGTGCCCAGCAGGTACCACGAGTGGATGAAATCAGAGGAGCTGCAGCGTTTGACAGCCTCGGAGCCGC
TGACCCTGGAGCAGGAGTATGCCATGCAGTGCAGCTGGCAGGAAGATGCAGACAAGTGTACCTTCATTGT
GCTGGATGCCGAGAAGTGGCAGGCCCAGCCAGGCGCCACCGAAGAGAGCTGCATGGTGGGAGATGTGAAC
CTCTTCCTCACAGATCTAGAAGACCTCACCTTGGGGGAGATCGAGGTCATGATTGCAGAGCCCAGCTGCA
GGGGTAAGGGCCTTGGCACTGAGGCCGTTCTCGCGATGCTGTCTTACGGAGTGACCACGCTAGGTCTGAC
CAAGTTTGAGGCTAAAATTGGGCAAGGAAATGAACCAAGCATCCGGATGTTCCAGAAACTTCACTTTGAG
CAGGTGGCTACGAGCAGTGTTTTTCAGGAGGTGACCCTCAGACTGACAGTGAGTGAGTCCGAGCATCAGT
GGCTTCTGGAGCAGACCAGCCACGTGGAAGAGAAGCCTTACAGAGATGGGTCGGCAGAGCCCTGCTGATG
GCTGGGCCTTGTGGGCAGCCACTCTGTGTGAGCAGGGTGTTGGGCCCATACACTTCAAAGACCAGAGCCC
TGCACTGGGAGAGTGCTCCTGGCCCAGGCTGGGAATCACCTTTCGAGGCCCTTCAGACTCTGGCGGGGCT
TGCTGTGGCCTCCCTCCAGCTAGTGGTGTGGCTGAGCAGACTCCAGGGCCAGGGCCAGTTCCCTTCTCCC

CTCCCGGCCAAACCCAGACCCAGACTCTAGGAAGCTGGAATGGAGGGCAGGGATCCATGGGAGATGTCGG
GATGAAGGTGGGAGCTGGAGGTGCAGGGGGACCTGGAACATGGATGGGAGTGGACAGGCCTTTCTCCTTA
GAGGCCAGAGGTGCTGCCCTGGCTGGGAGTGAAGCTCCAGGCACTACCAGCTTTCCTGATTTTCCCGTTT
GGTCCATGTGAAGAGCTACCACGAGCCCCAGCCTCACAGTGTCCACTCAAGGGCAGCTTGGTCCTCTTGT
CCTGCAGAGGCAGGCTGGTGTGACCCTGGGAACTTGACCCGGGAACAACAGGTGGTCCAGAGTGAGTGTG
GCCTGGCCCCTCAACCTAGTGTCCGTCCTCCTCTCTCCTGGAGCCAGTCTTGAGTTTAAAGGCATTAGTG
TTAGATACAGCTCCTTGTGGCTGGAAAACACCCCTCTGCTGATAAAGCTCAGGGGGCACTGAGGAAGCAG
AGGCCCCTTGGGGGTGCCCTCCTGAAGAGAGCGTCAGGCCATCAGCTCTGTCCCTCTGGTGCTCCCACGT
CTGTTCCTCACCCTCCATCTCTGGGAGCAGCTGCACCTGACTGGCCACGCGGGGGCAGTGGAGGCACAGG
CTCAGGGTGGCCGGGCTACCTGGCACCCTATGGCTTACAAAGTAGAGTTGGCCCAGTTTCCTTCCACCTG
AGGGGAGCACTCTGACTCCTAACAGTCTTCCTTGCCCTGCCATCATCTGGGGTGGCTGGCTGTCAAGAAA
GGCCGGGCATGCTTTCTAAACACAGCCACAGGAGGCTTGTAGGGCATCTTCCAGGTGGGGAAACAGTCTT
AGATAAGTAAGGTGACTTGCCTAAGGCCTCCCAGCACCCTTGATCTTGGAGTCTCACAGCAGACTGCATG
TGAACAACTGGAACCGAAAACATGCCTCAGTATAAAACAAACATTATAAAACGAAAAAAAAAAAAAAAAA
AAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_001711.3| Homo sapiens biglycan (BGN), mRNA
AGCCTCCCGCCCGCCGCCTCTGTCTCCCTCTCTCCACAAACTGCCCAGGAGTGAGTAGCTGCTTTCGGTC
CGCCGGACACACCGGACAGATAGACGTGCGGACGGCCCACCACCCCAGCCCGCCAACTAGTCAGCCTGCG
CCTGGCGCCTCCCCTCTCCAGGTCCATCCGCCATGTGGCCCCTGTGGCGCCTCGTGTCTCTGCTGGCCCT
GAGCCAGGCCCTGCCCTTTGAGCAGAGAGGCTTCTGGGACTTCACCCTGGACGATGGGCCATTCATGATG
AACGATGAGGAAGCTTCGGGCGCTGACACCTCGGGCGTCCTGGACCCGGACTCTGTCACACCCACCTACA
GCGCCATGTGTCCTTTCGGCTGCCACTGCCACCTGCGGGTGGTTCAGTGCTCCGACCTGGGTCTGAAGTC
TGTGCCCAAAGAGATCTCCCCTGACACCACGCTGCTGGACCTGCAGAACAACGACATCTCCGAGCTCCGC
AAGGATGACTTCAAGGGTCTCCAGCACCTCTACGCCCTCGTCCTGGTGAACAACAAGATCTCCAAGATCC
ATGAGAAGGCCTTCAGCCCACTGCGGAAGCTGCAGAAGCTCTACATCTCCAAGAACCACCTGGTGGAGAT
CCCGCCCAACCTACCCAGCTCCCTGGTGGAGCTCCGCATCCACGACAACCGCATCCGCAAGGTGCCCAAG
GGAGTGTTCAGCGGGCTCCGGAACATGAACTGCATCGAGATGGGCGGGAACCCACTGGAGAACAGTGGCT
TTGAACCTGGAGCCTTCGATGGCCTGAAGCTCAACTACCTGCGCATCTCAGAGGCCAAGCTGACTGGCAT
CCCCAAAGACCTCCCTGAGACCCTGAATGAACTCCACCTAGACCACAACAAAATCCAGGCCATCGAACTG
GAGGACCTGCTTCGCTACTCCAAGCTGTACAGGCTGGGCCTAGGCCACAACCAGATCAGGATGATCGAGA
ACGGGAGCCTGAGCTTCCTGCCCACCCTCCGGGAGCTCCACTTGGACAACAACAAGTTGGCCAGGGTGCC
CTCAGGGCTCCCAGACCTCAAGCTCCTCCAGGTGGTCTATCTGCACTCCAACAACATCACCAAAGTGGGT
GTCAACGACTTCTGTCCCATGGGCTTCGGGGTGAAGCGGGCCTACTACAACGGCATCAGCCTCTTCAACA
ACCCCGTGCCCTACTGGGAGGTGCAGCCGGCCACTTTCCGCTGCGTCACTGACCGCCTGGCCATCCAGTT
TGGCAACTACAAAAAGTAGAGGCAGCTGCAGCCACCGCGGGGCCTCAGTGGGGGTCTCTGGGGAACACAG
CCAGACATCCTGATGGGGAGGCAGAGCCAGGAAGCTAAGCCAGGGCCCAGCTGCGTCCAACCCAGCCCCC
CACCTCGGGTCCCTGACCCCAGCTCGATGCCCCATCACCGCCTCTCCCTGGCTCCCAAGGGTGCAGGTGG
GCGCAAGGCCCGGCCCCCATCACATGTTCCCTTGGCCTCAGAGCTGCCCCTGCTCTCCCACCACAGCCAC
CCAGAGGCACCCCATGAAGCTTTTTTCTCGTTCACTCCCAAACCCAAGTGTCCAAGGCTCCAGTCCTAGG
AGAACAGTCCCTGGGTCAGCAGCCAGGAGGCGGTCCATAAGAATGGGGACAGTGGGCTCTGCCAGGGCTG
CCGCACCTGTCCAGACACACATGTTCTGTTCCTCCTCCTCATGCATTTCCAGCCTTTCAACCCTCCCCGA
CTCTGCGGCTCCCCTCAGCCCCCTTGCAAGTTCATGGCCTGTCCCTCCCAGACCCCTGCTCCACTGGCCC
TTCGACCAGTCCTCCCTTCTGTTCTCTCTTTCCCCGTCCTTCCTCTCTCTCTCTCTCTCTCTCTCTCT
CTTTCTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTGTCTTGTGCTTCCTCAGACCTT
TCTCGCTTCTGAGCTTGGTGGCCTGTTCCCTCCATCTCTCCGAACCTGGCTTCGCCTGTCCCTTTCACTC
CACACCCTCTGGCCTTCTGCCTTGAGCTGGGACTGCTTTCTGTCTGTCCGGCCTGCACCCAGCCCCTGCC
CACAAAACCCCAGGGACAGCGGTCTCCCCAGCCTGCCCTGCTCAGGCCTTGCCCCCAAACCTGTACTGTC
CCGGAGGAGGTTGGGAGGTGGAGGCCCAGCATCCCGCGCAGATGACACCATCAACCGCCAGAGTCCCAGA
CACCGGTTTTCCTAGAAGCCCCTCACCCCCACTGGCCCACTGGTGGCTAGGTCTCCCCTTATCCTTCTGG
TCCAGCGCAAGGAGGGGCTGCTTCTGAGGTCGGTGGCTGTCTTTCCATTAAAGAAACACCGTGCAACGTG
AAAAAAAAAAAAAAAAAAAAA

NM_003998.2| Homo sapiens nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 (NFKB1), mRNA
GTGAGAGAGTGAGCGAGACAGAAAGAGAGAGAAGTGCACCAGCGAGCCGGGGCAGGAAGAGGAGGTTTCG
CCACCGGAGCGGCCCGGCGACGCGCTGACAGCTTCCCCTGCCCTTCCCGTCGGTCGGGCCGCCAGCCGCC
GCAGCCCTCGGCCTGCACGCAGCCACCGGCCCCGCTCCCGGAGCCCAGCGCCGCCGAGGCCGCAGCCGCC
CGGCCAGTAAGGCGGCGCCGCCGCCCGGCCACCGCGCGCCCTGCGCTTCCCTCCGCCCGCGCTGCGGCCA
TGGCGCGGCGCTGACTGGCCTGGCCCGGCCCCGCCGCGCTCCCGCTCGCCCCGACCCGCACTCGGGCCCG
CCCGGGCTCCGGCCTGCCGCCGCCTCTTCCTTCTCCAGCCGGCAGGCCCGCGCCGCTTAGGAGGGAGAGC

CCACCCGCGCCAGGAGGCCGAACGCGGACTCGCCACCCGGCTTCAGAATGGCAGAAGATGATCCATATTT
GGGAAGGCCTGAACAAATGTTTCATTTGGATCCTTCTTTGACTCATACAATATTTAATCCAGAAGTATTT
CAACCACAGATGGCACTGCCAACAGCAGATGGCCCATACCTTCAAATATTAGAGCAACCTAAACAGAGAG
GATTTCGTTTCCGTTATGTATGTGAAGGCCCATCCCATGGTGGACTACCTGGTGCCTCTAGTGAAAAGAA
CAAGAAGTCTTACCCTCAGGTCAAAATCTGCAACTATGTGGGACCAGCAAAGGTTATTGTTCAGTTGGTC
ACAAATGGAAAAAATATCCACCTGCATGCCCACAGCCTGGTGGGAAAACACTGTGAGGATGGGATCTGCA
CTGTAACTGCTGGACCCAAGGACATGGTGGTCGGCTTCGCAAACCTGGGTATACTTCATGTGACAAAGAA
AAAAGTATTTGAAACACTGGAAGCACGAATGACAGAGGCGTGTATAAGGGGCTATAATCCTGGACTCTTG
GTGCACCCTGACCTTGCCTATTTGCAAGCAGAAGGTGGAGGGGACCGGCAGCTGGGAGATCGGGAAAAAG
AGCTAATCCGCCAAGCAGCTCTGCAGCAGACCAAGGAGATGGACCTCAGCGTGGTGCGGCTCATGTTTAC
AGCTTTTCTTCCGGATAGCACTGGCAGCTTCACAAGGCGCCTGGAACCCGTGGTATCAGACGCCATCTAT
GACAGTAAAGCCCCCAATGCATCCAACTTGAAAATTGTAAGAATGGACAGGACAGCTGGATGTGTGACTG
GAGGGGAGGAAATTTATCTTCTTTGTGACAAAGTTCAGAAAGATGACATCCAGATTCGATTTTATGAAGA
GGAAGAAAATGGTGGAGTCTGGGAAGGATTTGGAGATTTTTCCCCCACAGATGTTCATAGACAATTTGCC
ATTGTCTTCAAAACTCCAAAGTATAAAGATATTAATATTACAAAACCAGCCTCTGTGTTTGTCCAGCTTC
GGAGGAAATCTGACTTGGAAACTAGTGAACCAAAACCTTTCCTCTACTATCCTGAAATCAAAGATAAAGA
AGAAGTGCAGAGGAAACGTCAGAAGCTCATGCCCAATTTTTCGGATAGTTTCGGCGGTGGTAGTGGTGCC
GGAGCTGGAGGCGGAGGCATGTTTGGTAGTGGCGGTGGAGGAGGGGGCACTGGAAGTACAGGTCCAGGGT
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TGCTGGGATGAAGCATGGAACCATGGACACTGAATCTAAAAAGGACCCTGAAGGTTGTGACAAAAGTGAT
GACAAAAACACTGTAAACCTCTTTGGGAAAGTTATTGAAACCACAGAGCAAGATCAGGAGCCCAGCGAGG
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GGATAACCTCTTTCTAGAGAAGGCTATGCAGCTTGCAAAGAGGCATGCCAATGCCCTTTTCGACTACGCG
GTGACAGGAGACGTGAAGATGCTGCTGGCCGTCCAGCGCCATCTCACTGCTGTGCAGGATGAGAATGGGG
ACAGTGTCTTACACTTAGCAATCATCCACCTTCATTCTCAACTTGTGAGGGATCTACTAGAAGTCACATC
TGGTTTGATTTCTGATGACATTATCAACATGAGAAATGATCTGTACCAGACGCCCTTGCACTTGGCAGTG
ATCACTAAGCAGGAAGATGTGGTGGAGGATTTGCTGAGGGCTGGGGCCGACCTGAGCCTTCTGGACCGCT
TGGGTAACTCTGTTTTGCACCTAGCTGCCAAAGAAGGACATGATAAAGTTCTCAGTATCTTACTCAAGCA
CAAAAAGGCAGCACTACTTCTTGACCACCCCAACGGGGACGGTCTGAATGCCATTCATCTAGCCATGATG
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CCGGGCGCACAGCACTGCACCTGGCTGTGGAGCACGACAACATCTCATTGGCAGGCTGCCTGCTCCTGGA
GGGTGATGCCCATGTGGACAGTACTACCTACGATGGAACCACACCCCTGCATATAGCAGCTGGGAGAGGG
TCCACCAGGCTGGCAGCTCTTCTCAAAGCAGCAGGAGCAGATCCCCTGGTGGAGAACTTTGAGCCTCTCT
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GATTTACTAGCACAAGGAGACATGAAACAGCTGGCTGAAGATGTGAAGCTGCAGCTGTATAAGTTACTAG
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CTTCCGGCTGAGTCCTGCTCCTTCCAAAACACTTATGGACAACTATGAGGTCTCTGGGGGTACAGTCAGA
GAGCTGGTGGAGGCCCTGAGACAAATGGGCTACACCGAAGCAATTGAAGTGATCCAGGCAGCCTCCAGCC
CAGTGAAGACCACCTCTCAGGCCCACTCGCTGCCTCTCTCGCCTGCCTCCACAAGGCAGCAAATAGACGA
GCTCCGAGACAGTGACAGTGTCTGCGACAGCGGCGTGGAGACATCCTTCCGCAAACTCAGCTTTACCGAG
TCTCTGACCAGTGGTGCCTCACTGCTAACTCTCAACAAAATGCCCCATGATTATGGGCAGGAAGGACCTC
TAGAAGGCAAAATTTAGCCTGCTGACAATTTCCCACACCGTGTAAACCAAAGCCCTAAAATTCCACTGCG
TTGTCCACAAGACAGAAGCTGAAGTGCATCCAAAGGTGCTCAGAGAGCCGGCCCGCCTGAATCATTCTCG
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ACAGATAGTATCTAGCAATCACAACACTGGCTGAGCGGATGCATCTGGGGATGAGGTTGCTTACTAAGCT
TTGCCAGCTGCTGCTGGATCACAGCTGCTTTCTGTTGTCATTGCTGTTGTCCCTCTGCTACGTTCCTATT
GTCATTAAAGGTATCACGGTCGCCACCTGGCATTCCTTCTGACCACAGCATCATTTTGCATTCAAATTAA
GGGTTAAGAAAAGAGATATTTTAAAATGAGAGTCACTTGATGTGCCATTTTAAAAAAAAAGGCATATTGC
TTTTTCTAATGTGGTTATTTCTCTGATTTGCAAAAAAAAAAAAAAAAAAAAAATACTTGTCAATATTTAA
ACATGGTTACAATCATTGCTGAAAATGGTATTTTCCCCCTTTTCTGCATTTTGCTATTGTAAATATGTTT
TTTAGATCAAATACTTTAAAGGAAAAAATGTTGGATTTATAAATGCTATTTTTTATTTTACTTTTATAAT
AAAAGGAAAAGCAAATTGATGACCTCAAAAAAAAAAAAAAAAAA

NM_002883.2| Homo sapiens Ran GTPase activating protein 1 (RANGAP1), mRNA
AAATCCTCCTCCTCCGCCATCATCCGCCGCGGTGCGGAGAGCAGGTGGTGCTGGAAGCGCGTGAGGCCGG
GAGCTCGAGAGAGCTAACAGACTAGCCGGCTGGACATCTGGACCGCTGGATCCGGAGGTGGCGACCCCGG
CCTGACCCGGACCCTAAATCCGTCCCCGCCCCAGAGGGCGGAGGCGCGCGCTCGATTCCCCCCACGCGGC
GGCGCCGCCTGTTTACGTCTGCAGATCTCCAGGGGAGCCCACCAGCCTAGTCAACATGGCCTCGGAAGAC
ATTGCCAAGCTGGCAGAGACACTTGCCAAGACTCAGGTGGCCGGGGGACAGCTGAGTTTCAAAGGCAAGA
GCCTCAAACTCAACACTGCAGAAGATGCTAAAGATGTGATTAAAGAGATTGAAGACTTTGACAGCTTGGA

GGCTCTGCGTCTGGAAGGCAACACAGTGGGCGTGGAAGCAGCCAGGGTCATCGCCAAGGCCTTAGAGAAG
AAGTCGGAGTTGAAGCGCTGCCACTGGAGTGACATGTTCACGGGAAGGCTGCGGACCGAGATCCCACCAG
CCCTGATCTCACTAGGGGAAGGACTCATCACAGCTGGGGCTCAGCTGGTGGAGCTGGACTTAAGCGACAA
CGCATTCGGGCCCGACGGTGTGCAAGGCTTCGAGGCCCTGCTCAAGAGCTCAGCCTGCTTCACCCTGCAG
GAACTCAAGCTCAACAACTGTGGCATGGGCATTGGCGGCGGCAAGATCCTGGCTGCAGCTCTGACCGAAT
GTCACCGGAAATCCAGTGCCCAAGGCAAGCCTCTGGCCCTGAAGGTCTTTGTGGCTGGCAGAAACCGTCT
GGAGAATGATGGCGCCACTGCCTTGGCAGAAGCTTTTAGGGTCATCGGGACCCTGGAGGAGGTCCACATG
CCACAGAATGGGATCAACCACCCTGGCATCACTGCCCTGGCCCAGGCTTTCGCTGTCAACCCCCTGCTGC
GGGTCATCAACCTGAATGACAACACCTTCACTGAGAAGGGCGCCGTGGCCATGGCCGAGACCTTGAAGAC
CTTGCGGCAGGTGGAGGTGATTAATTTTGGGGACTGCCTGGTGCGCTCCAAGGGTGCAGTTGCCATTGCA
GATGCCATCCGCGGCGGCCTGCCCAAGCTAAAGGAGCTGAACTTGTCATTCTGTGAAATCAAGAGGGATG
CTGCCCTGGCTGTTGCTGAGGCCATGGCAGACAAAGCTGAGCTGGAGAAGCTGGACCTGAATGGCAACAC
CCTGGGAGAAGAAGGCTGTGAACAGCTTCAGGAGGTGCTGGAGGGCTTCAACATGGCCAAGGTGCTGGCG
TCCCTCAGTGATGACGAGGACGAGGAGGAGGAGGAGGAAGGAGAAGAGGAAGAAGAGGAAGCAGAAGAAG
AGGAGGAGGAAGATGAGGAAGAGGAGGAAGAAGAGGAGGAGGAGGAGGAAGAAGAGCCTCAGCAGCGAGG
GCAGGGAGAGAAGTCAGCCACGCCCTCACGGAAGATTCTGGACCCTAACACTGGGGAGCCAGCTCCCGTG
CTGTCCTCCCCACCTCCTGCAGACGTCTCCACCTTCCTGGCTTTTCCCTCTCCAGAGAAGCTGCTGCGCC
TAGGGCCCAAGAGCTCCGTGCTGATAGCCCAGCAGACTGACACGTCTGACCCCGAGAAGGTGGTCTCTGC
CTTCCTAAAGGTGTCATCTGTGTTCAAGGACGAAGCTACTGTGAGGATGGCAGTGCAGGATGCAGTAGAT
GCCCTGATGCAGAAGGCTTTCAACTCCTCGTCCTTCAACTCCAACACCTTCCTCACCAGGCTGCTCGTGC
ACATGGGTCTGCTCAAGAGTGAAGACAAGGTCAAGGCCATTGCCAACCTGTACGGCCCCCTGATGGCGCT
GAACCACATGGTGCAGCAGGACTATTTCCCCAAGGCCCTTGCACCCCTGCTGCTGGCGTTCGTGACCAAG
CCCAACAGCGCCCTGGAATCCTGCTCCTTCGCCCGCCACAGTCTGCTGCAGACGCTGTACAAGGTCTAGA
CTCAAAGCCTCTCCCATCCCTTGGCCTGGACCAGTGAGCTGGGGAGGGACTCGGATGAACTGAGGCGCAG
CCTACGCCATTGCCTTGGACAGGACTCTGGCCACAGGCAGGGCGGGTCTGTGTCCCATGTGTCCTGTCAG
TCCCCTGAGTATGTGTGTGGGTGTGGCGCATGTGCAGGTCTGTGCCTCCTGTCGGGATTTGGGTTTTAAC
GTCTTCTGCTGGCCCAGCCCTGCTCTGTTGTGGGGAGTTGGCCCCCAGGGGAAAGGGCTGTGAGCTGCTC
CGCCATTAAACTCACCTCCACCTGAGGGCGCTCTGCTGATCTCCGCCTGGGCCCTGATGGCCGTCCCCAC
CCACCTGCCTTCCGGCCCGGCTCCCTGGCGGAGCCAGAACCCAGGGAGTTGCCCGCGTGCTGTCCTTCCC
CTCTGTGTTGTGATTGGGTTGTTTCCTGCCCTGCCTGGGGCTGCTTCTCGTCACCAAGCCCTGGTCCTGC
GGCAGCTGTCACCCCTACCATCCATACCACTGTGCTGACCGCTCAGCCTGAAGAGCAGAGAATGCCATGG
GTGGGACTGTGGGGGTCGGATCGTGGGGTTGTTGGCAGAGGGCAACCCTGGGCCCCACACCGTGTGGACA
GGCAGACACCAGATTGTCCAGGAGCAGGAGCTGCTGGGACTGCGCTGGCCCCGGACCTAGTGGGCCTTCT
CCTGGCTGCTGAGATGTCGTCTGTGACTGGCCTGGCTGGAGGGGGAGTGTTGACAACCCAAAGCTGTTCT
CCAGTCTGGGGAGGGAGAGGCAGGGTCCCCAATGTCCGAGCTGCATCTGGACGCTGCTCTTAAAGGACCT
CCTGGGGCAGGGGAGCGGTAGGGTCTGGACTGGGCAGATGCTGTATGACCTCCCTGAGCACCCGTGACTG
CCCCATGCTTTCCCCTTTGTGCTCTGTGTGTGTCTGGGCTGTGCCCGGGGGCTTCACAAATAAAGTCGTG
TGGCAGCTTCAAAAAAAAAAAAAAAAAAA

NM_003600.2| Homo sapiens aurora kinase A (AURKA), transcript variant 2, mRNA
ACAAGGCAGCCTCGCTCGAGCGCAGGCCAATCGGCTTTCTAGCTAGAGGGTTTAACTCCTATTTAAAAAG
AAGAACCTTTGAATTCTAACGGCTGAGCTCTTGGAAGACTTGGGTCCTTGGGTCGCAGGTGGGAGCCGAC
GGGTGGGTAGACCGTGGGGGATATCTCAGTGGCGGACGAGGACGGCGGGGACAAGGGGCGGCTGGTCGGA
GTGGCGGAGCGTCAAGTCCCCTGTCGGTTCCTCCGTCCCTGAGTGTCCTTGGCGCTGCCTTGTGCCCGCC
CAGCGCCTTTGCATCCGCTCCTGGGCACCGAGGCGCCCTGTAGGATACTGCTTGTTACTTATTACAGCTA
GAGGCATCATGGACCGATCTAAAGAAAACTGCATTTCAGGACCTGTTAAGGCTACAGCTCCAGTTGGAGG
TCCAAAACGTGTTCTCGTGACTCAGCAATTTCCTTGTCAGAATCCATTACCTGTAAATAGTGGCCAGGCT
CAGCGGGTCTTGTGTCCTTCAAATTCTTCCCAGCGCATTCCTTTGCAAGCACAAAAGCTTGTCTCCAGTC
ACAAGCCGGTTCAGAATCAGAAGCAGAAGCAATTGCAGGCAACCAGTGTACCTCATCCTGTCTCCAGGCC
ACTGAATAACACCCAAAAGAGCAAGCAGCCCCTGCCATCGGCACCTGAAAATAATCCTGAGGAGGAACTG
GCATCAAAACAGAAAAATGAAGAATCAAAAAAGAGGCAGTGGGCTTTGGAAGACTTTGAAATTGGTCGCC
CTCTGGGTAAAGGAAAGTTTGGTAATGTTTATTTGGCAAGAGAAAAGCAAAGCAAGTTTATTCTGGCTCT
TAAAGTGTTATTTAAAGCTCAGCTGGAGAAAGCCGGAGTGGAGCATCAGCTCAGAAGAGAAGTAGAAATA
CAGTCCCACCTTCGGCATCCTAATATTCTTAGACTGTATGGTTATTTCCATGATGCTACCAGAGTCTACC
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GAGAACTGCTACTTATATAACAGAATTGGCAAATGCCCTGTCTTACTGTCATTCGAAGAGAGTTATTCAT
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CAGTACATGCTCCATCTTCCAGGAGGACCACTCTCTGTGGCACCCTGGACTACCTGCCCCCTGAAATGAT
TGAAGGTCGGATGCATGATGAGAAGGTGGATCTCTGGAGCCTTGGAGTTCTTTGCTATGAATTTTTAGTT
GGGAAGCCTCCTTTTGAGGCAAACACATACCAAGAGACCTACAAAAGAATATCACGGGTTGAATTCACAT

TCCCTGACTTTGTAACAGAGGGAGCCAGGGACCTCATTTCAAGACTGTTGAAGCATAATCCCAGCCAGAG
GCCAATGCTCAGAGAAGTACTTGAACACCCCTGGATCACAGCAAATTCATCAAAACCATCAAATTGCCAA
AACAAAGAATCAGCTAGCAAACAGTCTTAGGAATCGTGCAGGGGGAGAAATCCTTGAGCCAGGGCTGCCA
TATAACCTGACAGGAACATGCTACTGAAGTTTATTTTACCATTGACTGCTGCCCTCAATCTAGAACGCTA
CACAAGAAATATTTGTTTTACTCAGCAGGTGTGCCTTAACCTCCCTATTCAGAAAGCTCCACATCAATAA
ACATGACACTCTGAAGTGAAAGTAGCCACGAGAATTGTGCTACTTATACTGGTTCATAATCTGGAGGCAA
GGTTCGACTGCAGCCGCCCCGTCAGCCTGTGCTAGGCATGGTGTCTTCACAGGAGGCAAATCCAGAGCCT
GGCTGTGGGGAAAGTGACCACTCTGCCCTGACCCCGATCAGTTAAGGAGCTGTGCAATAACCTTCCTAGT
ACCTGAGTGAGTGTGTAACTTATTGGGTTGGCGAAGCCTGGTAAAGCTGTTGGAATGAGTATGTGATTCT
TTTTAAGTATGAAAATAAAGATATATGTACAGACTTGTATTTTTTCTCTGGTGGCATTCCTTTAGGAATG
CTGTGTGTCTGTCCGGCACCCCGGTAGGCCTGATTGGGTTTCTAGTCCTCCTTAACCACTTATCTCCCAT
ATGAGAGTGTGAAAAATAGGAACACGTGCTCTACCTCCATTTAGGGATTTGCTTGGGATACAGAAGAGGC
CATGTGTCTCAGAGCTGTTAAGGGCTTATTTTTTTAAAACATTGGAGTCATAGCATGTGTGTAAACTTTA
AATATGCAAATAAATAAGTATCTATGTCTAAAAAAA

NM_006430.2| Homo sapiens chaperonin containing TCP1, subunit 4 (delta) (CCT4), mRNA
CTTCTCCGCCTCCGCCTCCTCCCGACGCCGGCGCCGCTTTCTGGAAGGTTCGTGAAGGCAGTGAGGGCTT
ACCGTTATTACACTGCGGCCGGCCAGAATCCGGGTCCATCCGTCCTTCCCGAGCCAACCCAGACACAGCG
GAGTTTGCCATGCCCGAGAATGTGGCACCCCGGAGCGGGGCGACTGCCGGGGCTGCCGGCGGCCGCGGGA
AAGGCGCCTATCAGGACCGCGACAAGCCAGCCCAGATCCGCTTCAGCAACATTTCCGCCGCCAAAGCGGT
TGCTGATGCTATTAGAACAAGCCTTGGACCAAAAGGAATGGATAAAATGATTCAAGATGGAAAAGGTGAT
GTAACCATTACAAATGATGGTGCTACCATTCTGAAACAAATGCAAGTATTACATCCAGCAGCCAGAATGC
TGGTGGAGCTGTCTAAGGCTCAAGATATAGAAGCAGGAGATGGCACCACATCAGTAGTCATCATTGCTGG
CTCCCTCTTAGATTCTTGTACCAAGCTTCTTCAGAAAGGGATTCATCCAACCATCATTTCTGAGTCATTC
CAGAAGGCCCTGGAAAAGGGCATTGAAATCTTGACTGACATGTCTCGACCTGTGGAACTGAGTGACAGAG
AAACTTTGTTAAATAGTGCAACCACTTCACTGAACTCAAAGGTGGTTTCTCAGTATTCAAGTCTGCTTTC
TCCAATGAGTGTAAATGCAGTGATGAAAGTGATTGACCCAGCCACAGCCACCAGTGTAGATCTTAGAGAT
ATTAAAATAGTTAAGAAGCTTGGTGGGACAATTGATGACTGTGAGTTGGTGGAAGGGCTGGTTCTCACCC
AAAAAGTGTCAAATTCTGGCATAACCAGAGTTGAAAAGGCCAAGATTGGGCTTATTCAGTTTTGCTTATC
TGCTCCCAAAACAGACATGGATAATCAAATAGTGGTTTCTGACTATGCCCAGATGGACCGAGTGCTGCGA
GAAGAGAGAGCCTATATTTTAAATTTAGTGAAGCAAATTAAAAAAACAGGATGTAATGTCCTTCTCATAC
AGAAATCTATTCTAAGAGATGCTCTTAGTGATCTTGCATTACACTTTCTGAATAAAATGAAGATCATGGT
GATTAAGGATATTGAAAGAGAAGACATTGAATTCATTTGTAAGACAATTGGAACCAAGCCAGTTGCTCAT
ATTGACCAATTTACTGCTGACATGCTGGGTTCTGCTGAGTTAGCTGAGGAGGTCAATTTAAATGGTTCTG
GCAAACTGCTCAAGATTACAGGCTGTGCCAGCCCTGGAAAAACAGTTACAATTGTTGTTCGTGGTTCTAA
CAAACTGGTGATTGAAGAAGCTGAGCGCTCCATTCATGATGCCCTATGTGTTATTCGTTGTTTAGTGAAG
AAGAGGGCTCTTATTGCAGGAGGTGGTGCTCCAGAAATAGAGTTGGCCCTACGATTAACTGAATATTCAC
GAACACTGAGTGGTATGGAATCCTACTGCGTTCGTGCTTTTGCAGATGCTATGGAGGTCATTCCATCTAC
ACTAGCTGAAAATGCCGGCCTGAATCCCATTTCTACAGTAACAGAACTAAGAAACCGGCATGCCCAGGGA
GAAAAAACTGCAGGCATTAATGTCCGAAAGGGTGGTATTTCCAACATTTTGGAGGAACTGGTTGTCCAGC
CTCTGTTGGTATCAGTCAGTGCTCTGACTCTTGCAACTGAAACTGTTCGGAGCATTCTGAAAATAGATGA
TGTGGTAAACACTCGATAATCTGGATAACTGACTAGCACCATTATGATCACCAGTATTGTGGCTGGAATG
GAAGAAGATCACCTTGGTGTTCCTTGTTTGGAAGATTATTTCCTCTGAATTTCTGGGCTTGGTCTTCCAG
TTGGCATTTGCCTGAAGTTGTATTGAAACAATTTAATGAAAATATTAAATATTTGGTTTCAAAAGGCAGA
TTTATCTTCTCCCAACATTCTGTTATTTCTGATACTTTTGAAAAACTAATAAAAACTAATAAAAGAAGCG
TAAAAAGTGAGTTTACATGTTGAGGAAAAAAATGGCCCAATATGCTCATCACTGATAAATGCTCCCTGGC
CTTAAAAACTACCAACATATAATATATATGCTGTCTTAAAAGTTAATGATCCAAGTGGCACCTCTCTGAA
CCTACTTTGGCTTGGGAGGCTGCCCAGTTAAAACAAAAATAAGTTAATGGTACAGAAAGAGAATGAAAAA
TGAAAGCCTCCTTTTATCCTATCATCCTAATTCCTTTTCCCAGTAATAATGACTGCTGTGTTGGATTCCT
TCTGCAAATAAAAGTGTATACATAAAAAAAAAAAAAAAA

NM_012388.2| Homo sapiens pallidin homolog (mouse) (PLDN), mRNA
ACACTGCGTCCGGGGCCAGACGACGATATCAGCGCGGGGTCCCCACAACGCCATGGGGCAGAGCCAACTC
TCGAGCGCGTGATCGAAGCCCGCAGTTTTTTCGCCCCCGTCACTTCCGGGTGCGACAATCTCTTCTGTCC
GGCCAGCCGCTGGAGTCGTTAGGTGCCGCCTTGCTTCTGACGAGCCACACGTTTGCTTCTTCCCTGTGTT
CCCAGCTGGAGGGACATGAGTGTCCCTGGGCCGTCGTCTCCGGACGGGGCCCTGACACGGCCACCCTACT
GCCTGGAGGCCGGGGAGCCGACGCCTGGTTTAAGTGACACTTCTCCAGATGAAGGGTTAATAGAGGACTT
GACTATAGAAGACAAAGCAGTGGAGCAACTGGCAGAAGGATTGCTTTCTCATTATTTGCCAGATCTGCAG
AGATCAAAACAAGCCCTCCAGGAACTCACACAGAACCAAGTTGTATTGTTAGACACACTGGAACAAGAGA
TTTCAAAATTTAAAGAATGTCATTCTATGTTGGATATTAATGCTTTGTTTGCTGAGGCTAAACACTATCA

TGCCAAGTTGGTGAATATAAGAAAAGAGATGCTGATGCTTCATGAAAAAACATCAAAGTTAAAAAAAAGA
GCACTTAAACTGCAGCAGAAGAGGCAAAAAGAAGAGTTGGAAAGGGAGCAGCAACGAGAGAAGGAGTTTG
AAAGAGAAAAGCAGTTAACTGCCAGACCAGCCAAAAGGATGTGAAAAGTTGTGTTTGTGTGTTTTCTTCT
CCTGTCCCATATTTGGGTTATGATGACTCAAGTGTAGACTGAAGTTGAGGTAGTGCCTTATGCCATTATG
TCATATGTTGAAATCCTTATTCCGGTATTACTGTGTCTCCATGCCTTTTTTCCAAGTAGCAGACGTCATG
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TATGAATTTTTTAGCTTCTTAATGAATATGGATTTAAAACTCTCCAGTTCTTATTTTATGAAATGACTTG
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CTGTTTAATCACACCTACCCCTGGAAAAGAGGTAAACCTTTTGAACAGTTGAATTTCATCAGAAGCTCTA
TAGCTTTTTGGTGAGAGGAAGTGATACTCTTTATTACAAGAAACAAGGAATTAACAAAAATAAGGAACTT
GTTGCTACCTTTCTTTTCTGTTGAACATTAAAAATAATTTGGTTTGTGGACTGGGCATGGTGGCTTATGG
AAAAGAGGTGAGCCTTTGTGAAGAACATAATGGAAAAGTGCATGTACGGGAAATAACAGCACTTTAGGTG
TAGTTCAGGTGAGCAAGATGCCAGATAGTTTCTACTAACCATATACTCTTTCTGCTTCTTAGCGTCGTTG
TCACTGCTTCTGTGGCTTTTGGGTTCAAAGTAGATTATATATATTCCTAAAGCTTGGTGGAGGTAGTGGA
GCTATCACACACTCAAATGAGCTTGTATATTTATAATACAATATAGTATTATAGTAACAGTTATCATACA
CTGAGATATAGTTTCTGTGCTTGTAAAAAATTCTTGAAGTGAGGCCGGGTGCGGTGGCTCATGCCTGTAA
TCCCAACGCTTTGGGACGCCGAGGCGGGTGGATCATGAGGTCAAAAGATTGAGACCATCCTGGCCAACAT
GGTGAAACCCCGTCTCTACTAGAAATACAAAAATTAGCTGGGTGTGGGGGCGTGCGCCTGTAGTCCCAGC
TACTTGGGAGGCTGAGGCAGGAGAATTGCTTGAACCCAGGAGGCAGAGGTTGCAGTAAGCCAAGATCGTG
CCACTGCACTCCAGCCTTGGTGACAGAGCAAGACTCCGTCTCCAAAAAAAAGAAAAAAAAATTTCTTGAA
GTGGAATGATGTACACCAAATACCCACTTTATAGATAATTACAGGGATGGTGAAAAATTAGTAAATAGCT
TCACAAAAGAGGCAGCGTGATACCTTTAGTGTTTTCTAGAATTTTTTTCTGGATATTATATGTAAGAATT
TATTGTTTGAGGGAAATACGAGGTTTCATTGTTTAAAATCAAGAGTGTATTGTTTACTGTTTTACAGTAT
TATTTAATATCCAACTGGTCTCCAATCTCCATTACATAGGGACTGTACAGAGCCTGTGAAGATGTATGAA
TGGTATCTTTTATAATCCACAATACATTTCTGGTAAGATGGAATCACTACCTCTGATCACATATCAGGCT
AACGATAGAGAAAGCATTAGGTTTAAGTTGAGGGCTAAGAACTGGTTTGTTTAAAGGTTTAAATTGTTGG
AAAAGAGCATTTGCCTTCTTTAATGAATGATGGTCTCACAATTCAGTTGACTTTGACCTAATATTTAAGA
ATATAAATATATTTTATTTGAAAGGCTGAGTGTTGCCCTCAAACACTTGCTAAAACTGCCTAGGGCAGGA
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CCCCAGTGATTCCAGTGCACCTGAAAAGTTGAGAACCACAGGCTTAGTGGTACGTTGGGGTTTCCCACGC
TAGGCTGCATAGCATTATAACCTGGGAACGTTAAAAAAAATACAGACATTCTTGATTCTTAGTGTAGTTA
TGATGTGGATCCTACGTATTTTTTAAAAAACTCTCCAGATAATTTTGATGTCACGAGTCACTAGTATAGA
CTATCTATCTAGTGAGTTGTATTATAAGCGATGAAATACTTTAAAAGCTACCCAGTAACTCTTGGGATTA
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GCAATAGATATAGAATTCTGGTTAGCTAATTGAACTTTTGTGACCTGTTTAAAAACAAATCAAATTTATT
GAACAAATGACAAGGCACAAATACTTTCAGAATTGTATTTCAGTTTTGAAGATTTAAAAAAAATGTGAAA
ATAAATATATATTAGTATCCTTTATTTTAAAGTCAATTGTAAACCAGAAGTACTGTTATTTTGGTGATTA
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CAAACAAACAACGTATCTTACTGACTAAACTGGAATGGAAATCTTATAATTAGTGTTGTATTTTTCTGCA
AAATAGTGAATTGGTACTTGGGAGACTTCTGTTCTTTGTTGAAAAAAATTGCCTTTGTTTTGAAATTATA
TCACCTGTGTTCTGAACTAAAATGATGGGAGTTCATTTTGTATTTACTTTGCAGTAAGTATCATGTAAAT
ATGCAAATGTTGAATGTAATATAGATTGTGTCCTCATTGAGTTTTTGGGTGATACAGTTTATAAATATAA
CAATTATACACCCTCATACATTCGTCTGATTTTGAGCTTGTAAGATGAGAACTGCTTTTTACACATTTAA
TAAAAAAAGGTCTCTGAATTAAAAAAAAAAAAAAAAAA

NM_001800.3| Homo sapiens cyclin-dependent kinase inhibitor 2D (p19, inhibits CDK4) (CDKN2D), transcript variant 1, mRNA
GGAGGGAGGGTGAGTTAGGGGGAGACCCGGCCCCCAAGGGGCGGGCGCCGGGCAGGGCCCCGCGGGCGGC
CGAGGGTTGGGCCCGGCTCCCAGCCCCTCGCCGTCCTCCGGCTGACAGGGGGAGGAGCCCGCCGGGAGGG
CCGGGGTCTCGGGCTGGGGAGCCGGGACGGGAGAGCAGCGCAGCCGGGTGCACCGCGGCCGCGCCCCGGG
AGGGCTGTTCGGGCCAGCGCCCGCCGGCTGCTCCGCGCTGACAGCGCCGGGCTGGGGCGGGGCGGGGGGC
TTTGCAGGCCGCCAGTGTCGACATGCTGCTGGAGGAGGTTCGCGCCGGCGACCGGCTGAGTGGGGCGGCG
GCCCGGGGCGACGTGCAGGAGGTGCGCCGCCTTCTGCACCGCGAGCTGGTGCATCCCGACGCCCTCAACC
GCTTCGGCAAGACGGCGCTGCAGGTCATGATGTTTGGCAGCACCGCCATCGCCCTGGAGCTGCTGAAGCA
AGGTGCCAGCCCCAATGTCCAGGACACCTCCGGTACCAGTCCAGTCCATGACGCAGCCCGCACTGGATTC
CTGGACACCCTGAAGGTCCTAGTGGAGCACGGGGCTGATGTCAACGTGCCTGATGGCACCGGGGCACTTC

CAATCCATCTGGCAGTTCAAGAGGGTCACACTGCTGTGGTCAGCTTTCTGGCAGCTGAATCTGATCTCCA
TCGCAGGGACGCCAGGGGTCTCACACCCTTGGAGCTGGCACTGCAGAGAGGGGCTCAGGACCTCGTGGAC
ATCCTGCAGGGCCACATGGTGGCCCCGCTGTGATCTGGGGTCACCCTCTCCAGCAAGAGAACCCCGTGGG
GTTATGTATCAGAAGAGAGGGGAAGAAACACTTTCTCTTCTTGTTTCTCCTGCCCACTGCTGCAGTAGGG
GAGGAGCACAGTTTGTGGCTTATAGGTGTTGGTTTTGGGGGTGTGAGTGTTTGGGGGACGTTTCTCATTT
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AGTTTCTTCTGCGCCTCAGGCTGCTGGGGCCTCAGACGAGACCCAAGGGCAGAGCATTTAAGAGTGAAGT
CATGACCTCCAGGGAGCCTAGAAGCTGGTGGCCTTGGCCGGCTGTGCTCAGAGACCTGAAGTGTGCACGT
TGCTTCAGGCATGGGGGGTGGGGGGAGCGTCCCAAATCAATAAGAAGGTAGAATGAGTTATGAGTTATTC
ATATTCTGTTGGAAGCTTGTTTTCCAGTCTCTTGTACAGCGTTTTAAAAGAAATGGATTCTATTTATTAT
GCTTTATTGGAAAAAATGTTGTAATAATTTAATGTTTTTACCCATTAAATTAAGACTTGTGCATGATCAA
AAAAAAAAAAAAAAA

NM_002452.3| Homo sapiens nudix (nucleoside diphosphate linked moiety X)-type motif 1 (NUDT1), transcript variant 1, mRNA
GGTCAGAGGCCACGCCCCCGGAAGCGGCGGTGCAGAACCCAGGGACCATGGGCGCCTCCAGGCTCTATAC
CCTGGTGCTGGTCCTGCAGCCTCAGCGAGTTCTCCTGGGCATGAAAAAGCGAGGCTTCGGGGCCGGCCGG
TGGAATGGCTTTGGGGGCAAAGTGCAAGAAGGAGAGACCATCGAGGATGGGGCTAGGAGGGAGCTGCAGG
AGGAGAGCGGTCTGACAGTGGACGCCCTGCACAAGGTGGGCCAGATCGTGTTTGAGTTCGTGGGCGAGCC
TGAGCTCATGGACGTGCATGTCTTCTGCACAGACAGCATCCAGGGGACCCCCGTGGAGAGCGACGAAATG
CGCCCATGCTGGTTCCAGCTGGATCAGATCCCCTTCAAGGACATGTGGCCCGACGACAGCTACTGGTTTC
CACTCCTGCTTCAGAAGAAGAAATTCCACGGGTACTTCAAGTTCCAGGGTCAGGACACCATCCTGGACTA
CACACTCCGCGAGGTGGACACGGTCTAGCGGGAGCCCAGGGCAGCCCCTGGGCAGGAGACGTGGCTGCTG
AACAGCCGCAAACCATCTTCACCTGGGGGCATTGAGTGGCGCAGAGCCGGGTTTCATCTGGAATTAACTG
GATGGAAGGGAAAATAAAGCTATCTAGCGGTGAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_003247.2| Homo sapiens thrombospondin 2 (THBS2), mRNA
GAGGAGGAGACGGCATCCAGTACAGAGGGGCTGGACTTGGACCCCTGCAGCAGCCCTGCACAGGAGAAGC
GGCATATAAAGCCGCGCTGCCCGGGAGCCGCTCGGCCACGTCCACCGGAGCATCCTGCACTGCAGGGCCG
GTCTCTCGCTCCAGCAGAGCCTGCGCCTTTCTGACTCGGTCCGGAACACTGAAACCAGTCATCACTGCAT
CTTTTTGGCAAACCAGGAGCTCAGCTGCAGGAGGCAGGATGGTCTGGAGGCTGGTCCTGCTGGCTCTGTG
GGTGTGGCCCAGCACGCAAGCTGGTCACCAGGACAAAGACACGACCTTCGACCTTTTCAGTATCAGCAAC
ATCAACCGCAAGACCATTGGCGCCAAGCAGTTCCGCGGGCCCGACCCCGGCGTGCCGGCTTACCGCTTCG
TGCGCTTTGACTACATCCCACCGGTGAACGCAGATGACCTCAGCAAGATCACCAAGATCATGCGGCAGAA
GGAGGGCTTCTTCCTCACGGCCCAGCTCAAGCAGGACGGCAAGTCCAGGGGCACGCTGTTGGCTCTGGAG
GGCCCCGGTCTCTCCCAGAGGCAGTTCGAGATCGTCTCCAACGGCCCCGCGGACACGCTGGATCTCACCT
ACTGGATTGACGGCACCCGGCATGTGGTCTCCCTGGAGGACGTCGGCCTGGCTGACTCGCAGTGGAAGAA
CGTCACCGTGCAGGTGGCTGGCGAGACCTACAGCTTGCACGTGGGCTGCGACCTCATAGACAGCTTCGCT
CTGGACGAGCCCTTCTACGAGCACCTGCAGGCGGAAAAGAGCCGGATGTACGTGGCCAAAGGCTCTGCCA
GAGAGAGTCACTTCAGGGGTTTGCTTCAGAACGTCCACCTAGTGTTTGAAAACTCTGTGGAAGATATTCT
AAGCAAGAAGGGTTGCCAGCAAGGCCAGGGAGCTGAGATCAACGCCATCAGTGAGAACACAGAGACGCTG
CGCCTGGGTCCGCATGTCACCACCGAGTACGTGGGCCCCAGCTCGGAGAGGAGGCCCGAGGTGTGCGAAC
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GCCCCGTGGATGGCTGTTTATCCAACCCCTGCTTCCCGGGAGCCCAGTGCAGCAGCTTCCCCGATGGGTC
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GGACTTGGACGGTGATGGACGGGGTGATATTTGTAAAGATGATTTTGACAATGACAACATCCCAGATATT
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AGCCAACTCGGACCCCGGCATCGCTGTAGGTTTTGACGAGTTTGGGTCTGTGGACTTCAGTGGCACATTC
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ACGGGGAACACGCCGGGGCAGGTGCGAACCTTATGGCACGACCCCAGGAACATTGGCTGGAAGGACTACA
CGGCCTATAGGTGGCACCTGACTCACAGGCCCAAGACTGGCTACATCAGAGTCTTAGTGCATGAAGGAAA
ACAGGTCATGGCAGACTCAGGACCTATCTATGACCAAACCTACGCTGGCGGGCGGCTGGGTCTATTTGTC
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TCTCTCTAGCAGCACCTCCTGTCCCTTGACCTTAACTCTGATGGTTCTTCACCTCCTGCCAGCAACCCCA
AACCCAAGTGCCTTCAGAGGATAAATATCAATGGAACTCAGAGATGAACATCTAACCCACTAGAGGAAAC
CAGTTTGGTGATATATGAGACTTTATGTGGAGTGAAAATTGGGCATGCCATTACATTGCTTTTTCTTGTT
TGTTTAAAAAGAATGACGTTTACATATAAAATGTAATTACTTATTGTATTTATGTGTATATGGAGTTGAA
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AAAGTTGTCACTATTCTTGAATTAGAGTTGCTCTACAATGACACACAAATCCCATTAAATAAATTATAAA
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GTGACGCGGTTCCAGATGTGGATTTGGCAAAACCTCATTTAAGTAAAAGGTTAGCAGAGCAAAGTGCGGT
GCTTTAGCTGCTGCTTGTGCCGCTGTGGCGTCGGGGAGGCTCCTGCCTGAGCTTCCTTCCCCAGCTTTGC
TGCCTGAGAGGAACCAGAGCAGACGCACAGGCCGGAAAAGGCGCATCTAACGCGTATCTAGGCTTTGGTA
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TTAGCTAGAACCTCATTTTTAAAGATTAACAACAGGAAATAAATTGTAAAAAAGGTTTTCTATACATGAA
AAAAAAAAAAAAAAAA

NM_000224.2| Homo sapiens keratin 18 (KRT18), transcript variant 1, mRNA
TCCGGGGCGGGGGCGGGGCCTCACTCTGCGATATAACTCGGGTCGCGCGGCTCGCGCAGGCCGCCACCGT
CGTCCGCAAAGCCTGAGTCCTGTCCTTTCTCTCTCCCCGGACAGCATGAGCTTCACCACTCGCTCCACCT
TCTCCACCAACTACCGGTCCCTGGGCTCTGTCCAGGCGCCCAGCTACGGCGCCCGGCCGGTCAGCAGCGC
GGCCAGCGTCTATGCAGGCGCTGGGGGCTCTGGTTCCCGGATCTCCGTGTCCCGCTCCACCAGCTTCAGG
GGCGGCATGGGGTCCGGGGGCCTGGCCACCGGGATAGCCGGGGGTCTGGCAGGAATGGGAGGCATCCAGA
ACGAGAAGGAGACCATGCAAAGCCTGAACGACCGCCTGGCCTCTTACCTGGACAGAGTGAGGAGCCTGGA
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GAGCAGCTCAACGGGATCCTGCTGCACCTTGAGTCAGAGCTGGCACAGACCCGGGCAGAGGGACAGCGCC
AGGCCCAGGAGTATGAGGCCCTGCTGAACATCAAGGTCAAGCTGGAGGCTGAGATCGCCACCTACCGCCG
CCTGCTGGAAGATGGCGAGGACTTTAATCTTGGTGATGCCTTGGACAGCAGCAACTCCATGCAAACCATC
CAAAAGACCACCACCCGCCGGATAGTGGATGGCAAAGTGGTGTCTGAGACCAATGACACCAAAGTTCTGA
GGCATTAAGCCAGCAGAAGCAGGGTACCCTTTGGGGAGCAGGAGGCCAATAAAAAGTTCAGAGTTCAAAA
AAAAAAAAAAAAAAA

NM_007263.3| Homo sapiens coatomer protein complex, subunit epsilon (COPE), transcript variant 1, mRNA
GACGTGTCTTTCAGGAAGAGGAGCTGGTGAGAAGACAGCGAAATGGCGCCTCCGGCCCCCGGCCCGGCCT
CCGGCGGCTCCGGGGAGGTAGACGAGCTGTTCGACGTAAAGAACGCCTTCTACATCGGCAGCTACCAGCA
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AGCTCCAGGCCGTGCGCATGTTTGCTGACTACCTCGCCCACGAGAGTCGGAGGGACAGCATCGTGGCCGA
GCTGGACCGAGAGATGAGCAGGAGCGTGGACGTGACCAACACCACCTTCCTGCTCATGGCCGCCTCCATC
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CCATGACAGTGCAGATCCTGCTGAAGCTGGACCGCCTGGACCTCGCCCGGAAGGAGCTGAAGAGAATGCA
GGACCTGGACGAGGATGCCACCCTCACCCAGCTCGCCACTGCCTGGGTCAGCCTGGCCACGGGTGGTGAG
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AGACAAGGATAGTGGCTACCCAGAGACGCTGGTCAACCTCATCGTCCTGTCCCAGCACCTGGGCAAGCCC
CCTGAGGTGACAAACCGATACCTGTCCCAGCTGAAGGATGCCCACAGGTCCCATCCCTTCATCAAGGAGT
ACCAGGCCAAGGAGAACGACTTTGACAGGCTGGTGCTACAGTACGCTCCCAGCGCCTGAGGCTGGCCCAG
AGCTGTCAGGACCATGAAGCCAGGACAGAGGCCAGGAGCCAGCCCTGCAGCCCTCCCCACCCGGCATCCA
CCTGCATCCCCTCTGGGGGCAGGAGCCCACCCCCAGCACCCCCATCTGTTAATAAATATCTCAACTCCAG
GGTGTTCCACCTGA

NM_004637.5| Homo sapiens RAB7A, member RAS oncogene family (RAB7A), mRNA
ACTTCCGCTCGGGGCGGCGGCGGTGGCGGAAGTGGGAGCGGGCCTGGAGTCTTGGCCATAAAGCCTGAGG
CGGCGGCAGCGGCGGAGTTGGCGGCTTGGAGAGCTCGGGAGAGTTCCCTGGAACCAGAACTTGGACCTTC
TCGCTTCTGTCCTCCGTTTAGTCTCCTCCTCGGCGGGAGCCCTCGCGACGCGCCCGGCCCGGAGCCCCCA
GCGCAGCGGCCGCGTTTGAAGGATGACCTCTAGGAAGAAAGTGTTGCTGAAGGTTATCATCCTGGGAGAT
TCTGGAGTCGGGAAGACATCACTCATGAACCAGTATGTGAATAAGAAATTCAGCAATCAGTACAAAGCCA
CAATAGGAGCTGACTTTCTGACCAAGGAGGTGATGGTGGATGACAGGCTAGTCACAATGCAGATATGGGA
CACAGCAGGACAGGAACGGTTCCAGTCTCTCGGTGTGGCCTTCTACAGAGGTGCAGACTGCTGCGTTCTG
GTATTTGATGTGACTGCCCCCAACACATTCAAAACCCTAGATAGCTGGAGAGATGAGTTTCTCATCCAGG
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CCTTCAACACAATTCCCCTCTCCTCTTCCAAACAAAACATACATTGATCTCTCACATCCAGCTGCCAAAA
GAAAACCCCATCAAACACAGTTACACCCCACATATCTCTCACACACACACACACACGCACACACACACAC
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AAAATTAAAATTCTCATTTTCTTTCTTTTTTTTCCCCCCTGCTCCACACTTCAAAACTCCCGTTAGATCA
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GGCGCTCCTTTCAGGGTTGTGGTATTCTTAGGTTAGCGGAGCTTTTTCCTCTTTTCCCCACCCATCTCCC
CAATATTGCCCATTATTAATTAACCTCTTTCTTTGGTTGGAACCCTGGCAGTTCTGCTCCCTTCCTAGGA
TCTGCCCCTGCATTGTAGCTTGCTTAACGGAGCACTTCTCCTTTTTTCCAAAGGTCTACATTCTAGGGTGT
GGGCTGAGTTCTTCTGTAAAGAGATGAACGCAATGCCAATAAAATTGAACAAGAACAATGATAAAAAAAA

XM_371237.1| PREDICTED: Homo sapiens cation channel, sperm associated 4 (CATSPER4), mRNA
ATGAGGGATAATGAAAAGGCCTGGTGGCAGCAATGGACCTCCCATACAGGCCTCGAGGGGTGGGGCGGGA
CTCAGGAGGACCGTATGGGGTTTGGAGGGGCAGTAGCTGCACTGAGGGGCCGCCCCTCTCCCCTGCAGAG
TACCATTCACGAGTCCTACGGTCGGCCAGAGGAGCAAGTGCTCATCAACCGCCAGGAAATCACGAACAAA
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GGACCAGAAACACTATGAGTTGTTCTCTACCATAGATGACATTGTGCTGACCATCCTTCTTTGTGAGGTT
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CACTCTTTGGTGCATTCGTGCCCAAGCATTTCCAGAACATACAGGTTGCGCTGTACACCCTCTTCATCTG
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ACTCAACATGATTGTGGAGGAGGTGCGCGCAATCCGCTTCAACCAGGAGCAGGAGTCAGAGGTGTTGAAC
AGGCGCTCGTCGACGAGCGGGTCGTTGGAGACTACGTCATCCAAGGACATCCGCCAGATGTCTCAACAGC
AAGACTTGCTCAGTGCGCTCGTTAGCATGGAAAAGGTTCATGACTCTAGCTCACAAATACTCCTTAAAAA
ACACAAGAGCAGCCACTGA

XM_372810.1| Homo sapiens plexin A2 (PLXNA2), mRNA
ATGGAGAAACAACTGAACCTTGAGGACTCTCAGCAGATAAACCAGATGAAGTGGATAAAGACTCGCTGTG
TGGAGAAGGCAGGGCAGCCCACGCAGAAAGGGAGAGGGGCCTCGCGCCGCCCTCAGCAGCCGGCGGCGGC
CGAGGTAGACCGAGCGGGGACGGAAGGACAGACCGACGTCGCCGAGCTGGAATCATGTGAGGGCCAACCG
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GACAAATGCCAACAGGCCTGGGAACCTAATCGATTTGCTGCCAGCATCAGCCAGTGTGTGAGCCTTGCAG
TGCATCCCAGCAGCATCTCAGTATCTGAGCACAGCCGGTTGGGAGCTGCTAGAGAACAGAGAGCCCACAT

GGAACAGAGAAGCACAAACTCTAGGGAACACGCAGCCTTTAGAAGCAGCCTCTTGATGAATGACAGCCAA
GGTGAGGAGCTAGCTATCTTCCACCAACCTAGTGAGCTCCTGGTAAACCTCCAGCTTAGCCTGGTAGTGA
GTGATGCTCCTGATCTATCTGCGGGTATCGCCTGTGCCTTTGGGAACCTGACAGAGGTGGAGGGGCAGGT
GTCCGGGAGCCAGGTCATCTGCATCTCACCTGGGCCCAAGGATGTCCCTGTCATCCCGCTGGATCAAGAC
TGGTTTGGGCTGGAGCTACAGCTGAGGTCCAAGGAGACAGGGAAGATATTTGTCAGCACCGAGTTCAAGT
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NM_014927.2| Homo sapiens connector enhancer of kinase suppressor of Ras
2 (CNKSR2), mRNA
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XM_165973.6| Homo sapiens ubiquitin specific protease 24 (USP24), mRNA
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XM_375851.1| PREDICTED: Homo sapiens KIAA0133 gene product (KIAA0133), mRNA
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NM_002894.2| Homo sapiens retinoblastoma binding protein 8 (RBBP8), transcript variant 1, mRNA

GACGTCGCGCGGGCTCCCGGGCGGGGCGGGTCCGGCCGCCTCCGAGCCCGGCCGGCAGCCCCCGGCCTTA
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CAAAGACTAGAAGAATTCTTCACCAAAAATCAACAGCTGAGGGAACAGCAGAAAGTCCTTCATGAAACCA
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TGAAAATGTTTGTGATATTTTGCTTTTGCACCTTTAAAACAATAAGGCGCTTTCATTTTGCACTCTAACT
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NM_006997.2| Homo sapiens transforming, acidic coiled-coil containing protein 2 (TACC2), transcript variant 4, mRNA
CCGGGGGGTTTAAGAGAAGAGCCTTTCGGACCACACCGGCGCTCACGCTCATACCCGCACGCCCCGGGCA
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CCCAGCCTTATTGGAGGAGACGCCCCTTGAGCCCGCTGTGGGGCCCAAAGCTGCCTGCCCTCTGGACTCA
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CAGACACCTGTGATGAGTCCGTTGACCCCTTTAAGACATCCTCTAAGACCCCCAGCTCACCTTCTAAATC
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CTCTCTCTGATCCACCTTCCCAGGACCCCACCCCAGCTGCTACACCAGAAACACCACCAGTGATCTCTGC
GGTGGTCCACGCCACAGATGAGGAAAAGCTGGCGGTCACCAACCAGAAGTGGACGTGCATGACAGTGGAC
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GTTCACCCACTGAGGAGTTGGATTACAGAAACTCCTATGAAATTGAATATATGGAGAAAATTGGCTCCTC
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CCTGTCAAGTCATCTCCCGTCCGCATGTCAGAGTCCCCGACGCCGTGTTCAGGGTCAAGTTTTGAAGAGA
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GCCCGACCTGGACTCTGCCCTCCAGATCGCCAGAGCAGAGATCATAACCAAGGAGAGAGAGGTCTCAGAA
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TCTTGCCTTAAGGAGTGTAAACTTGATCTGCATTTGCTGATTTGTTTTTAAAAAAACAAGAAATGCATGT
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NM_005441.2| Homo sapiens chromatin assembly factor 1, subunit B (p60) (CHAF1B), mRNA
CGGGAAGCGGCGCGCGCTGCGCGGGAGGTGACGGTGCCTCTGACTGTCCGGGTCCCTCCAGCATTTTGCA
GCTTTCTCCTGTCTTGAAGAAGTAGAACGGTGCCCGAGAAACGTTTTTCCCCTTCGAGACTCAGGAGGAT
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ACGGCTGGGAGGATCCACAGACTGGCGTCTGCCGGCGTGGACACCAATGTCAGGATCTGGAAGGTAGAAA
AGGGACCAGATGGAAAAGCCATCGTGGAATTTTTGTCCAATCTTGCTCGTCATACCAAAGCCGTCAATGT
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GTGAATGATAACAAGGAGCCGGAGCAGATCGCTTTTCAGGATGAGGACGAGGCCCAGCTGAACAAGGAGA
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CAGCAGAACCCAAGACCCCAGCAGCCCCGGCACGACTCCCCCTCAGGCCAGACAGGCCCCAGCCCCAACA
GTCATCAGGGACCCTCCCTCCATCACTCCTGCTGTCAAAAGCCCCTTGCCGGGGCCTTCGGAGGAGAAGA
CCCTGCAGCCCAGTAGTCAAAACACAAAAGCCCACCCATCCCGGAGGGTCACTCTGAACACACTGCAAGC
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CCAACCAGTGTGATTTCCACCCCTTCTACAGAAGAAATTCAGTCAGAGACGCCTGGAGACGCTCAGGGCA
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AGTGAGTCCTCCCTGGCATCCTCGTGAAAGTGCACACACTTCATGGAGGGACTCCTTTTCAATAAGAATT
AGGAAGATGAGAAAGTAATTTGAGATTTTACTCTGTCGAATTTTAGAGTATTTGAAGTGATTGTTAGATT
TCACTTCTAAGGAGTTGATTGATTAAACTTTGGAAGGTAAAAAAAAAAAAAAAAAAA

NM_007264.3| Homo sapiens G protein-coupled receptor 182 (GPR182), mRNA
CAGCCTCCTCACAGCTCCCCATAGCCTGGACCTGCCGGCCCTCCCTCCAGGACCGAGGGGCTCCCAAGGG
AAACTCAGGCGTGTGCTGGTCCCAATGTCAGTGAAACCCAGCTGGGGGCCTGGCCCCTCGGAGGGGGTCA
CCGCAGTGCCTACCAGTGACCTTGGAGAGATCCACAACTGGACCGAGCTGCTTGACCTCTTCAACCACAC
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ATGTTTGTGGTTGGGCTGGTGGAGAACCTCCTGGTGATATGCGTCAACTGGCGCGGCTCAGGCCGGGCAG
GGCTGATGAACCTCTACATCCTCAACATGGCCATCGCGGACCTGGGCATTGTCCTGTCTCTGCCCGTGTG
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AAAAAAAAAA

NM_001277.2| Homo sapiens choline kinase alpha (CHKA), transcript variant 1, mRNA
GGCAGAGGAGCGAGTGCAGCGGCCAGCAGCACATCCCCGCTCCACAGTCGCCGCAGTCGCCGCAGCCGCC
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AATGTCCTGCGGGATACAGTCTTAGCTGACTTTTTTTTACTCTGAACTCTTATTTGAATTGTTTTTTGTG
CATATATTTCTGCTACCACAGAGATTGTACTATACAAATAAAAAAATAAAAACCCAAAAAAAAAAAAAAA
AAA

NM_003150.3| Homo sapiens signal transducer and activator of transcription 3 (acute-phase response factor) (STAT3), transcript variant 2, mRNA
GGTTTCCGGAGCTGCGGCGGCGCAGACTGGGAGGGGGAGCCGGGGGTTCCGACGTCGCAGCCGAGGGAAC
AAGCCCCAACCGGATCCTGGACAGGCACCCCGGCTTGGCGCTGTCTCTCCCCCTCGGCTCGGAGAGGCCC
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CTACAGTGACAGCTTCCCAATGGAGCTGCGGCAGTTTCTGGCCCCTTGGATTGAGAGTCAAGATTGGGCA
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GCAGAAAACTCTCACGGACGAGGAGCTGGCTGACTGGAAGAGGCGGCAACAGATTGCCTGCATTGGAGGC
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CCTGGTGTGAATTATTCAGGGTGTCAGATCACATGGGCTAAATTTTGCAAAGAAAACATGGCTGGCAAGG
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NM_017431.2| Homo sapiens protein kinase, AMP-activated, gamma 3 non-catalytic subunit (PRKAG3), mRNA

TTGGTCTGGGGCTGGCCACATGGAGCCCGGGCTGGAGCACGCACTGCGCAGGACCCCTTCCTGGAGCAGC
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NM_002737.2| Homo sapiens protein kinase C, alpha (PRKCA), mRNA
GGCCGCAGCTCCCCGGCGGAGGCAAGAGGTGGTTGGGGGGGACCATGGCTGACGTTTTCCCGGGCAACGA
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NM_006116.2| Homo sapiens mitogen-activated protein kinase kinase kinase 7 interacting protein 1 (MAP3K7IP1), transcript variant alpha, mRNA
ACTCGCTGCTCTGCGGGGAGGCGGGCGCTCCCGCAGGGGTTCCTCCAAGATGGCGGCGCAGAGGAGGAGC
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NM_005060.3| Homo sapiens RAR-related orphan receptor C (RORC),
transcript variant 1, mRNA
GCCAGGTGCTCCCGCCTTCCACCCTCCGCCCTCCTCCCTCCCCTGGGCCCTGCTCCCTGCCCTCCTGGGC
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GCCAGCGCTGTAACGCGGCCTACTCCTGCACCCGTCAGCAGAACTGCCCCATCGACCGCACCAGCCGAAA
CCGATGCCAGCACTGCCGCCTGCAGAAATGCCTGGCGCTGGGCATGTCCCGAGATGCTGTCAAGTTCGGC
CGCATGTCCAAGAAGCAGAGGGACAGCCTGCATGCAGAAGTGCAGAAACAGCTGCAGCAGCGGCAACAGC
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GGCCTCCTGAAAGCCTCAGGCTCTGGGCCCTCATATTCCAACAACTTGGCCAAGGCAGGGCTCAATGGGG
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CAGCCAGCTGACCCCTGACCGATGTGGACTTCGTTTTGAGGAACACAGGCATCCTGGGCTTGGGGAACTG
GGACAGGGCCCAGACAGCTACGGCAGCCCCAGTTTCCGCAGCACACCGGAGGCACCCTATGCCTCCCTGA
CAGAGATAGAGCACCTGGTGCAGAGCGTCTGCAAGTCCTACAGGGAGACATGCCAGCTGCGGCTGGAGGA
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CATGAACCCTGGAGGGTGGTCCCCACCAGCTCTTTGGAAGTGAGCAGATGCTGCGGCTGGCTTTCTGTCA
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CAATGATGGGCAGTCGTGGAGTTGGGGGGGTTGGGGGTGGGATTGTGTCCTCTAAGGGGACGGGTTCATC
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AAAA

NM_173537.2| Homo sapiens GTF2I repeat domain containing 2 (GTF2IRD2), mRNA
GAAAGAAAGAGAAAAAGGAGGGCGAGTGGCGAGCAGGGGCCTCGGCCGCCACCCACACGCCCCGAAGCGT
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GCCTGTTGAAGAAGAGTCCTCCTCAGAGACCAGGATGGTGGTGACATTCCTCGTGTCTGCCCTCGAATCC
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TATTACATTGTGTGCCTCACTATTCATTAAAAATCAAGAAAGTTTTATTGT

NM_000454.4| Homo sapiens superoxide dismutase 1, soluble (SOD1), mRNA
GTTTGGGGCCAGAGTGGGCGAGGCGCGGAGGTCTGGCCTATAAAGTAGTCGCGGAGACGGGGTGCTGGTT
TGCGTCGTAGTCTCCTGCAGCGTCTGGGGTTTCCGTTGCAGTCCTCGGAACCAGGACCTCGGCGTGGCCT
AGCGAGTTATGGCGACGAAGGCCGTGTGCGTGCTGAAGGGCGACGGCCCAGTGCAGGGCATCATCAATTT
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GTAATCTTAAAAGTGTAATTGTGTGACTTTTTCAGAGTTGCTTTAAAGTACCTGTAGTGAGAAACTGATT
TATGATCACTTGGAAGATTTGTATAGTTTTATAAAACTCAGTTAAAATGTCTGTTTCAATGACCTGTATT
TTGCCAGACTTAAATCACAGATGGGTATTAAACTTGTCAGAATTTCTTTGTCATTCAAGCCTGTGAATAA
AAACCCTGTATGGCACTTATTATGAGGCTATTAAAAGAATCCAAATTCAAACTAAAAAAAAAAAAAAAAA
A

NM_033547.3| Homo sapiens integrator complex subunit 4 (INTS4), mRNA
CGGAAGCTGAGAGGGCCCGCGGGTAGGCATGGCGGCGCACCTTAAGAAGCGGGTTTATGAGGAATTCACG
AAAGTGGTTCAGCCACAGGAGGAAATTGCTACTAAGAAACTCCGACTAACAAAACCAAGTAAATCTGCAG
CACTCCACATAGATCTGTGTAAAGCTACCTCCCCAGCAGATGCTTTGCAATACTTGCTCCAGTTTGCCAG
GAAGCCTGTCGAGGCGGAAAGCGTAGAGGGAGTAGTCAGGATTCTCTTGGAACATTATTACAAGGAGAAT
GATCCATCTGTGAGACTGAAAATTGCATCATTGTTGGGTTTATTATCAAAGACAGCAGGATTTTCACCAG
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CCCCGACTCATGACATCCAAACCTGCAGAAGTGGTCAAAATTCTACAGACCATGCTGCGACAGAGTGCCT
TTCTGCATCTCCCGCTTCCAGAGCAGATCCACAAAGCCTCAGCCACCATCATCGAGCCAGCGGGCGAGTC
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CCGTGGCCTAGAGGGCCCTTCTTAGGTGTCAGAATGAGCCAAGCCTGAAGCACTTCACCTGGAATTGATG
TGTAGGCTTAAGGAGTATGTGACCCTTACAGTCTCATCTGGTATCAAACACAGGATAAATTGTTTCTTCA
TTAAAAAATAAAAAACCTTCAAGTCTACTTACCCTTCTCCTGTCCACAATAAAGTTGAGAAAACACCAAA
AAAAAAAAAAAA

NM_002626.4| Homo sapiens phosphofructokinase, liver (PFKL), transcript variant 2, mRNA
GACGGCGACGCGGCGCAGGCGGCGGGAGTGCGAGCTGGGCCCGTGTTTCGGCCGCCGCCATGGCCGCGGT
GGACCTGGAGAAGCTGCGGGCGTCGGGCGCGGGCAAGGCCATCGGCGTCCTGACCAGCGGCGGCGACGCG
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TCTACGAGGGCTATGAGGGCCTCGTGGAGGGAGGTGAGAACATCAAGCAGGCCAACTGGCTGAGCGTCTC
CAACATCATCCAGCTGGGCGGCACTATCATTGGCAGCGCTCGCTGCAAGGCCTTTACCACCAGGGAGGGG
CGCCGGGCAGCGGCCTACAACCTGGTCCAGCACGGCATCACCAACCTGTGCGTCATCGGCGGGGATGGCA
GCCTCACAGGTGCCAACATCTTCCGCAGCGAGTGGGGCAGCCTGCTGGAGGAGCTGGTGGCGGAAGGTAA
GATCTCAGAGACTACAGCCCGGACCTACTCGCACCTGAACATCGCGGGCCTAGTGGGCTCCATCGATAAC
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AAATAAAATCACCCTGACTGTGGGGTGCATCGGTCTCCGGAGAAAACAAAAAAA

NM_079834.2| Homo sapiens secretory carrier membrane protein 4 (SCAMP4), mRNA

GGTGGGTTGGGCCGGGCCGGTTGCTAAGACTTGGCGAAGCGCTGCGCTCGCGCCCGGATCCCTCAGGCGG
CTGCAGGCTTCAGCCTGCGCTGGTTGGTGAAACAGAGATGTCAGAAAAGGAGAACAACTTCCCGCCACTG
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CTTAGAAAAAAAAAAAAAAAAAAAAAA

NM_016337.2| Homo sapiens Enah/Vasp-like (EVL), mRNA
GAGGGTCTGTGGTCCTCTGATCAACATAGGCTGGTGGGAGTACAGGACTCGCCTCCTCAGGGTTCCCTGT
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NM_013246.2| Homo sapiens cardiotrophin-like cytokine factor 1 (CLCF1), mRNA
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XM_371214.3| PREDICTED: Homo sapiens KIAA0450 gene product (KIAA0450), mRNA
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XM_046751.7| PREDICTED: Homo sapiens protein tyrosine phosphatase, receptor type, f polypeptide (PTPRF), interacting protein (liprin), alpha 4 (PPFIA4), mRNA

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XM_032397.5| PREDICTED: Homo sapiens DKFZP564I122 protein (DKFZP564I122), mRNA
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GGCAGCTCTCCTCATTTTCCCCAAAACAGGGAAAGCAAGGAGTAAATTCCTCTTAAAATCAAAAGCTAAT
AATATGCTTCCTAAAATAAAGACTCATCAAGGTCTCA

XM_373989.2| PREDICTED: Homo sapiens hypothetical LOC388959 (LOC388959), mRNA
ATGTCGTTTCAGTTCTCAGAGAACTTGGATCCGAGCGGAGGGTGGATGTTTGGGGTCCAACACATTGGTT
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TCCCCAAACTTTCCCACCCCACCTTCGAGGCCGCAGAAAGGCGCCGCTCCGAGGCCTAGGAGGGCGCGGG
CGGCGGTGGGGAGGCTGGGACCTGGACCTTGCCGGCGCACTGCGGAGAGGGAGCCGCAGAGAGTTCCGCT
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TTAAAGTCTATTTTCCACTCGACTGCTCCGAAAAGCCCCCGCGCGCCGACGCCGTTCCGGCCCCCTTGGG
CGAGGTGGGGAGGGGCGGCCAGGGGTGGGGAGGCGAGGCGGCCGCACCCAGCACTCCTCCGCGTCCTGGG
AACCCCGGGGCCTGGCACGGCTGA

XM_496642.1| PREDICTED: Homo sapiens ubiquitin specific protease 19 (USP19), mRNA
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CTGTGCCAAGGAGGAGATGGCAGTGGCAGCAGATGCTGCAACCTTGGTGGATGAGCCCGAGTCGATGGTG
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CAGCCTGCCAGCGGAAGCAACAGTCGGAGGATGAAAAGCTGAAGCGCTGTACCCGGTGCTACCGTGTGGG
CTACTGCAACCAGCTCTGCCAGAAAACCCACTGGCCTGACCACAAGGGCCTCTGCCGACCTGAGAACATT
GGCTACCCCTTCCTGGTCAGTGTACCTGCCTCACGCCTCACTTATGCCCGCCTCGCTCAGTTGCTAGAGG
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AGTGAAGCTATGAATGCCCACACACCCCAGTTCTTCATCTATAAAATTGATTCATCCAACCGAGAGCAGC
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CCTCTCAGGTTGGGACTTCTTGTCAGCTTGGACCCCTGACCAGTGGGCTTTGGCTTCTCCAGCCGCCTCC
AGTGCTGCGTGATTTGATTCTGTTGTACCTTCAATTCTTCTGACCCGCATTATAAACATTATAATTTTAT
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AATAAGTAAGTAGACTT

XM_209700.7| PREDICTED: Homo sapiens similar to FLJ00052 protein (LOC285647), mRNA
ATGGCCATGGCTGACATTGAGGAGGGAGCTGGCAGAGGGAATGATGGCCATGGCAGACGGCGAGTACCGA
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XM_044622.6| PREDICTED: Homo sapiens collagen, type XIV, alpha 1 (undulin) (COL14A1), mRNA
ATGCGGTACTGGTTGCTTCCACCTTTTTTGGCAATTGTTTATTTCTGCACCATTGTCCAAGGTCAAGTGG
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CTCCTTATCTACGGGGATTACTTTGTACATGCAGATAAGTTTTCGCAAACCTATTTCCATTTTCTTTTGT
AAGCAAATAAAACTTTAAAACAA

XM_058426.9| PREDICTED: Homo sapiens hypothetical protein FLJ00012 (FLJ00012), mRNA
ATGATTCACTTTCGAGGGTACCCACAAGAGATGTGTCTGCTCCTCCAGACGCCGCCCAATGGGGTGAAGC
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TCATCATTAGTGGCCACAATGACCAGAAGATCCGGTTCTGGGACAGCAGGGGGCCCCACTGCACCCAGGT
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CGAGACAACACACTCAAGGTCATCGACCTGCGTGTCAGCAACATCCGCCAGGTGTTCAGGGCCGATGGCT
TCAAGTGTGGTTCTGACTGGACCAAAGCTGTGTTCAGCCCGGACAGAAGCTATGCACTGGCAGGCTCCTG
TGATGGGGCCCTTTACATCTGGGATGTGGACACCGGGAAACTGGAGAGCAGACTACAGGGACCCCATTGC
GCTGCCGTCAACGCCGTGGCCTGGTGCTACTCCGGGAGCCACATGGTGAGCGTGGACCAGGGCAGGAAGG
TTGTGCTCTGGCAGTAGGGCCACGACCTGCCTGCCTGGGCTGGAGCTCTTGCCCGAAGCCTGAAGCTTCC
TTCGGCGCCATGCAGGGGTTGGGGTTGGGACTGGAGCTGGCCTTGGGATTTAATGGGGAAGAAGGCCTGG
CAGGACCTGGCCTGTTTGTTTAAAAATGAAGTATGGGTTGGGGGATTACGCTAGTTTTTCTTTGTATTTT
TATCTCTATCTCCTCACTTTTTCTCCCAAAGTAGAAAAAAATGATATCTGAA

XM_061930.7| PREDICTED: Homo sapiens similar to Homeobox protein DBX1 (LOC120237), mRNA
ATGATGTTCCCCGGCCTCCTCGCGCCCCCCGCCGGGTACCCTAGCCTCCTGCGGCCCACGCCCACCTTGA
CGCTGCCCCAGTCCTTGCAGTCGGCATTTTCCGGCCACTCCAGCTTCCTGGTGGAGGATCTGATCCGCAT
CAGCCGACCCCCCGCCTACCTGCCCCGCAGCGTGCCCACCGCCAGCATGTCGCCGCCCAGGCAGGGGGCC
CCCACGGCCCTCACCGACACGGGGGCCTCGGACCTGGGCTCCCCGGGTCCCGGCAGCCGACGGGGCGGCT
CTCCGCCGACTGCCTTCTCCCCTGCCAGCGAGACGACGTTTCTGAAGTTTGGAGTGAACGCCATCCTCTC
CTCGGGGCCCAGAACAGAAACATCCCCAGCCTTGCTCCAGAGCGTCCCTCCCAAGACCTTCGCCTTTCCC
TACTTCGAAGGGTCTTTTCAGCCTTTCATCAGATCTTCTTATTTCCCAGCTTCCTCCAGCGTGGTGCCCA
TCCCCGGGACCTTCTCCTGGCCGCTGGCCGCGCGCGGGAAGCCTCGGCGGGGCATGCTGCGTCGAGCAGT
CTTCTCCGACGTGCAGCGGAAGGCGCTGGAGAAGATGTTCCAGAAGCAGAAGTACATCAGCAAGCCCGAC
CGCAAGAAGCTGGCGGCCAAGCTGGGCCTGAAAGACTCGCAGGACACAGCCCGAAGGACAGAGCCTGCAC
CCGACTCCACTCCCCGCCCCCGTGCGTCCCCCGGAGCCCCTGATCTGACCTCTGCTTCTCCCTTCTTCTC
GCCGGCCGGGACATTCCAGGTGAAAATCTGGTTCCAGAACCGACGCATGAAATGGCGGAACTCCAAGGAG
CGCGAACTCCTGTCTAGCGGGGGCTGTCGCGAGCAGACCCTGCCCACCAAGCTCAATCCGCACCCGGACC
TCAGCGACGTGGGCCAGAAGGGCCCTGGGAACGAAGAGGAGGAGGAGGGCCCGGGCAGCCCCAGCCACCG
CCTGGCCTACCACGCGTCCTCCGACCCCCAGCACCTGCGGGACCCGCGGCTGCCAGGGCCGCTGCCCCCC
TCGCCCGCGCACTCGAGCAGTCCCGGGAAACCTTCGGACTTCTCAGATTCCGAGGAGGAAGAGGAGGGCG
AGGAACAGGAGGAAATCACCGTGTCCTAG

NM_018101.2| Homo sapiens cell division cycle associated 8 (CDCA8), mRNA
GTGGAGTTTGAATTGGGTGGCGGTTGACTGTAGAGCCGCTCTCTCTCACTGGCACAGCGAGGTTTTGCTC
AGCCCTTGTCTCGGGACCGCAGCCTCCGCCGAGCGCCATGGCTCCTAGGAAGGGCAGTAGTCGGGTGGCC
AAGACCAACTCCTTACGGAGGCGGAAGCTCGCCTCCTTTCTGAAAGACTTCGACCGTGAAGTGGAAATAC
GAATCAAGCAAATTGAGTCAGACAGGCAGAACCTCCTCAAGGAGGTGGATAACCTCTACAACATCGAGAT
CCTGCGGCTCCCCAAGGCTCTGCGCGAGATGAACTGGCTTGACTACTTCGCCCTTGGAGGAAACAAACAG
GCCCTGGAAGAGGCGGCAACAGCTGACCTGGATATCACCGAAATAAACAAACTAACAGCAGAAGCTATTC
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GCCGATTGGAGGTGTCCATGGTCAAACCAACTCCAGGCCTGACACCCAGGTTTGACTCAAGGGTCTTCAA
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GCTGACAGCAAAGAGATCTTCCTCACTGTGCCAGTGGGCGGCGGAGAGAGCCTGCGATTATTGGCCAGTG
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TCTCGCCCAAATCTGCAGCAGCATACGGACCCACAAATGAGACACCAAAGTTGACAGGATGGACTTTTAA
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TCAGTCTCCCAGCTGAATCCTCCCTGCTCTCTGAGCTGCTGCCTTTTGCCTCCTGCAACTCAACATCCTC
TTCACCCTGCCCTGCCTGCAGTTGAGGGGGCGAAGAAGAACCCTGTGTTCTCAGGAAGACTGCCTCCACC
ACCGCTACCCAGAGAACCTCTGCATCTGGCATTTCTGCTCTCTATGCTTGAGACCGGGAGGTTTAGGCTC
AGATAAGTGAGCTCTGGGCCATGAGAGGGTAGGTCCAGAAGGTGGGGGGAACTGTACAGATCAGCAGAGC
AGGACAGTTGGCAGCAGTGACCTCAGTAGGGAACATGTCCGTCTACCCTCTCGCACTCATGACACCTCCC
CCTACCAGCCCTCCTCTTCCTCCTCCTCCTCCTCCTGTGGGAGGTGGTCAGTGGGACTTAGGGATCTTTC
ACCTGCTGTGCCCAGTAGTTCTGAAGTCTGCTTGTGGAGCAGTGTTTTATGTTTATCCCTGTTTACTGAA

GACCAAATACTGGTTTGGAGACAACTTCCATGTCTTGCTCTTCTACCTCCCTAGTTAGTGGAAATTTGGA
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TCCATGGTCCAGTGGTCCCTGCCAAGTCTGTAGACTTCAGAGAGCACTTCTCTCTTATGGGGTTCATGGG
AACAGGGGTGGGTGTGACTTGCTTGGTGGCCTCATTCCATGTGTGCCTGTGCCTGGGGCATGGACTTTGT
TAAGCAGAGTCAGCAGTGAGGTCCTCATTCTCCAGCCAGCCTCTCTGCCCTGGAGAATCATGTGCTATGT
TCTAAGAATTTGAGAACTAGAGTCCTCATCCCCAGGCTTGAAGGCACATGGCTTTCTCATGTAGGGCTCT
CTGTGGTATTTGTTATTATTTTGCAACAAGACCATTTTAGTAAAACAGTCCTGTTCAAGTTGTATTCTTT
TAAGTTCTTTTATTCTCCTTTCCCTGAGATTTTTGTATATATTGTTCTGAGTAATGGTATCTTTGAGCTG
ATTGTTCTAATCAGAGCTGGTACCTACTTTCAATAAATTCTGGTTTTGTGTTTTCTTTTGTAAAAAAAAA
AAAAAAAAA

NM_002541.2| Homo sapiens oxoglutarate (alpha-ketoglutarate) dehydrogenase (lipoamide) (OGDH), nuclear gene encoding mitochondrial protein, transcript variant 1, mRNA

ATACGCCGGGGCGGGGCCGAGAGTTTGGAGCCCCGGAGTGGGGTGTCGGCGCCTCATTCGGGTGGAGCTG
AGCCGGAGACAGGCAGTTGTGAAAAACTTCAGGACAAAAATGTTTCATTTAAGGACTTGTGCTGCTAAGT
TGAGGCCATTGACGGCTTCCCAGACTGTTAAGACATTTTCACAAAACAGACCAGCAGCAGCTAGGACATT
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CTGGCAGTGCAGTCGCTCATCAGGGCATATCAGATACGAGGGCACCATGTAGCACAGCTGGACCCCCTGG
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GAATCAGCACTTCCTCTGCGGGAGATCATCCGTCGGCTGGAGATGGCCTACTGCCAGCATATTGGGGTGG
AGTTCATGTTCATCAATGACCTGGAGCAGTGCCAGTGGATCCGGCAGAAGTTTGAGACCCCTGGGATCAT
GCAGTTCACAAATGAGGAGAAACGGACCCTGCTGGCCAGGCTTGTGCGGTCCACCAGGTTTGAGGAGTTC
CTACAGCGGAAGTGGTCCTCTGAGAAGCGCTTTGGTCTAGAAGGCTGCGAGGTACTGATCCCTGCCCTCA
AGACCATCATTGACAAGTCTAGTGAGAATGGCGTGGACTACGTGATCATGGGCATGCCACACAGAGGGCG
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GAGGCAGCTGATGAGGGCTCCGGAGATGTGAAGTACCACCTGGGCATGTATCACCGCAGGATCAATCGTG
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GATGGGCAAGACCAAAGCCGAACAGTTTTACTGTGGCGACACTGAAGGGAAAAAGGTCATGTCCATCCTG
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GCTCATGTACAAGCAGATCCGCAAGCAGAAGCCTGTGTTACAGAAGTACGCTGAGCTGCTGGTGTCGCAG
GGTGTGGTCAACCAGCCTGAGTATGAGGAGGAAATTTCCAAGTATGATAAGATCTGTGAGGAAGCTTTTG
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CCTGGACGGGCAGCCCAGGAGCATGTCCTGCCCCTCCACGGGTCTGACGGAGGATATTCTGACACACATC
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CTCGTGGGGAAATGGTGAAGAACCGGACTGTGGACTGGGCTCTAGCGGAGTACATGGCGTTTGGCTCGCT
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TGACCCAGATGTCCTGCCAGACCTTAAAGAAGCCAACTTCGACATCAATCAGCTATATGACTGCAATTGG
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GGTGCAGAAGTACCCCAATGCTGAGCTGGCCTGGTGCCAGGAGGAGCACAAGAACCAAGGCTACTATGAC
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CCCATGACTGCCCCTTGCTTCTCAACTAAAGAATAGTGCCTCAGCGCTGCCCACACCACCGCCCTCCTCG

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GAGGAAAGGTAGCCCCCGAGGGATGTCCTTGGGGAGGGGTCAGCTCTGGCCACAATCCTCCCCACCAGTC
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AGGCAGCACCTGGAGCCCACAGAGTCTGTGTGTAGCCAGGAAGCCCCGCTCAGGTAGCCACCACCGGGGC
ACTGGCTGCTCTGTCTTGGTCCTGTTAACCCTCCACCTCCTCTCTTGGACTCCCTCCCCACCCCAACCAC
TCTTTCTTTCTCCTTTAACCCAATGGAGACTTTCTGATGCATCGTTTTCTTTGCTGTGCCAAAGCAGGTC
AGAAGAGGGAGAGGAGGGGCTGGGGGTGAGGGGCCAGGCCATGGCCAAGGGGCCAGCTGCCCCTCATTTA
TCACTCTGACCTTCACAGGGACAGATCTGATTTATTTATTTTGGTTAAAAAAAAAAAAAAGGAACAGAAA
CAACTTTGCATTGCATTGGCTTGACCCATAAACTAAGTTATATCCGTGGGCAAAAAA

NM_006107.2| Homo sapiens cisplatin resistance-associated overexpressed protein (CROP), transcript variant 2, mRNA
CTGTGTGTAGGAGGGATTTCGGCCTGAGAGCGGGCCGAGGAGATTGGCGACGGTGTCGCCCGTGTTTTCG
TTGGCGGGTGCCTGGGCTGGTGGGAACAGCCGCCCGAAGGAAGCACCATGATTTCGGCCGCGCAGTTGTT
GGATGAGTTAATGGGCCGGGACCGAAACCTAGCCCCGGACGAGAAGCGCAGCAACGTGCGGTGGGACCAC
GAGAGCGTTTGTAAATATTATCTCTGTGGTTTTTGTCCTGCGGAATTGTTCACAAATACACGTTCTGATC
TTGGTCCGTGTGAAAAAATTCATGATGAAAATCTACGAAAACAGTATGAGAAGAGCTCTCGTTTCATGAA
AGTTGGCTATGAGAGAGATTTTTTGCGATACTTACAGAGCTTACTTGCAGAAGTAGAACGTAGGATCAGA
CGAGGCCATGCTCGTTTGGCATTATCTCAAAACCAGCAGTCTTCTGGGGCCGCTGGCCCAACAGGCAAAA
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TTTCAATCTTGGACTTTGAAAACAGGATTAAACGTTAGTATTCGTGTGAATCAGACTAAGTGGGATTTCA
TTTTTACAACTCTGCTCTACTTAGCCTTTGGATTTAGAAGTAAAAATAAAGTATCTCTGACTTTCTGTTA
CAAAGTTGATTGTCTCTGTCATTGAAAAGTTTTAGTATTAATCTTTTTTCTAATAAAGTTATTGACTCTGA
AAAAAAAAAAAA

NM_002826.4| Homo sapiens quiescin Q6 sulfhydryl oxidase 1 (QSOX1), transcript variant 1, mRNA
GGAGGCAGGCGGTGCCGCGGCGCCGGGACCCGACTCATCCGGTGCTTGCGTGTGGTGGTGAGCGCAGCGC
CGAGGATGAGGAGGTGCAACAGCGGCTCCGGGCCGCCGCCGTCGCTGCTGCTGCTGCTGCTGTGGCTGCT
CGCGGTTCCCGGCGCTAACGCGGCCCCGCGGTCGGCGCTCTATTCGCCTTCCGACCCGCTGACGCTGCTG
CAGGCGGACACGGTGCGCGGCGCGGTGCTGGGCTCCCGCAGCGCCTGGGCCGTGGAGTTCTTCGCCTCCT
GGTGCGGCCACTGCATCGCCTTCGCCCCGACGTGGAAGGCGCTGGCCGAAGACGTCAAAGCCTGGAGGCC
GGCCCTGTATCTCGCCGCCCTGGACTGTGCTGAGGAGACCAACAGTGCAGTCTGCAGAGACTTCAACATC
CCTGGCTTCCCGACTGTGAGGTTCTTCAAGGCCTTTACCAAGAACGGCTCGGGAGCAGTATTTCCAGTGG
CTGGTGCTGACGTGCAGACACTGCGGGAGAGGCTCATTGACGCCCTGGAGTCCCATCATGACACGTGGCC
CCCAGCCTGTCCCCCACTGGAGCCTGCCAAGCTGGAGGAGATTGATGGATTCTTTGCGAGAAATAACGAA
GAGTACCTGGCTCTGATCTTTGAAAAGGGAGGCTCCTACCTGGGTAGAGAGGTGGCTCTGGACCTGTCCC
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CCCCCGAAGCTGCACCCTGGCCTCAGAGCTGCACCAGGCCAGGAGCCTCCTGAGCACATGGCAGAGCTTC
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GTCTCTAGTGCCTTGCCCTGGCTTAGCTGCAGGAGAAGATGGCTGCTTTCACTTCCCCCCATTGAGCTCT
GCTCCCTCTGAGCCTGGTCTTTTGTCCTTTTTTATTTTGGTCTCCAAGATGAATGCTCATCTTTGGAGGG
TGCCAGGTAGAAGCTAGGGAGGGGAGTGTCTTCTCTCTCCAGGTTTCACCTTCCAGTGTGCAGAAGTTAG
AAGGGTCTGGCGGGGGCAGTGCCTTACACATGCTTGATTCCCACGCTACCCCCTGCCTTGGGAGGTGTGT
GGAATAAATTATTTTTGTTAAGGCAA

NM_005384.2| Homo sapiens nuclear factor, interleukin 3 regulated (NFIL3), mRNA

ACGTAGCGCGGCGCTCGGAACTGACCTACTAACACACATCTCTCCGCGCGGCCACGGCGCCCGCGGACCC
GGCGCGCCCGCCCGCCTCCCGCGCCGCGCCCTCGCCGCCGCCCGCCTCCCGCCGCGGCCCCGGAGGCCCG
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GAGCAGGAACACGATAACGAAGGAGGCCCAACTTCATTCAATAAGGAGCCTGACGGATTTATCCCAGACG
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AAAACTCAGAATAGTTTCAAAACTGGAGTTGTTGAAATGAAAGACAGTGGCTACAAAGTTTCTGACCCAG
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GTGATGTGCACATGTTGTCTGTTTGGTGTCTTTTTGTGCACAGATTATGATGAAGATTAGATTGTGTTAT
CACTCTGCCTGTGTATAGTCAGATAGTCCATGCGAAGGCTGTATATATTGAACATTATTTTTGTTGTTCT
ATTATAAAGTGTGTAAGTTACCAGTTTCAATAAAGGATTGGTGACAAACACAGAAAAAAAAAAAAAAAAA
AAAA

NM_005658.3| Homo sapiens TNF receptor-associated factor 1 (TRAF1), mRNA
GTAAAGTGGTGGCACCTGACCGGAGCATGTAGCCACCTATGACTTCCTGAGTGGGGTGGTGTGGGGAGGG
CCCTGAGGAGTCTGTGGTCCTAGGCCTTGGATTTCATCAGGGCTTTCCTGCTCCTTCTGGTTGCCTCAGT
CCCAGGCAAGATAGGGTCCCAGAATCTTACAGCCCAGAGGCCACCTGTTGCAACTTCTTTTTTACAGATG
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TGAATGATTTCTTTTCCAACAAAGCTCTATGTTAAGTGC

NM_003400.3| Homo sapiens exportin 1 (CRM1 homolog, yeast) (XPO1), mRNA
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NM_018226.3| Homo sapiens arginyl aminopeptidase (aminopeptidase B)-like 1 (RNPEPL1), mRNA

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GGGGTATCACGGTGATGGTCCCATTAAACTTCCACTCTGCAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
AAAAAAAA

NM_007165.4| Homo sapiens splicing factor 3a, subunit 2, 66kDa (SF3A2), mRNA
GGAAGTGCTTTCTTTGCCCGCCGTTCGCCAAACGAAGTCGTGGAGGTGGCGAAACGAGGAGGAGATAACG
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GCCCAGCGCCAGGTGCTCTTGCCTTTTCCCACTGAGAGAAGGCTGCTCTTTTGTACTGCCCCCCGCTCAT
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NM_005262.2| Homo sapiens growth factor, augmenter of liver regeneration (GFER), mRNA
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NM_012341.2| Homo sapiens GTP binding protein 4 (GTPBP4), mRNA
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CCCCCTTTCATCCCTGAAGGAGTGGTGGCTCGCAGGAAGAGGATGGAAACTGAGGAGTCCAGGAAGAAGA
GGGAACGAGATCTTGAGCTGGAAATGGGAGATGATTATATTTTGGATCTTCAGAAGTACTGGGATTTAAT
GAATTTGTCTGAAAAACATGATAAGATACCAGAAATCTGGGAAGGCCATAATATAGCTGATTATATTGAT
CCAGCCATCATGAAGAAATTGGAAGAATTAGAAAAAGAAGAAGAGCTGAGAACAGCTGCTGGAGAGTATG
ACAGTGTATCTGAGAGTGAAGACGAAGAGATGCTGGAAATCCGACAGCTGGCAAAGCAAATTCGAGAGAA
AAAGAAGTTGAAAATTCTGGAGTCCAAAGAAAAGAATACACAGGGACCCAGGATGCCGCGAACTGCTAAG
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CCCGTCCTCTGTGGCCCGGAGTGGGAGTTGCTCTCGAACTCCACGTGACGTTTCTGGTCTTAGGGATGTC
AAGATGGTGAAGAAAGCCAAGACTATGATGAAGAATGCTCAGAAGAAGATGAATCGGTTGGGGAAGAAAG
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AAAGGACAGGAGATAGTATCCGTTTGGTTGGCGTGGCTTCGCTAGAGTGTTGCTGTTTATTTCCTGGTTT
GGCACAGTATGGTTTCATGAAATTGGAGCTCTGTATAAACTGAAAAAGACAAAATAAGTAAAGCACTTGT
TGCTTTGCTGAAAACTATGGTTAACCCTATATAGGTGTGGGAAATTTTTGTCACTGCATAATATTACAAA
TATTTTGAGTAGACAGTGTTTCCACATTTAATGGAGTATCAGTTGCTTCAGATTTTCAGAACTGGGAAGA
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CACGTTTATGAGTGTGTCGGAATCCCGTGCTTAAAATACGCTCTTAAATTATTTTCTAGTCTTATTTTAC
AATGTCTCATTGTAGTCTGTCTTCAACTATTTTATCCAAAATAAACCTCCAGAAGAAAGTAGTTTTCATT
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AAAAAAAAAAAAAAAAA

NM_000136.2| Homo sapiens Fanconi anemia, complementation group C (FANCC), mRNA
AGAATGCACTGCTGACACGTGTGCGCGCGCGGCTCCACTGCCGGGCGACCGCGGGAAAATTCCAAAAA
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CAGTGCTGGACAGGCTGCTGTGAAGGGACATCACCTTTTCGCTTTTTCCAAGATGGCTCAAGATTCAGTA
GATCTTTCTTGTGATTATCAGTTTTGGATGCAGAAGCTTTCTGTATGGGATCAGGCTTCCACTTTGGAAA
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CCTGGTTTGCTTAAAAATATGGTTTTATCATTAGCGTCTGAACTCAGAGAGAATCATCTTAATGGATTTA
ACACTCAAAGGCGAATGGCTCCCGAGCGAGTGGCGTCCCTGTCACGAGTTTGTGTCCCACTTATTACCCT
GACAGATGTTGACCCCCTGGTGGAGGCTCTCCTCATCTGTCATGGACGTGAACCTCAGGAAATCCTCCAG
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CACCCTGCCATATTCCGGGTTGTTGATGAGATGTTCAGGTGTGCACTCCTGGAAACCGATGGGGCCCTGG
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GCATGCTGGTAAAACCACTACCCTCAGAGAGAGCCAAAAATACAGAAGAGGCGGAGAGCGCCCCTCCAAC
CAGGCTGTTATTCCCCTGGACTCCGTGACATCTGTGGAATTTTTTAGCTCTTTAAAATCTGTAATTTGTT
GTCTATTTTTTCATTCTAAATAAAACTTCAGTTTGCACCTAAAAAAAAAAAAAAAAAAAAA

NM_006103.3| Homo sapiens WAP four-disulfide core domain 2 (WFDC2), mRNA
CACCTGCACCCCGCCCGGGCATAGCACCATGCCTGCTTGTCGCCTAGGCCCGCTAGCCGCCGCCCTCCTC
CTCAGCCTGCTGCTGTTCGGCTTCACCCTAGTCTCAGGCACAGGAGCAGAGAAGACTGGCGTGTGCCCCG
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ATTAACTTTCCCCAGCTCGGCCTCTGTCGGGACCAGTGCCAGGTGGACAGCCAGTGTCCTGGCCAGATGA
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GCTGAGCAGTGAGGAGAGAAAGTTTCTGCCTGGCCCTGCATCTGGTTCCAGCCCACCTGCCCTCCCCTTT
TTCGGGACTCTGTATTCCCTCTTGGGCTGACCACAGCTTCTCCCTTTCCCAACCAATAAAGTAACCACTT
TCAGCAAAAA

NM_020461.3| Homo sapiens tubulin, gamma complex associated protein 6
(TUBGCP6), mRNA
ACTTTCCACGGGTGAGGCCTGGAGAACGGGTGGACGTGCAGGCCAGAACCCGGGACCCACCCGGCTGAGC
CCCCAGACTCTCCGCTGCCCACGACCCTCGGAGTGGCACCCTCTCCTGGCCGTCTCCTCTCGTGGGTCCC
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CGAGTGTGGATCAGTGGGAAGACGAAGGATTCCAGTCAGCGTCCAACTTGACTCCTGATTCCCAGTCTGA
GCCCAGCGTGACCCCAGACGTGGACCTGTGGGAAGCCGCACTTACCTATGAGGCCAGCAAGCGGAGGTGC
TGGGAGCGAGTTGGCTGCCCCCCTGGCCACAGAGAGGAGCCTTACCTGACGGAGGCGGGAAGGGACGCTT
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TAGGGGAGTCTGTGTCAGATGTGGCTCCCACTCGGCCACGGTGGAACATCCATGGACATGTGTCCAACGC
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CGTGCTCAAGTTCCGCAGCCAGCTCATCTCCCAGGCCTGGGGGCCCCCTGGGGGCCCGCGGGGTGCAGAG
CACCCCAACTTTGCACTCATGCAGCAGTCCTACAACACCTTCAAGTACTACTCCCACTTTCTCTTCAAAG
TGGTGACCAAGCTGGTGAACCGCGGCTACCAGCCCCACCTGGAGGACTTTCTGCTGCGCATCAACTTCAA
CAACTACTACCAGGACGCCTGAGGCTGCTCTGCGGGGGACGTGCACAATAAAGGTGTTCTCGGACCCTGA
AAAAAAAAAAAAAAAAAAAA

NM_002779.3| Homo sapiens pleckstrin and Sec7 domain containing (PSD), mRNA
ATGTAACAGGTCATGTGGCAGCCTCCTAGGGACACGCGTGGGGTCTGGAGCGGGACACGCGTGTGCAGCC
GGGCCTTACGTGTGGCTCAAGGGGCGGTGGGCGAGAGAGACGTGGACACAAGTGGGGTGGGGCACGTGAG
GGTCTGGGGAATAGAACCAGGGGTTCCATGCGCCCTCCTCCTTGTCTGGGGCGGGTCTTAGGTTGGCGGG
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CACCAGCTCCCGCCCCACTTGCCCCTCTTGAACCGGATTCTGGTACCAGCTCTGCTGCTGATGGTCCTTG
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GATTTGGGGCTGAGCTGGTTTCCCCTCCTTCCTCCCCAAGGGCTTACTCTTTCTCTTGCTGGAGGTGGAG
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GGGCCTTGCCCTTTGTACAGCCTCCACCCCCATTAAAACTGCTCTGGACTTGC

NM_014756.2| Homo sapiens cytoskeleton associated protein 5 (CKAP5), transcript variant 2, mRNA

ACTGCGGGGTGTGCGGCGGCCCAAGCGGTTTCAAACGGCTTAGAGCAGGCCGCTTGGTTCTGACCCAGCT
GAGGAAATACTCTTAATTCTAAGGAAAACCTGGAAGCACAATGGGAGATGACAGTGAGTGGTTGAAACTG
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CACTGATTCCAATGCAGTGGTTCAATTGAAAGGATTAGAAGCTGCACTTGTTTATGTTGAAAATGCCCAT
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AGAGCTCCTGAAAGGCTTGGACAATAAGAATCCCAAGATCATAGTGGCCTGTATAGAGACACTGAGGAAA
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AGTCTCGAGAGAAGGCTGTTCGAGATGAAGCCAAACTAATTGCTGTGGAGATTTACAGATGGATTCGGGA
TGCTCTGAGACCCCCATTACAAAATATAAACTCTGTTCAGTTGAAAGAACTAGAAGAAGAATGGGTCAAA
CTGCCAACAAGTGCTCCTAGACCTACTCGATTTCTTCGTTCCCAACAAGAACTAGAAGCTAAATTGGAAC
AACAACAGTCTGCTGGTGGAGATGCTGAAGGAGGTGGTGATGATGGTGATGAGGTGCCACAAATAGATGC
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TAAAAATCCAACCATCAAGCAGCAGACATCTCTTTTTATTGCAAGAAGTTTCCGCCACTGCACTGCTTCT
ACCCTGCCAAAGAGCTTGCTAAAGCCCTTTTGTGCTGCACTACTTAAGCACATCAATGATTCTGCTCCTG
AAGTCAGAGATGCCGCATTTGAAGCATTGGGTACTGCTTTGAAGGTGGTTGGCGAGAAAGCAGTAAACCC
ATTCCTAGCTGATGTGGACAAACTCAAGCTTGATAAGATCAAAGAATGTTCAGAAAAGGTAGAACTGATA
CATGGTAAGAAAGCTGGACTAGCTGCTGATAAGAAGGAATTCAAACCTCTGCCTGGAAGGACTGCTGCTT
CAGGGGCTGCAGGAGATAAGGACACAAAGGACATTTCTGCACCCAAACCAGGACCTCTAAAAAAGGCACC
TGCTGCTAAGGCTGGTGGGCCACCAAAAAAGGGGAAACCAGCTGCACCAGGAGGCGCAGGGAATACTGGA
ACCAAGAACAAGAAAGGACTGGAGACTAAAGAAATAGTGGAGCCTGAGCTCTCGATAGAAGTATGTGAAG
AAAAAGCTTCAGCTGTTCTTCCCCCTACCTGTATACAGCTTCTTGACAGCAGTAACTGGAAAGAAAGGCT
GGCTTGTATGGAAGAGTTCCAGAAGGCTGTTGAGCTAATGGACCGAACTGAAATGCCATGCCAGGCATTA
GTGAGGATGCTAGCCAAGAAACCTGGATGGAAAGAAACTAATTTTCAGGTGATGCAAATGAAGCTTCATA
TAGTTGCTTTGATTGCCCAGAAGGGAAATTTTTCCAAAACGTCAGCTCAGGTTGTATTAGATGGCCTTGT
GGACAAGATTGGAGATGTGAAATGTGGGAACAATGCAAAAGAAGCTATGACAGCAATAGCCGAAGCCTGT
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TCCCTATCATCACAGTCCTTGGAGACAGCAAGAACAATGTTCGAGCTGCTGCCCTAGCGACTGTGAATGC
TTGGGCAGAACAGACTGGCATGAAGGAATGGCTGGAAGGAGAAGATCTTTCTGAAGAGCTCAAAAAGGAA
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CAGACCTTATCCTTTGTGTTCCTCATCTCTACTCCTGCCTAGAAGATCGAAATGGAGATGTGCGAAAGAA
GGCCCAAGATGCCTTGCCATTCTTCATGATGCATTTAGGATATGAAAAAATGGCCAAGGCTACTGGGAAA
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TTGTCGTCAAGGTTGGAGAACCAAAGGATGTCATTCGTAAAGATGTTCGTGCCATCCTGAACCGGATGTG
CCTTGTCTACCCAGCTAGCAAGATGTTTCCCTTTATCATGGAAGGAACCAAATCCAAAAACTCTAAGCAG
AGAGCAGAGTGCCTGGAAGAGCTGGGATGTCTGGTTGAGTCCTATGGCATGAATGTTTGCCAACCAACCC
CAGGAAAAGCCTTAAAGGAAATAGCTGTTCACATAGGAGACCGTGACAATGCTGTACGCAATGCTGCACT
CAACACCATTGTAACGGTGTACAATGTACATGGGGATCAGGTGTTCAAACTGATTGGAAATCTTTCTGAA
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CGCCGAGAATTCCAGCTGGATCTAGATGAGATTGAGAATGACAATGGTACAGTCCGATGTGAAATGCCAG
AACTTGTTCAGCACAAACTGGATGACATTTTTGAGCCAGTCCTTATTCCTGAACCCAAGATCCGGGCTGT
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AGTGGTGACATCAACACAAGTATCCAAGCTCTGACACAGCTGTTTCAGATAGAGAGCCTTGCCCGGGAGG
CCTCCACTGGAGTACTAAAAGACCTAATGCATGGCCTCATCACCTTAATGCTGGATTCTCGGATTGAAGA
TCTTGAGGAAGGACAACAGGTCATCCGCTCTGTGAACCTCTTGGTGGTGAAGGTTCTGGAGAAGTCAGAC
CAGACCAACATCCTGAGTGCCCTACTTGTTTTGCTCCAAGACAGCCTGCTAGCAACAGCCAGTTCTCCCA
AATTCTCAGAGCTTGTTATGAAGTGTCTCTGGAGAATGGTTCGACTGTTGCCTGATACCATCAATAGCAT
TAACCTAGACAGAATTCTTCTGGATATCCACATTTTCATGAAGGTCTTCCCCAAAGAGAAACTGAAGCAA
TGCAAAAGTGAATTTCCCATAAGGACCCTAAAGACCCTGCTACACACCTTATGCAAATTAAAAGGGCCCA
AGATCCTGGACCACCTAACGATGATCGACAACAAAAACGAGTCTGAGCTGGAGGCCCATCTCTGCCGGAT
GATGAAGCACAGTATGGACCAGACTGGGAGCAAGTCTGATAAGGAAACAGAAAAGGGAGCATCTCGAATA
GATGAAAAATCATCAAAGGCCAAAGTGAATGATTTCTTAGCTGAGATTTTTAAGAAGATTGGCTCTAAAG

AAAACACTAAAGAGGGACTAGCAGAGTTATATGAATATAAGAAGAAATACTCAGATGCTGACATTGAACC
ATTTCTGAAAAATTCCTCACAGTTCTTCCAGAGCTATGTCGAAAGAGGCCTTCGGGTGATTGAGATGGAG
AGGGAGGGCAAAGGTCGTATTTCCACTTCAACAGGCATCTCCCCTCAGATGGAAGTCACATGTGTGCCCA
CGCCCACAAGCACAGTGTCCTCCATAGGTAACACAAATGGGGAAGAAGTGGGGCCATCTGTCTACTTGGA
AAGGCTAAAGATCCTCCGACAGCGATGTGGTCTGGACAACACAAAGCAAGATGACCGACCTCCTTTGACC
TCTTTGCTCTCCAAACCAGCAGTTCCTACTGTCGCCTCTTCCACAGACATGCTCCACAGCAAACTCTCTC
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TTAAAATGGCCTCAGCAGGCCTAGTGTATACAAACTGGTTGTATGTATCATGCCGTGGAGCTAGGGGGAG
GAGTCATTGTGGCACAAGTATTTGTACATACTCTGCTTCTCTCTGTCAGCGTCCTGCTGCTCTAGAAGAC
TGTCCGTGGATGAGTTTAGTGTACAGACTTGTAAACAGCTGCCCCCTCTCTGCTCAGTCTAGTTCCCAGA
TCCTTTTCTTTTCTTTTTAATTGCTCATTTGTAAAATTGTCCTAATCTTTCCTAGCTTTTTAATAGTTAA
TATTAGAAACTCTTTAATAGTTTTCCTTTCAGTTTGTGAGCTCTTCTCTGTCGCCCTGAAGGGTCACTGT
ATTCTGTATGAATGCATGGCATGATACAACTAATTTAAGAGTCTTTTATAAATAAAGTTTGCATTAACTA
TACCTGACAAAAAAAAAAAAAAAA

NM_003804.3| Homo sapiens receptor (TNFRSF)-interacting serine-threonine kinase 1 (RIPK1), mRNA
ATGCAACCAGACATGTCCTTGAATGTCATTAAGATGAAATCCAGTGACTTCCTGGAGAGTGCAGAACTGG
ACAGCGGAGGCTTTGGGAAGGTGTCTCTGTGTTTCCACAGAACCCAGGGACTCATGATCATGAAAACAGT
GTACAAGGGGCCCAACTGCATTGAGCACAACGAGGCCCTCTTGGAGGAGGCGAAGATGATGAACAGACTG
AGACACAGCCGGGTGGTGAAGCTCCTGGGCGTCATCATAGAGGAAGGGAAGTACTCCCTGGTGATGGAGT
ACATGGAGAAGGGCAACCTGATGCACGTGCTGAAAGCCGAGATGAGTACTCCGCTTTCTGTAAAAGGAAG
GATAATTTTGGAAATCATTGAAGGAATGTGCTACTTACATGGAAAAGGCGTGATACACAAGGACCTGAAG
CCTGAAAATATCCTTGTTGATAATGACTTCCACATTAAGATCGCAGACCTCGGCCTTGCCTCCTTTAAGA
TGTGGAGCAAACTGAATAATGAAGAGCACAATGAGCTGAGGGAAGTGGACGGCACCGCTAAGAAGAATGG
CGGCACCCTCTACTACATGGCGCCCGAGCACCTGAATGACGTCAACGCAAAGCCCACAGAGAAGTCGGAT
GTGTACAGCTTTGCTGTAGTACTCTGGGCGATATTTGCAAATAAGGAGCCATATGAAAATGCTATCTGTG
AGCAGCAGTTGATAATGTGCATAAAATCTGGGAACAGGCCAGATGTGGATGACATCACTGAGTACTGCCC
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TAAAGAAAGAGTATTCAAACGAAAATGCAGTTGTGAAGAGAATGCAGTCTCTTCAACTTGATTGTGTGGC
AGTACCTTCAAGCCGGTCAAATTCAGCCACAGAACAGCCTGGTTCACTGCACAGTTCCCAGGGACTTGGG
ATGGGTCCTGTGGAGGAGTCCTGGTTTGCTCCTTCCCTGGAGCACCCACAAGAAGAGAATGAGCCCAGCC
TGCAGAGTAAACTCCAAGACGAAGCCAACTACCATCTTTATGGCAGCCGCATGGACAGGCAGACGAAACA
GCAGCCCAGACAGAATGTGGCTTACAACAGAGAGGAGGAAAGGAGACGCAGGGTCTCCCATGACCCTTTT
GCACAGCAAAGACCTTACGAGAATTTTCAGAATACAGAGGGAAAAGGCACTGCTTATTCCAGTGCAGCCA
GTCATGGTAATGCAGTGCACCAGCCCTCAGGGCTCACCAGCCAACCTCAAGTACTGTATCAGAACAATGG
ATTATATAGCTCACATGGCTTTGGAACAAGACCACTGGATCCAGGAACAGCAGGTCCCAGAGTTTGGTAC
AGGCCAATTCCAAGTCATATGCCTAGTCTGCATAATATCCCAGTGCCTGAGACCAACTATCTAGGAAATA
CACCCACCATGCCATTCAGCTCCTTGCCACCAACAGATGAATCTATAAAATATACCATATACAATAGTAC
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ATAAACACCTGGACCCAATCAGGGAAAATCTGGGAAAGCACTGGAAAAACTGTGCCCGTAAACTGGGCTT
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ATGCTCCAAAAGTGGGTGATGAGGGAAGGCATAAAGGGAGCCACGGTGGGGAAGCTGGCCCAGGCGCTCC
ACCAGTGTTCCAGGATCGACCTTCTGAGCAGCTTGATTTACGTCAGCCAGAACTAACCCTGGATGGGCTA
CGGCAGCTGAAGTGGACGCCTCACTTAGTGGATAACCCCAGAAAGTTGGCTGCCTCAGAGCATTCAGAAT
TCTGTCCTCACTGATAGGGGTTCTGTGTCTGCAGAAATTTTGTTTCCTGTACTTCATAGCTGGAGAATGG
GGAAAGAAATCTGCAGCAAAGGGGTCTCACTCTGTTGCCAGGCTGGTCTCAAACTTCTGGACTCAAGTGA
TCCTCCCGCCTCGGCCTTCCAAAGTGCTGGGATATCAGGCACTGAGCCACTGCGCCCAGCCAACAATCCG
CTCTGAGGAAAGCGTAAGCAGGAAGACCTCTTAATGGCATAGCACCAATAAAAAAATGACTCCTAGTTGT
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TTGTTGAAGCAAGAAGAAAAACTCAGAGAATATAAAATCAACTTTTAAAATTGTGTGCTCTCTTCTTCAC
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GTTGCAAGGAATTGGTACTAACCGTGACTACAACAAAAATTCTTGATTGACTTTTAAAGTTATTTCCTGG
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TGAGACCAGAACCTCAGGGCTGATACTGTTGAGTGGCTTCCTCGGTTTACTCTGTGTACTGTGAAAGTAT
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CCAGCCTTTCAAAGCCCACCTGAAACCTGGGGGTGGATGAAAGAACTAGAATAGAAGACTGAAGCTGGGT
AGGCCGCTCAGTGTCCACTGGCATTTTGCTAAACCGACAAGGAAGGCTGTGTGCTTAGCTCTCCCCAGAG
GGAGGGCGAGAAGGGTGTGGTGATGGTCAATCTGGCTGTCGGAACAGATTCTGGTGTCTTGGGCTGATAA
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GCAGAGCAGGCTGTCTCAGCCTTGCCACTGTCGGCATCTCGGCCTGGGTAATTCTGTTGTGGGGACTGTC
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AGCCCCCCGCCCCAGTTGTGACAATAGTCTCTAAACATTGTCAAATGGTCCAAGGAAAGGGGAAAATTGC
CCCGGTTGAGAAGAGCACTGCTGTAAAGTAATGAGCCTCGGCTCTCCTGTCTGCACCTGTCCGGTTACTA
CTTGGCCACCACGCAGCCTTGGCTCCTACAGCCCAAAAGGGAGAATGGAGGGAGGCTCCAGGCTTTGCTG
GAGGGGCCTGGGTGAGTTCTGTTTGCTCCTTGTACCACCATCCAAATGGTGTTATCAAATCTCTTAGATT
CCAAAGAGGTTGAATAATTAATGTTCAAAGGCAAGAGGGCAAGGCATTTTTTAACACTTTTTAAAATAAA
AATTTATACCACAA

NM_002060.2| Homo sapiens gap junction protein, alpha 4, 37kDa (GJA4), mRNA
CAGCAGGGCTCCCGCGGGCGTCACTCCGGCCATCGTCCCCACCTCCACCTGGGCCGCCCGGCAGGCAGGC
GACGGAGGCCCGGGAGCCATGGGTGACTGGGGCTTCCTGGAGAAGTTGCTGGACCAGGTCCAGGAGCACT
CGACCGTGGTGGGTAAGATCTGGCTGACGGTGCTCTTCATCTTCCGCATCCTCATCCTGGGCCTGGCCGG
CGAGTCAGTGTGGGGTGACGAGCAATCAGATTTCGAGTGTAACACGGCCCAGCCAGGCTGCACCAACGTC
TGCTATGACCAGGCCTTCCCCATCTCCCACATCCGCTACTGGGTGCTGCAGTTCCTCTTCGTCAGCACAC
CCACCCTGGTCTACCTGGGCCATGTCATTTACCTGTCTCGGCGAGAAGAGCGGCTGCGGCAGAAGGAGGG
GGAGCTGCGGGCACTGCCGGCCAAGGACCCACAGGTGGAGCGGGCGCTGGCGGCCGTAGAGCGTCAGATG
GCCAAGATCTCGGTGGCAGAAGATGGTCGCCTGCGCATCCGCGGAGCACTGATGGGCACCTATGTCGCCA
GTGTGCTCTGCAAGAGTGTGCTAGAGGCAGGCTTCCTCTATGGCCAGTGGCGCCTGTACGGCTGGACCAT
GGAGCCCGTGTTTGTGTGCCAGCGAGCACCCTGCCCCTACCTCGTGGACTGCTTTGTCTCTCGCCCCACG
GAGAAGACCATCTTCATCATCTTCATGTTGGTGGTTGGACTCATCTCCCTGGTGCTTAACCTGCTGGAGT
TGGTGCACCTGCTGTGTCGCTGCCTCAGCCGGGGGATGAGGGCACGGCAAGGCCAAGACGCACCCCCGAC
CCAGGGCACCTCCTCAGACCCTTACACGGACCAGGTCTTCTTCTACCTCCCCGTGGGCCAGGGGCCCTCA
TCCCCACCATGCCCCACCTACAATGGGCTCTCATCCAGTGAGCAGAACTGGGCCAACCTGACCACAGAGG
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GGTGCCCACCAGGCACTACGGCTTCCTCTCCAGAATGTGGCTTTGCCTGAGCACAGACAGAGTCAGCATG
GAATGCTCTTGGCCAAGGGTACTGGGGGCCCTCTGGCCTTTTGCAGCTGATCCAGAGGAACCCAGAGCCA
ACTTACCCCAACCTCACCCTATGGAACAGTCACCTGTGCGCAGGTTGTCCTCAAACCCTCTCCTCACAGG
AAAAGGCGGATTGAGGCTGCTGGGTCAGCCTTGATCGCACAGACAGAGCTTGTGCCGGATTTGGCCCTGT
CAAGGGGACTGGTGCCTTGTTTTCATCACTCCTTCCTAGTTCTACTGTTCAAGCTTCTGAAATAAACAGG
ACTTGATCACAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
AAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_006169.2| Homo sapiens nicotinamide N-methyltransferase (NNMT), mRNA
GAGGAGGTGCTTGCCAGACACTGGGTCATGGCAGTGGTCGGTGAAGCTGCAGTTGCCTAGGGCAGGGATG
GAGAGAGAGTCTGGGCATGAGGAGAGGGTCTCGGGATGTTTGGCTGGACTAGATTTTACAGAAAGCCTTA
TCCAGGCTTTTAAAATTACTCTTTCCAGACTTCATCTGAGACTCCTTCTTCAGCCAACATTCCTTAGCCC
TGAATACATTTCCTATCCTCATCTTTCCCTTCTTTTTTTTCCTTTCTTTTACATGTTTAAATTTAAACCA
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GTCCTGTCTCTCTGAACTTTGGGGTCCTATGCATTAAATAATTTTCCTGACGAGCTCAAGTGCTCCCTCT
GGTCTACAATCCCTGGCGGCTGGCCTTCATCCCTTGGGCAAGCATTGCATACAGCTCATGGCCCTCCCTC
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CACCTATCTAAGCCATTTTAACCCTCGGGATTACCTAGAAAAATATTACAAGTTTGGTTCTAGGCACTCT
GCAGAAAGCCAGATTCTTAAGCACCTTCTGAAAAATCTTTTCAAGATATTCTGCCTAGACGGTGTGAAGG
GAGACCTGCTGATTGACATCGGCTCTGGCCCCACTATCTATCAGCTCCTCTCTGCTTGTGAATCCTTTAA
GGAGATCGTCGTCACTGACTACTCAGACCAGAACCTGCAGGAGCTGGAGAAGTGGCTGAAGAAAGAGCCA
GAGGCCTTTGACTGGTCCCCAGTGGTGACCTATGTGTGTGATCTTGAAGGGAACAGAGTCAAGGGTCCAG
AGAAGGAGGAGAAGTTGAGACAGGCGGTCAAGCAGGTGCTGAAGTGTGATGTGACTCAGAGCCAGCCACT

GGGGGCCGTCCCCTTACCCCCGGCTGACTGCGTGCTCAGCACACTGTGTCTGGATGCCGCCTGCCCAGAC
CTCCCCACCTACTGCAGGGCGCTCAGGAACCTCGGCAGCCTACTGAAGCCAGGGGGCTTCCTGGTGATCA
TGGATGCGCTCAAGAGCAGCTACTACATGATTGGTGAGCAGAAGTTCTCCAGCCTCCCCCTGGGCCGGGA
GGCAGTAGAGGCTGCTGTGAAAGAGGCTGGCTACACAATCGAATGGTTTGAGGTGATCTCGCAAAGTTAT
TCTTCCACCATGGCCAACAACGAAGGACTTTTCTCCCTGGTGGCGAGGAAGCTGAGCAGACCCCTGTGAT
GCCTGTGACCTCAATTAAAGCAATTCCTTTGACCTGTCA

NM_006006.4| Homo sapiens zinc finger and BTB domain containing 16 (ZBTB16), transcript variant 1, mRNA
GCAGCAGAGAGGAGTTGAGGGCGATGAGAGCGGGTACTGCGAACTGCCGGGCGATGCTGTCGCTGCCGCC
GTGATACGGAGAGCAACAGTTCCCCAGCAACACCCCTCCCCGACACAGGCACACACCCCCCGACAGGCAC
GCACACCCACCCCACAGTGCCCGGCTCGGCTGCGCCTCCTCTATTGGCCCAGGAAGCCCACCCAGCCCCG
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GAGACCATCCAGGCCTCAGACGACAATGACACGGAGGCCACCATGGCCGATGGCGGGGCCGAGGAAGAAG
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GACCACCCCTACGAGTGTGAGTTCTGTGGCAGCTGCTTCCGGGATGAGAGCACACTCAAGAGCCACAAAC
GCATCCACACGGGTGAGAAACCCTACGAGTGCAATGGCTGTGGCAAGAAGTTCAGCCTCAAGCATCAGCT
GGAGACGCACTATAGGGTGCACACAGGTGAGAAGCCCTTTGAGTGTAAGCTCTGCCACCAGCGCTCCCGG
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CAGAGTACTGCCCCAGCCTCTCCTCCATGCAGAAGCACATGAAGGGCCACAAGCCCGAGGAGATCCCGCC
CGACTGGAGGATAGAGAAGACGTACCTCTACCTGTGCTATGTGTGAAGGGAGGCCCGCGGCGGTGGAGCC
GAGCGGGGAGCCAGGAAAGAAGAGTTGGAGTGAGATGAAGGAAGGACTATGACAAATAAAAAAGGAAAAG
AAAAAAAAAAACAGAAGGAAAAGGAAAAAAAAAAAA

NM_000190.3| Homo sapiens hydroxymethylbilane synthase (HMBS), transcript variant 1, mRNA
CCGGAAGTGACGCGAGGCTCTGCGGAGACCAGGAGTCAGACTGTAGGACGACCTCGGGTCCCACGTGTCC
CCGGTACTCGCCGGCCGGAGCCCCCGGCTTCCCGGGGCCGGGGGACCTTAGCGGCACCCACACACAGCCT
ACTTTCCAAGCGGAGCCATGTCTGGTAACGGCAATGCGGCTGCAACGGCGGAAGAAAACAGCCCAAAGAT
GAGAGTGATTCGCGTGGGTACCCGCAAGAGCCAGCTTGCTCGCATACAGACGGACAGTGTGGTGGCAACA
TTGAAAGCCTCGTACCCTGGCCTGCAGTTTGAAATCATTGCTATGTCCACCACAGGGGACAAGATTCTTG
ATACTGCACTCTCTAAGATTGGAGAGAAAAGCCTGTTTACCAAGGAGCTTGAACATGCCCTGGAGAAGAA
TGAAGTGGACCTGGTTGTTCACTCCTTGAAGGACCTGCCCACTGTGCTTCCTCCTGGCTTCACCATCGGA
GCCATCTGCAAGCGGGAAAACCCTCATGATGCTGTTGTCTTTCACCCAAAATTTGTTGGGAAGACCCTAG
AAACCCTGCCAGAGAAGAGTGTGGTGGGAACCAGCTCCCTGCGAAGAGCAGCCCAGCTGCAGAGAAAGTT
CCCGCATCTGGAGTTCAGGAGTATTCGGGGAAACCTCAACACCCGGCTTCGGAAGCTGGACGAGCAGCAG
GAGTTCAGTGCCATCATCCTGGCAACAGCTGGCCTGCAGCGCATGGGCTGGCACAACCGGGTGGGGCAGA
TCCTGCACCCTGAGGAATGCATGTATGCTGTGGGCCAGGGGGCCTTGGGCGTGGAAGTGCGAGCCAAGGA
CCAGGACATCTTGGATCTGGTGGGTGTGCTGCACGATCCCGAGACTCTGCTTCGCTGCATCGCTGAAAGG
GCCTTCCTGAGGCACCTGGAAGGAGGCTGCAGTGTGCCAGTAGCCGTGCATACAGCTATGAAGGATGGGC

AACTGTACCTGACTGGAGGAGTCTGGAGTCTAGACGGCTCAGATAGCATACAAGAGACCATGCAGGCTAC
CATCCATGTCCCTGCCCAGCATGAAGATGGCCCTGAGGATGACCCACAGTTGGTAGGCATCACTGCTCGT
AACATTCCACGAGGGCCCCAGTTGGCTGCCCAGAACTTGGGCATCAGCCTGGCCAACTTGTTGCTGAGCA
AAGGAGCCAAAAACATCCTGGATGTTGCACGGCAGCTTAACGATGCCCATTAACTGGTTTGTGGGGCACA
GATGCCTGGGTTGCTGCTGTCCAGTGCCTACATCCCGGGCCTCAGTGCCCCATTCTCACTGCTATCTGGG
GAGTGATTACCCCGGGAGACTGAACTGCAGGGTTCAAGCCTTCCAGGGATTTGCCTCACCTTGGGGCCTT
GATGACTGCCTTGCCTCCTCAGTATGTGGGGGCTTCATCTCTTTAGAGAAGTCCAAGCAACAGCCTTTGA
ATGTAACCAATCCTACTAATAAACCAGTTCTGAAGGTGTAAAAAAAAAAAAAAAAA

NM_002842.2| Homo sapiens protein tyrosine phosphatase, receptor type, H (PTPRH), mRNA
CTAGGCCTGGGACTCCTGGGTCCCCGGCAGTGTCTGGAGGCATGGCTGGGGCTGGCGGGGGCCTCGGGGT
CTGGGGGAACCTGGTGCTGCTGGGCCTGTGCAGCTGGACAGGGGCCAGGGCGCCTGCCCCCAACCCAGGG
AGGAACCTGACAGTGGAGACTCAGACCACCAGCTCCATCTCCCTGAGCTGGGAGGTCCCCGATGGCCTAG
ACTCACAGAACTCCAACTACTGGGTTCAGTGTACTGGAGACGGCGGCACAACAGAGACTCGAAACACAAC
AGCCACCAACGTCACCGTGGATGGCCTTGGACCCGGGTCATTGTATACGTGTTCTGTGTGGGTGGAGAAA
GACGGAGTAAATAGCTCTGTGGGGACTGTCACTACTGCCACAGCTCCCAACCCAGTGAGGAACCTGAGAG
TGGAGGCTCAGACCAACAGCTCCATCGCCCTGACCTGGGAGGTCCCCGACGGCCCAGACCCACAGAACTC
CACCTACGGGGTTGAGTACACTGGAGATGGTGGCAGAGCAGGGACTCGAAGCACAGCACACACTAACATC
ACCGTGGATGGACTTGAACCCGGGTGTTTGTATGCGTTTTCCATGTGGGTGGGAAAGAATGGAATCAACA
GCTCCCGGGAGACTCGAAATGCCACCACAGCTCACAACCCAGTGAGGAACCTGAGAGTGGAGGCTCAGAC
CACCAGCTCCATCTCCCTGAGCTGGGAGGTCCCCGATGGCACAGACCCACAGAACTCGACCTACTGCGTT
CAGTGCACTGGAGATGGTGGCAGAACAGAGACTCGAAACACAACAGACACCAGAGTCACCGTGGATGGCC
TTGGACCCGGGTCATTGTATACGTGTTCTGTGTGGGTGGAGAAAGACGGAGTAAATAGCTCTGTGGAGAT
TGTCACTAGTGCCACAGCTCCCAACCCAGTGAGAAACCTGACAGTGGAGGCTCAGACCAACAGCTCCATC
GCCCTGACCTGGGAGGTCCCCGATGGCCCAGACCCACAGAACTCCACCTACGGGGTTGAGTACACTGGAG
ATGGTGGCAGAGCAGGGACTCGAAGCACAGCACACACCAACATCACCGTGGATAGACTTGAACCCGGGTG
TTTGTATGTGTTTTCCGTGTGGGTGGGGAAGAATGGAATCAACAGCTCCCGGGAGACTCGAAATGCCACC
ACAGCCCCCAACCCAGTGAGAAACCTCCATATGGAGACTCAGACCAACAGCTCCATCGCCCTATGCTGGG
AAGTCCCCGATGGCCCATACCCTCAGGACTACACCTACTGGGTAGAGTACACTGGAGACGGTGGTGGCAC
AGAGACCCGAAACACAACAAATACCAGTGTGACAGCTGAGAGACTTGAGCCCGGAACCTTGTACACATTC
TCTGTATGGGCAGAAAAAAATGGAGCACGTGGCTCCAGGCAGAATGTCAGCATCTCCACAGTCCCCAACG
CAGTGACAAGCCTCAGCAAGCAGGACTGGACCAACAGCACCATTGCTTTGCGCTGGACAGCTCCCCAGGG
CCCAGGCCAGTCTTCCTACAGCTACTGGGTCTCATGGGTCAGGGAAGGCATGACTGACCCCAGGACCCAA
AGCACCTCAGGTACTGACATCACCCTAAAGGAACTGGAAGCTGGCAGCCTGTACCACCTCACCGTCTGGG
CCGAGAGGAATGAGGTCAGAGGCTATAACAGCACCCTCACTGCAGCCACTGCTCCCAATGAGGTCACAGA
TCTCCAGAATGAAACTCAGACTAAGAACTCAGTCATGCTGTGGTGGAAGGCCCCTGGAGACCCCCACTCT
CAGTTGTACGTATACTGGGTCCAGTGGGCCAGCAAGGGACATCCCCGGAGGGGGCAAGATCCCCAAGCGA
ATTGGGTCAACCAGACCAGCAGGACCAATGAGACGTGGTACAAAGTGGAGGCCCTGGAACCCGGGACGTT
GTACAATTTCACCGTGTGGGCAGAGAGGAATGACGTAGCCAGTTCCACGCAGAGCCTCTGTGCGTCCACA
TACCCAGACACAGTCACCATCACTTCCTGTGTCAGCACCTCAGCGGGCTATGGAGTCAACTTGATCTGGT
CCTGCCCCCAGGGAGGCTACGAGGCCTTTGAGTTGGAGGTGGGAGGACAGCGGGGCTCCCAGGACAGATC
TTCATGTGGGGAGGCTGTGTCTGTGTTGGGTCTCGGGCCGGCTCGGTCCTACCCAGCCACCATCACGACC
ATCTGGGACGGAATGAAGGTCGTGTCTCACTCTGTGGTCTGCCACACCGAGAGTGCAGGGGTCATTGCCG
GAGCCTTTGTGGGCATCCTCCTGTTTCTCATCCTCGTGGGCCTGCTGATTTTCTTCCTGAAGAGGAGGAA
TAAGAAGAAGCAGCAGAAACCAGAACTCAGGGATCTGGTCTTTAGCTCCCCAGGGGACATCCCAGCTGAA
GACTTCGCTGACCACGTCAGGAAGAATGAGAGGGACAGCAACTGTGGTTTTGCAGACGAGTACCAGCAAC
TCTCCCTGGTGGGCCACAGCCAGTCTCAGATGGTGGCTTCGGCTTCAGAGAACAACGCCAAGAACCGCTA
CAGAAATGTGCTGCCCTATGACTGGTCCCGGGTGCCCCTGAAGCCCATCCATGAGGAGCCAGGCTCTGAC
TACATCAATGCCAGCTTCATGCCCGGTCTCTGGAGCCCCCAGGAGTTCATTGCAACCCAGGGTCCCCTGC
CACAGACAGTGGGTGACTTCTGGCGCCTGGTGTGGGAACAGCAGAGCCACACCCTGGTCATGCTGACCAA
CTGCATGGAGGCCGGCCGGGTGAAGTGTGAGCATTACTGGCCTCTGGACTCGCAGCCCTGCACCCATGGG
CACCTGCGGGTAACCCTGGTAGGTGAGGAAGTGATGGAGAACTGGACGGTGCGGGAACTGCTGCTCCTCC
AGGTGGAGGAGCAGAAGACACTGTCTGTGCGCCAATTCCACTACCAGGCCTGGCCGGATCACGGCGTTCC
CTCCTCCCCAGACACCTTGCTGGCTTTCTGGAGGATGCTTCGGCAGTGGCTGGATCAGACCATGGAGGGA
GGCCCACCCATTGTGCACTGCAGTGCTGGCGTGGGTCGCACAGGAACCCTCATTGCCCTGGACGTCCTGC
TCCGGCAGCTGCAGTCCGAGGGTCTCCTTGGGCCCTTCAGCTTTGTAAGGAAGATGAGAGAGAGTCGGCC
GTTGATGGTGCAGACTGAGGCTCAGTACGTATTCCTGCATCAGTGCATCCTGCGGTTCCTCCAACAGTCA
GCCCAGGCCCCAGCCGAGAAGGAAGTCCCGTATGAGGATGTCGAAAACCTCATCTACGAGAACGTGGCCG
CCATCCAGGCCCACAAGTTGGAGGTCTAAGTGACGAGGGGGCTGGGTCGGCAGCCCAGGCATCCTCAAGC
TCTGGACACCCACTTGAGCCCAGATTCCTGGAAGAGCAGAGGGCTGGGCTCCCAGACTCCTGGGTGCTGT

GGGAGGAGGGGGCTGGTATCCCAAACTCTGGTTTCCCCAGGAGAGAGTGGTCTGGTGGGCTTCAGATGAG
TCCTATGGGAGCTGGGGATCTGGATTCCTGGTTCCCTGAAGGAGGAGAGGGATGATAGCTTGGATTCCCT
AGGTCTTTCCAGGATGCAGAAAGAAACAGGCTGGGGCCTGGATTCTGAGGCAGGAAGGAATTTGGGTCTG
GAGTTCTGGCTACTTGAGGACCAAAGGCAGGAAGGATCCTGCCTTGATTTTACTTCAGAAACCAAATCAG
TCTTCTATAATCTGGGGTCGGAGGGAGTCCCTGTGCCCAAGGTCTCTCTGCACCCCACCATCCACATGTA
TTTTTCCTTCTATCCCATAATTTATTAAATCACTGTTCTCCCCAG

NM_003931.2| Homo sapiens WAS protein family, member 1 (WASF1), transcript variant 1, mRNA
AGCAGTGTTCTCTTCGTCCCCCTCCCCCAAACTGAGGATTGGGCAATACCACAGAACCTCAGGAAAGGGG
GGAAGAGCGAGCTTCGGCCCCACTAATGGGGGAGTGGGCGGAGGCTGGATTTCCCACCTCGGCTGCACCT
GGGCACTGGAGGCTGAAGAGGAAAGTGAGAATCTGAAGTTTTGAGACCTCTGACTGGCCAGGAATAGCTC
CTGGGGCGGGGGGCAAGGATGGGACCATAGGCGGAAAGAGTCTCGCGGTCCCCCCTGCTTCTGGCGCGGG
TCCCTGCGCCCGGTTGTGGAGCGTCTCGCGCGGGGAGGGGGCGGGGGGAACGGCAGCTCGCGGTGTTGTT
CACTCGCGCGTCGAGCACACGGTGGGTCCGGCGGCGGGTTGGCGCCCCAGGCGGCGTTCCCTGTGGCCTG
GCGCCTGGGCCGCTGCCCTGAGCGGGTTCCGCCCCAGAGCCCGACCCTCCTGGGGGCTCTAGGCGGAGTC
CCGCGAGCCGAGGGGGACCGGCGACCGCTGCCGAAGCATGAAGAAGGGGTAAGGCGTGAGCCCCCAAGAT
TTCACGCATGCCCCCTAGCTTCGGTACTCTGACACCTTCTCTTGCACTTGCGGATGATGAACTGGAATAA
CGATGAAAGAAAGCACATCCGATCTCAACATTCACGTCCTGCCCTATAACCGATTAATTAATTGATCCCC
AGCTAGACTAGTGTTGGAGAAATCAGCATGTTAAAACAACTGTTGATGATAGCTGTTGGAGTAAAGTTGC
AGTGGAAGCTATGGCTGCAAAATCGTTAAAATCTTCAAGGTGAACTGGCACAAAGGTTAATCTCAAGATG
CCGCTAGTGAAAAGAAACATCGATCCTAGGCACTTGTGCCACACAGCACTGCCTAGAGGCATTAAGAATG
AACTGGAATGTGTAACCAATATTTCCTTGGCAAATATAATTAGACAACTAAGTAGCCTAAGTAAATATGC
TGAAGATATATTTGGAGAATTATTCAATGAAGCACATAGTTTTTCCTTCAGAGTCAACTCATTGCAAGAA
CGTGTGGACCGTTTATCTGTTAGTGTTACACAGCTTGATCCAAAGGAAGAAGAATTGTCTTTGCAAGATA
TAACAATGAGGAAAGCTTTCCGAAGTTCTACAATTCAAGACCAGCAGCTTTTCGATCGCAAGACTTTGCC
TATTCCATTACAGGAGACGTACGATGTTTGTGAACAGCCTCCACCTCTCAATATACTCACTCCTTATAGA
GATGATGGTAAAGAAGGTCTGAAGTTTTATACCAATCCTTCGTATTTCTTTGATCTATGGAAAGAAAAAA
TGTTGCAAGATACAGAGGATAAGAGGAAGGAAAAGAGGAAGCAGAAGCAGAAAAATCTAGATCGTCCTCA
TGAACCAGAAAAAGTGCCAAGAGCACCTCATGACAGGCGGCGAGAATGGCAGAAGCTGGCCCAAGGTCCA
GAGCTGGCTGAAGATGATGCTAATCTCTTACATAAGCATATTGAAGTTGCTAATGGCCCAGCCTCTCATT
TTGAAACAAGACCTCAGACATACGTGGATCATATGGATGGATCTTACTCACTTTCTGCCTTGCCATTTAG
TCAGATGAGTGAGCTTCTGACTAGAGCTGAGGAAAGGGTATTAGTCAGACCACATGAACCACCTCCACCT
CCACCAATGCATGGAGCAGGAGATGCAAAACCGATACCCACCTGTATCAGTTCTGCTACAGGTTTGATAG
AAAATCGCCCTCAGTCACCAGCTACAGGCAGAACACCTGTGTTTGTGAGCCCCACTCCCCCACCTCCTCC
ACCACCTCTTCCATCTGCCTTGTCAACTTCCTCATTAAGAGCTTCAATGACTTCAACTCCTCCCCCTCCA
GTACCTCCCCCACCTCCACCTCCAGCCACTGCTTTGCAAGCTCCAGCAGTACCACCACCTCCAGCTCCTC
TTCAGATTGCCCCTGGAGTTCTTCACCCAGCTCCTCCTCCAATTGCACCTCCTCTAGTACAGCCCTCTCC
ACCAGTAGCTAGAGCTGCCCCAGTATGTGAGACTGTACCAGTTCATCCACTCCCACAAGGTGAAGTTCAG
GGGCTGCCTCCACCCCCACCACCGCCTCCTCTGCCTCCACCTGGCATTCGACCATCATCACCTGTCACAG
TTACAGCTCTTGCTCATCCTCCCTCTGGGCTACATCCAACTCCATCTACTGCCCCAGGTCCCCATGTTCC
ATTAATGCCTCCATCTCCTCCATCACAAGTTATACCTGCTTCTGAGCCAAAGCGCCATCCATCAACCCTA
CCTGTAATCAGTGATGCCAGGAGTGTGCTACTGGAAGCAATACGAAAAGGTATTCAGCTACGCAAAGTAG
AAGAGCAGCGTGAACAGGAAGCTAAGCATGAACGCATTGAAAACGATGTTGCCACCATCCTGTCTCGCCG
TATTGCTGTTGAATATAGTGATTCGGAAGATGATTCAGAATTTGATGAAGTAGATTGGTTGGAGTAAGAA
AAATGCATTGATAAATATTACAAAACTGAATGCAAATGTCCTTTGTGGTGCTTGTTCCTTGAAAATGTTT
GGTCATTCTAGTGTTTTGCTTTCTTTTCCTTATAATAAATGACCCTTTTCCTCCATAACTTTTGATTTCT
AAGGAAAATATTAGCATACATTTCAAACTAAATGTTTTACAGTGGCTTATCTTTTTTTTCCCCCTGAAAA
GACTAATTTGGTCAAATAAACCACTAAGTATTAAGCATGGACAGCTGTTGTTAGAGTAGCAGATTCAGTT
TTTTGATATATCTTAATTGTGTACTTTGTGAATTTTAATTTAAAGAAAGCAACTGAAATTGAAATCTTGA
GGGCAGCTGTGTCTACTAATGAGCCTTATTCCATTTCCTGATGTTTTAAAAGAAGAAACACTGCCTTGAT
TATACGAATACACTCAGAAAGTACATTTAGCTTGTAGTGTTGAATTCTCTTAAAGGAATGCTTGAATTTT
TTCATTATTGTTTTATTGTTTTTATATACTTGCCTTATTTGAATGTTTAGCAGTATCCCCTTCCCACTTA
TATATTGTGTGATATGATTTTGCTTGCCTATAGGAGTTAAAAACTTTTCCATGTGAAATACTCTGACTTA
AACATACATGTAACTTACATAACTGTTAAGAATAACAGTCTGATTTAATAAATGGTTCATTTTAAAAGTT
AAAAAAAAAA

NM_001069.2| Homo sapiens tubulin, beta 2A (TUBB2A), mRNA
GCGGCAGGTCTCTGCGCAGCCCAGCCCGCCGGTCCACGCCGCGCACCGCTCCGAGGGCCAGCGCCACCCG
CTCCGCAGCCGGCACCATGCGCGAGATCGTGCACATCCAGGCGGGCCAGTGCGGCAACCAGATCGGCGCC
AAGTTTTGGGAGGTCATCAGCGATGAGCATGGGATCGACCCCACAGGCAGTTACCATGGAGACAGTGACT

Figure 64 (Continued) 175/290

TGCAGCTGGAGAGAATCAACGTGTACTACAATGAGGCTGCTGGTAACAAATATGTACCTCGGGCCATCCT
GGTGGATCTGGAGCCTGGCACCATGGACTCTGTCAGGTCTGGACCCTTCGGCCAGATCTTCAGACCAGAC
AACTTCGTGTTCGGCCAGAGTGGAGCCGGGAATAACTGGGCCAAGGGCCACTACACAGAGGGAGCCGAGC
TGGTCGACTCGGTCCTGGATGTGGTGAGGAAGGAGTCAGAGAGCTGTGACTGTCTCCAGGGCTTCCAGCT
GACCCACTCTCTGGGGGGCGGCACGGGGTCCGGGATGGGCACCCTGCTCATCAGCAAGATCCGGGAAGAG
TACCCAGACCGCATCATGAACACCTTCAGCGTCATGCCCTCACCCAAGGTGTCAGACACGGTGGTGGAGC
CCTACAACGCCACCCTCTCTGTCCACCAGCTGGTGGAAAACACAGATGAAACCTACTCCATTGATAACGA
GGCCCTGTATGACATCTGCTTCCGCACCCTGAAGCTGACCACCCCCACCTACGGGGACCTCAACCACCTG
GTGTCGGCCACCATGAGCGGGGTCACCACCTGCCTGCGCTTCCCGGGCCAGCTGAACGCAGACCTGCGCA
AGCTGGCGGTGAACATGGTGCCCTTCCCTCGCCTGCACTTCTTCATGCCCGGCTTCGCGCCCCTGACCAG
CCGGGGCAGCCAGCAGTACCGGGCGCTCACGGTGCCCGAGCTCACCCAGCAGATGTTCGACTCCAAGAAC
ATGATGGCCGCCTGCGACCCGCGCCACGGCCGCTACCTGACGGTGGCTGCCATCTTCCGGGGCCGCATGT
CCATGAAGGAGGTGGACGAGCAGATGCTCAACGTGCAGAACAAGAACAGCAGCTACTTCGTGGAGTGGAT
CCCCAACAACGTGAAGACGGCCGTGTGCGACATCCCGCCCCGCGGCCTGAAGATGTCGGCCACCTTCATC
GGCAACAGCACGGCCATCCAGGAGCTGTTCAAGCGCATCTCCGAGCAGTTCACGGCCATGTTCCGGCGCA
AGGCCTTCCTGCACTGGTACACGGGCGAGGGCATGGACGAGATGGAGTTCACCGAGGCCGAGAGCAACAT
GAACGACCTGGTGTCCGAGTACCAGCAGTACCAGGACGCCACGGCCGACGAACAAGGGGAGTTCGAGGAG
GAGGAGGGCGAGGACGAGGCTTAAAAACTTCTCAGATCAATCGTGCATCCTTAGTGAACTTCTGTTGTCC
TCAAGCATGGTCTTTCTACTTGTAAACTATGGTGCTCAGTTTTGCCTCTGTTAGAAATTCACACTGTTGA
TGTAATGATGTGGAACTCCTCTAAAAATTACAGTATTGTCTGTGAAGGTATCTATACTAATAAAAAAGCA
TGTGTAGAAAA

NM_002385.2| Homo sapiens myelin basic protein (MBP), transcript variant 2, mRNA
GGACAACACCTTCAAAGACAGGCCCTCTGAGTCCGACGAGCTCCAGACCATCCAAGAAGACAGTGCAGCC
ACCTCCGAGAGCCTGGATGTGATGGCGTCACAGAAGAGACCCTCCCAGAGGCACGGATCCAAGTACCTGG
CCACAGCAAGTACCATGGACCATGCCAGGCATGGCTTCCTCCCAAGGCACAGAGACACGGGCATCCTTGA
CTCCATCGGGCGCTTCTTTGGCGGTGACAGGGGTGCGCCCAAGCGGGGCTCTGGCAAGGTACCCTGGCTA
AAGCCGGGCCGGAGCCCTCTGCCCTCTCATGCCCGCAGCCAGCCTGGGCTGTGCAACATGTACAAGGACT
CACACCACCCGGCAAGAACTGCTCACTACGGCTCCCTGCCCCAGAAGTCACACGGCCGGACCCAAGATGA
AAACCCCGTAGTCCACTTCTTCAAGAACATTGTGACGCCTCGCACACCACCCCCGTCGCAGGGAAAGGGG
GCCGAAGGCCAGAGACCAGGATTTGGCTACGGAGGCAGAGCGTCCGACTATAAATCGGCTCACAAGGGAT
TCAAGGGAGTCGATGCCCAGGGCACGCTTTCCAAAATTTTTAAGCTGGGAGGAAGAGATAGTCGCTCTGG
ATCACCCATGGCTAGACGCTGAAAACCCACCTGGTTCCGGAATCCTGTCCTCAGCTTCTTAATATAACTG
CCTTAAAACTTTAATCCCACTTGCCCCTGTTACCTAATTAGAGCAGATGACCCCTCCCCTAATGCCTGCG
GAGTTGTGCACGTAGTAGGGTCAGGCCACGGCAGCCTACCGGCAATTTCCGGCCAACAGTTAAATGAGAA
CATGAAAACAGAAAACGGTTAAAACTGTCCCTTTCTGTGTGAAGATCACGTTCCTTCCCCCGCAATGTGC
CCCCAGACGCACGTGGGTCTTCAGGGGGCCAGGTGCACAGACGTCCCTCCACGTTCACCCCTCCACCCTT
GGACTTTCTTTTCGCCGTGGCTGCGGCACCCTTGCGCTTTTGCTGGTCACTGCCATGGAGGCACACAGCT
GCAGAGACAGAGAGGACGTGGGCGGCAGAGAGGACTGTTGACATCCAAGCTTCCTTTGTTTTTTTTTCCT
GTCCTTCTCTCACCTCCTAAAGTAGACTTCATTTTTCCTAACAGGATTAGACAGTCAAGGAGTGGCTTAC
TACATGTGGGAGCTTTTGGTATGTGACATGCGGGCTGGGCAGCTGTTAGAGTCCAACGTGGGGCAGCACA
GAGAGGGGGCCACCTCCCCAGGCCGTGGCTGCCCACACACCCCAATTAGCTGAATTCGCGTGTGGCAGAG
GGAGGAAAAGGAGGCAAACGTGGGCTGGGCAATGGCCTCACATAGGAAACAGGGTCTTCCTGGAGATTTG
GTGATGGAGATGTCAAGCAGGTGGCCTCTGGACGTCACCGTTGCCCTGCATGGTGGCCCCAGAGCAGCCT
CTATGAACAACCTCGTTTCCAAACCACAGCCCACAGCCGGAGAGTCCAGGAAGACTTGCGCACTCAGAGC
AGAAGGGTAGGAGTCCTCTAGACAGCCTCGCAGCCGCGCCAGTCGCCCATAGACACTGGCTGTGACCGGG
CGTGCTGGCAGCGGCAGTGCACAGTGGCCAGCACTAACCCTCCCTGAGAAGATAACCGGCTCATTCACTT
CCTCCCAGAAGACGCGTGGTAGCGAGTAGGCACAGGCGTGCACCTGCTCCCGAATTACTCACCGAGACAC
ACGGGCTGAGCAGACGGCCCCGTGGATGGAGACAAAGAGCTCTTCTGACCATATCCTTCTTAACACCCGC
TGGCATCTCCTTTCGCGCCTCCCTCCCTAACCTACTGACCCACCTTTTGATTTTAGCGCACCTGTGATTG
ATAGGCCTTCCAAAGAGTCCCACGCTGGCATCACCCTCCCCGAGGACGGAGATGAGGAGTAGTCAGCGTG
ATGCCAAAACGCGTCTTCTTAATCCAATTCTAATTCTGAATGTTTCGTGTGGGCTTAATACCATGTCTAT
TAATATATAGCCTCGATGATGAGAGAGTTACAAAGAACAAAACTCCAGACACAAACCTCCAAATTTTTCA
GCAGAAGCACTCTGCGTCGCTGAGCTGAGGTCGGCTCTGCGATCCATACGTGGCCGCACCCACACAGCAC
GTGCTGTGACGATGGCTGAACGGAAAGTGTACACTGTTCCTGAATATTGAAATAAAACAATAAACTTTTA
ATGGTAAAAAAAAAAAAAAAAAAAAA

NM_003271.4| Homo sapiens tetraspanin 4 (TSPAN4), transcript variant 5, mRNA
AAAGCTGCGGCGGCGGTTCGCGTTTCTCGTGTCCGCTTGACTGACAGCTGCGCGGCGGGAGCGGGCGGCG

Figure 64 (Continued)

CGAGCGGGAGGCGGCGGCGCAGAGCTTGGGGCTTCCTTGGTCGCACCCACCACCTGCCTGCCCACTGGTC
AGCCTTCAGGGACCCTGAGCACCGCCTGGTCTCTTTCCTGTGGCCAGCCCAGAACTGAAGCGCTGCGGCA
TGGCGCGCGCCTGCCTCCAGGCCGTCAAGTACCTCATGTTCGCCTTCAACCTGCTCTTCTGGCTGGGAGG
CTGTGGCGTGCTGGGTGTCGGCATCTGGCTGGCCGCCACACAGGGGAGCTTCGCCACGCTGTCCTCTTCC
TTCCCGTCCCTGTCGGCTGCCAACTTGCTCATCATCACCGGCGCCTTTGTCATGGCCATCGGCTTCGTGG
GCTGCCTGGGTGCCATCAAGGAGAACAAGTGCCTCCTGCTCACTTTCTTCCTGCTGCTGCTGCTGGTGTT
CCTGCTGGAGGCCACCATCGCCATCCTCTTCTTCGCCTACACGGACAAGATTGACAGGTATGCCCAGCAA
GACCTGAAGAAAGGCTTGCACCTGTACGGCACGCAGGGCAACGTGGGCCTCACCAACGCCTGGAGCATCA
TCCAGACCGACTTCCGCTGCTGTGGCGTCTCCAACTACACTGACTGGTTCGAGGTGTACAACGCCACGCG
GGTACCTGACTCCTGCTGCTTGGAGTTCAGTGAGAGCTGTGGGCTGCACGCCCCCGGCACCTGGTGGAAG
GCGCCGTGCTACGAGACGGTGAAGGTGTGGCTTCAGGAGAACCTGCTGGCTGTGGGCATCTTTGGGCTGT
GCACGGCGCTGGTGCAGATCCTGGGCCTGACCTTCGCCATGACCATGTACTGCCAAGTGGTCAAGGCAGA
CACCTACTGCGCGTAGGCCGCCCACCGCCCGCTTCTCTGCCAAAAGGACGCCCACGGGGAGATGGCCGCA
CCCACAGCTGCCTTTCCCACCACCAGCCTCGGTGCTCTGCCCCATGCTGGGAGGAGGGAGGGAGGGACAG
GTGCCTGGAGCCCCCGGAACCCTGTTTCTGGAAGGCCCTAGCTCAGGTGGCTTCAGGGCCTCCGGACCCC
CCCTGGGAGGGGTGGCCACGTGCTGGCTGCGGAACCCAGGGCAGGGGTGGGAGGGGCCTCCAGCACTTTT
TATATTTACGTATTCTCCAAAGCAGTGTTCACACGGGAGCCAGCCTGTGGCCCCCAGCCTCCTGGAAAAC
AGGTTGGCGCTGGAGGAGCCGGGTCTTGGCATCCTGGAGGTGGCCCCACTGGTCCTGGTGCTCCAGGCGG
GGCCGTGGACCCCTCACCTACATTCCATAGTGGGCCCGTGGGGCTCCTGGTGCATCTTAATAAAGTGTGA
GCAGCAACCTTGAAAAAAAAAAAAAAAAA

NM_001813.2| Homo sapiens centromere protein E, 312kDa (CENPE), mRNA
TAAATTTAAAGGCGGGGCGGCCTGTGAGCCCTGAAGTGCCGGCCGCGGAGGGTCCTGGCCATTTTCCTGG
GACCAGTTCAGCCTGATAGGATGGCGGAGGAAGGAGCCGTGGCCGTCTGCGTGCGAGTGCGGCCGCTGAA
CAGCAGAGAAGAATCACTTGGAGAAACTGCCCAAGTTTACTGGAAAACTGACAATAATGTCATTTATCAA
GTTGATGGAAGTAAATCCTTCAATTTTGATCGTGTCTTTCATGGTAATGAAACTACCAAAAATGTGTATG
AAGAAATAGCAGCACCAATCATCGATTCTGCCATACAAGGCTACAATGGTACTATATTTGCCTATGGACA
GACTGCTTCAGGAAAAACATATACCATGATGGGTTCAGAAGATCATTTGGGAGTTATACCCAGGGCAATT
CATGACATTTTCCAAAAAATTAAGAAGTTTCCTGATAGGGAATTTCTCTTACGTGTATCTTACATGGAAA
TATACAATGAAACCATTACAGATTTACTCTGTGGCACTCAAAAAATGAAACCTTTAATTATTCGAGAAGA
TGTCAATAGGAATGTGTATGTTGCTGATCTCACAGAAGAAGTTGTATATACATCAGAAATGGCTTTGAAA
TGGATTACAAAGGGAGAAAAGAGCAGGCATTATGGAGAAACAAAAATGAATCAAAGAAGCAGTCGTTCTC
ATACCATCTTTAGGATGATTTTGGAAAGCAGAGAGAAGGGTGAACCTTCTAATTGTGAAGGATCTGTTAA
GGTATCCCATTTGAATTTGGTTGATCTTGCAGGCAGTGAAAGAGCTGCTCAAACAGGCGCTGCAGGTGTG
CGGCTCAAGGAAGGCTGTAATATAAATCGAAGCTTATTTATTTTGGGACAAGTGATCAAGAAACTTAGTG
ATGGACAAGTTGGTGGTTTCATAAATTATCGAGATAGCAAGTTAACACGAATTCTCCAGAATTCCTTGGG
AGGAAATGCAAAGACACGTATTATCTGCACAATTACTCCAGTATCTTTTGATGAAACACTTACTGCTCTC
CAGTTTGCCAGTACTGCTAAATATATGAAGAATACTCCTTATGTTAATGAGGTATCAACTGATGAAGCTC
TCCTGAAAAGGTATAGAAAAGAAATAATGGATCTTAAAAAACAATTAGAGGAGGTTTCTTTAGAGACGCG
GGCTCAGGCAATGGAAAAAGACCAATTGGCCCAACTTTTGGAAGAAAAAGATTTGCTTCAGAAAGTACAG
AATGAGAAAATTGAAAACTTAACACGGATGCTGGTGACCTCTTCTTCCCTCACGTTGCAACAGGAATTAA
AGGCTAAAAGAAAACGAAGAGTTACTTGGTGCCTTGGCAAAATTAACAAAATGAAGAACTCAAACTATGC
AGATCAATTTAATATACCAACAAATATAACAACAAAAACACATAAGCTTTCTATAAATTTATTACGAGAA
ATTGATGAATCTGTCTGTTCAGAGTCTGATGTTTTCAGTAACACTCTTGATACATTAAGTGAGATAGAAT
GGAATCCAGCAACAAAGCTACTAAATCAGGAGAATATAGAAAGTGAGTTGAACTCACTTCGTGCTGACTA
TGATAATCTGGTATTAGACTATGAACAACTACGAACAGAAAAAGAAGAAATGGAATTGAAATTAAAAGAA
AAGAATGATTTGGATGAATTTGAGGCTCTAGAAAGAAAAACTAAAAAAGATCAAGAGATGCAACTAATTC
ATGAAATTTCGAACTTAAAGAATTTAGTTAAGCATGCAGAAGTATATAATCAAGATCTTGAGAATGAACT
CAGTTCAAAAGTAGAGCTGCTTAGAGAAAAGGAAGACCAGATTAAGAAGCTACAGGAATACATAGACTCT
CAAAAGCTAGAAAATATAAAAATGGACTTGTCATACTCATTGGAAAGCATTGAAGACCCAAAACAAATGA
AGCAGACTCTGTTTGATGCTGAAACTGTAGCCCTTGATGCCAAGAGAGAATCAGCCTTTCTTAGAAGTGA
AAATCTGGAGCTGAAGGAGAAAATGAAAGAACTTGCAACTACATACAAGCAAATGGAAAATGATATTCAG
TTATATCAAAGCCAGTTGGAGGCAAAAAAGAAAATGCAAGTTGATCTGGAGAAAGAATTACAATCTGCTT
TTAATGAGATAACAAAACTCACCTCCCTTATAGATGGCAAAGTTCCAAAAGATTTGCTCTGTAATTTGGA
ATTGGAAGGAAAGATTACTGATCTTCAGAAAGAACTAAATAAAGAAGTTGAAGAAAATGAAGCTTTGCGG
GAAGAAGTCATTTTGCTTTCAGAATTGAAATCTTTACCTTCTGAAGTAGAAAGGCTGAGGAAAGAGATAC
AAGACAAATCTGAAGAGCTCCATATAATAACATCAGAAAAAGATAAATTGTTTTCTGAAGTAGTTCATAA
GGAGAGTAGAGTTCAAGGTTTACTTGAAGAAATTGGGAAAACAAAAGATGACCTAGCAACTACACAGTCG
AATTATAAAAGCACTGATCAAGAATTCCAAAATTTCAAAACCCTTCATATGGACTTTGAGCAAAAGTATA
AGATGGTCCTTGAGGAGAATGAGAGAATGAATCAGGAAATAGTTAATCTCTCTAAAGAAGCCCAAAAATT
TGATTCGAGTTTGGGTGCTTTGAAGACCGAGCTTTCTTACAAGACCCAAGAACTTCAGGAGAAAACACGT

Figure 64 (Continued)

GAGGTTCAAGAAAGACTAAATGAGATGGAACAGCTGAAGGAACAATTAGAAAATAGAGATTCTACGCTGC
AAACTGTAGAAAGGGAGAAAACACTGATTACTGAGAAACTGCAGCAAACTTTAGAAGAAGTAAAAACTTT
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NM_001223.3| Homo sapiens caspase 1, apoptosis-related cysteine peptidase (interleukin 1, beta, convertase) (CASP1), transcript variant beta, mRNA
GGGAGGAGAGAAAAGCCATGGCCGACAAGGTCCTGAAGGAGAAGAGAAAGCTGTTTATCCGTTCCATGGG
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NM_004501.3| Homo sapiens heterogeneous nuclear ribonucleoprotein U (scaffold attachment factor A) (HNRNPU), transcript variant 2, mRNA
CTCGCGCCAGGCGAGTCTCCGCGTCTCCCTCGCGAACTCGGTGAAAGGAATTGGCGCCGTTCGACACCAG
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NM_013276.2| Homo sapiens sedoheptulokinase (SHPK), mRNA
GATTGGCAGGCGACTCCCCGCCCCTGCCCCGCCCCCCTGGGCGAAGAGCGCGGACTTGTGGGGCCGCTGG
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GCCTGATCACTCTGGCCAAATGTCAGATACTAAAGCCCATTCTTGGCCGGGCATGTTGGCTCCCGCCTGT
AATCCCAGCACTTTGGGAAGCCCAAGTGGGTGAATCACCTGAGGTCAGGAGTTCAAGACCAGCCTGACCA
ACATGGGGATACCCCGTCTCTACTAAAAATACAAGCCGGGCGTGGTGGCGCATGCCTGTAATCCCAGCTA
CTCAGGAGGCTGAGGCAGGAAAATCACTTGAACTCGGGAGGCAGAGGTTGCAGTGAGCTGAGATCGCGCC
ATTGCACTCCAGCCTGGGTGACAGAGCAAGACTCTGTCTCATAAATAAATACAAAGCCCATTCTTCCAGA
GTCTTGTGCCTTAAATAAAACACACCTCTCTGCTGTGGGAAGACTGTGCAATGGCACAGCCGCAGAGCTT
GGTTTGGGAGGTTGAAGTGCTCTGGGGAGAATTCGTAGATCATCCTCAGAAAAGCCTTGCCCTGGTGTTC
TACCAGAAAAACGTCTCCCAATCACCCAGGAAAGCTGTCCACAGTAGTCCCCCCTTATCCACGGTGTCAC
TTTCCATGGGTTCAGTTATCTGCGGTCAACCACGGTCTGACAATATTAAATGGAAAATTCTTCAAACAGT
TCCCAAGTTTTCCCTTGTGCATTGTTCTGAGCAGTGTGATGGAGAGTCTCTGCCGTGCCATCTGGGATGC
AAACCGTCCCTGTGTTCCCCACGTCCAGGCCGTAGATGCTCCCCGCCGGTCAGTCACTTAGTCGTCAGAT
CGCCCGTCCTGGTATCACAGTGCTTCTGTTCAGGTCAGTCTTTTTTTACTTCATTAGGGCCCCAAAGCAC
CAGAGTAGTGATGCTGGTAGTGATGCTGGCTGTTCGGATATGCCAAAGAGAAACCGTAAGGTGCTTCCTT
TAAGTGAAAAGATGGGAAGAAAACAGTCATATGCTGAATCTTCTATCTGTGAAATTCT

NM_002744.4| Homo sapiens protein kinase C, zeta (PRKCZ), transcript variant 1, mRNA
GCCCCGCGCGCCGCCGGAGTTCCGCGGAGTTGACCGGGTCGGCGCCGTCGGTCCTGAGCGCTGCCTTCCG
CGTTCCGCCGCGGCCCCACCTGGAGCCCCCGCCCCGCGCCATGGCCGGAGCTCCCGGGGCGCAGCGCTGA
CGGCGGCGGGGGGGAGCGCGCCATGCCCAGCAGGACCGGCCCCAAGATGGAAGGGAGCGGCGGCCGCGTCC
GCCTCAAGGCGCATTACGGGGGGGACATCTTCATCACCAGCGTGGACGCCGCCACGACCTTCGAGGAGCT
CTGTGAGGAAGTGAGAGACATGTGTCGTCTGCACCAGCAGCACCCGCTCACCCTCAAGTGGGTGGACAGC
GAAGGTGACCCTTGCACGGTGTCCTCCCAGATGGAGCTGGAAGAGGCTTTCCGCCTGGCCCGTCAGTGCA
GGGATGAAGGCCTCATCATTCATGTTTTCCCGAGCACCCCTGAGCAGCCTGGCCTGCCATGTCCGGGAGA
AGACAAATCTATCTACCGCCGGGGAGCCAGAAGATGGAGGAAGCTGTACCGTGCCAACGGCCACCTCTTC
CAAGCCAAGCGCTTTAACAGGAGAGCGTACTGCGGTCAGTGCAGCGAGAGGATATGGGGCCTCGCGAGGC
AAGGCTACAGGTGCATCAACTGCAAACTGCTGGTCCATAAGCGCTGCCACGGCCTCGTCCCGCTGACCTG
CAGGAAGCATATGGATTCTGTCATGCCTTCCCAAGAGCCTCCAGTAGACGACAAGAACGAGGACGCCGAC
CTTCCTTCCGAGGAGACAGATGGAATTGCTTACATTTCCTCATCCCGGAAGCATGACAGCATTAAAGACG
ACTCGGAGGACCTTAAGCCAGTTATCGATGGGATGGATGGAATCAAAATCTCTCAGGGGCTTGGGCTGCA
GGACTTTGACCTAATCAGAGTCATCGGGCGCGGGAGCTACGCCAAGGTTCTCCTGGTGCGGTTGAAGAAG
AATGACCAAATTTACGCCATGAAAGTGGTGAAGAAAGAGCTGGTGCATGATGACGAGGATATTGACTGGG
TACAGACAGAGAAGCACGTGTTTGAGCAGGCATCCAGCAACCCCTTCCTGGTCGGATTACACTCCTGCTT
CCAGACGACAAGTCGGTTGTTCCTGGTCATTGAGTACGTCAACGGCGGGGACCTGATGTTCCACATGCAG
AGGCAGAGGAAGCTCCCTGAGGAGCACGCCAGGTTCTACGCGGCCGAGATCTGCATCGCCCTCAACTTCC
TGCACGAGAGGGGGATCATCTACAGGGACCTGAAGCTGGACAACGTCCTCCTGGATGCGGACGGGCACAT
CAAGCTCACAGACTACGGCATGTGCAAGGAAGGCCTGGGCCCTGGTGACACAACGAGCACTTTCTGCGGA
ACCCCGAATTACATCGCCCCCGAAATCCTGCGGGGAGAGGAGTACGGGTTCAGCGTGGACTGGTGGGCGC
TGGGAGTCCTCATGTTTGAGATGATGGCCGGGCGCTCCCCGTTCGACATCATCACCGACAACCCGGACAT
GAACACAGAGGACTACCTTTTCCAAGTGATCCTGGAGAAGCCCATCCGGATCCCCCGGTTCCTGTCCGTC
AAAGCCTCCCATGTTTTAAAAGGATTTTTAAATAAGGACCCCAAAGAGAGGCTCGGCTGCCGGCCACAGA
CTGGATTTTCTGACATCAAGTCCCACGCGTTCTTCCGCAGCATAGACTGGGACTTGCTGGAGAAGAAGCA
GGCGCTCCCTCCATTCCAGCCACAGATCACAGACGACTACGGTCTGGACAACTTTGACACACAGTTCACC
AGCGAGCCCGTGCAGCTGACCCCAGACGATGAGGATGCCATAAAGAGGATCGACCAGTCAGAGTTCGAAG
GCTTTGAGTATATCAACCCATTATTGCTGTCCACCGAGGAGTCGGTGTGAGGCCGCGTGCGTCTCTGTCG
TGGACACGCGTGATTGACCCTTTAACTGTATCCTTAACCACCGCATATGCATGCCAGGCTGGGCACGGCT
CCGAGGGCGGCCAGGGACAGACGCTTGCGCCGAGACCGCAGAGGGAAGCGTCAGCGGGCGCTGCTGGGAG
CAGAACAGTCCCTCACACCTGGGCCCGGGCAGGCCAGCTTCGTGCTGGAGGAACTTGCTGCTGTGCCTGC
GTCGCGGCGGATCCGCGGGGACCCTGCCGAGGGGGCTGTCATGCGGTTTCCAAGGTGCACATTTTCCACG
GAAACAGAACTCGATGCACTGACCTGCTCCGCCAGGAAAGTGAGCGTGTAGCGTCCTGAGGAATAAAATG
TTCCGATGATGTGGAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_002152.2| Homo sapiens histidine rich calcium binding protein (HRC), mRNA
CTCCGGAGACAGCCTCAGAGGGGTGTCCAGAGCCAGGCAGCAAGGACCAGAGGAAGGTGGACAGAGACCG

AAAGGACAAAGGAGAGACACAGAGAAAAAAAGAGACCAACCCCAAAGGGACAAAGACCCCAACGTTTGTC
CACATTGCCGCAGAGGCAGCTGGAGCCAGTGCTGCCTGTCCGTCCCCATGGGCCACCATAGGCCATGGCT
GCACGCTTCTGTCCTCTGGGCTGGGGTGGCCAGCCTGCTCCTCCCCCCGGCCATGACCCAGCAGCTCAGA
GGGGATGGGCTAGGCTTCAGAAACCGGAACAACAGCACTGGAGTCGCCGGGCTCTCCGAGGAGGCATCAG
CAGAGCTTCGCCACCACCTCCACAGCCCTAGAGACCATCCAGATGAGAACAAGGATGTTTCCACAGAGAA
TGGGCATCATTTCTGGAGCCACCCAGACCGTGAAAAGGAGGATGAAGATGTCTCCAAGGAATATGGGCAC
CTACTCCCAGGCCACAGGTCCCAAGACCACAAAGTCGGAGATGAGGGTGTCTCAGGTGAGGAGGTCTTTG
CAGAGCATGGTGGGCAGGCCCGTGGGCACAGAGGCCACGGGAGTGAAGACACGGAAGACTCAGCTGAGCA
CAGGCACCACCTCCCCAGCCACAGGAGCCACAGCCATCAAGACGAGGATGAGGATGAAGTTGTGTCCAGT
GAGCATCACCATCATATCCTCAGGCATGGACACCGAGGCCATGATGGGGAAGATGATGAAGGAGAAGAGG
AGGAGGAGGAGGAGGAGGAGGAAGAGGAGGCCTCCACTGAGTATGGACACCAGGCCCACAGGCACCGAGG
CCATGGGAGTGAAGAGGATGAGGATGTCTCAGATGGACACCATCATCATGGCCCCAGCCACAGGCACCAA
GGCCATGAAGAAGATGACGATGATGATGATGATGATGATGATGATGATGATGATGATGTCTCCATTG
AATATAGACACCAGGCTCACAGGCACCAAGGCCACGGGATTGAAGAGGATGAAGATGTCTCAGATGGACA
CCATCATCGCGACCCCAGCCACAGGCACCGAAGCCATGAAGAAGATGACAATGATGATGATGATGTCTCC
ACTGAGTATGGACACCAGGCCCACAGGCACCAAGACCACAGAAAGGAAGAGGTTGAGGCTGTCTCAGGTG
AACACCACCATCATGTCCCTGACCACAGGCACCAAGGCCACAGAGACGAGGAAGAAGATGAGGATGTGTC
CACTGAACGTTGGCACCAGGGTCCCCAACATGTCCACCATGGCCTTGTAGATGAGGAAGAGGAAGAAGAG
GAGATCACAGTCCAGTTCGGCCACTATGTTGCAAGCCACCAACCTCGAGGCCACAAGAGTGATGAAGAGG
ACTTCCAAGATGAGTATAAAACAGAAGTCCCTCACCATCACCACCACAGAGTCCCCAGGGAGGAAGATGA
GGAGGTCTCTGCTGAGCTTGGCCACCAGGCCCCCAGCCACAGGCAAAGCCACCAAGATGAAGAAACTGGC
CATGGTCAAAGAGGGTCCATCAAAGAGATGAGCCATCACCCCCCAGGACACACAGTGGTCAAGGATAGAA
GCCATTTGAGGAAGGATGATTCTGAGGAGGAAAAGGAGAAGGAAGAGGACCCCGGCTCCCATGAGGAAGA
CGATGAAAGTTCAGAGCAGGGAGAGAAAGGCACCCATCATGGCAGCCGGGACCAGGAAGATGAGGAGGAT
GAGGAGGAAGGTCATGGCCTCAGCCTGAACCAGGAGGAGGAAGAAGAGGAAGACAAGGAGGAGGAGGAGG
AGGAAGAAGACGAGGAGAGGAGGGAAGAGAGGGCTGAGGTTGGGGCCCCACTGAGCCCAGACCACAGCGA
GGAGGAAGAGGAGGAGGAGGAGGGGCTGGAGGAAGATGAGCCCCGCTTCACCATCATCCCCAACCCACTG
GACAGGAGAGAGGAGGCTGGAGGTGCCTCCAGCGAGGAGGAAAGCGGTGAGGACACAGGTCCACAGGATG
CTCAGGAGTATGGGAACTACCAGCCAGGGTCCCTGTGTGGCTACTGCTCCTTCTGCAATCGATGCACTGA
ATGTGAGAGCTGTCACTGTGATGAGGAGAACATGGGTGAGCACTGCGACCAGTGCCAGCACTGTCAGTTC
TGCTATCTCTGCCCGCTGGTCTGCGAAACGGTCTGCGCTCCAGGAAGCTACGTTGACTATTTCTCCTCGT
CCCTTTATCAGGCCCTGGCAGACATGCTGGAAACGCCGGAACCCTGACCCAGTCGCTCGACTGCGACGCA
GGTGTACCTCCCCCTGCCTTCCAACCCCTTTCCACGAGCTATATTTATTTCTCCGAATAAACGTGCTCCC
CGAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_000609.4| Homo sapiens chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1) (CXCL12), transcript variant 2, mRNA
GCACTTTCACTCTCCGTCAGCCGCATTGCCCGCTCGGCGTCCGGCCCCCGACCCGCGCTCGTCCGCCCGC
CCGCCCGCCCGCCCGCGCCATGAACGCCAAGGTCGTGGTCGTGCTGGTCCTCGTGCTGACCGCGCTCTGC
CTCAGCGACGGGAAGCCCGTCAGCCTGAGCTACAGATGCCCATGCCGATTCTTCGAAAGCCATGTTGCCA
GAGCCAACGTCAAGCATCTCAAAATTCTCAACACTCCAAACTGTGCCCTTCAGATTGTAGCCCGGCTGAA
GAACAACAACAGACAAGTGTGCATTGACCCGAAGCTAAAGTGGATTCAGGAGTACCTGGAGAAAGCTTTA
AACAAGAGGTTCAAGATGTGAGAGGGTCAGACGCCTGAGGAACCCTTACAGTAGGAGCCCAGCTCTGAAA
CCAGTGTTAGGGAAGGGCCTGCCACAGCCTCCCCTGCCAGGGCAGGGCCCCAGGCATTGCCAAGGGCTTT
GTTTTGCACACTTTGCCATATTTTCACCATTTGATTATGTAGCAAAATACATGACATTTATTTTTCATTT
AGTTTGATTATTCAGTGTCACTGGCGACACGTAGCAGCTTAGACTAAGGCCATTATTGTACTTGCCTTAT
TAGAGTGTCTTTCCACGGAGCCACTCCTCTGACTCAGGGCTCCTGGGTTTTGTATTCTCTGAGCTGTGCA
GGTGGGGAGACTGGGCTGAGGGAGCCTGGCCCCATGGTCAGCCCTAGGGTGGAGAGCCACCAAGAGGGAC
GCCTGGGGGTGCCAGGACCAGTCAACCTGGGCAAAGCCTAGTGAAGGCTTCTCTCTGTGGGATGGGATGG
TGGAGGGCCACATGGGAGGCTCACCCCCTTCTCCATCCACATGGGAGCCGGGTCTGCCTCTTCTGGGAGG
GCAGCAGGGCTACCCTGAGCTGAGGCAGCAGTGTGAGGCCAGGGCAGAGTGAGACCCAGCCCTCATCCCG
AGCACCTCCACATCCTCCACGTTCTGCTCATCATTCTCTGTCTCATCCATCATCATGTGTGTCCACGACT
GTCTCCATGGCCCCGCAAAAGGACTCTCAGGACCAAAGCTTTCATGTAAACTGTGCACCAAGCAGGAAAT
GAAAATGTCTTGTGTTACCTGAAAACACTGTGCACATCTGTGTCTTGTTTGGAATATTGTCCATTGTCCA
ATCCTATGTTTTTGTTCAAAGCCAGCGTCCTCCTCTGTGACCAATGTCTTGATGCATGCACTGTTCCCCC
TGTGCAGCCGCTGAGCGAGGAGATGCTCCTTGGGCCCTTTGAGTGCAGTCCTGATCAGAGCCGTGGTCCT
TTGGGGTGAACTACCTTGGTTCCCCCACTGATCACAAAAACATGGTGGGTCCATGGGCAGAGCCCAAGGG
AATTCGGTGTGCACCAGGGTTGACCCCAGAGGATTGCTGCCCCATCAGTGCTCCCTCACATGTCAGTACC
TTCAAACTAGGGCCAAGCCCAGCACTGCTTGAGGAAAACAAGCATTCACAACTTGTTTTTGGTTTTTAAA
ACCCAGTCCACAAAATAACCAATCCTGGACATGAAGATTCTTTCCCAATTCACATCTAACCTCATCTTCT
TCACCATTTGGCAATGCCATCATCTCCTGCCTTCCTCCTGGGCCCTCTCTGCTCTGCGTGTCACCTGTGC

Figure 64 (Continued)

TTCGGGCCCTTCCCACAGGACATTTCTCTAAGAGAACAATGTGCTATGTGAAGAGTAAGTCAACCTGCCT
GACATTTGGAGTGTTCCCCTTCCACTGAGGGCAGTCGATAGAGCTGTATTAAGCCACTTAAAATGTTCAC
TTTTGACAAAGGCAAGCACTTGTGGGTTTTTGTTTTGTTTTTCATTCAGTCTTACGAATACTTTTGCCCT
TTGATTAAAGACTCCAGTTAAAAAAAATTTTAATGAAGAAAGTGGAAAACAAGGAAGTCAAAGCAAGGAA
ACTATGTAACATGTAGGAAGTAGGAAGTAAATTATAGTGATGTAATCTTGAATTGTAACTGTTCTTGAAT
TTAATAATCTGTAGGGTAATTAGTAACATGTGTTAAGTATTTTCATAAGTATTTCAAATTGGAGCTTCAT
GGCAGAAGGCAAACCCATCAACAAAAATTGTCCCTTAAACAAAAATTAAAATCCTCAATCCAGCTATGTT
ATATTGAAAAAATAGAGCCTGAGGGATCTTTACTAGTTATAAAGATACAGAACTCTTTCAAAACCTTTTG
AAATTAACCTCTCACTATACCAGTATAATTGAGTTTTCAGTGGGGCAGTCATTATCCAGGTAATCCAAGA
TATTTTAAAATCTGTCACGTAGAACTTGGATGTACCTGCCCCCAATCCATGAACCAAGACCATTGAATTC
TTGGTTGAGGAAACAAACATGACCCTAAATCTTGACTACAGTCAGGAAAGGAATCATTTCTATTTCTCCT
CCATGGGAGAAAATAGATAAGAGTAGAAACTGCAGGGAAAATTATTTGCATAACAATTCCTCTACTAACA
ATCAGCTCCTTCCTGGAGACTGCCCAGCTAAAGCAATATGCATTTAAATACAGTCTTCCATTTGCAAGGG
AAAAGTCTCTTGTAATCCGAATCTCTTTTTGCTTTCGAACTGCTAGTCAAGTGCGTCCACGAGCTGTTTA
CTAGGGATCCCTCATCTGTCCCTCCGGGACCTGGTGCTGCCTCTACCTGACACTCCCTTGGGCTCCCTGT
AACCTCTTCAGAGGCCCTCGCTGCCAGCTCTGTATCAGGACCCAGAGGAAGGGGCCAGAGGCTCGTTGAC
TGGCTGTGTGTTGGGATTGAGTCTGTGCCACGTGTTTGTGCTGTGGTGTGTCCCCCTCTGTCCAGGCACT
GAGATACCAGCGAGGAGGCTCCAGAGGGCACTCTGCTTGTTATTAGAGATTACCTCCTGAGAAAAAAGGT
TCCGCTTGGAGCAGAGGGGCTGAATAGCAGAAGGTTGCACCTCCCCCAACCTTAGATGTTCTAAGTCTTT
CCATTGGATCTCATTGGACCCTTCCATGGTGTGATCGTCTGACTGGTGTTATCACCGTGGGCTCCCTGAC
TGGGAGTTGATCGCCTTTCCCAGGTGCTACACCCTTTTCCAGCTGGATGAGAATTTGAGTGCTCTGATCC
CTCTACAGAGCTTCCCTGACTCATTCTGAAGGAGCCCCATTCCTGGGAAATATTCCCTAGAAACTTCCAA
ATCCCCTAAGCAGACCACTGATAAAACCATGTAGAAAATTTGTTATTTTGCAACCTCGCTGGACTCTCAG
TCTCTGAGCAGTGAATGATTCAGTGTTAAATGTGATGAATACTGTATTTTGTATTGTTTCAATTGCATCT
CCCAGATAATGTGAAAATGGTCCAGGAGAAGGCCAATTCCTATACGCAGCGTGCTTTAAAAAATAAATAA
GAAACAACTCTTTGAGAAACAACAATTTCTACTTTGAAGTCATACCAATGAAAAAATGTATATGCACTTA
TAATTTTCCTAATAAAGTTCTGTACTCAAATGTAGCCACCAA

NM_024046.3| Homo sapiens CaM kinase-like vesicle-associated (CAMKV), mRNA
GTGCGAATCCGGTGCCGGCAAGCGGCTGGCGATGCTGGAGGTTCGCTAGCCGAAGCGGCTGCATCTGGCG
CCGCGTCTGCCCCGCGTGCTCGGAGCGGATTCTGCCCGCCGTCCCCGGAGCCCTCGGCGCCCCGCTGAGC
CCGCGATCACTTCCTCCCTGTGACCAACCGGCGCTGCAGGTTAGAGCCTGGCAATGCCGTTTGGGTGTGT
GACTCTGGGCGACAAGAAGAACTATAACCAGCCATCGGAGGTGACTGACAGATATGATTTGGGACAGGTC
ATCAAGACTGAGGAGTTTTGTGAAATCTTCCGGGCCAAGGACAAGACGACAGGCAAGCTGCACACCTGCA
AGAAGTTCCAGAAGCGGGACGGCCGCAAGGTGCGGAAAGCTGCCAAGAACGAGATAGGCATCCTCAAGAT
GGTGAAGCATCCCAACATCCTACAGCTGGTGGATGTGTTTGTGACCCGCAAGGAGTACTTTATCTTCCTG
GAGCTGGCCACGGGGAGGGAGGTGTTTGACTGGATCCTGGACCAGGGCTACTACTCGGAGCGAGACACAA
GCAACGTGGTACGGCAAGTCCTGGAGGCCGTGGCCTATTTGCACTCACTCAAGATCGTGCACAGGAATCT
CAAGCTGGAGAACCTGGTTTACTACAACCGGCTGAAGAACTCGAAGATTGTCATCAGTGACTTCCATCTG
GCTAAGCTAGAAAATGGCCTCATCAAGGAGCCCTGTGGGACCCCCGAGTATCTGGCCCCAGAGGTGGTAG
GCCGGCAGCGGTATGGACGCCCTGTGGACTGCTGGGCCATTGGAGTCATCATGTACATCCTGCTTTCAGG
CAATCCACCTTTCTATGAGGAGGTGGAAGAAGATGATTATGAGAACCATGATAAGAATCTCTTCCGCAAG
ATCCTGGCTGGTGACTATGAGTTTGACTCTCCATATTGGGATGATATTTCGCAGGCAGCCAAAGACCTGG
TCACAAGGCTGATGGAGGTGGAGCAAGACCAGCGGATCACTGCAGAAGAGGCCATCTCCCATGAGTGGAT
TTCTGGCAATGCTGCTTCTGATAAGAACATCAAGGATGGTGTCTGTGCCCAGATTGAAAAGAACTTTGCC
AGGGCCAAGTGGAAGAAGGCTGTCCGAGTGACCACCCTCATGAAACGGCTCCGGGCACCAGAGCAGTCCA
GCACGGCTGCAGCCCAGTCGGCCTCAGCCACAGACACTGCCACCCCCGGGGCTGCAGGTGGGGCCACAGC
TGCAGCTGCGAGTGGAGCTACCTCAGCCCCTGAGGGTGATGCTGCTCGTGCTGCAAAGAGTGATAATGTG
GCCCCCGCAGACCGTAGTGCCACCCCAGCCACAGATGGAAGTGCCACCCCAGCCACTGATGGCAGTGTCA
CCCCAGCCACCGATGGAAGCATCACTCCAGCCACTGATGGGAGTGTCACCCCAGCCACTGACAGGAGCGC
TACTCCAGCCACTGATGGGAGAGCCACACCAGCCACAGAAGAGAGCACTGTGCCCACCACCCAAAGCAGT
GCCATGCTGGCCACCAAGGCAGCTGCCACCCCTGAGCCGGCTATGGCCCAGCCGGACAGCACAGCCCCAG
AGGGCGCCACAGGCCAGGCTCCACCCTCTAGTAAAGGGGAAGAGGCTGCTGGTTATGCCCAGGAGTCTCA
AAGGGAGGAGGCCAGCTGAGTAGGCAGCCTGGTGAGGGGGGGCAGGGGATGGGCAGGAGGGTGGGAGAGT
GGATGAGGGGCTTCTCACTGTACATAGAGTCACTGGCATGATGCCCTCGCTCCCCCATGCCCCCACATCC
CAGTGGGGCATAACTAGGGGTCACGGGAGAGCAGTCTCGTCTCCTGTGTGTATGTGTGTGAGTGGTGGGC
AGGCCAGTGGCAGGGCCGGCCCCAGCCCCTGCATGGATTCCTTGTGGCTTTTCTGTCTTTTGCTAGCTTC
ACCAGTTTCTGTTCCTTGTGGGATGCTGCTCTAGGGATACTCAGGGGGCTCCTGCTCTCCTTCCCCTTCC
CTTCTTGCCTCACCATTCCCCTAGGCAGGCCCTGCAGGTCCCACACTCTCCCAGGCCCTAAACTTGGGCG
GCCTTGCCCTGAGAGCTGGTCCTCCAGCGAGGCCCTGTCAGCGGTCTTAGGCTCCTGCACATGAAGGTGT

GTGCCTGTGGTGTGTGGGCTGCTCTAGGAGCAGATACAGGCTGGTATAGAGGATGCAGAAAGGTAGGGCA
GTATGTTTAAGTCCAGACTTGGCACATGGCTAGGGATACTGCTCACTAGCTGTGGAGGTCCTCAGGAGTG
GAGAGAATGAGTAGGAGGGCAGAAGCTTCCATTTTTGTCCTTCCTAAGACCCTGTTATTTGTGTTATTTC
CTGCCTTTCCGAGTCCTGCAGTGGGCTGCCCTGTACCCTGAACCTCATGAGCCTCTAAGGGAAAGGAGGA
ACAATTAGGACGTGGCAATGAGACCTGGCAGGGCAGAGTACAAGCCCAGCACCCAGTGTCCCAGCCTTAC
TGGGTCCTTACCCTGGGCCAAACAGGGAGGGCTGATACCTCCTTGCTCTTCCTAGATGCCCACCTCCTAC
AATCTCAGCCCACAAGTCCTCTCCACCCTAGGGGGCTTGCTGCATGGCAATAACTCATAATCTGATTTGG
AGGTTTGCCCTTTACAGGGGCAGATTTTCTGCTCAGTTCAACAATGAAATGAAGAGGAACTCCCTCTTTC
TACAGCTCACTTCTATCAGAGGCCCAGGTGCCTCAGAGCCACATTGAGTTGCTTTTTCTGGGATGAGGAA
GTAGGGTTAAACTCCCCAGTTTCCTGAGGGAGGCTCCTGACAGGTGCCCTTTGTCAGACCCTACCACAGC
CTGGATAGGCAGCCACATTGGTCCTCGCCCTTGCTCGGCACTCCGTGGTGGTCCTGCCCTTCTCCCTGCA
TGCCTGTGGGTCTGCTCTGGTGTGTGAAGGTCGGTGGGTTAACTGTGTGCCTACTGAACCTGGCAAATAA
ACATCACCCTGCAAAGCC

NM_005388.3| Homo sapiens phosducin-like (PDCL), mRNA
TGACCCACTGCTCTTCCTCTTAAGAAAGTGCTCCATTCCTTCCGGCGCCCGGAGCTGCTGGCCCAAAGGG
ATCCGGAGCGAGCTAGGGCAGACACCATGACCACCCTTGATGATAAGTTGCTGGGGGAGAAACTGCAGTA
CTACTATAGCAGCAGTGAGGATGAGGACAGTGACCACGAGGACAAGGACCGAGGCAGATGTGCCCCAGCC
AGCAGTTCTGTGCCTGCAGAGGCTGAGCTGGCAGGCGAAGGCATCTCAGTTAACACAGGCCCAAAAGGTG
TGATCAATGACTGGCGCCGCTTCAAGCAGTTGGAGACAGAGCAGAGGGAGGAGCAGTGCCGGGAGATGGA
AAGGCTGATCAAGAAGCTGTCAATGACTTGCAGGTCCCATCTGGATGAAGAGGAGGAGCAACAGAAACAG
AAAGACCTCCAGGAGAAGATCAGTGGGAAGATGACTCTGAAGGAGTTTGCCATAATGAATGAGGACCAAG
ATGATGAAGAGTTTCTGCAGCAGTACCGGAAGCAGCGAATGGAAGAGATGCGGCAGCAGCTTCACAAGGG
GCCCCAATTCAAGCAGGTTTTTGAGATCTCCAGTGGAGAAGGGTTTTTAGACATGATTGATAAAGAACAG
AAAAGCATTGTCATCATGGTTCATATTTATGAGGATGGCATTCCAGGGACCGAAGCCATGAATGGTTGCA
TGATCTGCCTTGCCGCAGAGTACCCAGCTGTCAAGTTCTGCAAGGTGAAGAGCTCAGTTATTGGCGCCAG
CAGTCAGTTCACCAGGAATGCCCTTCCTGCCCTGCTGATCTATAAGGGGGGTGAATTGATCGGCAATTTT
GTTCGTGTTACTGACCAGCTGGGGGATGATTTCTTTGCTGTGGACCTTGAAGCTTTTCTCCAGGAATTTG
GATTACTCCCAGAAAAGGAAGTCTTGGTGCTGACATCTGTGCGTAACTCTGCCACGTGTCACAGTGAGGA
TAGCGACCTGGAAATAGATTGAACTGATAGTCTAGTTGCATAGATTTCTCATTGTTTGGGTTGGAATACA
CGTCATTGTTTATTTTTGTTCCTTTGTCTTCTGGCTTTTCAGCTGTTCTTTGTAGTCCCTTTTATTATGC
ATAAAATAAAGAAATTCTTAGATTAAATCAGAATGCTGAATAACCTTGTAGCTAGCAATAAGGTGACTTA
CAGTTGTATAAACAGGAAGCCAGGCTTTTGAACTGTTTACTTAAGATTCTGTGGTGTGACATCTCTGTTA
TTGTTTCCAGTCAATATTTACAAAGCATCCTAAAGACAGGGTCTTGGAAATTGTCTTCAGATGATCTTAG
AGGTCTCTGCCAAGTCTGAGAGTATAATTCTGTAGGTATTGTGTTATTTGCAACGTAAATAGTGCATTTT
CTTAATCAAATGATTGTAAATTATATTTACTTGTAATCAGTTCCATAGCTTTAGACGGTGGTTAGATTTT
TTTTTTCCCCACCAGGGTCTTGTTTAAAGGGGTGAGCCACCGCACCCAGTCCTGAGGGGTGGCCTCTGCT
GCTGGATTTCATGTCTTCCTCCAGCATGACTAAGTCTGGAACAGCAGGAAGGGTTGATGCTTACTGACCT
GGTGATGTTAGAAGACAAGTAGTTTATGGATTTAAACATTAGAGCTGGAGTGGGGCTGGAAATCTTTGTA
AAGGAAGTTCTTTCAGTAAGATGCCCCTGCTTGTCTTTGTCTCTTTTTTGTTTAACAAGGTAACTTTTTG
TTTAACAAGGTAACTTTTTGTTTAACCTAGATTTTTTTTAAAACTTTTTTTTTTTTTCATATTGGAAAAG
TAATTCATATTCAGTAGAGGAAAACTGACCAAAACAGAAGCAAAAATAAGAAAATTAAAATAATCTCTAA
TCCTACTACCTAGAATAAAACACTATTAATATTTTGGTCTGTTTCCTGCCAAGGTGTTTTCTGTGTATAC
ATGGATATTTTGTTTGTTTTTAAACAAAACGATGGGATCATTCTGAACATACTGTTCTATAGTATGGTCA
GCTAATAATATATCAGACCTTTTTTTATATTATTAAATATTCTACAACTTTTTAAAAATGTCTATTAATA
TTCCATCGTATAGATGTGATATAATTTGCTTGATGGTTGTCTCTTAAAAAGAAAGATAGCAAATACTTTT
TTTAAATTACAAAAGTGATAGATGTTCATTGTAGAAAATGTAATAAACACTGTTAAGACTTAAAAGCCAT
ATAATTCCACCAACCAAAATTAATCCCTTTTGTCATATTTCTAGTCATTTTTATAGCCTTTTTTTTCTATG
TATTTATAATAATTATCATTTGCGTTTTTTTCCTTTTTTTTAACTTTAAAAATGTATATTCTAGGGTCAGG
GGAAATGTAATCTGGAATTAAATATTAGCCTTAAAATTCACAATTTTGATTTTCCTGGCTTTTCAGGAAT
TGACTAACTGTAAAAGAGTCTTGAAAGTATTTAGTCAACAAACAGAGTGCATTTTTTTTTTTTTTTGACTA
AGAAAGCTCGTTGTAGTAGAAAGGGTGGAATGTATTGAAAATTATTAGAAGCAGGGAAGTATTGTTAGTC
TAGCTTATTTCCTTTCAGTCTTTTTTCAATATTTTTATAAACATTGAGTACTTACTGAATTTAGTTCTGT
GCTCTTCCTTATTTAGTGTTGTATCATAAATACTTTGATGTTTCAAACATTCTAAATAAATAATTTTCAG
TGGCTTCATAATAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_000360.3| Homo sapiens tyrosine hydroxylase (TH), transcript variant 2, mRNA
CGGACCTCCACACTGAGCCATGCCCACCCCCGACGCCACCACGCCACAGGCCAAGGGCTTCCGCAGGGCC
GTGTCTGAGCTGGACGCCAAGCAGGCAGAGGCCATCATGTCCCCGCGGTTCATTGGGCGCAGGCAGAGCC
TCATCGAGGACGCCCGCAAGGAGCGGGAGGCGGCGGTGGCAGCAGCGGCCGCTGCAGTCCCCTCGGAGCC

CGGGGACCCCCTGGAGGCTGTGGCCTTTGAGGAGAAGGAGGGGAAGGCCGTGCTAAACCTGCTCTTCTCC
CCGAGGGCCACCAAGCCCTCGGCGCTGTCCCGAGCTGTGAAGGTGTTTGAGACGTTTGAAGCCAAAATCC
ACCATCTAGAGACCCGGCCCGCCCAGAGGCCGCGAGCTGGGGGCCCCCACCTGGAGTACTTCGTGCGCCT
CGAGGTGCGCCGAGGGGACCTGGCCGCCCTGCTCAGTGGTGTGCGCCAGGTGTCAGAGGACGTGCGCAGC
CCCGCGGGGCCCAAGGTCCCCTGGTTCCCAAGAAAAGTGTCAGAGCTGGACAAGTGTCATCACCTGGTCA
CCAAGTTCGACCCTGACCTGGACTTGGACCACCCGGGCTTCTCGGACCAGGTGTACCGCCAGCGCAGGAA
GCTGATTGCTGAGATCGCCTTCCAGTACAGGCACGGCGACCCGATTCCCCGTGTGGAGTACACCGCCGAG
GAGATTGCCACCTGGAAGGAGGTCTACACCACGCTGAAGGGCCTCTACGCCACGCACGCCTGCGGGGAGC
ACCTGGAGGCCTTTGCTTTGCTGGAGCGCTTCAGCGGCTACCGGGAAGACAATATCCCCCAGCTGGAGGA
CGTCTCCCGCTTCCTGAAGGAGCGCACGGGCTTCCAGCTGCGGCCTGTGGCCGGCCTGCTGTCCGCCCGG
GACTTCCTGGCCAGCCTGGCCTTCCGCGTGTTCCAGTGCACCCAGTATATCCGCCACGCGTCCTCGCCCA
TGCACTCCCCTGAGCCGGACTGCTGCCACGAGCTGCTGGGGCACGTGCCCATGCTGGCCGACCGCACCTT
CGCGCAGTTCTCGCAGGACATTGGCCTGGCGTCCCTGGGGGCCTCGGATGAGGAAATTGAGAAGCTGTCC
ACGCTGTACTGGTTCACGGTGGAGTTCGGGCTGTGTAAGCAGAACGGGGAGGTGAAGGCCTATGGTGCCG
GGCTGCTGTCCTCCTACGGGGAGCTCCTGCACTGCCTGTCTGAGGAGCCTGAGATTCGGGCCTTCGACCC
TGAGGCTGCGGCCGTGCAGCCCTACCAAGACCAGACGTACCAGTCAGTCTACTTCGTGTCTGAGAGCTTC
AGTGACGCCAAGGACAAGCTCAGGAGCTATGCCTCACGCATCCAGCGCCCCTTCTCCGTGAAGTTCGACC
CGTACACGCTGGCCATCGACGTGCTGGACAGCCCCCAGGCCGTGCGGCGCTCCCTGGAGGGTGTCCAGGA
TGAGCTGGACACCCTTGCCCATGCGCTGAGTGCCATTGGCTAGGTGCACGGCGTCCCTGAGGGCCCTTCC
CAACCTCCCCTGGTCCTGCACTGTCCCGGAGCTCAGGCCCTGGTGAGGGGCTGGGTCCCGGGTGCCCCCC
ATGCCCTCCCTGCTGCCAGGCTCCCACTGCCCCTGCACCTGCTTCTCAGCGCAACAGCTGTGTGTGCCCG
TGGTGAGGTTGTGCTGCCTGTGGTGAGGTCCTGTCCTGGCTCCCAGGGTCCTGGGGGCTGCTGCACTGCC
CTCCGCCCTTCCCTGACACTGTCTGCTGCCCCAATCACCGTCACAATAAAAGAAACTGTGGTCTCTA

XM_377774.3| PREDICTED: Homo sapiens kinesin family member 5C, transcript variant 1 (KIF5C), mRNA
TATTGCACTACATCACTGAGTTCCCGTATGCCAAGTTATGGCCACTTACATCCACGTCAGCAAAACTGGT
GCCGAGGGAGCTGTTCTTGACGAAGCTAAAAATATCAATAAGTCTTTGTCTGCTCTTGGAAATGTGATCT
CTGCTTTGGCAGAAGGGACAAAAACACATGTGCCATACCGGGACAGCAAGATGACTCGGATTCTTCAGGA
CTCTTTGGGTGGGAACTGCAGAACCACCATCGTCATTTGCTGTTCTCCTTCTGTCTTCAATGAGGCTGAG
ACCAAGTCCACACTGATGTTCGGACAGAGAGCTAAGACCATCAAGAATACAGTCTCTGTGAACCTAGAAC
TGACAGCAGAAGAATGGAAGAAGAAATATGAAAAAGAGAAAGAGAAAAACAAGACTTTGAAGAATGTTAT
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GCCAAGGACCAGAAGAACCTGGAGCCTTGTGATAACACCCCCATCATAGACAATATTGCTCCTGTTGTTG
CTGGCATCTCTACAGAGGAGAAAGAGAAGTACGATGAGGAGATCTCCAGTCTCTACAGACAACTGGATGA
CAAGGATGATGAAATTAACCAGCAGAGCCAGCTGGCTGAAAAGCTGAAGCAACAGATGTTGGATCAGGAT
GAGCTTTTAGCTTCCACAAGAAGAGACTATGAGAAGATACAGGAGGAGCTGACACGTCTCCAGATTGAAA
ATGAGGCAGCCAAGGATGAGGTGAAAGAAGTTCTCCAGGCCCTGGAGGAGCTGGCTGTCAATTATGACCA
GAAATCACAGGAAGTGGAGGATAAGACCCGGGCCAATGAGCAGCTGACAGACGAGCTGGCCCAGAAAACG
ACTACATTGACAACCACACAGAGAGAGCTGAGCCAGCTACAAGAGCTTAGCAACCACCAGAAGAAAAGGG
CAACTGAGATCCTGAATTTGCTGTTGAAAGATCTGGGGGAGATAGGTGGAATTATTGGCACCAATGATGT
GAAAACTTTGGCAGATGTGAATGGAGTCATTGAGGAGGAGTTTACCATGGCCCGCCTGTACATCAGCAAG
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CAAGTCTCTGACAGACTACATGCAGAACATGGAACAGAAGAGGAGGCAGCTAGAAGAGTCCCAGGACTCG
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AGGAACATCTGACGCGGTTGCAGGATGCTGAAGAAATGAAGAAGGCGCTGGAGCAGCAGATGGAGAGCCA
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AAATAGAGGACCAAGAGAGAGAAATGAAGCTGGAAAAGCTCTTATTGCTCAACGATAAAAGGGAACAAGC
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CTCTTTGTCCAGGATCTGACCACCCGAGTTAAAAAAAGTGTGGAGTTGGACAACGATGATGGAGGGGGCA
GTGCTGCCCAGAAGCAGAAAATTTCCTTCTTGGAGAATAACCTGGAGCAGCTCACCAAAGTTCACAAGCA
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GAGCGCGTCAAGGCTCTGGAGAGCGCGCTGAAGGAGGCCAAGGAGAACGCCATGCGGGACCGTAAGCGCT
ACCAGCAGGAGGTGGATCGTATCAAGGAGGCCGTGCGGGCCAAGAACATGGCCAGAAGGGCCCATTCAGC
CCAGATCGCCAAGCCCATCCGCCCCGGACACTACCCGGCCTCATCTCCAACGGCCGTCCATGCCATTCGA
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TGAGAACCATTACCACCGACTCGGCCTCCGGGGTGTTGGGTGGTTTCTGGGTGGTTCCTGGAGCCTCCTC
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GTGGAAATGTGGCTGCCCCTCTCCTCACTACTTCCTCTCTGATCGTTCTGAAGCTTGCATTGGGAATGGC
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TGATAAAAGGGTCCTTGTGGGGCAGGTTTTGCAAAGCTTATCAGGTAATAACATATGCCACATAACCTTG
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TAATATCCATCATAAAATAGATTGTTTTAGATTCTTTCCAGGGTGATTTTTCCCTGGGTACCCCGTTTCT
ACTTCTAAAGAATTGCTTGGCACTTTCATGTTTCAAAGGGAAACATTCGCTTGTAGTTCCATTTTACTTG
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ATTGCTATGTATAATAAGCCAGTTATTATATCAGGACCATGTTCTCTGTAGGCCACTTTCTAAAAAAGCC
ACATATGTGCAATTTTCAGGTTTTTAGACTATTGCTCCCTGTACTTTAAATGTAAAAACCACACTTCTGA
ACAACTAAGCTCATGAATATGATTTTGGTTATATGCAGCTTTTGACTAGCATGTATTGTGTCTTTTTCTC
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TGTGCAAACATATCATCTCTTCAAATGCTGCACACTTTGCTTTTGTTAAACAGCAGGTAGTAGACAGAAC
AATAACAGTTTCGCGTTAAGACTTTTAAAGGAAATAGAATCGTGATTAAGAAATCAGAATTTATAGATAT
ATTGGGATAAATGAAGAAATAAAAATGTTTGTCTAGAATGTAGCATCTAGTGACTTTTTAAAGCCCTAAC
GTTTACATAAAGAAGCTCTAGTTCTTATAGAAATAACAAAGCAAATAAAAGTTCTTAACAATCCCCTCTT
TCGAAGTGCATTTTTTTAAAGCAGGGCAGGAGACATTTGGACTCTAGCTATATGACATACTGGGAAAGGC
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TGTCCTGTGTACTGCAGATGTGTGTTCTCTGGGCTTTATGTATCTGTACAGTAGCTTTCACATTAAAAAA
ATTGTGGACAAACTTGTCCGGGGGGTTTGAGGGGAGAATGGTGGTTTATATCAATAACGATGCTGTACTA
TAGTCCATGTAACAAAAGATCTGGAAGTCACCCTCCTCTGGCCCACGGAAAATTTTGGTAATCTTCTAGG
TTCTAAAATGAAGATGTATGGGTACTCTGGCAGACTGCATGTTGTATAATTTGAAAAATACTAAAAGTGG
AAAATAAAATTGAATTAAA

NM_012394.3| Homo sapiens prefoldin subunit 2 (PFDN2), mRNA
GCAAGGCGGGGGCTGAGCTGCGCCGGCCGGAAACCCAGCAGGCGGCGAAGATGGCGGAGAACAGCGGTCG
CGCCGGCAAGAGCAGCGGGAGCGGCGCGGGGAAGGGGGCGGTGTCCGCAGAGCAGGTGATTGCTGGCTTC
AACCGCCTTCGGCAGGAACAGCGAGGCCTGGCATCCAAAGCAGCTGAGTTGGAGATGGAGTTGAATGAGC
ACAGCCTAGTGATCGATACACTGAAGGAGGTAGATGAAACTCGTAAGTGCTACCGCATGGTTGGAGGAGT
GCTGGTGGAGCGAACTGTCAAAGAGGTGCTGCCCGCTTTGGAGAACAACAAGGAGCAGATACAGAAGATC
ATTGAGACACTGACACAGCAGCTTCAGGCAAAGGGAAAAGAACTAAATGAATTCCGGGAAAAGCACAACA
TTCGTCTCATGGGAGAAGATGAGAAGCCAGCAGCCAAGGAAAACTCAGAAGGGGCTGGGGCTAAGGCCAG
CTCAGCTGGAGTGTTGGTCTCCTAGGGACCAAGGCCTTTGCATTTTTTTCTACCCTGACTCCCACTTCTA
ATTTCTCTTTATTGTTATTATTATTATTTTCTCTGCTATTGTAATATTTTTTTGTTAATTAAATGTTTTG

GTCAGAATCTTAAAAAAAAAAAAAAAAAAAAAAAA

NM_080745.3| Homo sapiens tripartite motif-containing 69 (TRIM69), transcript variant b, mRNA
GGGGAGCTATGAACCTTAAGATTAGACCACTAACTCGAATCTAAATGAGCTGCCCTTGTCTCCTACAAAA
GAAAAGTTGGGCAGGTAGGGTATTCTAATGAGGGTTTCTCTTTCTCTTAAGCAAATGATGATCAAAGTTA
ACTGACAAACTGTCACGGAATCTGCCAGACCTCACTCTGGCCTTGCTGCTTCTCTCCAGCTCCTGAACTT
TTCTTTCTTCCATCATGCTCTGAGCCCATTCCTTGAAAACTAAAAGGTCCCTGACTCCCAGTCTGCAGCC
ATCCTGGGCCTGCTGAGCTCTGATTCAAGTGCCTGCCTCTGCCCCTTGGTGGGCTGAAGCTTCATGGAGG
AGGAGCTTGCCATCCAACAGGGTCAACTGGAGACAACTCTGAAGGAGCTTCAGACCCTGAGGAACATGCA
GAAGGAAGCTATTGCTGCTCACAAGGAAAACAAGCTACATCTGCAGCAACATGTGTCCATGGAGTTTCTA
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GAGCATTCAGGCAAAGACGGAACAACAGAACTCCTTCGACTTTCTCAAAGACATCACAACTCTCTTACAT
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AGGTGGGCATATACCTGGATTATGAAGGAGGACAGTTGTCCTTCTACAATGCTAAAACCATGACTCACAT
TTACACCTTCAGTAACACTTTCATGGAGAAACTTTATCCCTACTTCTGCCCCTGCCTTAATGATGGTGGA
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TATTAAAGAGGTATTGAAATATTTAAAAAAAAAA

NM_013326.3| Homo sapiens chromosome 18 open reading frame 8 (C18orf8), mRNA
CCGCGCCGGGCCCAGAGCCGCAGCCGCAGCCGCCGCTACAGTCCGGGCCGGGCTCCACCGCGCATCCTGC
TCCACTCTGGCGACCGCCCCCGGGGCCCCCGCCGCGGGCGCGGCGCCCGCCATGGGCGAGGAGGACTACT
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AGGAATCCCATCTCATTTAGAATGGATGACAAAGGAGAAGTGAAGTGCATTAAGTTTTCCTTAGAAAATA
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CACGGTCCTGGAGAATGTCCTGCAGCCTTTTCACTTTAGGGCTGGCACTATGTCGAAGCTGCCCAAATTT
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CCATATACGGGCAGCTGTATGTTCTCTTCTTGAGGCATCATTCTCGGACCTCCAACAGCACAGGAGCGGA
GGTGGTCCTCTATCATCTACCACGAGAAGGTGCCTGTAAAAAGATGCACATATTGAAGTTAAATAGGACG
GGAAAGTTTGCCCTGAACGTGGTGGACAACCTGGTAGTCGTGCATCATCAGGATACAGAGACATCGGTAA
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TATCCTCCTGCTCATCAGCTATCTCTGGACATGCTGAAGCGACTTTCAACAGCAAATGATGAAATAGTAG
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CGCTTTTTTGAACAGCGAAACCAGCGTTTGCGAGGGAGCCCCAATTTCACACCAGGGGAACACTGTGAAG
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CTTGCTGTTTTTTTTATATAAAAATGTGTACAAAGTTAATTTATTGCATTAATAAAGCTCTTTAAACTATA
AAA

NM_006238.3| Homo sapiens peroxisome proliferator-activated receptor delta (PPARD), transcript variant 1, mRNA
GCGGAGCGTGTGACGCTGCGGCCGCCGCGGACCTGGGGATTAATGGGAAAAGTTTTGGCAGGAGCGGGAG
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GGGCATGCACGTGATACTCACACAGTGGCTTCTGCTCACCAACAGATGAAGACAGATGCACCAACGAGGC
TGATGGGAACCACCCTGTAGAGGTCCATCTGCGTTCAGACCCAGACGATGCCAGAGCTATGACTGGGCCT
GCAGGTGTGGCGCCGAGGGGAGATCAGCCATGGAGCAGCCACAGGAGGAAGCCCCTGAGGTCCGGGAAGA
GGAGGAGAAAGAGGAAGTGGCAGAGGCAGAAGGAGCCCCAGAGCTCAATGGGGGACCACAGCATGCACTT
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GAAGCTGGTGGCAGGGCTGACTGCAAACGAGGGGAGCCAGTACAACCCACAGGTGGCCGACCTGAAGGCC
TTCTCCAAGCACATCTACAATGCCTACCTGAAAAACTTCAACATGACCAAAAAGAAGGCCCGCAGCATCC
TCACCGGCAAAGCCAGCCACACGGCGCCCTTTGTGATCCACGACATCGAGACATTGTGGCAGGCAGAGAA
GGGGCTGGTGTGGAAGCAGTTGGTGAATGGCCTGCCTCCCTACAAGGAGATCAGCGTGCACGTCTTCTAC
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TCTGTCCTCCTTTCTTATTCTGTGAGATGTTTTGTATTATTTCACCAGCAGCATAGAACAGGACCTCTGC
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CACCCAGTGTCCTTCCATCTTCACACTGGTTTGCCAGGCCAATGTTGCTGATGGCCCCCTGCACTGGCCG
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CCTCCCCTGCTCCCAGGTTGAGGTGCGCTCACCTCAGAGCAGGGCCAAAGCACAGCTGGGCATGCCATGT
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CACTCTCTGCTGGCAGGATTCTTCCCGCTCCCCACCTACCCAGCTGATGGGGGTTGGGGTGCTTCCTTTC
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CCCGACCCGGGTCTGGTGCTGAGGATACAGCTCTTCTCAGTGTCTGAACAATCTCCAAAATTGAAATGTA
TATTTTTGCTAGGAGCCCCAGCTTCCTGTGTTTTTAATATAAATAGTGTACACAGACTGACGAAACTTTA
AATAAATGGGAATTAAATATTTAA

NM_001783.3| Homo sapiens CD79a molecule, immunoglobulin-associated alpha (CD79A), transcript variant 1, mRNA
TCCACTCACAGCCTGAAGCATACCCGGCAGGGGCTGTCCCCAGGCCCAACAAGCAAAGGGCCCAGTAGCG
AGGGCCACTGGAGCCCATCTCCGGGGGGCTGGGCAGGAAGTAGGGTGGGGTTTGGGGTAGGGATCTGGTA

CCCTGGGACTGCTGCAACTCAAACTAACCAACCCACTGGGAGAAGATGCCTGGGGGTCCAGGAGTCCTCC
AAGCTCTGCCTGCCACCATCTTCCTCCTCTTCCTGCTGTCTGCTGTCTACCTGGGCCCTGGGTGCCAGGC
CCTGTGGATGCACAAGGTCCCAGCATCATTGATGGTGAGCCTGGGGGAAGACGCCCACTTCCAATGCCCG
CACAATAGCAGCAACAACGCCAACGTCACCTGGTGGCGCGTCCTCCATGGCAACTACACGTGGCCCCCTG
AGTTCTTGGGCCCGGGCGAGGACCCCAATGGTACGCTGATCATCCAGAATGTGAACAAGAGCCATGGGGG
CATATACGTGTGCCGGGTCCAGGAGGGCAACGAGTCATACCAGCAGTCCTGCGGCACCTACCTCCGCGTG
CGCCAGCCGCCCCCCAGGCCCTTCCTGGACATGGGGGAGGGCACCAAGAACCGAATCATCACAGCCGAGG
GGATCATCCTCCTGTTCTGCGCGGTGGTGCCTGGGACGCTGCTGCTGTTCAGGAAACGATGGCAGAACGA
GAAGCTCGGGTTGGATGCCGGGGATGAATATGAAGATGAAAACCTTTATGAAGGCCTGAACCTGGACGAC
TGCTCCATGTATGAGGACATCTCCCGGGGCCTCCAGGGCACCTACCAGGATGTGGGCAGCCTCAACATAG
GAGATGTCCAGCTGGAGAAGCCGTGACACCCCTACTCCTGCCAGGCTGCCCCCGCCTGCTGTGCACCCAG
CTCCAGTGTCTCAGCTCACTTCCCTGGGACATTCTCCTTTCAGCCCTTCTGGGGGCTTCCTTAGTCATAT
TCCCCCAGTGGGGGGTGGGAGGGTAACCTCACTCTTCTCCAGGCCAGGCCTCCTTGGACTCCCCTGGGGG
TGTCCCACTCTTCTTCCCTCTAAACTGCCCCACCTCCTAACCTAATCCCCCCGCCCCGCTGCCTTTCCCA
GGCTCCCCTCACCCCAGCGGGTAATGAGCCCTTAATCGCTGCCTCTAGGGGAGCTGATTGTAGCAGCCTC
GTTAGTGTCACCCCCTCCTCCCTGATCTGTCAGGGCCACTTAGTGATAATAAATTCTTCCCAACTGCA

NM_003302.2| Homo sapiens thyroid hormone receptor interactor 6 (TRIP6), mRNA
GACGTCATCGGAGGCGTGGTCGTCCCCAAAATTAGGGAGGAAGAGGAAAAAAAAAAAGCCAGAAAAAGTTT
TCTTTTCTGGAGTCCCAAACGAGGTGCGGGACGGAAGAGGGGGTGAAGGCCAGAGGCTCGGGGCTTCAAG
ACCGCTGTCTGGAGTCCCCCTTTCCAGGCCATGTCGGGGCCCACCTGGCTGCCCCCGAAGCAGCCGGAGC
CCGCCAGAGCCCCTCAGGGGAGGGCGATCCCCCGCGGCACCCCGGGGCCACCACCGGCCCACGGAGCAGC
ACTCCAGCCCCACCCCAGGGTCAATTTTTGCCCCCTTCCATCTGAGCAGTGTTACCAGGCCCCAGGGGGA
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TACCGCACGGGCTCCCTGAAGCCAAATCCAGCCTCGCCGCTCCCAGCGTCTCCCTATGGGGGCCCCACTC
CAGCCTCTTACACTACCGCCAGCACCCCGGCTGGCCCAGCCTTCCCCGTGCAAGTGAAGGTGGCACAGCC
AGTGAGGGGCTGCGGCCCACCCAGGCGGGGAGCCTCTCAGGCCTCTGGGCCCCTCCCGGGCCCCCACTTT
CCTCTCCCAGGCCGAGGTGAAGTCTGGGGGCCTGGCTATAGGAGCCAGAGAGAGCCAGGGCCAGGGGCCA
AAGAGGAAGCTGCTGGGGTCTCTGGCCCTGCAGGAAGAGGAAGAGGAGGCGAGCACGGGCCCCAGGTGCC
CCTGAGCCAGCCTCCAGAGGATGAGCTGGATAGGCTGACGAAGAAGCTGGTTCACGACATGAACCACCCG
CCCAGCGGGGAGTACTTTGGCCAGTGTGGTGGCTGCGGAGAAGATGTGGTTGGGGATGGGGCTGGGGTTG
TGGCCCTTGATCGCGTCTTTCACGTGGGCTGCTTTGTATGTTCTACATGCCGGGCCCAGCTTCGCGGCCA
GCATTTCTACGCCGTGGAGAGGAGGGCATATTGCGAGGGCTGCTACGTGGCCACCCTGGAGAAATGTGCC
ACGTGCTCCCAGCCCATCCTGGACCGGATCCTGCGGGCTATGGGGAAGGCCTACCACCCTGGCTGCTTCA
CCTGCGTGGTGTGTCACCGCGGCCTCGACGGCATCCCCTTCACAGTGGATGCTACGAGCCAGATCCACTG
CATTGAGGACTTTCACAGGAAGTTTGCCCCAAGATGCTCAGTGTGCGGTGGGGCCATAATGCCTGAGCCA
GGTCAGGAGGAGACTGTGAGAATTGTTGCTCTGGATCGAAGTTTTCACATTGGCTGTTACAAGTGCGAGG
AGTGTGGGCTGCTGCTCTCCTCTGAGGGCGAGTGTCAGGGCTGCTACCCGCTGGATGGGCACATCTTGTG
CAAGGCCTGCAGCGCCTGGCGCATCCAGGAGCTCTCAGCCACCGTCACCACTGACTGCTGAGTCTTCCTA
GAAGTACCTGCTGGGTTCTCAGTTCCAGTTCCCATCCTTTGATTGATCACTCTCCCTGACATCCACCTGT
ATGACTTTGTCACCAAATGCTGTCTTCTCTTTCTCCAATCAAGAAATAATAATCCCTCGAGTTTACAAAA
CAAAAAAAAAAA

NM_004126.3| Homo sapiens guanine nucleotide binding protein (G protein), gamma 11 (GNG11), mRNA
GAGAACACAAAGTAAAACTTCTGCTGTTCCCAGTCCTGCAGCCCCAGCCCAGCCGTCCCCCGCGGGCGTG
GCAGGTCCTGAGTCAGGGCCTGTCCGTGGCCAGCATCCCTGCTGGGCGATTGAGCAGCGGGAAGCTGCTT
GGACCCAGTCTCAAACTTAACCCTCATCTAGCACCCGGGCAGGCCTCCTGGGTTGCAGGGACTTGAGAAA
AGGCAGAGTTCTCAGGTCCTAGGAAGCTGGGGCACGCTGGCGTGACAAGCGTCCCGGAGAAAGCCAAGCC
CTCGGGGAGCTGGGGACCGCAGCAGGGCTGCAGTCACATCCTGCGCGGGTGGGCGGCGGGCCAGGCCTTC
AGTTGTTTCGGGACGCGCCGAGCTTCGCCGCTCTTCCAGCGGCTCCGCTGCCAGAGCTAGCCCGAGCCCG
GTTCTGGGGCGAAAATGCCTGCCCTTCACATCGAAGATTTGCCAGAGAAGGAAAAACTGAAAATGGAAGT
TGAGCAGCTTCGCAAAGAAGTGAAGTTGCAGAGACAACAAGTGTCTAAATGTTCTGAAGAAATAAAGAAC
TATATTGAAGAACGTTCTGGAGAGGATCCTCTAGTAAAGGGAATTCCAGAAGACAAGAACCCCTTTAAAG
AAAAAGGCAGCTGTGTTATTTCATAAATAACTTGGGAGAAACTGCATCCTAAGTGGAAGAACTAGTTTGT
TTTAGTTTTCCCAGATAAAACCAACATGCTTTTTAAGGAAGGAAGAATGAAATTAAAAGGAGACTTTCTT
AAGCACCATATAGATAGGGTTATGTATAAAAGCATATGTGCTACTCATCTTTGCTCACTATGCAGTCTTT
TTTAAGAGAGCAGAGAGTATCAGATGTACAATTATGGAAATAAGAACATTACTTGAGCATGACACTTCTT

TCAGTATATTGCTTGATGCTTCAAATAAAGTTTTGTCTTGGGAAAAAAAAAAAA

NM_004849.2| Homo sapiens ATG5 autophagy related 5 homolog (S. cerevisiae) (ATG5), mRNA
GTGACGTCATCTCCGGGCGCCGAGGGTGACTGGACTTGTGGTGCGCTGCCAGGGCTCCGCAGCGTTGCCG
GTTGTATTCGCTGGATACCAGAGGGCGGAAGTGCAGCAGGGTTCAGCTCCGACCTCCGCGCCGGTGCTTT
TTGCGGCTGCGCGGGCTTCCTGGAGTCCTGCTACCGCGTCCCCGCAGGACAGTGTGTCAGGCGGGCAGCT
TGCCCCGCCGCCCCACCGGAGCGCGGAATCTGGGCGTCCCCACCAGTGCGGGGAGCCGGAAGGAGGAGCC
ATAGCTTGGAGTAGGTTTGGCTTTGGTTGAAATAAGAATTTAGCCTGTATGTACTGCTTTAACTCCTGGA
AGAATGACAGATGACAAAGATGTGCTTCGAGATGTGTGGTTTGGACGAATTCCAACTTGTTTCACGCTAT
ATCAGGATGAGATAACTGAAAGGGAAGCAGAACCATACTATTTGCTTTTGCCAAGAGTAAGTTATTTGAC
GTTGGTAACTGACAAAGTGAAAAAGCACTTTCAGAAGGTTATGAGACAAGAAGACATTAGTGAGATATGG
TTTGAATATGAAGGCACACCACTGAAATGGCATTATCCAATTGGTTTGCTATTTGATCTTCTTGCATCAA
GTTCAGCTCTTCCTTGGAACATCACAGTACATTTTAAGAGTTTTCCAGAAAAAGACCTTCTGCACTGTCC
ATCTAAGGATGCAATTGAAGCTCATTTTATGTCATGTATGAAAGAAGCTGATGCTTTAAAACATAAAAGT
CAAGTAATCAATGAAATGCAGAAAAAAGATCACAAGCAACTCTGGATGGGATTGCAAAATGACAGATTTG
ACCAGTTTTGGGCCATCAATCGGAAACTCATGGAATATCCTGCAGAAGAAAATGGATTTCGTTATATCCC
CTTTAGAATATATCAGACAACGACTGAAAGACCTTTCATTCAGAAGCTGTTTCGTCCTGTGGCTGCAGAT
GGACAGTTGCACACACTAGGAGATCTCCTCAAAGAAGTTTGTCCTTCTGCTATTGATCCTGAAGATGGGG
AAAAAAAGAATCAAGTGATGATTCATGGAATTGAGCCAATGTTGGAAACACCTCTGCAGTGGCTGAGTGA
ACATCTGAGCTACCCGGATAATTTTCTTCATATTAGTATCATCCCACAGCCAACAGATTGAAGGATCAAC
TATTTGCCTGAACAGAATCATCCTTAAATGGGATTTATCAGAGCATGTCACCCTTTTGCTTCAATCAGGT
TTGGTGGAGGCAACCTGACCAGAAACACTTCGCTGCTGCAAGCCAGACAGGAAAAAGATTCCATGTCAGA
TAAGGCAACTGGGCTGGTCTTACTTTGCATCACCTCTGCTTTCCTCCACTGCCATCATTAAACCTCAGCT
GTGACATGAAAGACTTACCGGACCACTGAAGGTCTTCTGTAAAATATAATGAAGCTGAAACCTTTGGCCT
AAGAAGAAAATGGAAGTATGTGCCACTCGATTTGTATTTCTGATTAACAAATAAACAGGGGTATTTCCTA
AGGTGACCATGGTTGAACTTTAGCTCATGAAAGTGGAAACATTGGTTTAATTTTCAAGAGAATTAAGAAA
GTAAAAGAGAAATTCTGTTATCAATAACTTGCAAGTAATTTTTTGTAAAAGATTGAATTACAGTAAACCC
ATCTTTCCTTAACGAAAATTTCCTATGTTTACAGTCTGTCTATTGGTATGCAATCTTGTAACTTTGATAA
TGAACAGTGAGAGATTTTTAAATAAAGCCTCTAAATATGTTTTGTCATTTAATAACATACAGTTTTGTCA
CTTTTCAAGTACTTTCTGACTCACATACAGTAGATCACTTTTTACTCTGTGTTACCATTTTGACTGGTCG
TCATTGGCATGGGGTGGATATAGGGCATAGGATTACTTGTCTCAGAAGCTGTCATAGAATTTCTTGCTGC
CAATTAAAAAACCTGTGTTCTTTACACACTACACGTATAAATATTGTAACTGTTCATCTTTGTTGTTTTA
TCACTGTAAGCCTGTCAAATCATAGTATCCTAAGCATCTGTAAATGCTAATTTTGCATTTTTGGAAAAAC
CCATTCCTTCCAAGCTAGTGTTTTTCATTGGCTCCAGGTCTAATTTTTCACTGTGGTCCCTGGCAGCCAG
TCTTTTGAAGTTTAAAGATTACCTGTCTCTTGACTGCAGTACCTTTTCTTTAATTTTTACCAAAAATATC
CAGAGGTTACTGGAGTTCTTATTCAATATAAGGAAAGTTTGCTGCACTTTATTACCAAGCCTCTGGGATT
TTACCAGTCAAACATATTTGTGCATTACATTTCATTTCTTGTGAGCTAGCTGGCTGTCCATATTGAATGT
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ATGAATGTTTTTCTTATAAAAAATTATACAAATGCTGAAGTGAGATTCTACTATTGTTCATTGCTTCCTT
TTCTTTTTCCTTTTGCGATTTTCACTGATTAATAGCACATTTCTTCACAAAATTAGATAAAGTTGGTCAA
AGACCAGATATTCTGGAATGGAAATTGTAAAGCTTAATCAAAAAGAATAGCCAGTACAGCATACAATCTC
AGAAACTTAGAAGCAAGTAGAAAATAATTGGTTGATGTAAACGAAAGTGCCATTTTAGTAAAGGCAGGAA
AAAAATAGCAATATTTGAGTTATGTAAGGATAAAAAATCCACTGACTTGTATTTTTGCACAAGAGGCTGG
TCTGAATATGATTGTTCACATTAAGAGTGTTTATTCGTCGGTTCATTTTGGGGATTTTCCCCCTTGATGT
TTTGACAGATTGAAGTGAGCTTTAGTGAGCAAAAGGATCAGAATGCAGGGAACACTAAGCTGTGATGAAG
AAAGTGTGGTAAAAAGCCAGAGTAGTTTTATACAGACAAAACCAGTGTCAGGCCTTTGCAGTAGGCTTGA
GTGAACTTCTGATCTAGATTTGAAAGTAAATTTTATGAAGACATTGCCCATTTTTACTTCCTCATTCATT
ATTGTACCAGCATCATAGCTTTATTACTCTAATCCCAGGTAAGTCAAGCCTACAATGCCCTAGAGGAAGA
GTAAAACCAGAAATTCATGCTGGCTTAAATAATCTATTTTTGTTTCTTTTCATTTGAATATTTAAATTTT
ATGGTTTATTAAAAAATTAAATAA

NM_032673.2| Homo sapiens polycomb group ring finger 1 (PCGF1), mRNA
GTGGCCGGCTGGGATCAGCCTTTAAGATGGCGTCTCCTCAGGGGGGCCAGATTGCGATCGCGATGAGGCT
TCGGAACCAGCTCCAGTCAGTGTACAAGATGGACCCGCTACGGAACGAGGAGGAGGTTCGAGTGAAGATC
AAAGACTTGAATGAACACATTGTTTGCTGCCTATGCGCCGGCTACTTCGTGGATGCCACCACCATCACAG
AGTGTCTTCATACTTTCTGCAAGAGTTGTATTGTGAAGTACCTCCAAACTAGCAAGTACTGCCCCATGTG
CAACATTAAGATCCACGAGACACAGCCACTGCTCAACCTCAAACTGGACCGGGTCATGCAGGACATCGTG
TATAAGCTGGTGCCTGGCTTGCAAGACAGTGAAGAGAAACGGATTCGGGAATTCTACCAGTCCCGAGGTT
TGGACCGGGTCACCCAGCCCACTGGGGAAGAGCCAGCACTGAGCAACCTCGGCCTCCCCTTCAGCAGCTT
TGACCACTCTAAAGCCCACTACTATCGCTATGATGAGCAGTTGAACCTGTGCCTGGAGCGGCTGAGTTCT

GGCAAAGACAAGAATAAAAGCGTCCTGCAGAACAAGTATGTCCGATGTTCTGTTAGAGCTGAGGTACGCC
ATCTCCGGAGGGTCCTGTGTCACCGCTTGATGCTAAACCCTCAGCATGTGCAGCTCCTTTTTGACAATGA
AGTTCTCCCTGATCACATGACAATGAAGCAGATATGGCTCTCCCGCTGGTTCGGCAAGCCATCCCCTTTG
CTTTTACAATACAGTGTGAAAGAGAAGAGGAGGTAGGGGCCAAGCCCCCACCCCATCCCACTCCCCTTCC
CTCCCCAGATATTTATGTGAAATGAACTGCAGCTTTATTTTTTGAAATAAAAACTTTTAAAAAGCAAAAA
AAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_000427.2| Homo sapiens loricrin (LOR), mRNA
CTCTCCTCACTCACCCTTCCTGGTGCTTTGGGCTCTCCTTCCTTCTCAGACAAGATGTCTTATCAGAAAA
AGCAGCCCACCCCTCAGCCCCCAGTGGACTGCGTGAAGACCTCTGGCGGCGGTGGCGGTGGCGGCGGCAG
CGGCGGTGGTGGCTGCGGCTTCTTCGGCGGCGGCGGCTCAGGGGGCGGTAGCAGCGGTTCTGGCTGCGGC
TACTCCGGCGGCGGTGGCTACTCTGGCGGCGGCTGCGGCGGGGGCTCCTCCGGCGGCGGGGGCGGGGGCG
GCATTGGAGGCTGCGGAGGGGGCTCCGGTGGGAGCGTCAAGTACTCCGGAGGCGGCGGCTCCTCCGGCGG
GGGCTCTGGCTGTTTCTCCAGCGGTGGGGGCGGCTCCGGCTGCTTCTCCTCCGGTGGCGGCGGCTCCTCC
GGGGGCGGCTCCGGCTGCTTCTCCAGCGGTGGGGGCGGCTCCTCCGGGGGCGGCTCCGGCTGCTTCTCCT
CCGGCGGCGGCGGCTTCTCGGGCCAGGCGGTCCAGTGCCAGAGCTACGGAGGCGTCTCTAGCGGCGGCTC
CTCCGGGGGCGGCTCCGGCTGCTTCTCCAGCGGCGGGGGCGGCGGCTCTGTCTGCGGCTACTCTGGCGGC
GGCTCTGGCTGCGGCGGAGGCTCCTCTGGCGGCAGCGGCTCCGGCTACGTCTCCTCGCAGCAGGTCACTC
AGACCTCGTGCGCGCCCCAGCCGAGTTACGGAGGGGGGTCGTCCGGCGGCGGCGGCAGCGGCGGAAGCGG
CTGCTTCTCCAGCGGCGGGGGCGGCGGGAGCTCCGGCTGCGGCGGCGGCTCCTCCGGGATTGGCAGCGGC
TGCATCATCAGTGGCGGGGGCTCCGTCTGCGGAGGTGGTTCCTCTGGAGGCGGCGGCGGCGGCTCCTCCG
TGGGTGGCTCCGGGAGTGGCAAGGGCGTCCCGATCTGCCACCAGACCCAGCAGAAGCAGGCGCCTACCTG
GCCGTCCAAATAGATCCCCCAGGGTACCACGGAGGCGAAGGAGTTGGAGGTGTTTTCCAGGGGCACCGAT
GGGCTTAGAGCTCTCATGATGCTACCCGAGGTTTGCAAATCCTTCATGTCTTAACCTACCTGGAAGAAGC
CATTGAGCTCTCCGGCTGCATCTAGTTCTGCTGTTTAGCCTCTTTGGTTTCTGTACAACTACCTCCCAAC
CCCAGTGCCTCAGTCAATAAATTTGCAAATTCATGAGAA

NM_016564.3| Homo sapiens cell cycle exit and neuronal differentiation 1 (CEND1), mRNA
AGCAGTGCATTGTGGGAAACTCCCGAGCTCTCCTCCGCGTTCGCAGCCGCCGTCATCCCGCGGAGGAGCG
CGCAGCCCCGGGGAGGCCGGAGGACGCGCCCATAGAATGCCCAGGGGCGACAAACTGTCCTGAGCCCCTC
TGGTGCAGCCACCTGCCTGTCCCATACGCCCCGCCCACCATGGAGTCCAGAGGGAAGTCAGCCAGCAGCC
CCAAGCCCGACACCAAGGTGCCCCAGGTCACCACCGAGGCCAAGGTACCCCCGGCAGCCGATGGGAAAGC
CCCCTTGACCAAGCCCTCGAAGAAGGAGGCCCCGGCCGAGAAGCAGCAGCCGCCAGCAGCCCCCACCACG
GCACCTGCCAAGAAGACCTCGGCCAAGGCCGACCCTGCCCTTCTCAACAACCACAGCAACCTGAAGCCAG
CCCCCACGGTCCCCAGCAGTCCCGATGCAACCCCGGAGCCCAAGGGTCCTGGGGACGGGGCGGAGGAAGA
TGAGGCTGCCAGTGGGGGGCCTGGGGGCCGAGGTCCCTGGTCCTGTGAGAACTTCAACCCCCTGCTGGTG
GCTGGGGGTGTGGCCGTGGCAGCCATAGCCCTGATTCTCGGTGTGGCCTTCCTGGTCCGGAAAAAATAAT
ACCTGGGGGCCAGGCGGGGGGCACGGAGCCACTTCCTGTACAGACCCGAGGAAGCCAGTGCATGCAGAGT
TCACCCTTACCTATTCGTACACACGCACATTCATTACACACCTACATACGCCCCCAACACACGCGCACGG
TGAAGAGGACGCCCGAGCCCACCCCTGCTGACCCAGGACTTCCCCAACCTCCAGGGCAGAAAGAGCCCAG
GCTCCGGGGTCCACAGCACAGGATGTGGGGCGAGGGCACAGCTGGGGAACGGCAAGAAAGGAATGGACCC
TGTGTGTGGCCCCCCCCACCCCTGGCGGCTGGGTGATCCTGGGCCCCCAGGGCTGGTCTGAGTGCAGGTG
GGGGTGCCGGGATGGGTTGGGCCTGGGCCCGGCCCTCGTGGGGACATTAAAGGGCGCGGTGGCTCCACTC
GCCCCATTTCTGCTCTTTGCGTACCCCCCGGGGGTCTGCCTGGGCGAATGCAGTGGGACGGACCTGGGGT
GGGCACTCACCTCAGCAGAGCCTCAATGCCACCTCCCCACACCACCCAAGCCTAGCTGGGTGGGAAGACG
GAGCCCTGAAGCCTGCGTGTGGAGGGGGTGCTGCCCAAGGGTCGCCTTCCCCAGCTTGTGCTCCAGGGGA
GCCCCGACGCCGGCGGGGAGGCAGGGATGTCGAGGGCCCCTCACAGCTGTGCCCGCCCTGCCCCGCTAGA
CTGCCCCTCCAGCTTGCACCCAGCACCACCTGAGTCTAACCAGCGTATAATGCAATAACAGGTAGAGTAG
AACTGCTTTTGGCGGCGACCGTCACACACTGTCCTCAGCCCACTGCACCTGGGGAAGCCCCTCGTGCAGC
CCCTCCTTAGCACATCCAGTCCCAGCGTGGCCCTTGGCGCGCAGCAGCAGGCGAACTCCCAGAAGGTGGT
GGTGGGCACTGTGAGAACGTGGCCTGCCCTCACTGGCCAGTGGCCACAGGAACCCCGGGCCTGAGTGGAG
GCTGAGTCTGAAATAAACTCTGTCGTCTGAAGGCTG

NM_012385.2| Homo sapiens nuclear protein, transcriptional regulator, 1 (NUPR1), transcript variant 2, mRNA
CCAGCTGGGTGAGCCTGGGAGGGAGGAGGTGAGTTGGGCTGGACTCAGGGACCGACTCTTCCCGTCTCAT
GACTGTGTTTACTGGGCTGGATTTTGGGAAGGGGCCAGATTGCATCAGACAGGGCCTGATGGGCTGGAGC
CAGACTGTGGTCTGAGGAGGAGACACAGCCTTATAAGCTGAGGGAGTGGAGAGGCCCGGGGCCAGGAAAG
CAGAGACAGACAAAGCGTTAGGAGAAGAAGAGAGGCAGGGAAGACAAGCCAGGCACGATGGCCACCTTCC
CACCAGCAACCAGCGCCCCCCAGCAGCCCCCAGGCCCGGAGGACGAGGACTCCAGCCTGGATGAATCTGA

CCTCTATAGCCTGGCCCATTCCTACCTCGGAGGTGGAGGCCGGAAAGGTCGCACCAAGAGAGAAGCTGCT
GCCAACACCAACCGCCCCAGCCCTGGCGGGCACGAGAGGAAACTGGTGACCAAGCTGCAGAATTCAGAGA
GGAAGAAGCGAGGGGCACGGCGCTGAGACAGAGCTGGAGATGAGGCCAGACCATGGACACTACACCCAGC
AATAGAGACGGGACTGCGGAGGAAGGAGGACCCAGGACAGGATCCAGGCCGGCTTGCCACACCCCCCACC
CCTAGGACTTATTCCCGCTGACTGAGTCTCTGAGGGGCTACCAGGAAAGCGCCTCCAACCCTAGCAAAAG
TGCAAGATGGGGAGTGAGAGGCTGGGAATGGAGGGGCAGAGCCAGGAAGATCCCCCAGAAAAGAAAGCTA
CAGAAGAAACTGGGGCTCCTCCAGGGTGGCAGCAACAATAAATAGACACGCACGGCAGCCACAGCTTGGG
TGTGTGTTCATCCTTGTTAAAAAAAAAAAAAAAAAAAAAAAA

NM_138296.2| Homo sapiens pre T-cell antigen receptor alpha (PTCRA), mRNA
TAGAAGGCAGTCTTGTGGGTGCCTCCTCCCCCAGCCGCAACTCAGGTCTGCAGCTGGGTCCTGCCTCCTT
CCGAGTGGGCCATGGCCGGTACATGGCTGCTACTTCTCCTGGCCCTTGGGTGTCCAGCCCTACCCACAGG
TGTGGGCGGCACACCCTTTCCTTCTCTGGCCCCACCAATCATGCTGCTGGTGGATGGAAAGCAGCAGATG
GTGGTGGTCTGCCTGGTCCTTGATGTTGCACCCCCTGGCCTTGACAGCCCCATCTGGTTCTCAGCCGGCA
ATGGCAGTGCACTGGATGCCTTCACCTATGGCCCTTCCCCAGCAACGGATGGCACCTGGACCAACTTGGC
CCATCTCTCCCTGCCTTCTGAGGAGCTGGCATCCTGGGAGCCTTTGGTCTGCCACACTGGGCCTGGGGCT
GAGGGTCACAGCAGGAGTACACAGCCCATGCATCTGTCAGGAGAGGCTTCTACAGCCAGGACCTGCCCCC
AGGAGCCTCTCAGGGGGACACCGGGTGGGGCGCTGTGGCTGGGGGTCCTGCGGCTGCTGCTCTTCAAGCT
GCTGCTGTTTGACCTGCTCCTGACCTGCAGCTGCCTGTGCGACCCCGCGGGCCCGCTGCCTTCCCCCGCA
ACCACCACCCGCCTGCGAGCCCTCGGCTCCCATCGACTGCACCCGGCCACGGAGACTGGGGGACGAGAGG
CCACCAGCTCACCCAGACCCCAGCCTCGGGACCGCCGCTGGGGTGACACCCCTCCGGGTCGGAAGCCCGG
GAGCCCAGTATGGGGGGAAGGGTCTTACCTCAGCAGTTACCCCACTTGCCCAGCACAGGCCTGGTGCTCA
AGATCTGCCCTCAGGGCTCCTTCCTCCAGTCTTGGAGCATTTTTTGCAGGTGACCTGCCTCCTCCTCTGC
AGGCTGGAGCTGCCTGAGGGCAGGGCTCTACCTCCCCTGCGTCACACTGTGTGAGGCTGTGTCTCTGCCA
TCCAAAAGGGGGCCCCTTGAGAATGGTGATCCACCCAGTTACAGGGGCATTTAGGGAGCAGATGACTGAG
AACATTAAAAAAGAACTTAAATGACACAGCAAAAAAAAAAAAAAAAA

NM_006377.3| Homo sapiens unc-13 homolog B (C. elegans) (UNC13B), mRNA
GTGGAATGACGGGAGGGAGTCCCGGCAGCTCCTACCTCCCAGCGATGGCGTCGCTGTGCCGCGCCCAGTC
CCCAGCCTGCCGGCCGGTACTCACCGCTACCCGGAGTTCGCTCAGACGGTGAGATTTGGGGCGGGTCCGA
GGCAGCGGCGGGACGCTACCTGCGACCGGGACCATGAGGAGCTGCCAGACCCGTGGGGCCGGTAACGAGA
GCAGTCGCGGCACCTGCTGAGAGGAAAGAGGGAGCGGTCCGGCGCGGCTGGGGCGCGGCAGAGGCTTGCC
CGATCCTCGGCCATGTCACTGCTCTGCGTGCGCGTTAAAAGGGCCAAATTCCAGGGTTCACCAGATAAAT
TTAACACATATGTGACCCTGAAAGTACAGAATGTGAAGAGCACAACTGTAGCAGTTCGTGGTGATCAGCC
TTCCTGGGAACAGGATTTCATGTTTGAGATTAGTCGCCTGGACCTGGGTCTAAGTGTGGAGGTATGGAAC
AAAGGACTGATCTGGGACACCATGGTGGGGACTGTGTGGATTGCGCTGAAGACTATTCGTCAGTCGGATG
AGGAAGGGCCTGGGGAATGGTCCACATTAGAGGCAGAGACGTTAATGAAAGACGATGAGATCTGTGGAAC
TAGAAACCCAACTCCTCATAAAATTTTGCTTGATACAAGATTTGAGTTGCCTTTTGATATCCCAGAGGAG
GAAGCCAGATATTGGACCTACAAATGGGAGCAAATCAATGCCTTGGGAGCTGACAATGAGTATTCTAGTC
AAGAAGAAAGCCAGAGGAAGCCATTGCCCACTGCTGCCGCCCAGTGTTCTTTTGAAGACCCTGATAGTGC
CGTCGATGACCGAGATAGTGACTATCGCAGTGAGACCAGCAACAGCTTCCCACCTCCTTACCATACAGCT
TCCCAGCCCAACGCTTCTGTGCACCAGTTCCCTGTGCCGGTGCGATCGCCACAGCAGCTGCTACTTCAAG
GCAGTTCCCGGGACTCTTGTAATGACTCTATGCAAAGTTATGACCTTGATTATCCAGAGCGGCGGGCTAT
CAGCCCCACCAGCAGCAGTAGGTATGGCTCCTCCTGTAATGTGAGTCAAGGAAGCTCTCAGCTAAGTGAA
CTAGACCAGTATCACGAACAAGATGACGACCATCGGGAGACGGACTCGATTCATTCTTGCCACAGCTCTC
ACAGCCTGTCCAGAGATGGCCAAGCAGGTTTTGGAGAACAAGAGAAACCCTTGGAGGTGACAGGTCAAGC
AGAGAAGGAGGCAGCATGTGAACCCAAGGAGATGAAAGAAGATGCCACAACCCACCCTCCCCCAGATCTG
GTGCTGCAAAAAGACCACTTCCTAGGTCCCCAGGAGAGTTTTCCTGAGGAGAATGCATCTTCACCATTTA
CCCAAGCCAGAGCACATTGGATCCGAGCAGTTACCAAGGTTCGACTCCAGCTGCAGGAGATTCCAGATGA
TGGTGACCCCTCTCTGCCTCAGTGGCTCCCGGAAGGGCCAGCCGGAGGGCTCTATGGCATTGACAGCATG
CCAGATTTACGCAGAAAGAAGCCACTGCCACTTGTCAGTGATCTGTCACTGGTCCAGTCTCGGAAGGCAG
GAATCACTTCTGCAATGGCTACACGCACTTCTCTTAAGGACGAAGAGCTGAAATCCCACGTGTATAAGAA
AACCCTGCAGGCCTTAATCTACCCCATTTCGTGCACCACTCCTCATAACTTTGAGGTCTGGACGGCCACT
ACCCCAACCTACTGCTATGAGTGTGAAGGCCTGCTCTGGGGCATTGCCCGGCAGGGCATGCGCTGCAGCG
AATGTGGAGTCAAGTGCCATGAGAAGTGCCAGGATCTGCTCAATGCTGACTGCCTGCAGCGGGCTGCAGA
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ATCCGAGAGCGAAATAAGCCAGAGATCTTTGAAGTTATCCGGGACGTCTTCACAGTGAACAAAGCTGCCC
ATGTGCAGCAGATGAAAACAGTGAAGCAGAGTGTACTGGATGGCACCTCCAAATGGTCAGCCAAGATCAC
CATTACTGTGGTGTGTGCCCAGGGCCTACAAGCCAAGGACAAAACAGGATCCAGTGACCCTTACGTGACT
GTGCAAGTCAGCAAAACTAAGAAGCGTACCAAGACCATTTTTGGAAACTTGAATCCTGTTTGGGAGGAGA
AGTTCCATTTTGAGTGCCACAACTCCTCTGACCGCATTAAGGTGCGTGTATGGGATGAGGATGATGACAT

CAAGTCAAGAGTAAAGCAACGCCTAAAGCGAGAGTCTGATGATTTCCTTGGCCAAACCATCATTGAGGTT
CGGACCCTAAGTGGCGAGATGGACGTCTGGTACAACTTGGAGAAGAGGACAGACAAATCAGCCGTCTCAG
GGGCTATCCGACTACAAATCAGTGTGGAGATCAAGGGGGAGGAGAAAGTAGCCCCATACCACGTGCAGTA
TACATGTCTCCATGAGAATCTTTTCCATTACCTCACAGACATTCAGGGCAGTGGAGGAGTCCGCATCCCT
GAAGCTCGAGGAGACGATGCCTGGAAGGTGTACTTTGATGAGACAGCCCAAGAAATTGTGGATGAATTTG
CCATGCGTTATGGCATTGAGTCCATATATCAGGCCATGACGCACTTTGCATGTTTATCATCCAAGTACAT
GTGTCCTGGTGTGCCAGCAGTGATGAGCACCTTACTGGCCAACATCAACGCCTACTATGCCCACACAACT
GCCTCTACCAATGTCTCTGCATCTGATCGCTTTGCAGCCTCCAACTTTGGGAAAGAGAGATTTGTAAAAC
TGCTGGACCAGCTACACAACTCACTGAGGATCGACCTCTCTACATACAGGAATAATTTCCCTGCTGGGAG
TCCTGAACGGCTTCAGGACTTAAAATCCACAGTGGATTTGCTGACCAGCATTACTTTCTTCAGAATGAAG
GTACAAGAACTGCAAAGCCCTCCAAGAGCCAGCCAGGTGGTAAAGGATTGTGTGAAGGCCTGTTTGAACT
CCACATATGAATATATCTTCAACAACTGCCACGACTTATACAGCCGCCAGTACCAGCTGAAGCAGGAGCT
ACCTCCAGAGGAACAAGGGCCCAGCATTCGGAACCTGGATTTCTGGCCCAAGCTCATCACACTCATCGTG
TCAATCATAGAGGAAGATAAGAATTCCTACACACCTGTTCTGAACCAGTTTCCTCAGGAGTTGAATGTGG
GAAAAGTCAGCGCAGAAGTGATGTGGCATTTGTTTGCCCAAGACATGAAATATGCATTGGAGGAGCATGA
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TACAATGGCTGGATGAGAATGAGGATGTATCCCTGGAATTCCTGCGTGGGGCCCTGGAACGAGATAAGAA
GGATGGATTCCAGCAGACATCAGAGCATGCACTCTTTTCCTGCTCTGTGGTGGATGTCTTCACACAACTC
AATCAGAGCTTTGAGATCATCCGGAAGCTGGAATGCCCAGACCCCAGCATCCTTGCCCACTACATGAGGA
GGTTTGCTAAGACCATCGGGAAGGTGCTGATGCAGTATGCAGACATCTTGTCAAAGGACTTCCCAGCCTA
TTGCACAAAGGAGAAACTGCCCTGCATCCTGATGAACAACGTGCAGCAACTGAGGGTCCAGCTGGAGAAA
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AACTGAATACGGTTCTGGATGAGCTCAGCATGGTGTTTGGAAACAGTTTCCAGGTACGGATTGATGAGTG
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TCAGCGGCTCAGGATGCAGATAGCGTACTCCGGCCTCTCATGGACTTCCTGGATGGCAACCTCACCCTCT
TTGCCACTGTGTGTGAGAAGACGGTTCTGAAGCGTGTACTGAAGGAGCTCTGGCGCGTGGTGATGAACAC
AATGGAGAGGATGATTGTTCTGCCCCCACTCACTGACCAGACGGGCACCCAGCTGATCTTCACTGCTGCC
AAGGAGCTGAGCCATCTTTCCAAACTCAAGGATCACATGGTACGAGAGGAAACACGGAATCTCACTCCAA
AGCAGTGTGCAGTCCTTGACCTCGCCCTGGACACCATCAAGCAATACTTCCATGCAGGAGGCAATGGGCT
GAAGAAAACCTTCCTGGAGAAGAGCCCAGATCTGCAGTCTCTACGCTATGCCCTGTCTCTGTACACACAG
ACTACTGACACTCTCATCAAGACCTTTGTGCGCTCGCAGACCACCCAAGGGTCTGGTGTGGACGATCCTG
TGGGAGAAGTCTCTATTCAGGTGGACTTGTTTACACACCCTGGTACTGGGGAGCACAAGGTCACAGTGAA
AGTGGTGGCTGCCAATGACCTCAAGTGGCAGACAGCGGGTATGTTCCGGCCTTTCGTGGAGGTGACTATG
GTTGGCCCACACCAAAGTGATAAGAAGAGGAAGTTCACAACCAAATCCAAAAGCAACAACTGGGCCCCCA
AGTACAATGAGACATTCCACTTCCTCCTGGGAAATGAGGAGGGGCCCGAGTCCTATGAGTTGCAGATATG
CGTGAAGGATTACTGCTTTGCCCGGGAAGATCGCGTGCTAGGGCTGGCTGTGATGCCTCTGAGGGATGTC
ACAGCCAAGGGCAGCTGTGCCTGCTGGTGCCCCTTGGGCCGGAAGATCCATATGGATGAGACAGGCCTGA
CCATTCTCCGGATTTTATCTCAGAGGAGCAATGACGAGGTGGCCCGAGAATTTGTGAAACTCAAATCAGA
GTCTCGTTCCACGGAGGAGGGGAGCTGAACACCTTCGACTCCTGTGCCAATCAGGCAGCAGCAATTTCAC
AAATCAGGGCCAGTGGGAGTTAGCTGTGTAACCGGCTTAGGGTCTTTGCAGTCAAGAGGCTGACCCCTTC
AGTTAAAGATATTTAAGGAAAAATTTGGGGTGGTGATAATATGGCTTTTCACAGAAAGGGTCATGAAGCC
CTGGCCCAACAGGACTGTGGTACTAGGGGCTGGGATGTGGGGTTACCACATGGAGAGATTTTCCATTAAG
AGAGAAGGACAAACATTTCTGAGAGTGTCAGCCATTCTTGGTAGACACCTCTCCACTCCTCATCCCACCT
CTACCCATCTCCATGCCACACCTTATCCAGTTAGACACATACATACCAATCATTAGAAGAACAAGTTTAG
AAGGTGTGGAACTTGTGCCTGGCTGGCTGGGTAGTCAGCTGAGCCTGTTGCTGAGCCCGGTGGTCTGGAT
TGGAGTATGGCCAGGGCAGGAGTACACAGAATAGAATTTAGACTGTCCCTTGAGTAGAATCCACTGATTT
TCTGTGGCTCCAGTGAGAACAAGGCTTTGAAACTGAACAAGATAACTTCTAGAAATGAACTGTACTAATC
CCTTTCCCCAGATTGTATCATGAGTAGAATCAGGTTCACGTGGTGCTTCAAAGCCCTGAGAAGAATATTT
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CTGTGGCCACTGCCTTCTAGCCGTCCTCCACCTTGCAGAAAGAATCTAGCCTTTGGTCTCTCTCTCTCTC
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GGACTTCCCAAGACTTGTTGAGAGATGTGACATGGTTCTTGGATTTCCTCTGTAGCAGCCTCCTGGACTT
CCTGAGGACTCGACATTGTCCACAGATGTACTGGCCATTACATGAAACAAGAAACCAAGCATCTTTGCTG
TTGTTAATTATTATATGTGCCATTGTTACAGGAGATTATAGGCTAGTCTGTAATAAATTATCTTATAGCA
AAAAAAAAAAAAAAAAAAAAAAAAAAAA

NM_005704.3| Homo sapiens protein tyrosine phosphatase, receptor type, U (PTPRU), transcript variant 3, mRNA
GCCGCGCCGCTCCGCTCCGGCTCGGGCTCCGGCTCGCCTCGGGCTGGGCTCGGGCTCCGGGGGCGGCGTC
CCCCGCGCCGGGCCCCGGGACGGGCGGCGACGCTCCAACCATGGCCCGTGCCCAGGCGCTCGTGCTGGCA
CTCACCTTCCAGCTCTGCGCGCCGGAGACCGAGACTCCGGCAGCTGGCTGCACCTTCGAGGAGGCAAGTG
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CCCTGGCACCCGGGCACCTGCGGACCTGCCCCACGGCTCCTACTTGATGGTCAACACTTCCCAGCATGCC
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ACTTCCTGTACAGCCGGGACGGGCACAGCCCGGGCACCCTGGGCGTCTACGTGCGCGTTAATGGGGGCCC
CCTGGGCAGTGCTGTGTGGAATATGACTGGATCCCACGGCCGTCAGTGGCACCAGGCTGAGCTGGCTGTC
AGCACTTTCTGGCCCAATGAATATCAGGTGCTGTTTGAGGCCCTCATCTCCCCAGACCGCAGGGGCTACA
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CGTGGAGGTCAACGCGGGCCAGAACGCGTCGTTCCAGTGCATGGCCGCGGGCAGAGCGGCCGAGGCCGAA
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GGCCCCGCGCGGCGCGGGCGTCTCTAACTTCGCGGAGCTCATCGTCAAGGAGCCCCCAACTCCCATCGCG
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GACATGATCTTCCTCAAGTGGGAGGAGCCCCAGGAGCCCAATGGTCTCATCACCCAGTATGAGATCAGCT
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CTACCGAGGCTTCTGGAACCCACCACTTGAGCCTAGGAAGGCCTATCTCATCTACTTCCAGGCAGCAAGC
CACCTGAAGGGGGAGACCCGGCTGAATTGCATCCGCATTGCCAGGAAAGCTGCCTGCAAGGAAAGCAAGC
GGCCCCTGGAGGTGTCCCAGAGATCGGAGGAGATGGGGCTTATCCTGGGCATCTGTGCAGGGGGGCTTGC
TGTCCTCATCCTTCTCCTGGGTGCCATCATTGTCATCATCCGCAAAGGGAGAGACCACTATGCCTACTCC
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CTCTGCCCGGCACGAGGTCCGCCAGTTCCACTTCACAGCGTGGCCAGAGCATGGCGTCCCCTACCATGCC
ACGGGGCTGCTGGCTTTCATCCGGCGCGTGAAGGCCTCCACCCCACCTGATGCCGGGCCCATTGTCATCC
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ACCTCCATCGTCATGCTCAACCAGCTGAACCAGTCCAACTCCGCCTGGCCCTGCCTGCAGTACTGGCCAG
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CACTCAGGGCCAGACCCACCATCCTGGACTGGCGAGGAAGATCAGTGCCTCCTGCTCTGCCCAAACACAC
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TGAGATATTTTGCTCACTATCCCTCCCCACTTGCTTCCCTGATATGTGCTCTGAGCTTCCCTGAACCAGG
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ACCTCGCCCCCCTCTTCCTTTAATCTTCAGGCCTCACTGGCCTGTCCTGCTCAGCTTGGGCCAGTGACAA
TCTGCAAGGCTGAACAACAGCCCCTGGGGTTGAGGCCCCTGTGGCTCCTGGTCAGGCTGCCCGTTGTGGG
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TTTGTTTCTTTTGTTATTTTTGGTTGGGTGGGTGGGAAGGTCTCTTTAAAATGGGGCAGGCCACACCCCC
ATTCCGTGCCTCAATTTCCCCATCTGTAAACTGTAGATATGACTACTGACCTACCTCGCAGGGGGCTGTG
GGGAGGCATAAGCTGATGTTTGTAAAGCGCTTTGTAAATAAACGTGCTCTCTGAATGCCACAGAAAAAAA
AAAAAAAAA

NM_000680.2| Homo sapiens adrenergic, alpha-1A-, receptor (ADRA1A), transcript variant 1, mRNA
GAATTCCGAATCATGTGCAGAATGCTGAATCTTCCCCCAGCCAGGACGAATAAGACAGCGCGGAAAAGCA
GATTCTCGTAATTCTGGAATTGCATGTTGCAAGGAGTCTCCTGGATCTTCGCACCCAGCTTCGGGTAGGG
AGGGAGTCCGGGTCCCGGGCTAGGCCAGCCCGGCAGGTGGAGAGGGTCCCCGGCAGCCCCGCGCGCCCCT
GGCCATGTCTTTAATGCCCTGCCCCTTCATGTGGCCTTCTGAGGGTTCCCAGGGCTGGCCAGGGTTGTTT
CCCACCCGCGCGCGCGCTCTCACCCCCAGCCAAACCCACCTGGCAGGGCTCCCTCCAGCCGAGACCTTTT
GATTCCCGGCTCCCGCGCTCCCGCCTCCGCGCCAGCCCGGGAGGTGGCCCTGGACAGCCGGACCTCGCCC
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CGTCAACCTGGCGGTGGCCGACCTCCTGCTCACCTCCACGGTGCTGCCCTTCTCCGCCATCTTCGAGGTC
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AACCATCGTCACCCAGAGGAGGGGTCTCATGGCTCTGCTCTGCGTCTGGGCACTCTCCCTGGTCATATCC
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CCTGATTTCAAGCCCTCTGAAACAGTTTTTAAAATAGTATTTTGGCTCGGATATCTAAACAGCTGCATCA
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CTTCTCGGAAGGCCAGCTCTTCTTGGAGGACAAGACAGGACCAATCAAAGAGGGGACCTGCTGGGAATGG
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ACCAGTTCAGAATGATACGGAACAGCATTTCCCTGCAGCTAATGCTTTCTTGGTCACTCTGTGCCCACTT
CAACGAAAACCACCATGGGAAACAGAATTTCATGCACAATCCAAAAGACTATAAATATAGGATTATGATT
TCATCATGAATATTTTGAGCACACACTCTAAGTTTGGAGCTATTTCTTGATGGAAGTGAGGGGATTTTAT
TTTCAGGCTCAACCTACTGACAGCCACATTTGACATTTATG

NM_002381.4| Homo sapiens matrilin 3 (MATN3), mRNA
AAATCCGAGCCTCGCGTGGGCTCCTGGCCCCCGACGGACACCACCAGGCCCACGGAGCCCACCATGCCGC

GCCCGGCCCCCGCGCGCCGCCTCCCGGGACTCCTCCTGCTGCTCTGGCCGCTGCTGCTGCTGCCCTCCGC
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CGCCGCCCCTCTCCTGCGGCTCCCGACGGCGCGCCCGCTTCCGGGACCAGCGAGCCTGGCCGCGCCCGCG
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CTGGTATTGAGCTCTATGCTGTGGGCGTGGACCGGGCAGACATGGCGTCCCTCAAGATGATGGCCAGTGA
GCCCCTAGAGGAGCATGTTTTCTACGTGGAGACCTATGGGGTCATTGAGAAACTTTCCTCTAGATTCCAG
GAAACCTTCTGTGCGCTGGACCCCTGTGTGCTTGGAACACACCAGTGCCAGCACGTCTGCATCAGTGATG
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ACCTGAAAATGTGGACAGCTTGGTGTACTTAATACTCATGCATTCTTTTGCACACCTGTTATTGCCAATG
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TATTATCATGTCTATTTTTGATGACTCATCAATTTTGTCTATTAAAGATATTTCTTTAAATTAAAAAAAA
AAAAAAAAAA

NM_032906.3| Homo sapiens phosphatidylinositol glycan anchor biosynthesis, class Y (PIGY), transcript variant 1, mRNA
ACCAGGGGGCGGGGCCTGCCCGGCTGGCCTGGACGAACGGGAAGCCGGGAGCTCGGCCACGGGTGGCGAG
GCTGCGGTGAGGCCTGGTCTCCGGCTGCCAGACCATGCTGAGTGGAGCACGCTGCAGGCTCGCCTCAGCG
CTGCGGGGAACGCGCGCGCCGCCGTCCGCGGTCGCCCGTAGGTGCCTGCACGCGTCGGGGTCGCGGCCTT
TGGCCGACCGGGGCAAGAAGACTGAGGAGCCGCCCCGCGACTTCGATCCGGCGCTGCTGGAGTTCCTGGT
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ACATGTTAAGGATGACTGAAATAATTCCAAAGGAGTGATGTTGGAATAGTCCCTCTAAGGGAGAGAAATG
CATTTGAACGAATGTGATATAAAACCACATAATCAAATAGAAACTTCATGTACTTACAAAAACTGAGTTT
GTAAAATTACCTTCATTTCTTTGACATTAAATGCTTATATTAGCAATAAACATGTTGACACTTTCCTATA
AAAAATAAACCAGTTTGCAGTAG

NM_001736.3| Homo sapiens complement component 5a receptor 1 (C5AR1), mRNA
CTTGGGCAGGAGGGACCTTCGATCCTCGGGGAGCCCAGGAGACCAGAACATGAACTCCTTCAATTATACC
ACCCCTGATTATGGGCACTATGATGACAAGGATACCCTGGACCTCAACACCCCTGTGGATAAAACTTCTA
ACACGCTGCGTGTTCCAGACATCCTGGCCTTGGTCATCTTTGCAGTCGTCTTCCTGGTGGGAGTGCTGGG
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ACTGGCCCTTTGGCGGGGCCGCCTGCAGCATCCTGCCCTCCCTCATCCTGCTCAACATGTACGCCAGCAT
CCTGCTCCTGGCCACCATCAGCGCCGACCGCTTTCTGCTGGTGTTTAAACCCATCTGGTGCCAGAACTTC
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CCTTCCTGTACCGGGTGGTCCGGGAGGAGTACTTTCCACCAAAGGTGTTGTGTGGCGTGGACTACAGCCA
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CCAGCCTCCCCCAATGGCAACATCTTGCAAAACTACAATGTAGTCTCATAACCAGGATATTGACATTGAT
ACAGTGAAGATACAGGACATTCTCATCACCACAGGGATCCCCAGGATGCCCACTTCCCTCCACCCCCACA
CCCCAGCCGTGTCCCTAACCCCTGGCAACCAGGAATCCACTCTCCATTTCTATAATGTTGTCATTTCAAG
AATGTTATTCAATGGAATCATATAGTATGTAACCTGTTTTGAGCTTAAAAAAAAAGTATACATGACTTTA
ATGAGGAAAATAAAAATGAATATTGAAATGTT

NM_003173.2| Homo sapiens suppressor of variegation 3-9 homolog 1 (Drosophila) (SUV39H1), mRNA
CTCGCGAGGCCGGCTAGGCCCGAATGTCGTTAGCCGTGGGGAAAGATGGCGGAAAATTTAAAAGGCTGCA
GCGTGTGTTGCAAGTCTTCTTGGAATCAGCTGCAGGACCTGTGCCGCCTGGCCAAGCTCTCCTGCCCTGC
CCTCGGTATCTCTAAGAGGAACCTCTATGACTTTGAAGTCGAGTACCTGTGCGATTACAAGAAGATCCGC
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AAGAACAGCTTCGTCATGGAGTACGTGGGAGAGATCATTACCTCAGAGGAGGCAGAGCGGCGGGGCCAGA
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CTTTGGCCTGGCTGGGCTCCCTGGCTCCCCTAAGAAGCGGGTCCGTATTGAATGCAAGTGTGGGACTGAG
TCCTGCCGCAAATACCTCTTCTAGCCCTTAGAAGTCTGAGGCCAGACTGACTGAGGGGGCCTGAAGCTAC
ATGCACCTCCCCCACTGCTGCCCTCCTGTCGAGAATGACTGCCAGGGCCTCGCCTGCCTCCACCTGCCCC
CACCTGCTCCTACCTGCTCTACGTTCAGGGCTGTGGCCGTGGTGAGGACCGACTCCAGGAGTCCCCTTTC
CCTGTCCCAGCCCCATCTGTGGGTTGCACTTACAAACCCCCACCCACCTTCAGAAATAGTTTTTCAACAT

CAAGACTCTCTGTCGTTGGGATTCATGGCCTATTAAGGAGGTCCAAGGGGTGAGTCCCAACCCAGCCCCA
GAATATATTTGTTTTTGCACCTGCTTCTGCCTGGAGATTGAGGGGTCTGCTGCAGGCCTCCTCCCTGCTG
CCCCAAAGGTATGGGGAAGCAACCCCAGAGCAGGCAGACATCAGAGGCCAGAGTGCCTAGCCCGACATGA
AGCTGGTTCCCCAACCACAGAAACTTTGTACTAGTGAAAGAAAGGGGGTCCCTGGGCTACGGGCTGAGGC
TGGTTTCTGCTCGTGCTTACAGTGCTGGGTAGTGTTGGCCCTAAGAGCTGTAGGGTCTCTTCTTCAGGGC
TGCATATCTGAGAAGTGGATGCCCACATGCCACTGGAAGGGAAGTGGGTGTCCATGGGCCACTGAGCAGT
GAGAGGAAGGCAGTGCAGAGCTGGCCAGCCCTGGAGGTAGGCTGGGACCAAGCTCTGCCTTCACAGTGCA
GTGAAGGTACCTAGGGCTCTTGGGAGCTCTGCGGTTGCTAGGGGCCCTGACCTGGGGTGTCATGACCGCT
GACACCACTCAGAGCTGGAACCAAGATCTAGATAGTCCGTAGATAGCACTTAGGACAAGAATGTGCATTG
ATGGGGTGGTGATGAGGTGCCAGGCACTGGGTAGAGCACCTGGTCCACGTGGATTGTCTCAGGGAAGCCT
TGAAAACCACGGAGGTGGATGCCAGGAAAGGGCCCATGTGGCAGAAGGCAAAGTACAGGCCAAGAATTGG
GGGTGGGGGAGATGGCTTCCCCACTATGGGATGACGAGGCGAGAGGGAAGCCCTTGCTGCCTGCCATTCC
CAGACCCCAGCCCTTTGTGCTCACCCTGGTTCCACTGGTCTCAAAAGTCACCTGCCTACAAATGTACAAA
AGGCGAAGGTTCTGATGGCTGCCTTGCTCCTTGCTCCCCCACCCCCTGTGAGGACTTCTCTAGGAAGTCC
TTCCTGACTACCTGTGCCCAGAGTGCCCCTACATGAGACTGTATGCCCTGCTATCAGATGCCAGATCTAT
GTGTCTGTCTGTGTGTCCATCCCGCCGACCCCCCAGACTAACCTCCAGGCATGGACTGAATCTGGTTCTC
CTCTTGTACACCCCTCAACCCTATGCAGCCTGGAGTGGGCATCAATAAAATGAACTGTCGACTGAACAAA
AAAAAAAAAAAAAAA

NM_016155.4| Homo sapiens matrix metallopeptidase 17 (membrane-inserted)
(MMP17), mRNA
CCGGCGGGGGCGCCGCGGAGAGCGGAGGGCGCCGGGCTGCGGAACGCGAAGCGGAGGGCGCGGGACCCTG
CACGCCGCCCGCGGGCCCATGTGAGCGCCATGCGGCGCCGCGCAGCCCGGGGACCCGGCCCGCCGCCCCC
AGGGCCCGGACTCTCGCGGCTGCCGCTGCCGCTGCTGCTGCTGCTGGCGCTGGGGACCCGCGGGGGCTGC
GCCGCGCCCGCACCCGCGCCGCGCGCCGAGGACCTCAGCCTGGGAGTGGAGTGGCTAAGCAGGTTCGGTT
ACCTGCCCCCGGCTGACCCCACAACAGGGCAGCTGCAGACGCAAGAGGAGCTGTCTAAGGCCATCACAGC
CATGCAGCAGTTTGGTGGCCTGGAGGCCACCGGCATCCTGGACGAGGCCACCCTGGCCCTGATGAAAACC
CCACGCTGCTCCCTGCCAGACCTCCCTGTCCTGACCCAGGCTCGCAGGAGACGCCAGGCTCCAGCCCCCA
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GTGGCGGGCAGCGCCGCCGACATCCAGATCGACTTCTCCAAGGCCGACCATAACGACGGCTACCCCTTCG
ACGGCCCCGGCGGCACCGTGGCCCACGCCTTCTTCCCCGGCCACCACCACACCGCCGGGGACACCCACTT
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CACGAGTTTGGCCACGCCATTGGGTTAAGCCATGTGGCCGCTGCACACTCCATCATGCGGCCGTACTACC
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CGGTGTGCGGGAGTCTGTGTCTCCCACGGCGCAGCCCGAGGAGCCTCCCCTGCTGCCGGAGCCCCCAGAC
AACCGGTCCAGCGCCCCGCCCAGGAAGGACGTGCCCCACAGATGCAGCACTCACTTTGACGCGGTGGCCC
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GTCCCTGCAGCCGGCACAGATGCACCGCTTCTGGCGGGGCCTGCCGCTGCACCTGGACAGCGTGGACGCC
GTGTACGAGCGCACCAGCGACCACAAGATCGTCTTCTTTAAAGGAGACAGGTACTGGGTGTTCAAGGACA
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CTTCTCCTGGGCCCACAATGACAGGACTTATTTCTTTAAGGACCAGCTGTACTGGCGCTACGATGACCAC
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ACGCCATGCGCTGGTCCGACGGTGCCTCCTACTTCTTCCGTGGCCAGGAGTACTGGAAAGTGCTGGATGG
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GCCGATGGATCTGTGGCTGCGGGCGTGGACGCGGCAGAGGGGCCCCGCGCCCCTCCAGGACAACATGACC
AGAGCCGCTCGGAGGACGGTTACGAGGTCTGCTCATGCACCTCTGGGGCATCCTCTCCCCCGGGGGCCCC
AGGCCCACTGGTGGCTGCCACCATGCTGCTGCTGCTGCCGCCACTGTCACCAGGCGCCCTGTGGACAGCG
GCCCAGGCCCTGACGCTATGACACACAGCGCGAGCCCATGAGAGGACAGAGGCGGTGGGACAGCCTGGCC
ACAGAGGGCAAGGACTGTGCCGGAGTCCCTGGGGGAGGTGCTGGCGCGGGATGAGGACGGGCCACCCTGG
CACCGGAAGGCCAGCAGAGGGCACTGCCCGCCAGGGCTGGGCAGGCTCAGGTGGCAAGGACGGAGCTGTC
CCCTAGTGAGGGACTGTGTTGACTGACGAGCCGAGGGGTGGCCGCTCCAGAAGGGTGCCCAGTCAGGCCG
CACCGCCGCCAGCCTCCTCCGGCCCTGGAGGGAGCATCTCGGGCTGGGGGCCCACCCCTCTCTGTGCCGG
CGCCACCAACCCCACCCACACTGCTGCCTGGTGCTCCCGCCGGCCCACAGGGCCTCCGTCCCCAGGTCCC
CAGTGGGGCAGCCCTCCCCACAGACGAGCCCCCCACATGGTGCCGCGGCACGTCCCCCCTGTGACGCGTT
CCAGACCAACATGACCTCTCCCTGCTTTGTAAAAAAAAAAAAAAAAAA

XM_114973.6| PREDICTED: Homo sapiens EPH receptor A6 (EPHA6), mRNA
GGCCCAAGTGAATAGTCCTCGCGCAAGCGGGACACTGTGGTGGATGCAATTCCCCTCGCCTCCAGCCGCG
AGGAGCTCCCCGGCGCCGCAGGCAGCGTCCTCCTCCGAAGCAGCTGCACCTGCAACTGGGCAGCCTGGAC
CCTCGTGCCCTGTTCCCGGGACCTCGCGCAGGGGGCGCCCCGGGACACCCCCTGCGGGCCGGGTGGAGGA

GGAAGAGGAGGAGGAGGAAGAAGACGTGGACAAGGACCCCCATCCTACCCAGAACACCTGCCTGCGCTGC
CGCCACTTCTCTTTAAGGGAGAGGAAAAGAGAGCCTAGGAGAACCATGGGGGGCTGCGAAGTCCGGGAAT
TTCTTTTGCAATTTGGTTTCTTCTTGCCTCTGCTGACAGCGTGGCCAGGCGACTGCAGTCACGTCTCCAA
CAACCAAGTTGTGTTGCTTGATACAACAACTGTACTGGGAGAGCTAGGATGGAAAACATATCCATTAAAT
GGGTGGGATGCCATCACTGAAATGGATGAACATAATAGGCCCATTCACACATACCAGGTATGTAATGTAA
TGGAACCAAACCAAAACAACTGGCTTCGTACAAACTGGATCTCCCGTGATGCAGCTCAGAAAATTTATGT
GGAAATGAAATTCACACTAAGGGATTGTAACAGCATCCCATGGGTCTTGGGGACTTGCAAAGAAACATTT
AATCTGTTTTATATGGAATCAGATGAGTCCCACGGAATTAAATTCAAGCCAAACCAGTATACAAAGATCG
ACACAATTGCTGCTGATGAGAGTTTTACCCAGATGGATTTGGGTGATCGCATCCTCAAACTCAACACTGA
AATTCGTGAGGTGGGGCCTATAGAAAGGAAAGGATTTTATCTGGCTTTTCAAGACATTGGGGCGTGCATT
GCCCTGGTTTCAGTCCGTGTTTTCTACAAGAAATGCCCCTTCACTGTTCGTAACTTGGCCATGTTTCCTG
ATACCATTCCAAGGGTTGATTCCTCCTCTTTGGTTGAAGTACGGGGTTCTTGTGTGAAGAGTGCTGAAGA
GCGTGACACTCCTAAACTGTATTGTGGAGCTGATGGAGATTGGCTGGTTCCTCTTGGAAGGTGCATCTGC
AGTACAGGATATGAAGAAATTGAGGGTTCTTGCCATGCTTGCAGACCAGGATTCTATAAAGCTTTTGCTG
GGAACACAAAATGTTCTAAATGTCCTCCACACAGTTTAACATACATGGAAGCAACTTCTGTCTGTCAGTG
TGAAAAGGGTTATTTCCGAGCTGAAAAAGACCCACCTTCTATGGCATGTACCAGGCCACCTTCAGCTCCT
AGGAATGTGGTTTTTAACATCAATGAAACAGCCCTTATTTTGGAATGGAGCCCACCAAGTGACACAGGAG
GGAGAAAAGATCTCACATACAGTGTAATCTGTAAGAAATGTGGCTTAGACACCAGCCAGTGTGAGGACTG
TGGTGGAGGACTCCGCTTCATCCCAAGACATACAGGCCTGATCAACAATTCCGTGATAGTACTTGACTTT
GTGTCTCACGTGAATTACACCTTTGAAATAGAAGCAATGAATGGAGTTTCTGAGTTGAGTTTTTCTCCCA
AGCCATTCACAGCTATTACAGTGACCACGGATCAAGATGCACCTTCCCTGATAGGTGTGGTAAGGAAGGA
CTGGGCATCCCAAAATAGCATTGCCCTATCATGGCAAGCACCTGCTTTTTCCAATGGAGCCATTCTGGAC
TACGAGATCAAGTACTATGAGAAAGAACATGAGCAGCTGACCTACTCTTCCACAAGGTCCAAAGCCCCCA
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GGACAGATTCTCGTGATAGCCACCGCCGCTGTTGGCGGATTCACTCTCCTCGTCATCCTCACTTTATTCT
TCTTGATCACTGGGAGATGTCAGTGGTACATAAAAGCCAAGATGAAGTCAGAAGAGAAGAGAAGAAACCA
CTTACAGAATGGGCATTTGCGCTTCCCGGGAATTAAAACTTACATTGATCCAGATACATATGAAGACCCA
TCCCTAGCAGTCCATGAATTTGCAAAGGAGATTGATCCCTCAAGAATTCGTATTGAGAGAGTCATTGGGG
CAGGTGAATTTGGAGAAGTCTGTAGTGGGCGTTTGAAGACACCAGGGAAAAGAGAGATCCCAGTTGCCAT
TAAAACTTTGAAAGGTGGCCACATGGATCGGCAAAGAAGAGATTTTCTAAGAGAAGCTAGTATCATGGGC
CAGTTTGACCATCCAAACATCATTCGCCTAGAAGGGGTTGTCACCAAAAGATCCTTCCCGGCCATTGGGG
TGGAGGCGTTTTGCCCCAGCTTCCTGAGGGCAGGGTTTTTAAATAGCATCCAGGCCCCGCATCCAGTGCC
AGGGGGAGGATCTTTGCCCCCCAGGATTCCTGCTGGCAGACCAGTAATGATTGTGGTGGAATATATGGAG
AATGGATCCCTAGACTCCTTTTTGCGGAAGCATGATGGCCACTTCACAGTCATCCAGTTGGTCGGAATGC
TCCGAGGCATTGCATCAGGCATGAAGTATCTTTCTGATATGGGTTATGTTCATCGAGACCTAGCGGCTCG
GAATATACTGGTCAATAGCAACTTAGTATGCAAAGTTTCTGATTTTGGTCTCTCCAGAGTGCTGGAAGAT
GATCCAGAAGCTGCTTATACAACAACTGGTGGAAAAATCCCCATAAGGTGGACAGCCCCAGAAGCCATCG
CCTACAGAAAATTCTCCTCAGCAAGCGATGCATGGAGCTATGGCATTGTCATGTGGGAGGTCATGTCCTA
TGGAGAGAGACCTTATTGGGAAATGTCTAACCAAGATGTCATTCTGTCCATTGAAGAAGGGTACAGACTT
CCAGCTCCCATGGGCTGTCCAGCATCTCTACACCAGCTGATGCTCCACTGCTGGCAGAAGGAGAGAAATC
ACAGACCAAAATTTACTGACATTGTCAGCTTCCTTGACAAACTGATCCGAAATCCCAGTGCCCTTCACAC
CCTGGTGGAGGACATCCTTGTAATGCCAGAGTCCCCTGGTGAAGTTCCGGAATATCCTTTGTTTGTCACA
GTTGGTGACTGGCTAGATTCTATAAAGATGGGGCAATACAAGAATAACTTCGTGGCAGCAGGGTTTACAA
CATTTGACCTGATTTCAAGAATGAGCATTGATGACATTAGAAGAATTGGAGTCATACTTATTGGACACCA
GAGACGAATAGTCAGCAGCATACAGACTTTACGTTTACACATGATGCACATACAGGAGAAGGGATTTCAT
GTATGAAAGTACCACAAGCACCTGTGTTTTGTGCCTCAGCATTTCTAAAATGAACGATATCCTCTCTACT
ACTCTCTCTTCTGATTCTCCAAACATCACTTCACAAACTGCAGTCTTCTGTTCAGACTATAGGCACACAC
CTTATGTTTATGCTTCCAACCAGGATTTTAAAATCATGCTACATAAATCCGTTCTGAATAACCTGCAACT
AAAACCCTGGCCCACTGCAGATTATTGCTACGCAATGCAACAGCTTTAAAACCTATCTCAGCCCATGAAT
GGAAAACAAATCCAAATCCGATCCTTGAAAAGCAAAGGCTCTAAAGAAGCTCTTCAGAAGAGACGGTAAA
GAATGAATTCTTTTACTTATCACCCAACCACATTTCTTAAAAATGTGTTTTGGTGTCTTTTCCTACCAAA
TTTCTGCTCTACAAGGCAGTCAGTTAAATCTCTCATTTCATAATTTTCACTGTGCTAGTTCTTTCTTTTC
TCCTCTTTTAATAAATTTTCTAAAACTTT

XM_209695.5| PREDICTED: Homo sapiens KIF4B, transcript variant 1 (LOC285643), mRNA
GTGCGCGCTCCGCCCTCGGGCCGCCTGCACTGCAATTGGTTGGTGTCTCTGGGAGGGATTTGAAACTTGG
CGGTTAAAGCTCCGGCTGGGACAGGGCGGCGGGAGACCCCGGGTGAACGGGGAAGGGACATTTAGTTTGA
GACGGTGCTGAGATAGGATCATGAAGGAAGAGGTGAAGGGAATTCCTGTAAGAGTGGCACTGCGTTGTCG
CCCTCTGGTCCCCAAAGAGATTAGCGAGGGCTGCCAGATGTGCCTTTCCTTCGTGCCCGGGGAGACTCAG

GTGGTGGTTGGTACTGATAAATCCTTCACCTACGATTTTGTGTTTGACCCCTGTACTGAGCAGGAAGAAG
TCTTCAATAAAGCAGTAGCGCCGCTCATAAAAGGCATATTTAAAGGATATAATGCAACGGTCCTGGCCTA
TGGGCAGACTGGCTCTGGAAAAACCTATTCAATGGGAGGTGCATACACTGCGGAGCAGGAGAATGAACCA
ACAGTTGGCATTATTCCTAGGGTAATACAACTGCTCTTCAAAGAAATTGATAAAAAGAGTGACTTTGAAT
TTACTCTGAAAGTGTCTTACTTAGAGATTTACAATGAAGAAATTTTGGATCTTCTATGCCCATCTCGTGA
GAAAGCTCAAATAAATATACGGGAGGATCCTAAGGAAGGCATAAAGATTGTGGGACTCACTGAGAAGACT
GTTTTAGTTGCCTTGGATACTGTTTCCTGTTTGGAGCAGGGCAACAACTCTAGGACTGTGGCCTCCACAG
CTATGAACTCCCAGTCGTCCCGATCTCATGCCATCTTTACAATCTCCATAGAGCAAAGAAAGAAAAGTGA
CAAGAATTGCAGCTTTCGCTCCAAGCTGCATCTTGTAGATCTCGCTGGATCAGAAAGACAGAAGAAAACC
AAGGCTGAAGGGGATCGTCTAAAAGAGGGTATTAATATTAACCGAGGCCTCCTATGCTTGGGAAATGTAA
TCAGTGCTCTTGGAGATGACAAAAAGGGTAGCTTTGTGCCCTACAGAGATTCCAAGTTAACTCGACTGCT
GCAAGATTCTCTAGGAGGTAACAGCCACACTCTTATGATAGCCTGTGTGAGTCCTGCTGACTCCAATCTA
GAGGAAACATTAAGTACCCTTCGCTATGCTGACAGAGCAAGAAAAATCAAGAACAAACCTATTGTTAATA
TTGATCCCCACACAGCTGAACTTAATCATCTAAAGCAACAGGTACAACAGCTACAAGTCTTGTTGCTACA
AGCCCATGGAGGTACCCTGCCTGGATCTATAAATGCAGAACCATCAGAGAATCTACAATCCCTGATGGAG
AAGAATCAGTCCCTGGTAGAGGAGAATGAAAAATTAAGTCGTTGTCTGAGCAAGGCAGCTGGTCAGACAG
CCCAGATGTTGGAGAGGATCATTTTGACAGAGCAAGTGAATGAAAAACTGAACGCCAAGCTAGAAGAGCT
CAGGCAGCATGTGGCCTGCAAGCTGGATCTTCAAAAGCTAGTGGAGACTTTGGAAGACCAGGAATTGAAA
GAAAATGTAGAGATAATTTGTAACCTGCAGCAACTGATTACCCAGTTATCAGATGAAACTGTTGCTTGCA
CGGCTGCAGCCATTGATACTGCGGTAGAAGAAGAAGCTCAAGTGGAAACCAGTCCAGAGACAAGCAGGTC
TTCTGACGCTTTTACCACTCAGCATGCTCTCCATCAAGCTCAGATGTCTAAGGAGGTGGTTGAGTTGAAT
AACGCCCTTGCACTGAAAGAGGCCCTAGTTAGGAAGATGACTCAGAACGACAACCAACTACAGCCCATTC
AGTTTCAATACCAGGATAACATAAAAAATCTAGAATTAGAAGTCATCAATCTGCAAAAGGAAAAGGAAGA
ATTGGTTCGTGAACTTCAGACAGCAAAGAAGAATGTCAACCAAGCCAAGCTGAGTGAGCACCGCCACAAA
CTTCTCCAGGAGCTGGAGGGTCAAATAGCTGATCTGAAGAAGAAACTGAATGAGCAGTCCAAACTTCTGA
AACTAAAGGAATCCACAGAGCGTACTGTCTCCAAGCTGAACCAAGAGATATGGATGATGAAAAACCAGCG
GGTACAGTTAATGCGTCAAATGAAAGAGGATGCTGAGAAGTTTAGACAATGGAAGCAGAAAAAAGACAAA
GAAGTAATACAGTTGAAAGAACGAGACCGTAAGAGGCAATATGAGCTGCTCAAACTTGAAAGAAACTTCC
AGAAACAATCCAGTGTGCTCAGACGTAAAACGGAAGAGGCAGCAGCTGCCAACAAGCGACTCAAGGATGC
TCTCCAGAAACAACGAGAGGTCACAGATAAGCGGAAAGAGACTCAGAGCCATGGAAAGGAAGGTATTGCA
GCTCGAGTGAGGAATTGGCTTGGAAATGAAATTGAGGTTATGGTCAGTACTGAGGAAGCCAAACGCCATC
TGAATGACCTCCTTGAAGACAGAAAGATCCTGGCTCAGGATGTGGTTCAACTCAAAGAAAAAAAGGAATC
TCGGGAGAATCCACCTCCTAAACTCCGGAAGTGTACATTCTCCCTTTCTGAGGTGCATGGTCAAGTTTTG
GAGTCAGAAGATTGTATTACAAAACAGATTGAAAGCCTAGAGACTGAAATGGAACTCAGGAGTGCTCAGA
TTGCTGACCTACAGCAGAAGCTGCTGGATGCAGAAAGTGAAGATAGGCCAAAACAATGCTGGGAGAATAT
TGCCACCATTCTGGAAGCCAAGTGTGCCCTGAAATATTTGATTGGAGAGCTGGTCTCCTCCAAAATACAT
GTCACCAAACTTGAAAACAGCCTGAGACAGAGCAAGGCCAGCTGTGCTGACATGCAGAAGATGCTATTTG
AGGAACAAAATCATTTTTCTGAGATAGAGACAGAGTTACAAGCTGAGCTGGTCAGAATGGAGCAACAGCA
CCAAGAGAAGGTGCTATACCTTGTCAGCCAGCTGCAGGAAAGCCAAATGGCAGAGAAGCAGTTAGAGAAA
TCAGCCAGTGAAAAGGAACAACAGCTGGTGAGCACACTGCAGTGTCAGGATGAAGAACTTGAGAAGATGC
GAGAAGTGTGTGAGCAAAATCAGCAGCTTCTCCAAGAGAATGAAATCATCAAGCAGAAACTGATCCTCCT
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ATCCCACCTAAGCCAAAACCTTCTCGTGTTAAAGAAAAGTTTCTGGAGCAAAGCATGGACATCGAGGATC
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GGATGATGAGGAATGGAAGCCAACAAAATTAGTCAAGGTGTCCAGGAAGAACATCCAAGGGTGTTCCTGC
AAGGGCTGGTGTGGGAACAAGCAGTGTGGGTGCAGGAAGCAAAAGTCAGACTGTGGTGTGGACTGTAGCT
GTGACCCCACAAAGTGTCGGAACCGCCAGCAAGGCAAGGATAGCTTGGGCACTGTTGAACAGACCCAGGA
TTCCGAAGGCTCCTTCAAACTGGAGGATCCTACCGAGGTGACCCCAGGATTGAGCTTCTTTAACCCTGTC
TGTGCCACCCCCAATAGCAAGATCCTGAAAGAGATGTGTGACATGGAGCAGGTGCTGTCAAAGAAGACTG
CTCCAGCTCCCTCCCCTTTTGACCTCCCAGAGTCGAAACATGGAGCAACAGAATACCAACAAAATAAGCC
TCCAGGGAAGAAAAAGAAACGAGCTCTGGCTAGCAACACCAGCTTCTTCTCTGGCTGCTCCCCTATCGAA
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CATTACTAAAAAGAAGGTAACCTTTGTTGGATGTTGTCCCTCAGTCTCCATCCCCAGACTACTGCTCTCT
GCTCTCTAGAAGGCTGCTAAACCACCTGCTGAAGAGAGAACCAACAGACTTTCCTAATGACTCATCAGGA
ACCAGTCCTCAGTATGATCAAGTTCCTTCTTATTTGTGAGCAGTTCAGGCTATCTCCTGATGGGGATGAG
GCCAAGGCTTTCTTATCTTTTGGTTGTCTCTGCTTAATGGAGGAGCCTGGCCTAGGATGGAGGTCTGGCT
CAGATCTTTCACTCCACCTCGGGAATGGGGTTGTGATCTTTCCTGTCCTGACCCCCTCTGAATTGTAAGT
GTTTCAATAGTACTCTTGATTGTATGCCATGTTGTTGAAGTAAATGAATTATTTTTAAATGTTAAGTAAA
TAAACCTTAGCCCATC

Figure 64 (Continued)

XM_374502.3| PREDICTED: Homo sapiens mucin 3B, cell surface associated (MUC3B), mRNA

ATGCTCTGTGCAGATGTAGTGGAGACCGAGGTGGGCATGGAAGTGTCTGTGGATCAGCAGTTCTCGCCGG
ACCTCAATGACAACACTTCCCAGGCCTACAGGGATTTCAACAAGACCTTCTGGAATCAGATGCAGAAGAT
TTTTGCAGACATGCAGGGCTTCACCTTCAAGGGTGTGGAGATCCTGTCCCTGAGGAATGGCAGCATCGTG
GTGGACTACCTGGTCCTGCTGGAGATGCCCTTCAGCCCCCAGCTGGAGAGCGAGTATGAGCAGGTGAAGA
CCACGCTGAAGGAGGGGCTCCAGAACGCCAGCCAGGATGCGAACAGCTGCCAGGACTCCCAGACCCTGTG
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CGCGCCGCTCCCACGGGCTATGAAGAGTTCTACTTCCCTCTGGTGGAGGCCACCCGGCTCCGCTGTGTCA
CCAAATGCACGTCGGGCGTGGACAACGCCATCGACTGTCACCAGGGCCAGTGCGTTCTAGAGACGAGCGG
TCCCGCGTGTCGCTGCTACTCCACCGACACGCACTGGTTCTCTGGCCCGCGCTGCGAGGTGGCCGTCCAC
TGGAGGGCGCTGGTCGGGGGCCTGACGGCCGGCGCCGCGCTGCTGGTGCTGCTGCTGCTGGCGCTGGGCG
TCCGGGCGGTGCGCTCCGGATGGTGGGGCGGCCAGCGCCGAGGCCGGTCCTGGGACCAGGACAGGAAATG
GTTCGAGACCTGGGATGAGGAAGTCGTGGGCACTTTTTCAAACTGGGGTTTCGAGGACGACGGAACAGAC
AAGGATACAAATTTCCATGTGGCCTTGGAGAACGTGGACACCACTATGAAGGTGCACATCAAGAGACCCG
AGATGACCTCGTCCTCAGTGTGA

NM_003292.2| Homo sapiens translocated promoter region (to activated MET oncogene) (TPR), mRNA

GCGCAAGAGGATCAGGGATAGCCTCTGAGCTCGGGTTCCCAGGGTTCGTAGCTTCCAACGGCTGCGCGCG
CACTTCGGTCGCGGGCGGTGAGGTGCTGTTGCTGAAACGCTGCCGCTGAGGGTGGACTCGATTTCCCAGG
GTCCCGCCGCGGGAGTCTCCGGCGGGCGGGCGCGCGAGCCACCGAGCGAGGTGATAGAGGCGGCGGCC
CAGGCGTCTGGGTCCTGCTGGTCTTCGCCTTTCTTCTCCGCTTCTACCCCGTCGGCCGCTGCCACTGGGG
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CAAGTCTGTCCAGAACAAACTTGAAAAGTTCCTTGCTGATCAGCAATCCGAGATCGATGGCCTGAAGGGG
CGGCATGAGAAATTTAAGGTGGAGAGCGAACAACAGTATTTTGAAATAGAAAAGAGGTTGTCCCACAGTC
AGGAGAGACTTGTGAATGAAACCCGAGAGTGTCAAAGCTTGCGGCTTGAGCTAGAGAAACTCAACAATCA
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CAATTTACAAGAACAAAGGAAGAATTAGAAGCTGAGAAAAGAGACTTAATTAGAACCAATGAGAGACTAT
CTCAAGAACTTGAATACTTAACAGAGGATGTTAAACGTCTGAATGAAAAACTTAAAGAAAGCAATACAAC
AAAGGGTGAACTTCAGTTAAAATTGGATGAACTTCAAGCTTCTGATGTTTCTGTTAAGTATCGAGAAAAA
CGCTTGGAGCAAGAAAAGGAATTGCTACATAGTCAGAATACATGGCTGAATACAGAGTTGAAAACCAAAA
CTGATGAACTTCTGGCTCTTGGAAGAGAAAAAGGGAATGAGATTCTAGAGCTTAAATGTAATCTTGAAAA
TAAAAAAGAAGAGGTTTCTAGACTGGAAGAACAAATGAATGGCTTAAAAACATCAAATGAACATCTTCAA
AAGCATGTGGAGGATCTGTTGACCAAATTAAAAGAGGCCAAGGAACAACAGGCCAGTATGGAAGAGAAAT
TCCACAATGAATTAAATGCCCACATAAAACTTTCTAATTTGTACAAGAGTGCCGCTGATGACTCAGAAGC
AAAGAGCAATGAACTAACCCGGGCAGTAGAGGAACTACACAAACTTTTGAAAGAAGCTGGTGAAGCCAAC
AAAGCAATACAAGATCATCTTCTAGAGGTGGAGCAATCCAAAGATCAAATGGAAAAAGAAATGCTTGAGA
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CATATTGTCTGAAGAAGAGCTTGCCGCCATGTCTCCTACTGCAGCAGCTGTAGCTAAGATAGTGAAACCT
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AGAAGCAAGGGGTAACCACGTAATTCGTGATGAGGAAGTAAGCTCTGCTGATATAAGTAGTTCATCTGAG
GTAATATCACAGCATCTAGTATCTTACAGAAATATTGAAGAGCTTCAACAACAAAATCAACGTCTCTTAG
TGGCCCTTAGAGAGCTTGGGGAAACCAGAGAAAGAGAAGAACAAGAAACAACTTCATCCAAAATCACTGA
GCTTCAGCTCAAACTTGAGAGTGCCCTTACTGAACTAGAACAACTCCGCAAATCACGACAGCATCAAATG
CAGCTTGTTGATTCCATAGTTCGTCAGCGTGATATGTACCGTATTTTATTGTCACAAACAACAGGAGTTG
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ACAGACTGTTTCCACTCCTGCTCCAGTACCTGTTATTGAATCAACAGAGGCTATAGAGGCTAAGGCTGCC
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AGCAGCTTGAGAAACTTCAAGAACAAGTTACAGATTTGCGATCACAAAATACCAAAATTTCTACCCAGCT
AGATTTTGCTTCTAAACGTTATGAAATGCTGCAAGATAATGTTGAAGGATATCGTCGAGAAATAACATCA
CTTCATGAGAGAAATCAGAAACTCACTGCCACAACTCAAAAGCAAGAACAGATTATCAATACGATGACTC
AAGATTTGAGAGGAGCAAATGAGAAGCTAGCTGTCGCAGAAGTAAGAGCAGAAAATTTGAAGAAGGAAAA
GGAAATGCTTAAATTGTCTGAAGTTCGTCTTTCTCAGCAAAGAGAGTCTTTGTTAGCTGAACAAAGGGGG
CAAAACTTACTGCTAACTAATCTGCAAACAATTCAGGGAATACTGGAGCGATCTGAAACAGAAACCAAAC
AAAGGCTTAGTAGCCAGATAGAAAAACTGGAACATGAGATCTCTCATCTAAAGAAGAAGTTGGAAAATGA
GGTGGAACAAAGGCATACACTTACTAGAAATCTAGATGTTCAACTTTTAGATACAAAGAGACAACTGGAT

Figure 64 (Continued)

ACAGAGACAAATCTTCATCTTAACACAAAAGAACTATTAAAAAATGCTCAAAAAGAAATTGCCACATTGA
AACAGCACCTCAGTAATATGGAAGTCCAAGTTGCTTCTCAGTCTTCACAGAGAACTGGTAAAGGTCAGCC
TAGCAACAAAGAAGATGTGGATGATCTTGTGAGTCAGCTAAGACAGACAGAAGAGCAGGTGAATGACTTA
AAGGAGAGACTCAAAACAAGTACGAGCAATGTGGAACAATATCAAGCAATGGTTACTAGTTTAGAAGAAT
CCCTGAACAAGGAAAAACAGGTGACAGAAGAAGTGCGTAAGAATATTGAAGTTCGTTTAAAAGAGTCAGC
TGAATTTCAGACACAGTTGGAAAAGAAGTTGATGGAAGTAGAGAAGGAAAAACAAGAACTTCAGGATGAT
AAAAGAAGAGCCATAGAGAGCATGGAACAACAGTTATCTGAATTGAAGAAAACACTTTCTAGTGTTCAGA
ATGAAGTACAAGAAGCTCTTCAGAGAGCAAGCACAGCTTTAAGTAATGAGCAGCAAGCCAGACGTGACTG
TCAGGAACAAGCTAAAATAGCTGTGGAAGCTCAGAATAAGTATGAGAGAGAATTGATGCTGCATGCTGCT
GATGTTGAAGCTCTACAAGCTGCGAAGGAGCAGGTTTCAAAAATGGCATCAGTCCGTCAGCATTTGGAAG
AAACAACACAGAAAGCAGAATCACAGTTGTTGGAGTGTAAAGCATCTTGGGAGGAAAGAGAGAGAATGTT
AAAGGATGAAGTTTCCAAATGTGTATGTCGCTGTGAAGATCTGGAGAAACAAAACAGATTACTTCATGAT
CAGATCGAAAAATTAAGTGACAAGGTCGTTGCCTCTGTGAAGGAAGGTGTACAAGGTCCACTGAATGTAT
CTCTCAGTGAAGAAGGAAAATCTCAAGAACAAATTTTGGAAATTCTCAGATTTATACGACGAGAAAAAGA
AATTGCTGAAACTAGGTTTGAGGTGGCTCAGGTTGAGAGTCTGCGTTATCGACAAAGGGTTGAACTTTTA
GAAAGAGAGCTGCAGGAACTGCAAGATAGTCTAAATGCTGAAAGGGAGAAAGTCCAGGTAACTGCAAAAA
CAATGGCTCAGCATGAAGAACTGATGAAGAAAACTGAAACAATGAATGTAGTTATGGAGACCAATAAAAT
GCTAAGAGAAGAGAAGGAGAGACTAGAACAGGATCTACAGCAAATGCAAGCAAAGGTGAGGAAACTGGAG
TTAGATATTTTACCCTTACAAGAAGCAAATGCTGAGCTGAGTGAGAAAAGCGGTATGTTGCAGGCAGAGA
AGAAGCTCTTAGAAGAGGATGTCAAACGTTGGAAAGCACGTAACCAGCATCTAGTAAGTCAACAGAAAGA
TCCAGATACAGAAGAATATCGGAAGCTCCTTTCTGAAAAGGAAGTTCATACTAAGCGTATTCAACAATTG
ACAGAAGAAATTGGTAGACTTAAAGCTGAAATTGCAAGATCAAATGCATCTTTGACTAACAACCAGAACT
TAATTCAGAGTCTGAAGGAAGATCTAAATAAAGTAAGAACTGAAAAGGAAACCATCCAGAAGGACTTAGA
TGCCAAAATAATTGATATCCAAGAAAAAGTCAAAACTATTACTCAAGTTAAGAAAATTGGACGTAGGTAC
AAGACTCAATATGAAGAACTTAAAGCACAACAGGATAAGGTTATGGAGACATCGGCTCAGTCCTCTGGAG
ACCATCAGGAGCAGCATGTTTCAGTCCAGGAAATGCAGGAACTCAAAGAAACGCTCAACCAAGCTGAAAC
AAAATCAAAATCACTTGAAAGTCAAGTAGAGAATCTGCAGAAGACATTATCTGAAAAAGAGACAGAAGCA
AGAAATCTCCAGGAACAGACTGTGCAACTTCAGTCTGAACTTTCACGACTTCGTCAGGATCTTCAAGATA
GAACCACACAGGAGGAGCAGCTCCGACAACAGATAACTGAAAAGGAAGAAAAAACCAGAAAGGCTATTGT
AGCAGCAAAGTCAAAAATTGCACACTTAGCTGGTGTAAAAGATCAGCTAACTAAAGAAAATGAGGAGCTT
AAACAAAGGAATGGAGCCTTAGATCAGCAGAAAGATGAATTGGATGTTCGCATTACTGCGCTAAAGTCCC
AATATGAAGGTCGAATTAGTCGCTTGGAAAGAGAACTCAGGGAGCATCAAGAGAGACACCTTGAGCAGAG
AGATGAGCCTCAAGAACCTTCTAATAAGGTCCCTGAACAGCAGAGACAGATCACATTGAAAACAACTCCA
GCTTCTGGTGAAAGAGGAATTGCCAGCACATCAGACCCACCAACAGCCAATATCAAGCCAACTCCTGTTG
TGTCTACTCCAAGTAAAGTGACAGCTGCAGCTATGGCTGGAAATAAGTCAACACCCAGGGCTAGTATCCG
CCCAATGGTTACACCTGCAACTGTTACAAATCCCACTACTACCCCAACAGCTACAGTGATGCCCACTACA
CAAGTGGAATCACAGGAAGCTATGCAGTCAGAAGGGCCTGTGGAACATGTTCCAGTTTTTGGAAGCACAA
GTGGATCCGTTCGTTCTACTAGTCCTAATGTCCAGCCTTCTATCTCTCAACCTATTTTAACTGTTCAGCA
ACAAACACAGGCTACAGCTTTTGTGCAACCCACTCAACAGAGTCATCCTCAGATTGAGCCTGCCAATCAA
GAGTTATCTTCAAACATAGTAGAGGTTGTTCAGAGTTCACCAGTTGAGCGGCCTTCTACTTCCACAGCAG
TATTTGGCACAGTTTCGGCTACCCCCAGTTCTTCTTTGCCAAAGCGTACACGTGAAGAGGAAGAGGATAG
CACCATAGAAGCATCAGACCAAGTCTCTGATGATACAGTGGAAATGCCTCTTCCAAAGAAGTTGAAAAGT
GTCACACCTGTAGGAACTGAGGAAGAAGTTATGGCAGAAGAAAGTACTGATGGAGAGGTAGAGACTCAGG
TATACAACCAGGATTCTCAAGATTCCATTGGAGAAGGAGTTACCCAGGGAGATTATACACCTATGGAAGA
CAGTGAAGAAACCTCTCAGTCTCTACAAATAGATCTTGGGCCACTTCAATCAGATCAGCAGACGACAACT
TCATCCCAGGATGGTCAAGGCAAAGGAGATGATGTCATTGTAATTGACAGTGATGATGAAGAAGAGGATG
ATGATGAAAATGATGGAGAACATGAGGATTATGAAGAGGATGAGGAAGATGATGATGATGATGAAGATGA
CACAGGGATGGGAGATGAGGGTGAAGATAGTAATGAAGGAACTGGTAGTGCCGATGGCAATGATGGTTAT
GAAGCTGATGATGCTGAGGGTGGTGATGGGACTGATCCAGGTACAGAAACAGAAGAAAGTATGGGTGGAG
GTGAAGGTAATCACAGAGCTGCTGATTCTCAAAACAGTGGTGAAGGAAATACAGGTGCTGCAGAATCTTC
TTTTTCTCAGGAGGTTTCTAGAGAACAACAGCCATCATCAGCATCTGAAAGACAGGCCCCTCGAGCACCT
CAGTCACCGAGACGCCCACCACATCCACTTCCCCCAAGACTGACCATTCATGCCCCACCTCAGGAGTTGG
GACCACCAGTTCAGAGAATTCAGATGACCCGAAGGCAGTCTGTAGGACGTGGCCTTCAGTTGACTCCAGG
AATAGGTGGCATGCAACAGCATTTTTTTGATGATGAAGACAGAACAGTTCCAAGTACTCCAACTCTTGTG
GTGCCACATCGTACTGATGGATTTGCTGAAGCAATTCATTCGCCGCAGGTTGCTGGTGTCCCTAGATTCC
GGTTTGGGCCACCTGAAGATATGCCACAAACAAGTTCTAGTCACTCTGATCTTGGCCAGCTTGCTTCTCA
AGGAGGTTTAGGAATGTATGAAACACCCCTGTTCCTAGCTCATGAAGAAGAGTCAGGTGGCCGAAGTGTT
CCCACTACTCCACTACAAGTAGCAGCCCCAGTGACTGTATTTACTGAGAGCACCACCTCTGATGCTTCGG
AACATGCCTCTCAATCTGTTCCAATGGTGACTACATCCACTGGCACTTTATCTACAACAAATGAAACAGC
AACAGGTGATGATGGAGATGAAGTATTTGTGGAGGCAGAATCTGAAGGTATTAGTTCAGAAGCAGGCCTA
GAAATTGATAGCCAGCAGGAAGAAGAGCCGGTTCAAGCATCTGATGAGTCAGATCTCCCCTCCACCAGCC

Figure 64 (Continued)

AGGATCCTCCTTCTAGCTCATCTGTAGATACTAGTAGTAGTCAACCAAAGCCTTTCAGACGAGTAAGACT
TCAGACAACATTGAGACAAGGTGTCCGTGGTCGTCAGTTTAACAGACAGAGAGGTGTGAGCCATGCAATG
GGAGGGAGAGGAGGAATAAACAGAGGAAATATTAATTAAATGGTCTGTAAACAATAACAACTGTGAATAA
GATTATCAAATCTGTTTTAGTGTAATGATTGTCAAGTTTAAAAACATTTTTATATATAAACTGGTATACT
CATGTCAATATTCTTTATTAATAAAATGTTTTTCAGTGTCAAAATTTATTATTCATTTCTTCATTAGTTG
ACTCCTCCTTTGCTCATCAGTCTAAGGACAGTTGTACCAGACTTTGGATAAGGTCTGCCCAGAACGAGTA
GTAATTGCTCTTGCTGTTCTACTAGGCACATCAATGTTATAGTATTGATCTAAATGGAAGAGAAAACATT
TTTTTAGTTAAAAAGAAAACAATGCCCAAACTAAAAAATAACTTATGTTGACTATTATGCTCAAAGACAA
TGTTTATCATTTTAATAGAGATGTTTTTACTAATTAATTTGAACTTTATAACAAAAAGAAAAACAATTGC
CTAGACTTTTCAGCTTTTTTGATGTTTCAAAAGATTGACATTTCACCATCTTTTTGTAAAATCAGGTTCA
GCTCTCCTTTATGAAGTAAACATTAAAGAGTAACCAAGTTTGAAAAATAATTTACTTGGGGTTATTCCTT
TTAAAAAATACATGCCAATGTCATTCATATTATGAAATTACAGGCAGAATAACTTAGATTTCTGGGCATT
TCAAAGAAAAGCATCCTGAGTAATATAATTTAATTAATAAAATTAGTTTCTCAGGAACTTCTTTCTGATC
TTACAGACTCTGCAGTGATGCAAATCATTATAACCTTGTGCCAAACAAGGTATCTGTTAAATGCCACAAA
TGATAGAAGTAAAATACTATTGTCAGTAGCAAGTTTACTCTAGTAACTGGATGTTTTATCGTAATCTCAT
GAAGGTTAGAGCAGAATTGAATTGCAGTGCCATCATTTTAATTGAAATTAAAGCAAAAGTCTTAACTCTT
TTCCACAGCAATTAGAATAAGTACCGTAGTGTAACTTCTCACATTCAGTCATCATTGCAGCCAGCATTTT
TACTTTATCTTCATGTTTTCACAAATGATATCACCTCCTTGGGAAACTGTTAGTTAATACCTTACCTTTA
GAAAAGGCATAGTAATCATAGCCGTCAGGTTTTCTGATGTTGGGCAGTGATATAGCTGAGGTAACCACAT
TTGGAAGTCCTCTCCACAGTATACTCACTTTAACTTCATTATGAAGGACACCTGTAAGTGGCATGTTTAA
TAAAAGATACCAGATTAAAAGGCAATGTACTATCTTGGAAAGAGCCAGACATCTGAGTTTTAATCTCAGT
TTTAGCCCTCTGATGTAGAACTATTGAGGGTTATAGACTGGTATATAATGTTCTTGGTAAGAAGTACTTG
ATAAATAGTATTGGTTATAACTAACAAACCTGAACAAACTGCTTTACTTACCCACAAGGAAAAAGAAAGT
ATTGGTCTTTGGTTATTCACTAAGGCAAGTGGATGAGTTTTTCATCAGTAAGCTTAAATTATTAGGGCTG
TTTGATCAGTATCCATATTTCATAAGCCTTACTGTATAAGAAACTGTATTACATCTACTTATGTTTAAGG
ATTTTTTTAACACAATAAAAATGTTACCTTTGTCTTGATAAGCCAGTCTGGCAGGTGAATATTGAATTCT
GATGGTGTGTGTTTGAGAAGGTCCTATAGCACGTTCAAAGCGACGTCTCCTAACCTGTGTCGTTTCTCCA
TACACTGGATAATTTAGAGCAGGCCTTCTTCCAGGGCACTTCTGTACAGGTTCCTGTTTATAAATATACT
GCTGAATGCTGCCACCTGTTATGTATTAGAATATCACATGGAAAATGAAAATTAATTTTAATACCCTCAG
AAAAGGTGGAAAACAACTTTTACAATGTATAGGAAACAGTTTTGTTCTCATTTTTCATATAATATATTGA
TATTAATAATGGCTATTAGTCAAGGGTATTATAAAAATAATTATTAAACTGAAATACTTGTTGAATGAAT
AGATGCAGCAAATTACATAGTTATATATTTAATTTCAATTGAAAGTGACAAGTGCTCAGTTTGGCAGCAC
ATATACTAAAACTGGAATGATACAGAGATTAGCATGGCCCTTGTGCAAGGATGACATGCACATTTGTGAA
GCGAAAGTAAATGACATTCTATCAGTGACCTGAAAACTCAAATGAATTGTGACTTGCCTGTGAAGAAATG
AAAATAAAAATTGAGGGCAATAAGAATACTACCCTCAATATTGATTTTTTTCACTGAAAATATTTGATTT
CAGCCATTAAAGATATCTTTTGACAGTAAAGTCAATAATAAATGAAAAAAAAAAAAAAAAAAAAAAA

NM_003000.2| Homo sapiens succinate dehydrogenase complex, subunit B, iron sulfur (Ip) (SDHB), nuclear gene encoding mitochondrial protein, mRNA
ACCCGGGAAACCGGAAGCCGCCTCCCACTTGGTTGCTCGTACGCGGCTAGTGGGTCCTCAGTGGATGTAG
GCTGGGCGCCGCGATGTTCGACGGGACACCGGCGGAGAGCGACCTCGGGGTTAAGGGGTGGGGCTGACGT
CAGGAGCCAAGATGGCGGCGGTGGTCGCCCTCTCCTTGAGGCGCCGGTTGCCGGCCACAACCCTTGGCGG
AGCCTGCCTGCAGGCCTCCCGAGGAGCCCAGACAGCTGCAGCCACAGCTCCCCGTATCAAGAAATTTGCC
ATCTATCGATGGGACCCAGACAAGGCTGGAGACAAACCTCATATGCAGACTTATGAAGTTGACCTTAATA
AATGTGGCCCCATGGTATTGGATGCTTTAATCAAGATTAAGAATGAAGTTGACTCTACTTTGACCTTCCG
AAGATCATGCAGAGAAGGCATCTGTGGCTCTTGTGCAATGAACATCAATGGAGGCAACACTCTAGCTTGC
ACCCGAAGGATTGACACCAACCTCAATAAGGTCTCAAAAATCTACCCTCTTCCACACATGTATGTGATAA
AGGATCTTGTTCCCGATTTGAGCAACTTCTATGCACAGTACAAATCCATTGAGCCTTATTTGAAGAAGAA
GGATGAATCTCAGGAAGGCAAGCAGCAGTATCTGCAGTCCATAGAAGAGCGTGAGAAACTGGACGGGCTC
TACGAGTGCATTCTCTGTGCCTGCTGTAGCACCAGCTGCCCCAGCTACTGGTGGAACGGAGACAAATATC
TGGGGCCTGCAGTTCTTATGCAGGCCTATCGCTGGATGATTGACTCCAGAGATGACTTCACAGAGGAGCG
CCTGGCCAAGCTGCAGGACCCATTCTCTCTATACCGCTGCCACACCATCATGAACTGCACAAGGACCTGT
CCTAAGGGTCTGAATCCAGGGAAAGCTATTGCAGAGATCAAGAAAATGATGGCAACCTATAAGGAGAAGA
AAGCTTCAGTTTAACTGTTTCCATGCTAAACATGATTTATAACCAGCTCAGAGCTGAACATAATTTATAT
CTAATTTGAGTTCCTTTAAAGATCTTGGTTTTCCATGAATACAGCATGTATAATAAAAATTTTAAGAAAT
AAATGTTATTCTACTTTATTAACAAAAAAAAAAAAAAAAA

NM_021954.3| Homo sapiens gap junction protein, alpha 3, 46kDa (GJA3), mRNA
GGGCTGCAGGGATGGCGATCAGGGCGGGGAGGTCGGGCCCTGGCGCGGGGCGCACGCGTGAAGCAGCACC

DEMANDE OU BREVET VOLUMINEUX

LA PRÉSENTE PARTIE DE CETTE DEMANDE OU CE BREVET COMPREND PLUS D'UN TOME.

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