US 20150017163A1

(19) **United States**

(12) **Patent Application Publication**

Patel et al.

(10) **Pub. No.: US 2015/0017163 A1**

(43) **Pub. Date: Jan. 15, 2015**

(54) **METHODS FOR TREATING OR PREVENTING OPHTHALMOLOGICAL CONDITIONS**

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(21) Appl. No.: **14/329,702**

(22) Filed: **Jul. 11, 2014**

Related U.S. Application Data

(60) Provisional application No. 61/845,938, filed on Jul. 12, 2013, provisional application No. 61/845,936, filed on Jul. 12, 2013, provisional application No. 61/845,935, filed on Jul. 12, 2013, provisional application No. 61/866,502, filed on Aug. 15, 2013, provisional application No. 61/866,503, filed on Aug. 15, 2013, provisional application No. 61/866,507, filed on Aug. 15, 2013, provisional application No. 61/911,854, filed on Dec. 4, 2013, provisional application No. 61/911,860, filed on Dec. 4, 2013, provisional application No. 61/911,894, filed on Dec. 4, 2013, provisional application No. 61/926,848, filed on Jan. 13, 2014, provisional application No. 61/926,812, filed on Jan. 13, 2014, provisional application No. 61/926,825, filed on Jan. 13, 2014, provisional application No. 61/931,135, filed on Jan. 24, 2014, provisional application No. 61/931,116, filed on Jan. 24, 2014, provisional application No. 61/931,125, filed on Jan. 24, 2014.

Publication Classification

(51) **Int. Cl.**

A61K 31/713	(2006.01)
A61K 9/00	(2006.01)
A61K 45/06	(2006.01)
A61K 39/395	(2006.01)
C12N 15/115	(2006.01)

(52) **U.S. Cl.**

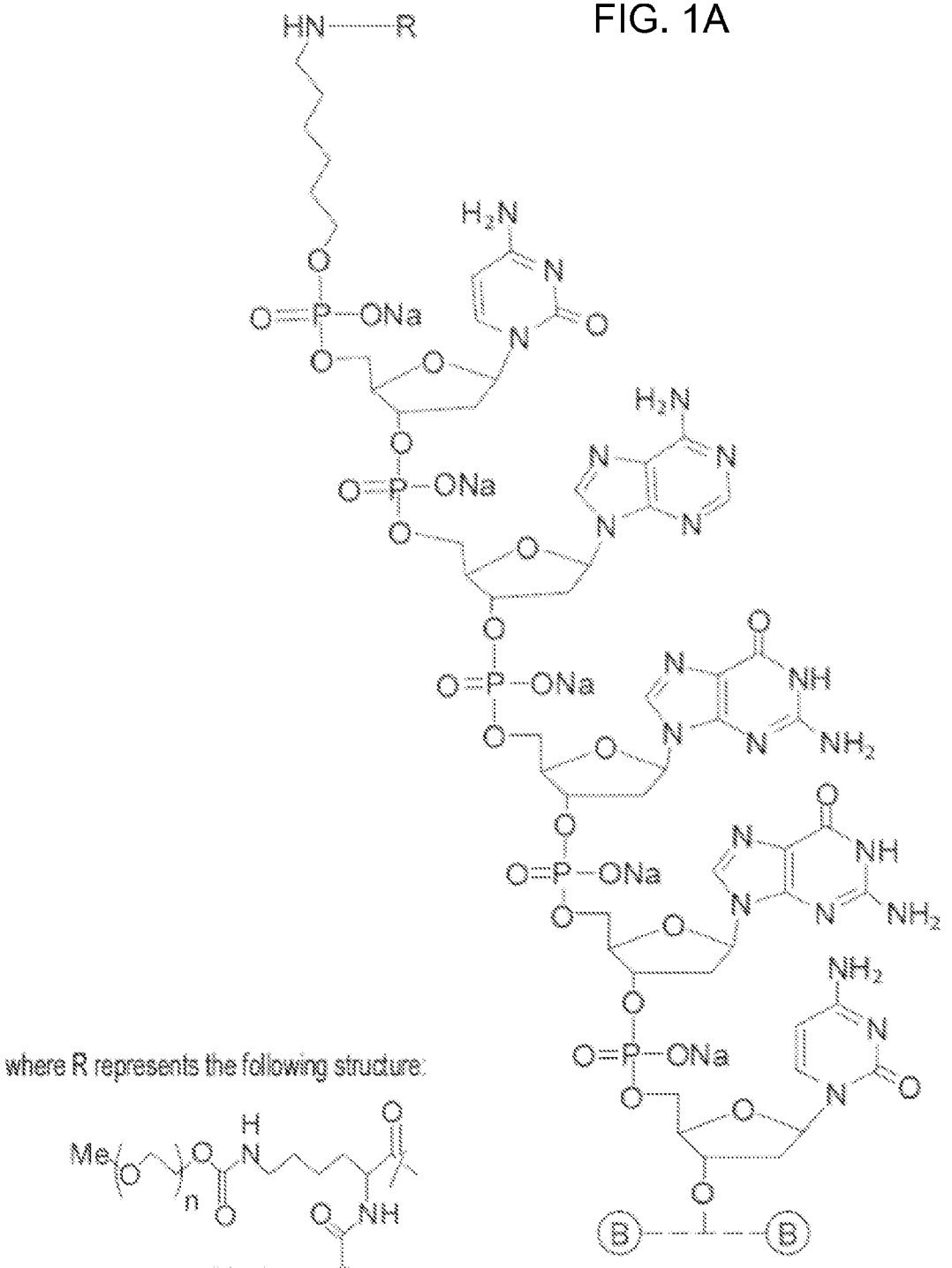
CPC ***A61K 31/713*** (2013.01); ***A61K 39/3955*** (2013.01); ***C12N 15/115*** (2013.01); ***A61K 45/06*** (2013.01); ***A61K 9/0048*** (2013.01); *C12N 2310/16* (2013.01); *C12N 2310/351* (2013.01); *C12N 2310/321* (2013.01); *C12N 2310/3521* (2013.01); *C12N 2310/3533* (2013.01); *C12N 2310/317* (2013.01); *C12N 2310/314* (2013.01)

USPC ..... **424/134.1**; 424/133.1; 514/44 A

(57) **ABSTRACT**

The present invention relates to methods for treating and preventing ophthalmological disease and disorders, comprising administering Antagonist A or another pharmaceutically acceptable salt thereof, optionally in combination with another treatment, to a subject in need thereof. The present invention also relates to methods for treating and preventing ophthalmological disease and disorders, comprising administering an anti-C5 agent (e.g., ARC1905), optionally in combination with another treatment, to a subject in need thereof.

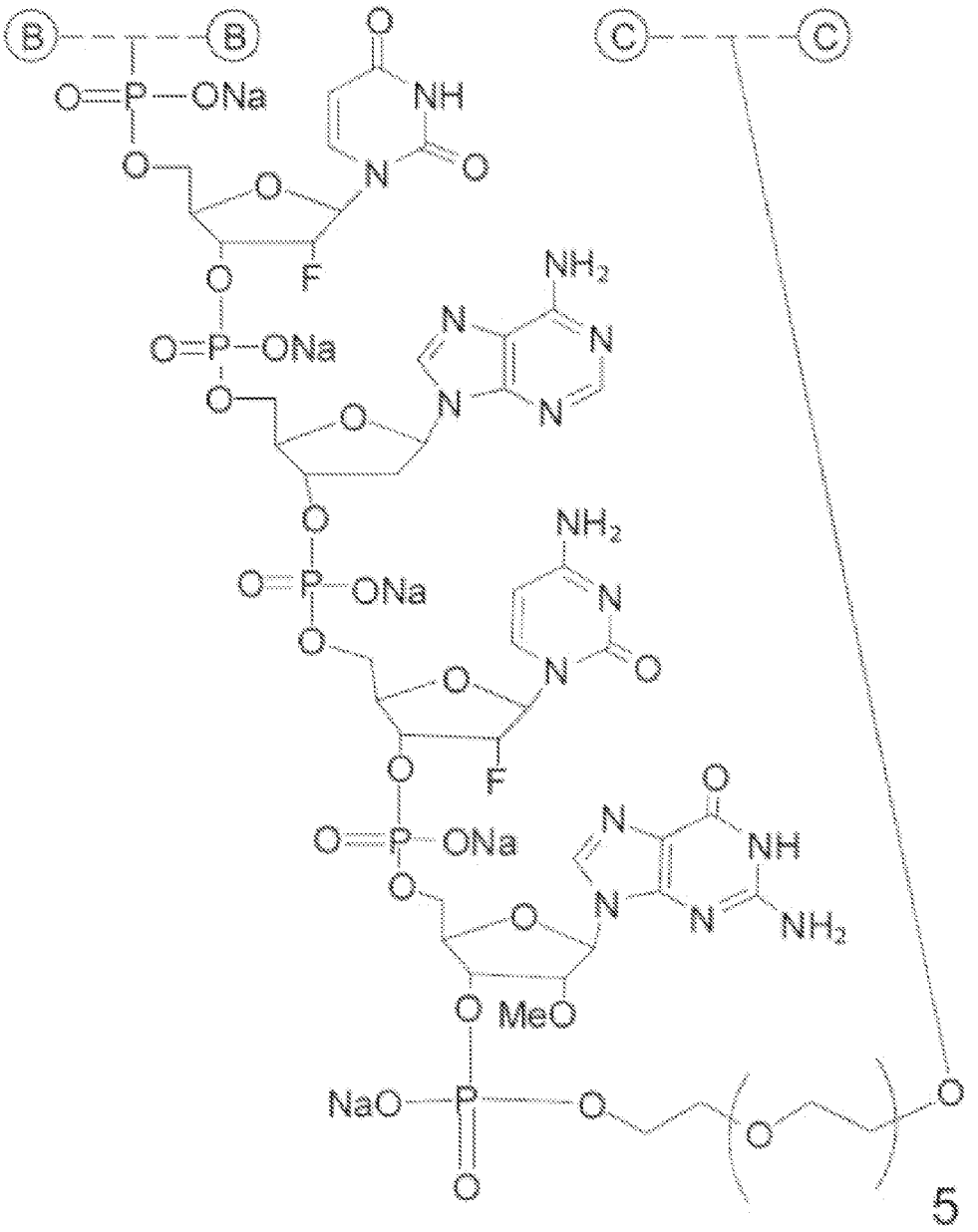
FIG. 1A



where R represents the following structure:

and n is about 450.

FIG. 1B



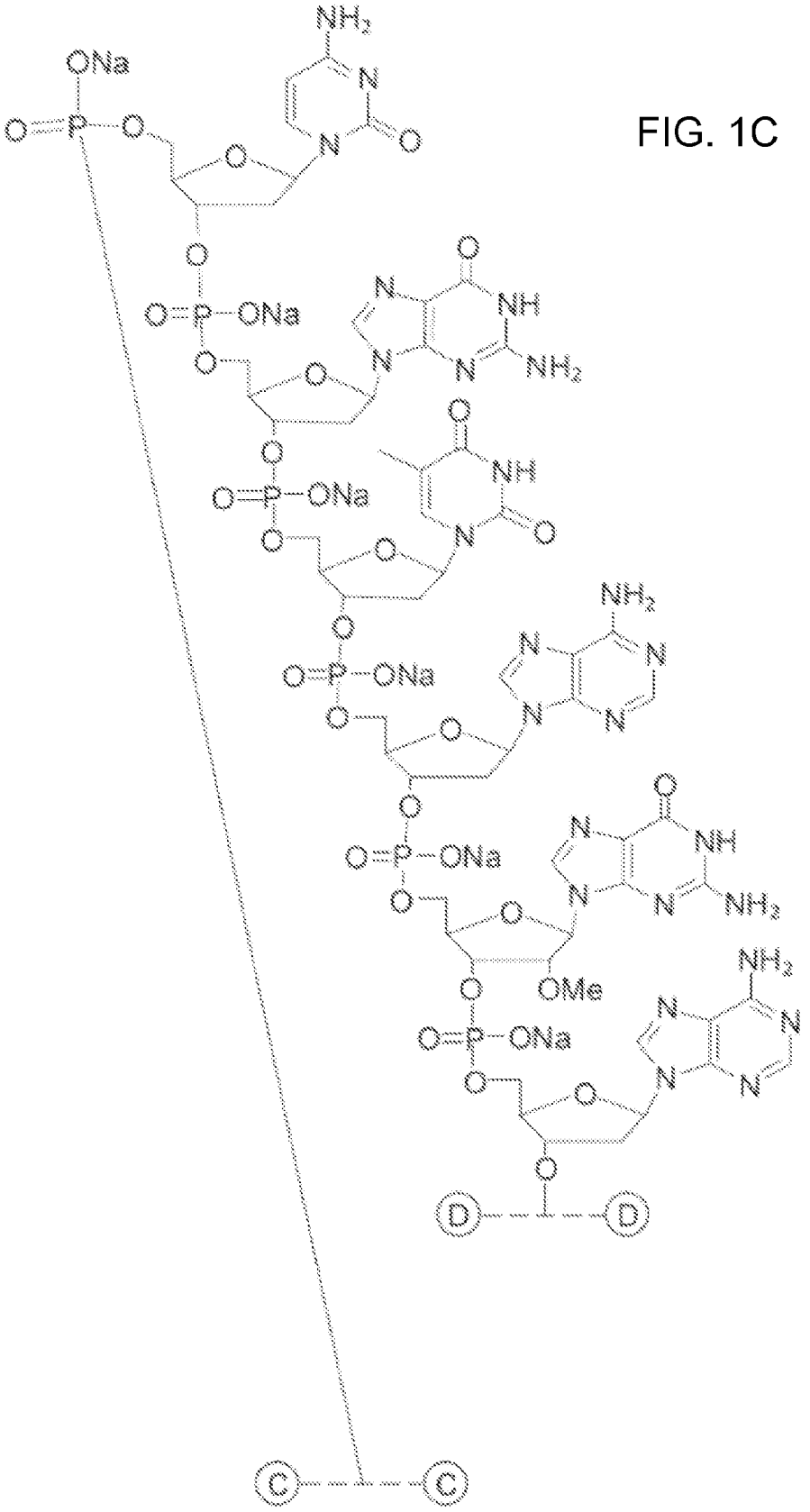


FIG. 1C

FIG. 1D

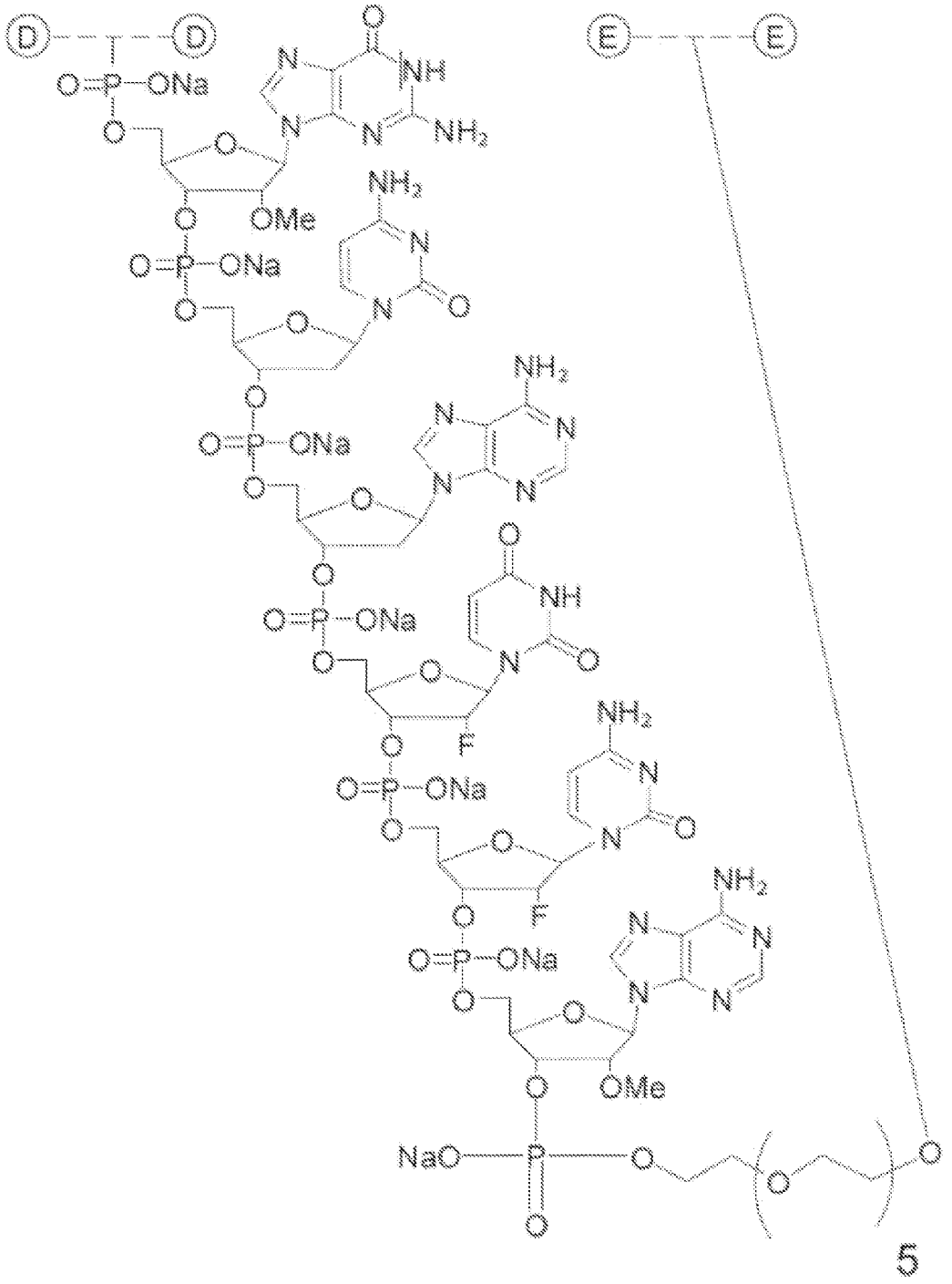


FIG. 1E

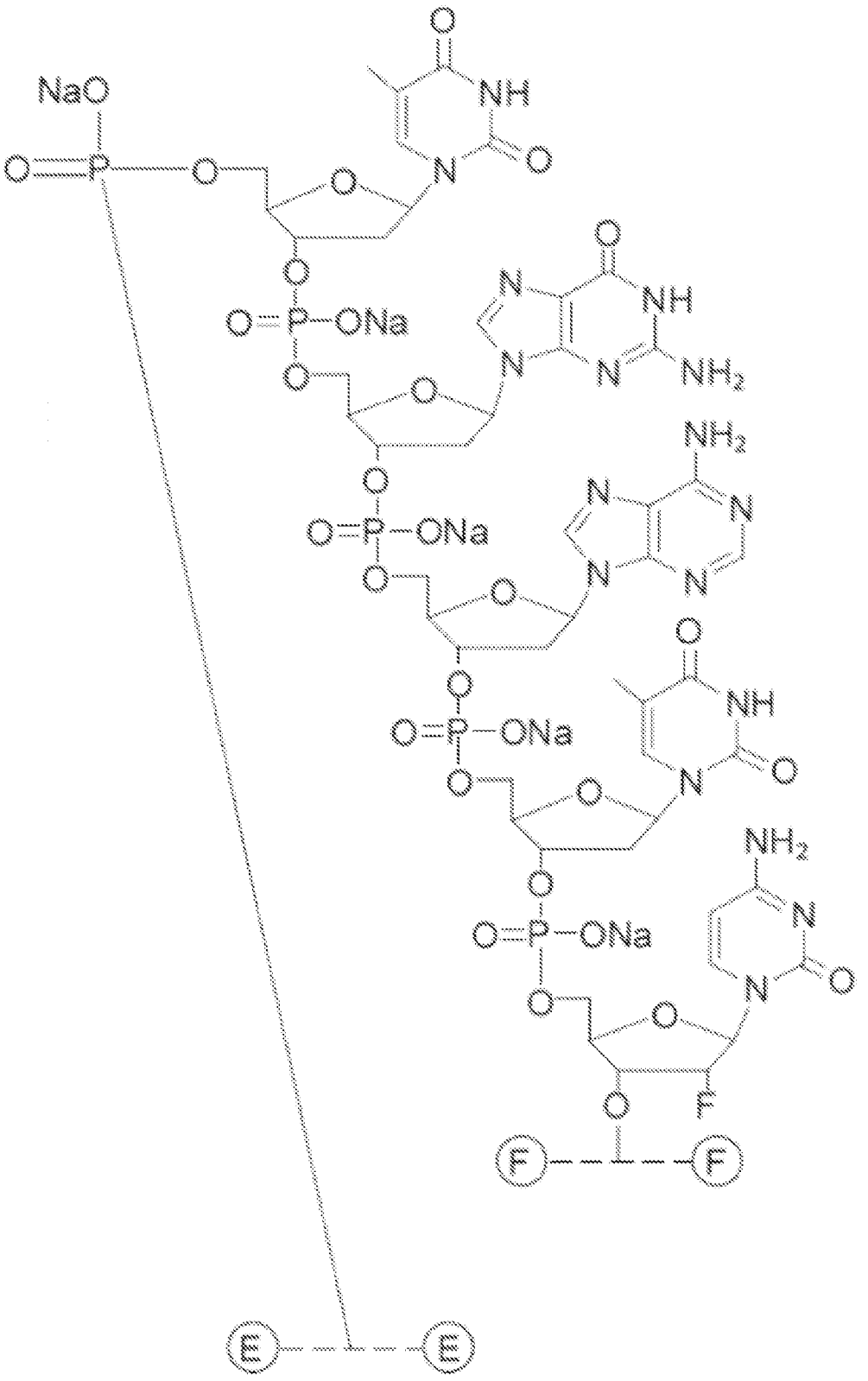

NaO
O
P
O
O
O
NH
N
O
O
P
ONa
O
N
NH
N
N
NH2
O
P
ONa
O
O
N
N
N
N
NH2
O
P
ONa
O
O
O
NH
N
O
O
P
ONa
O
O
N
NH2
O
F
F
F
E
E

FIG. 1F

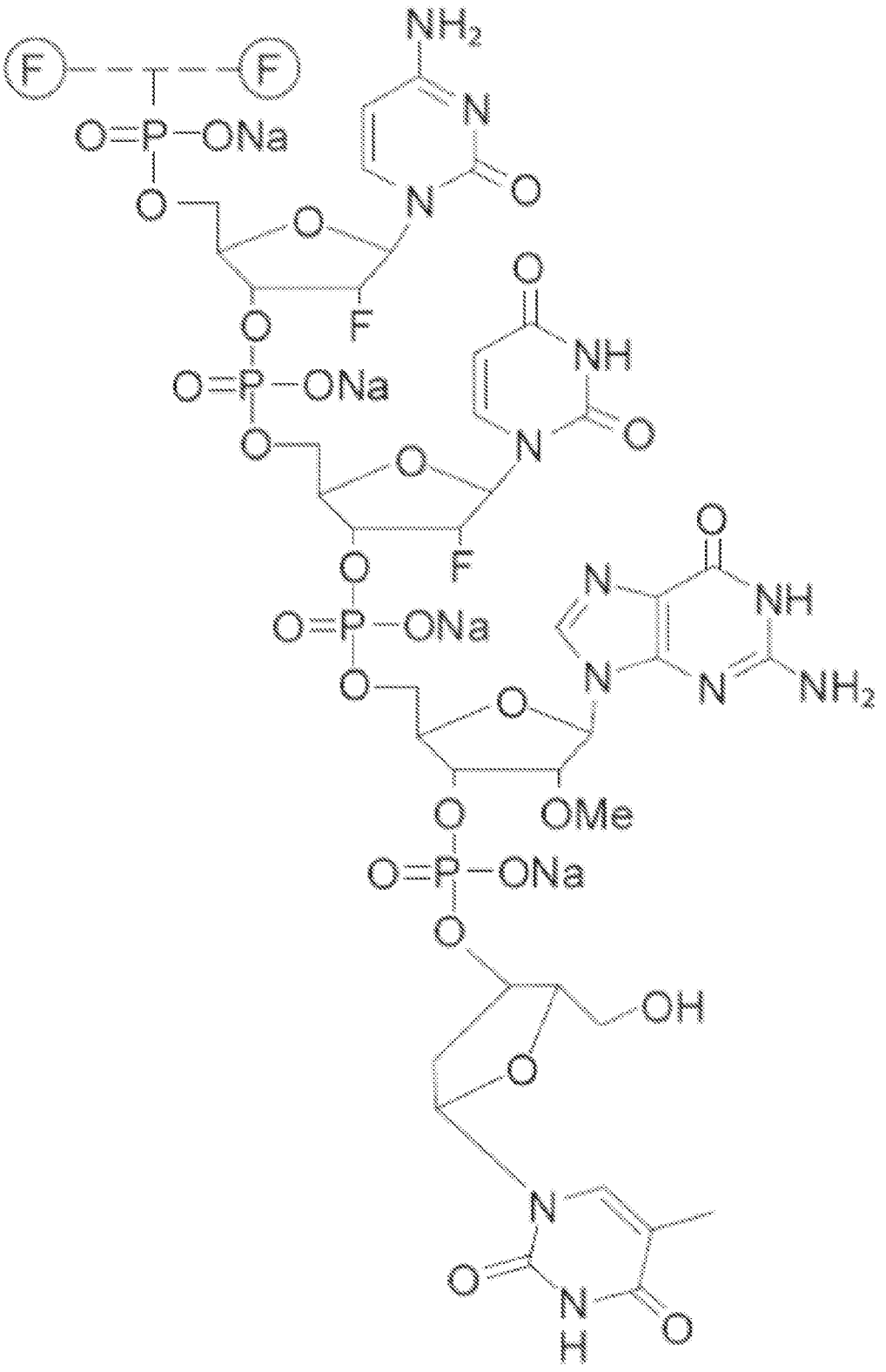

NH2
F
F
O=P–ONa
O
N
O
O
F
O=P–ONa
O
NH
O
N
O
O
F
O=P–ONa
N
NH
O
N
N
NH2
O
OMe
O=P–ONa
O
OH
O
N
O
N
H
O

FIG. 2

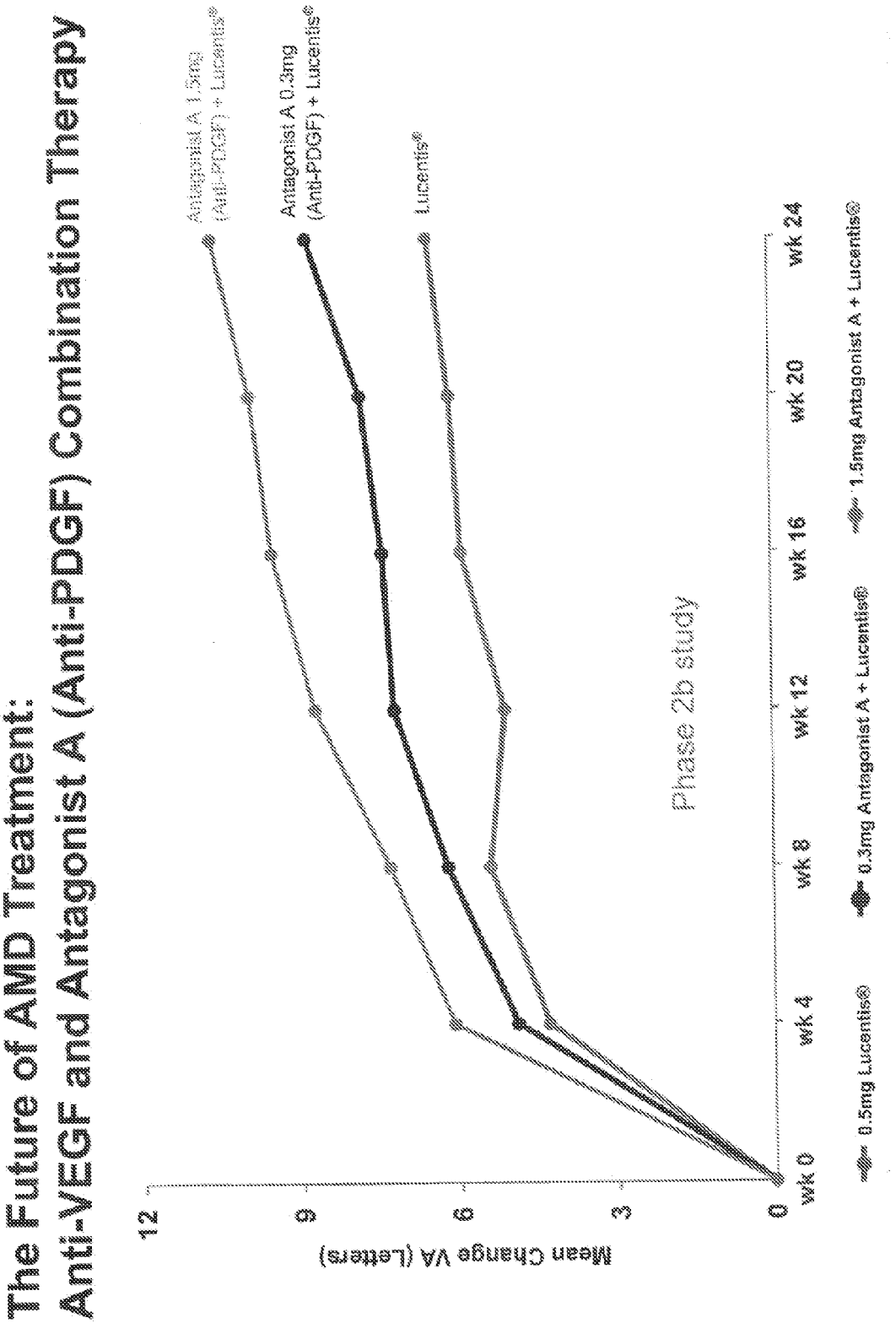

The Future of AMD Treatment:
Anti-VEGF and Antagonist A (Anti-PDGF) Combination Therapy
Mean Change VA (Letters)
12
9
6
3
0
wk 0
wk 4
wk 8
wk 12
wk 16
wk 20
wk 24
Phase 2b study
Antagonist A 1.5mg (Anti-PDGF) + Lucentis®
Antagonist A 0.3mg (Anti-PDGF) + Lucentis®
Lucentis®
0.5mg Lucentis®
0.3mg Antagonist A + Lucentis®
1.5mg Antagonist A + Lucentis®

FIG. 3

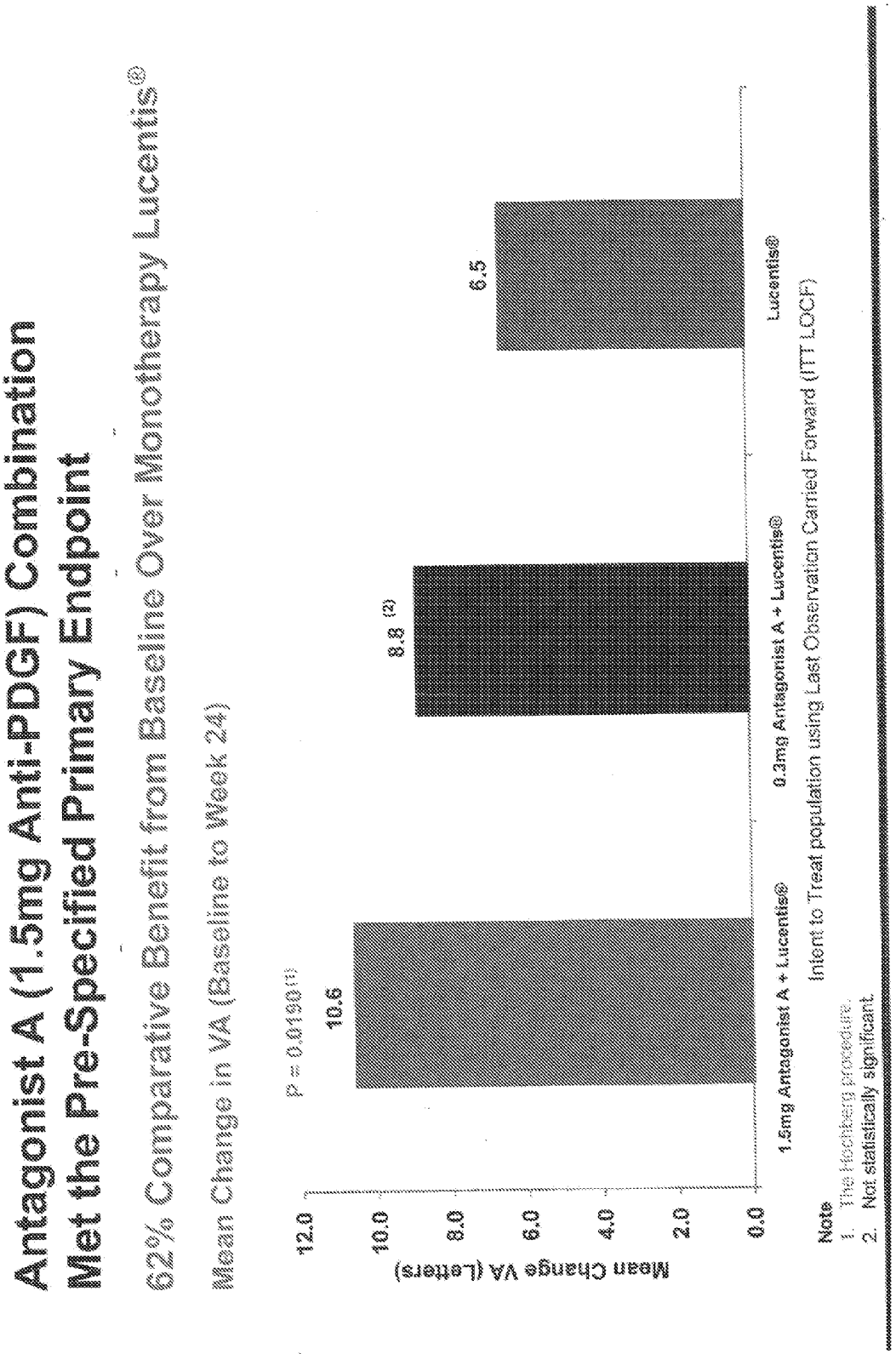

Antagonist A (1.5mg Anti-PDGF) Combination
Met the Pre-Specified Primary Endpoint
62% Comparative Benefit from Baseline Over Monotherapy Lucentis®
Mean Change in VA (Baseline to Week 24)
P = 0.0190(1)
10.6
8.8 (2)
6.5
Mean Change VA (Letters)
12.0
10.0
8.0
6.0
4.0
2.0
0.0
1.5mg Antagonist A + Lucentis®
0.3mg Antagonist A + Lucentis®
Lucentis®
Intent to Treat population using Last Observation Carried Forward (ITT LOCF)
Note
1. The Hochberg procedure.
2. Not statistically significant.

FIG. 4

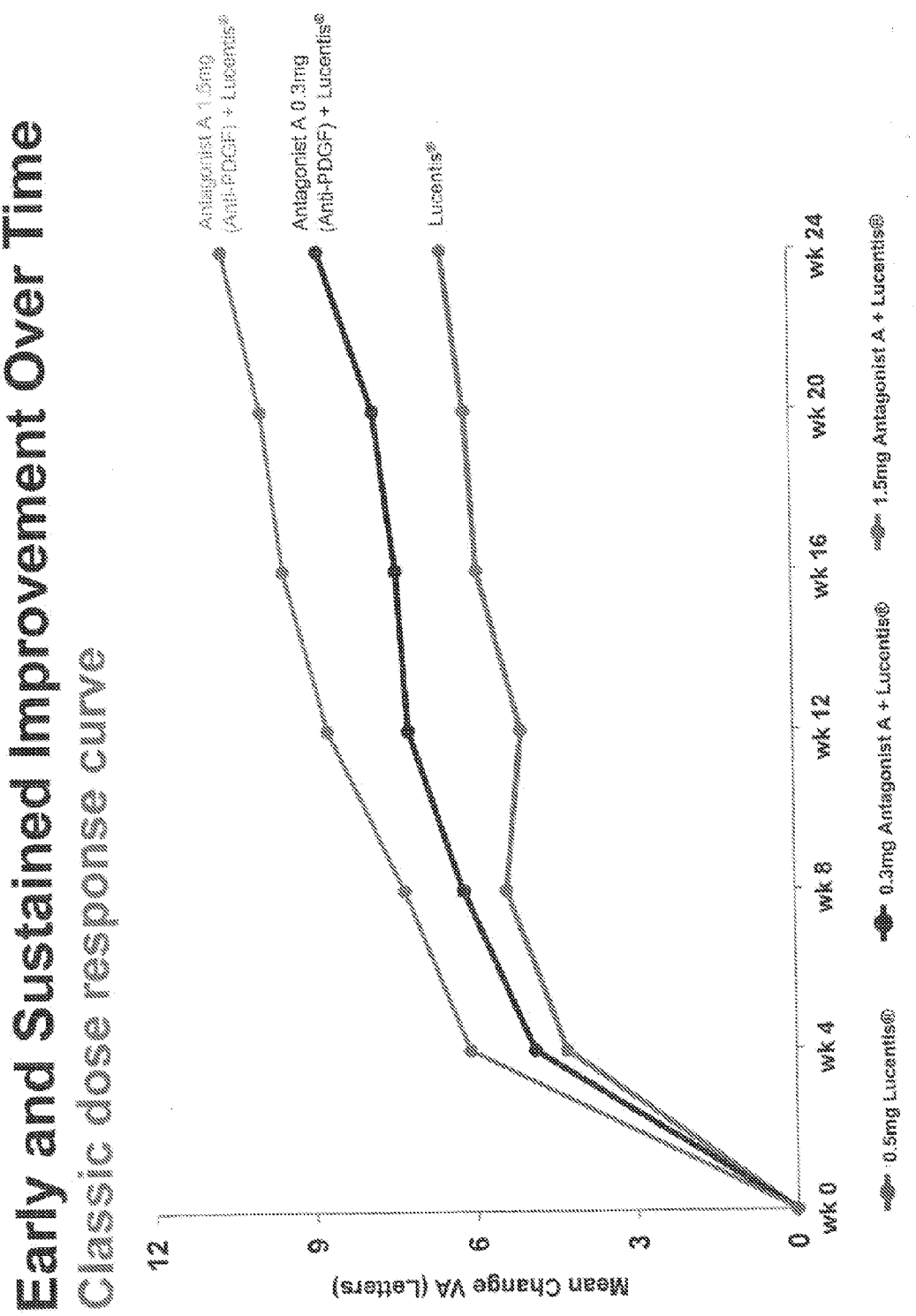

Early and Sustained Improvement Over Time
Classic dose response curve
Mean Change VA (Letters)
12
9
6
3
0
wk 0
wk 4
wk 8
wk 12
wk 16
wk 20
wk 24
Antagonist A 1.5mg (Anti-PDGF) + Lucentis®
Antagonist A 0.3mg (Anti-PDGF) + Lucentis®
Lucentis®
0.5mg Lucentis®
0.3mg Antagonist A + Lucentis®
1.5mg Antagonist A + Lucentis®

FIG. 5A

Increased Efficiancy Independent of Baseline Lesion Size

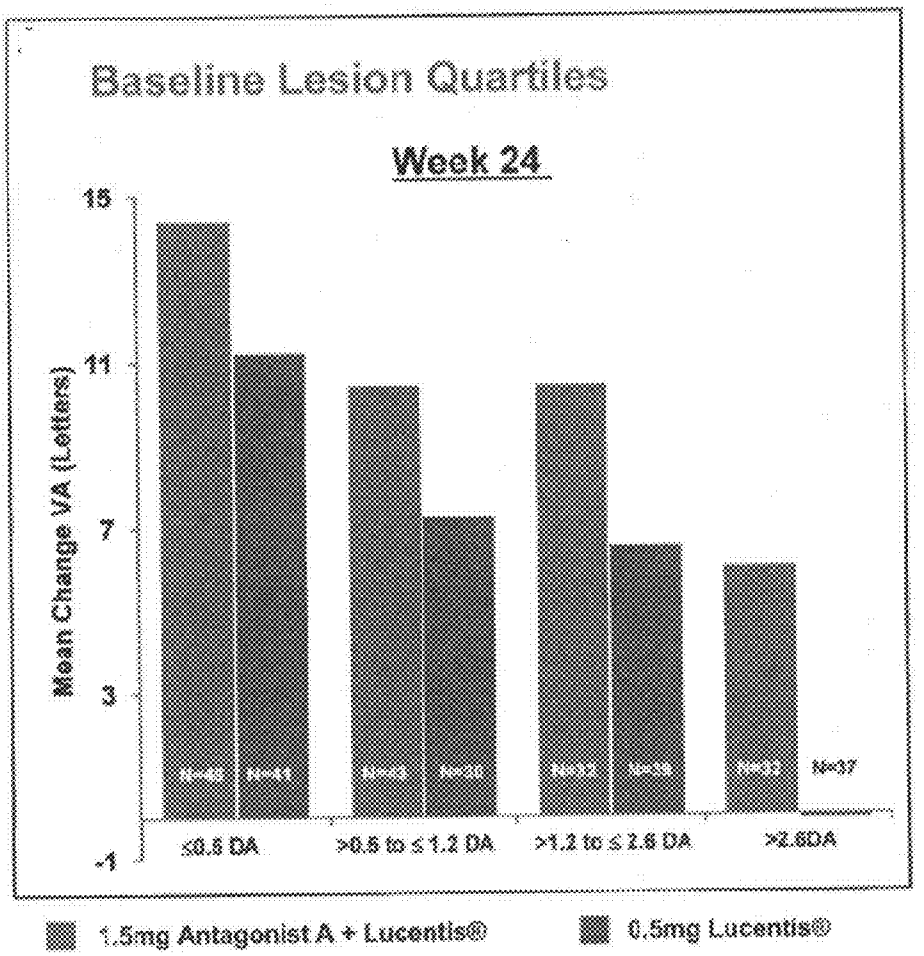


FIG. 5B

Increased Efficiancy Independent of Baseline Vision

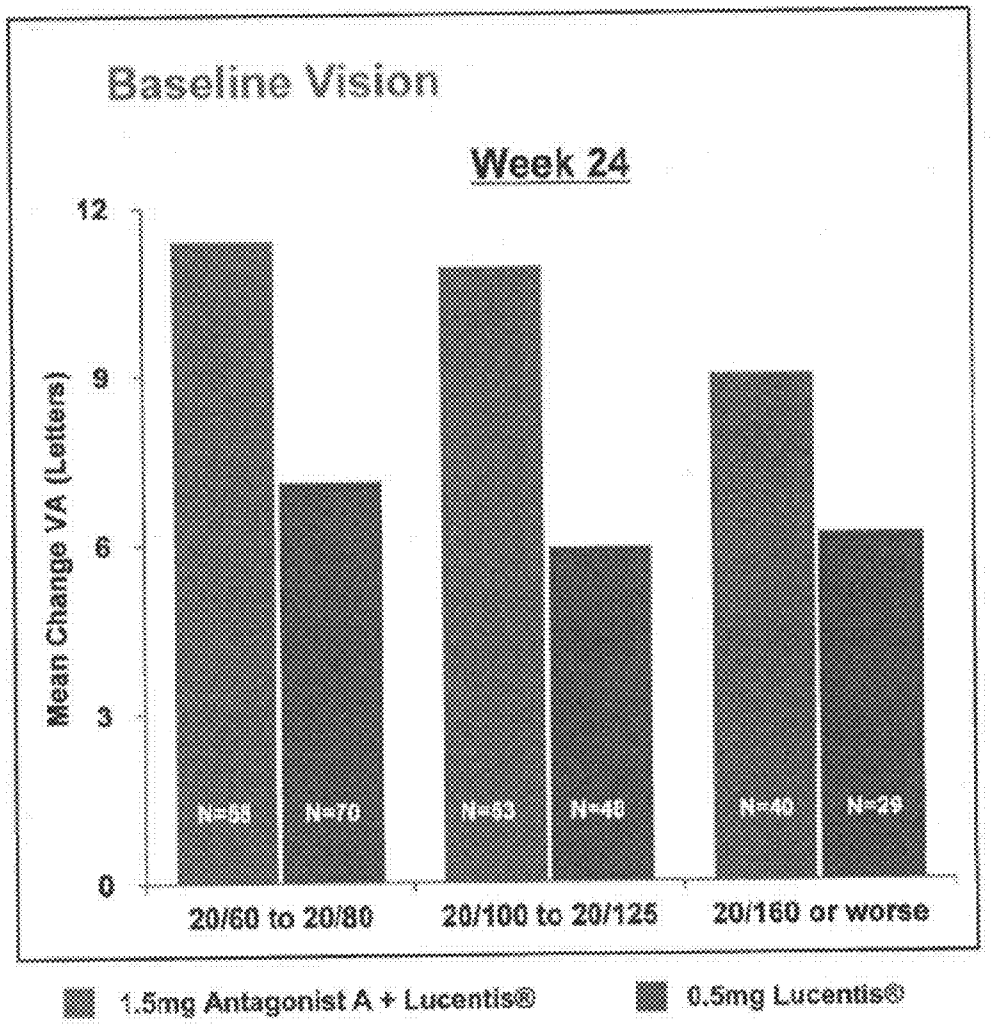
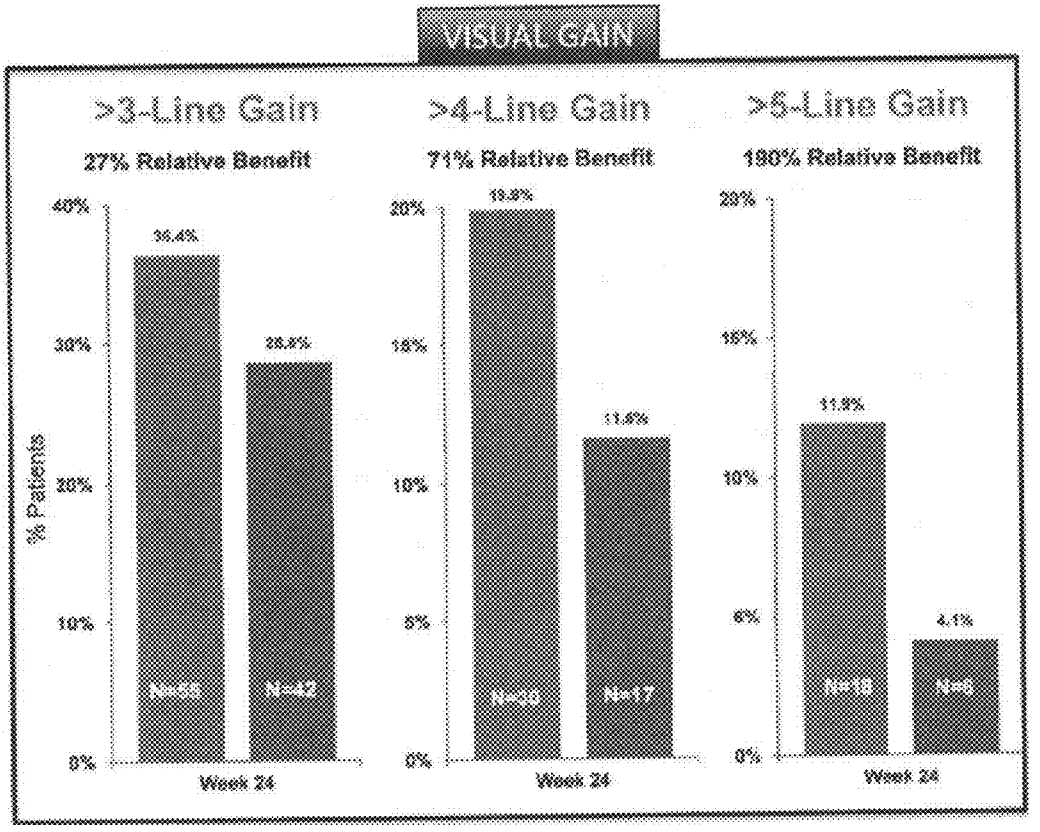


FIG. 6A

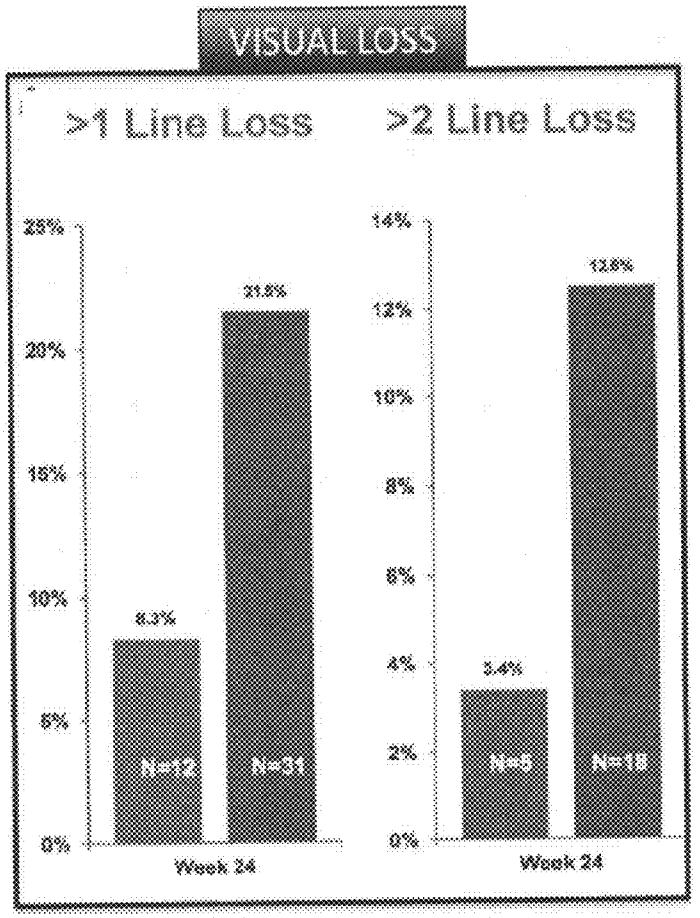
Antagonist A Combination Group had Greater Proportion of Patients With Significant Visual Gain



Intent to Treat population using Last Observation Carried Forward (ITT LOCF)

FIG. 6B

Antagonist A Combination Group had Fewer Patients With Visual Loss



Intent to Treat population using Last Observation Carried Forward (ITT LOCF)

FIG. 7A

Improved Final Visual Acuity Outcome in Antagonist A 1.5 mg Combination Arm: 20/40 or Better

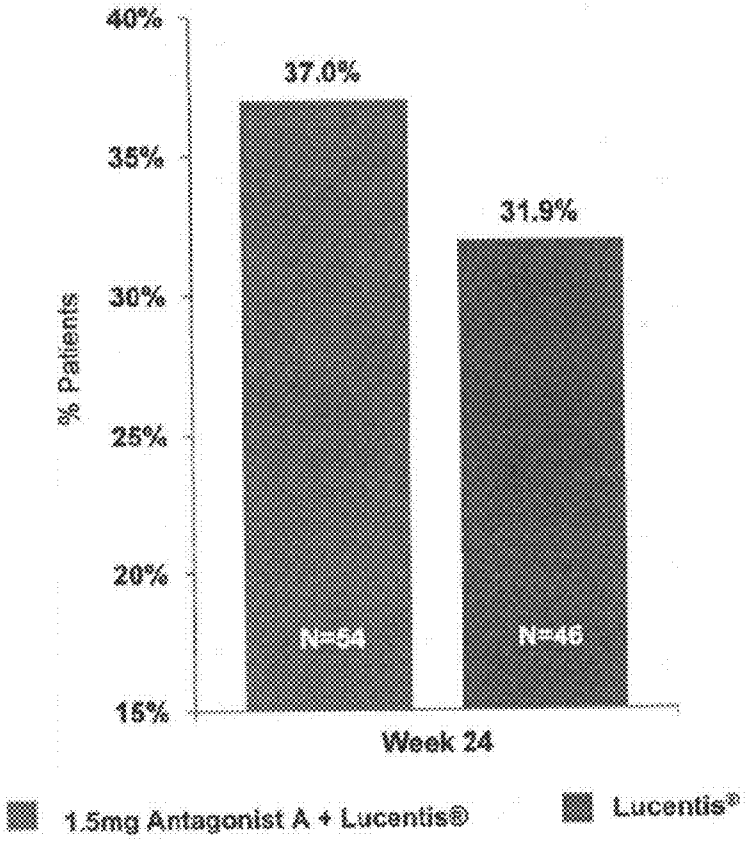


FIG. 7B

Improved Final Visual Acuity Outcome in Antagonist A 1.5 mg Combination Arm: 20/25 or Better

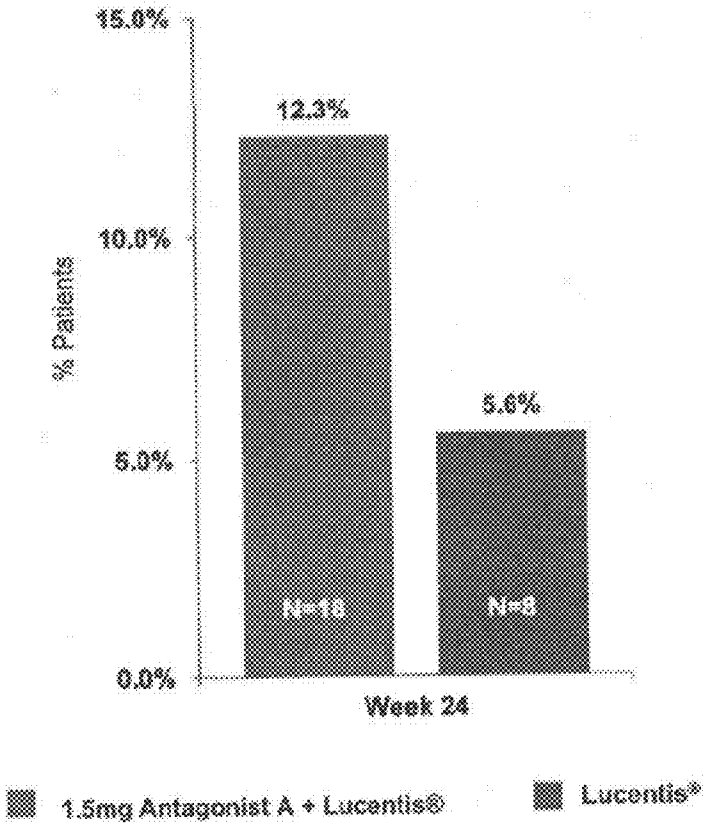


FIG. 7C

Improved Final Visual Acuity Outcome in Antagonist A 1.5 mg Combination Arm: 20/200 or Worse

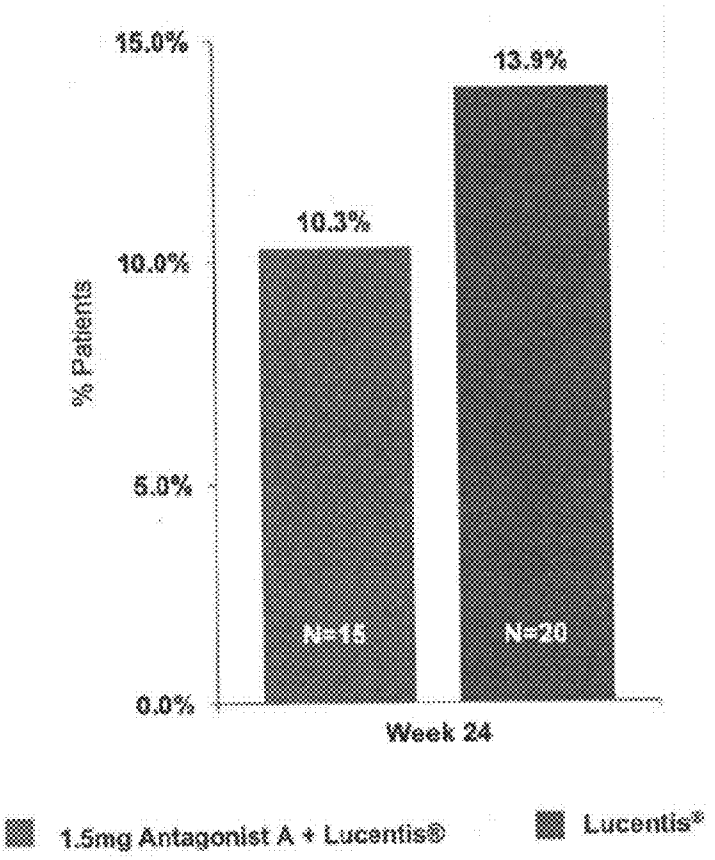


FIG. 8A

Increased Reduction in CNV Size in Small and Large Baseline CNV in Antaognist A (1.5 mg) Combination Arm

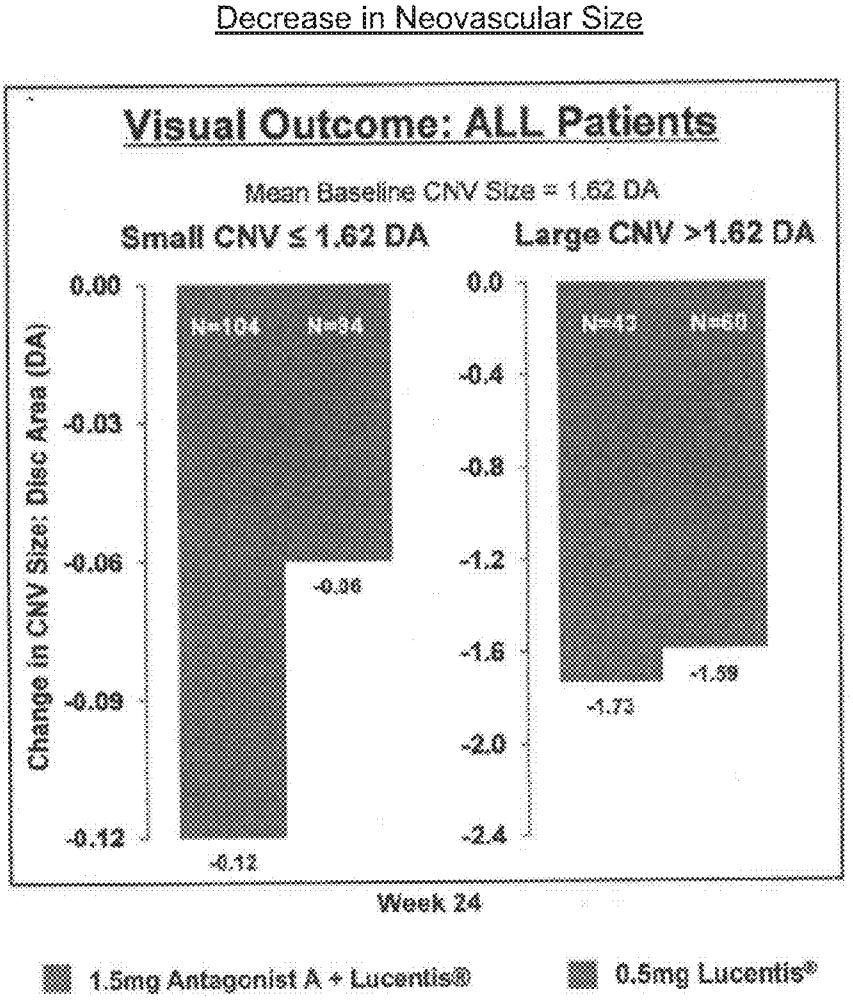


FIG. 8B

Increased Reduction in CNV Size in Small and Large Baseline CNV in Antaognist A (1.5 mg) Combination Arm

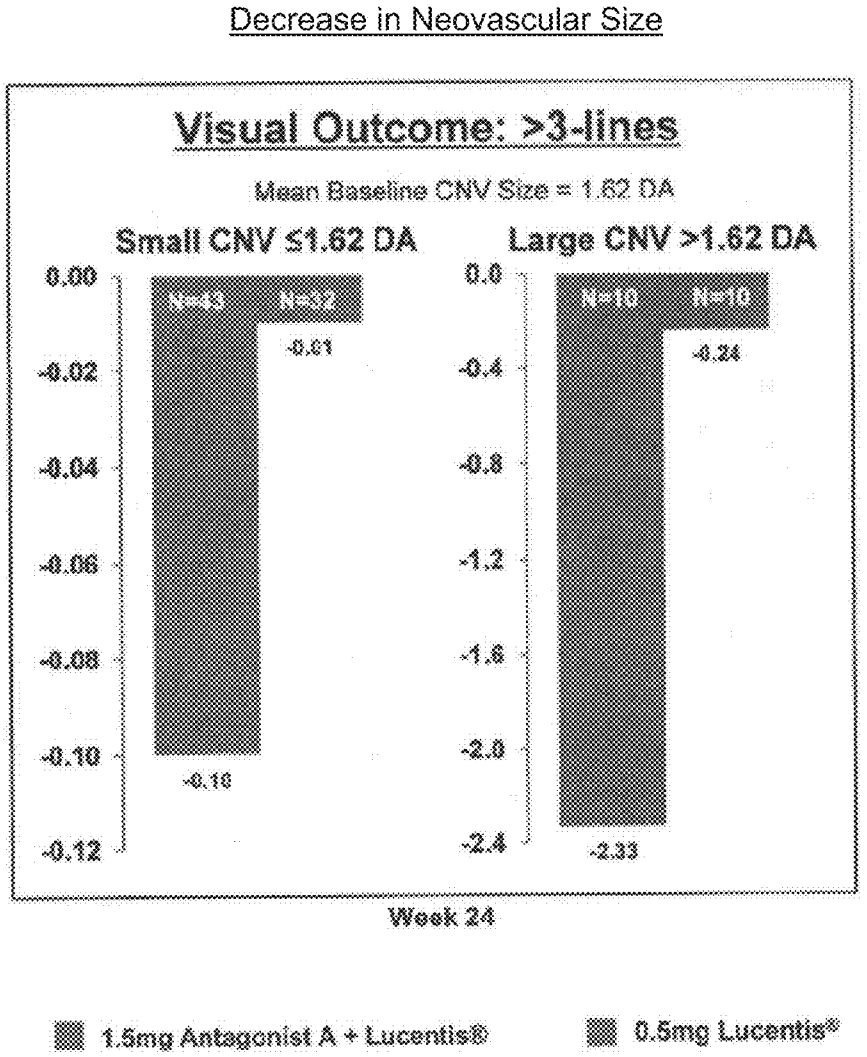


FIG. 9

ARC1905 Phase 2a Dry AMD (GA) Trial

- No adverse events considered to be related to ARC1905

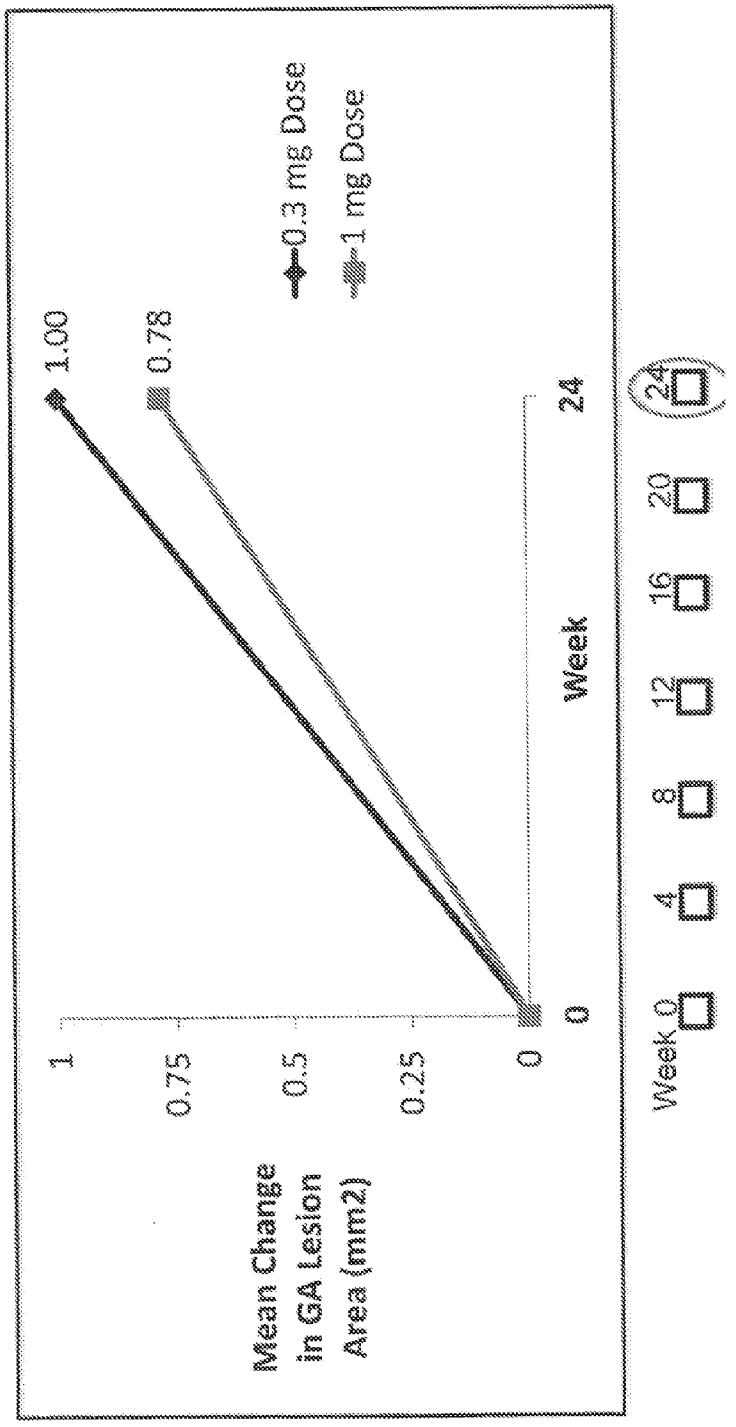


FIG. 10

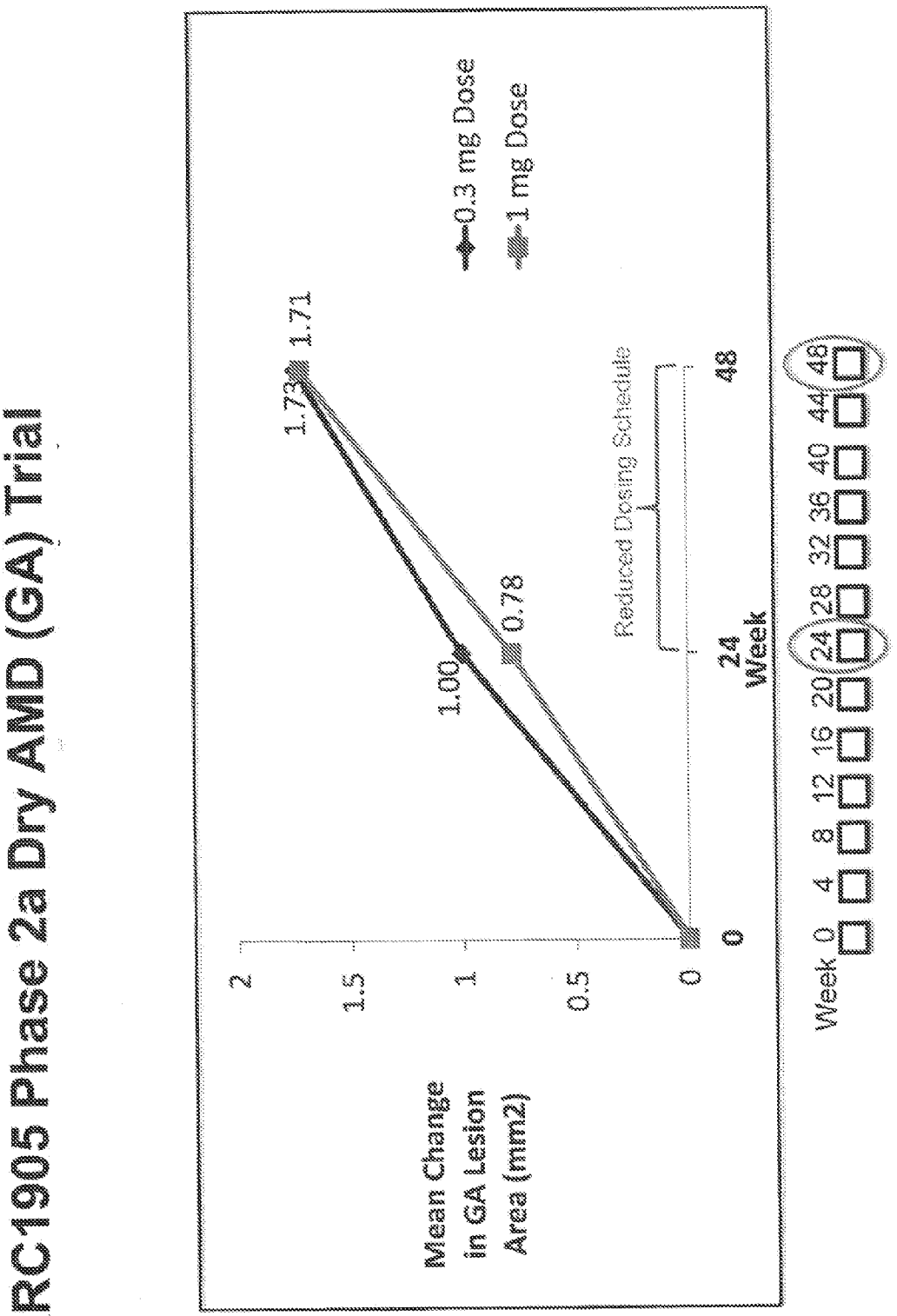

ARC1905 Phase 2a Dry AMD (GA) Trial
Mean Change in GA Lesion Area (mm2)
2
1.5
1
0.5
0
1.73
1.71
1.00
0.78
0.3 mg Dose
1 mg Dose
Reduced Dosing Schedule
24 Week
48
Week 0 4 8 12 16 20 24 28 32 36 40 44 48

FIG. 11

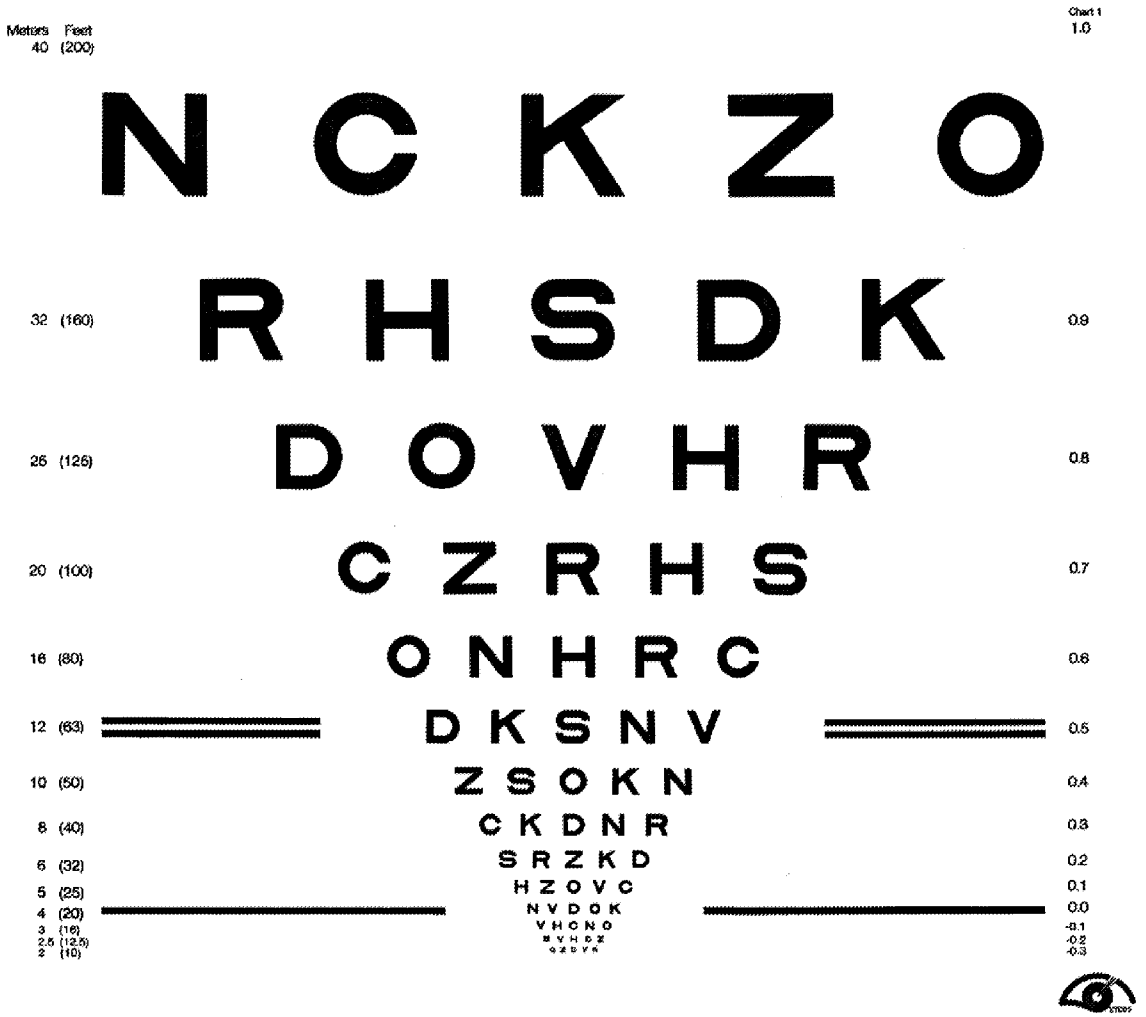

Meters Feet
40 (200)
Chart 1
1.0
N C K Z O
32 (160)
R H S D K
0.9
26 (126)
D O V H R
0.8
20 (100)
C Z R H S
0.7
16 (80)
O N H R C
0.6
12 (63)
D K S N V
0.5
10 (50)
Z S O K N
0.4
8 (40)
C K D N R
0.3
6 (32)
S R Z K D
0.2
5 (25)
H Z O V C
0.1
4 (20)
N V D O K
0.0
3 (16)
V H C N O
-0.1
2.5 (12.5)
-0.2
2 (10)
-0.3

FIG. 12

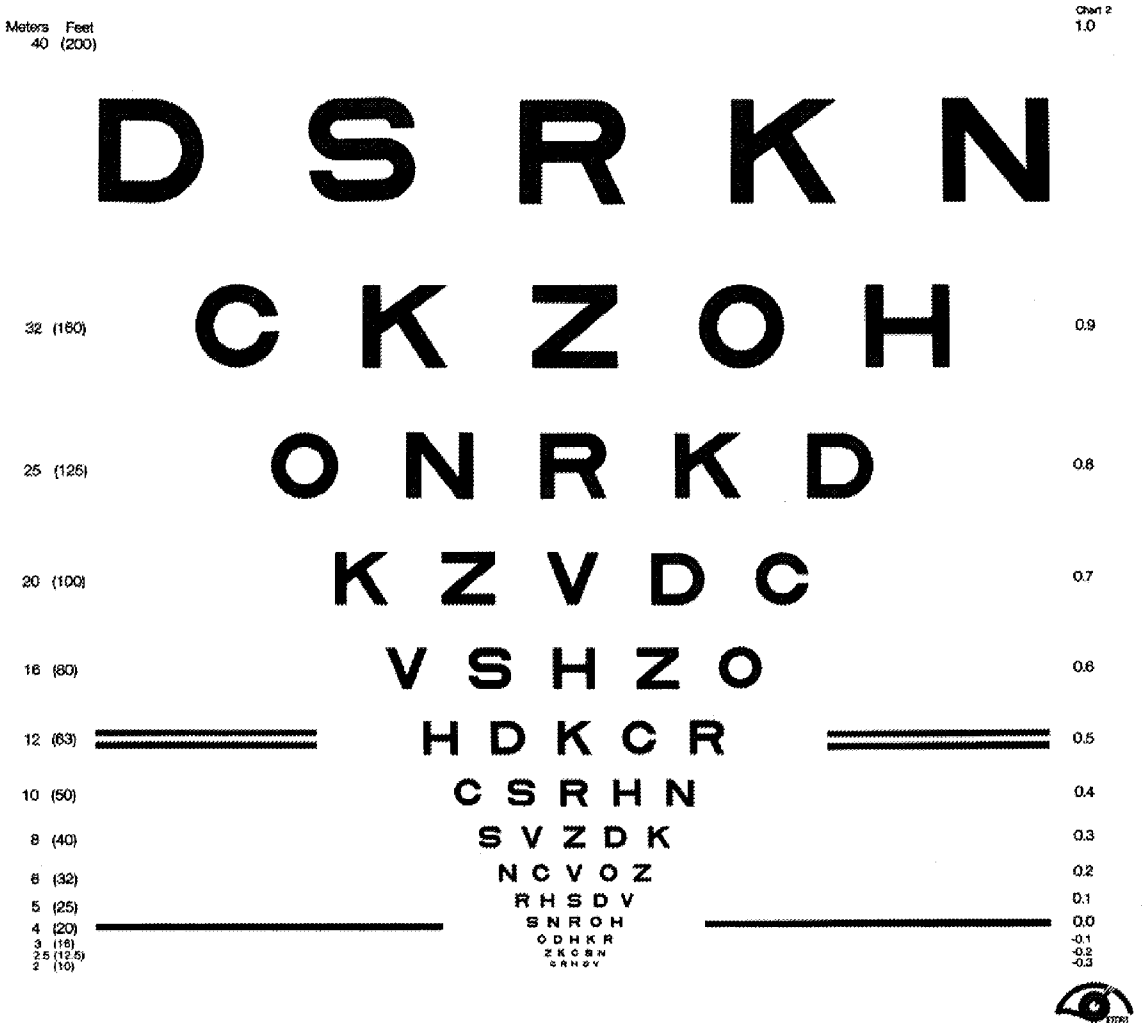
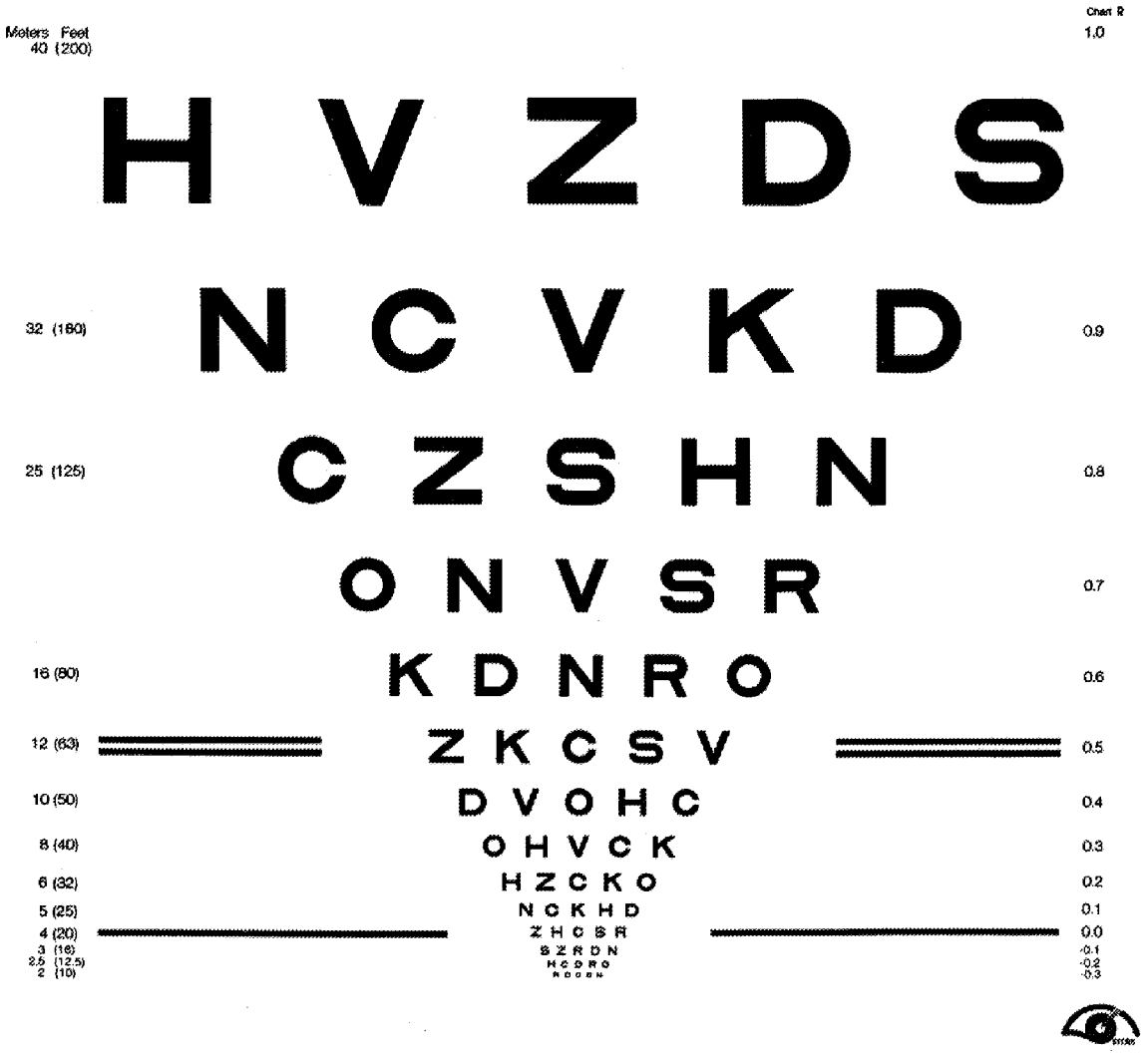

Meters Feet
40 (200)
Chart 2
1.0
D S R K N
32 (160)
C K Z O H
0.9
25 (125)
O N R K D
0.8
20 (100)
K Z V D C
0.7
16 (80)
V S H Z O
0.6
12 (63)
H D K C R
0.5
10 (50)
C S R H N
0.4
8 (40)
S V Z D K
0.3
6 (32)
N C V O Z
0.2
5 (25)
R H S D V
0.1
4 (20)
S N R O H
0.0
3 (16)
O D H K R
-0.1
2.5 (12.5)
Z K C S N
-0.2
2 (10)
C R N O V
-0.3

FIG. 13


Meters Feet
40 (200)
Chart R
1.0
H V Z D S
32 (160)
N C V K D
0.9
25 (125)
C Z S H N
0.8
O N V S R
0.7
16 (80)
K D N R O
0.6
12 (63)
Z K C S V
0.5
10 (50)
D V O H C
0.4
8 (40)
O H V C K
0.3
6 (32)
H Z C K O
0.2
5 (25)
N C K H D
0.1
4 (20)
Z H C S R
0.0
3 (16)
2.5 (12.5)
2 (10)
-0.1
-0.2
-0.3

METHODS FOR TREATING OR PREVENTING OPHTHALMOLOGICAL CONDITIONS

CROSS REFERENCE TO RELATED APPLICATIONS

[0001] This application claims the benefit of U.S. provisional application Nos. 61/845,938, filed Jul. 12, 2013, 61/845,935, filed Jul. 12, 2013, 61/845,936, filed Jul. 12, 2013, 61/866,502, filed Aug. 15, 2013, 61/866,503, filed Aug. 15, 2013, 61/866,507, filed Aug. 15, 2013, 61/911,854, filed Dec. 4, 2013, 61/911,860, filed Dec. 4, 2013, 61/911,894, filed Dec. 4, 2013, 61/926,812, filed Jan. 13, 2014, 61/926,825, filed Jan. 13, 2014, 61/926,848, filed Jan. 13, 2014, 61/931,116, filed Jan. 24, 2014, 61/931,125, filed Jan. 24, 2014, and 61/931,135, filed Jan. 24, 2014, each of which is incorporated by reference herein in its entirety.

SEQUENCE LISTING

[0002] The Sequence Listing associated with this application is provided in text format in lieu of a paper copy, and is hereby incorporated by reference into the specification. The name of the text file containing the Sequence Listing is OPHT_012_06US_SeqList_ST25.txt. The text file is about 372 KB, was created on Jul. 10, 2014, and is being submitted electronically via EFS-Web.

FIELD OF THE INVENTION

[0003] This invention relates to methods and compositions useful for the treatment or prevention of an ophthalmological disease or disorder, comprising administration of an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof.

BACKGROUND OF THE INVENTION

[0004] Various disorders of the eye are characterized, caused by, or result in choroidal, retinal or iris neovascularization or retinal edema. One of these disorders is macular degeneration. Age-related macular degeneration (AMD) is a disease that affects approximately one in ten Americans over the age of 65. One type of AMD, "wet-AMD," accounts only for approximately 10% of age-related macular degeneration cases but results in approximately 90% of cases of legal blindness from macular degeneration in the elderly. Another disorder of the eye is diabetic retinopathy. Diabetic retinopathy can affect up to 80% of all patients having diabetes for 10 years or more and is the third leading cause of adult blindness, accounting for almost 7% of blindness in the USA. Other disorders include hypertensive retinopathy, central serous chorioretinopathy, cystoid macular edema, Coats disease and ocular or adnexal neoplasms such as choroidal hemangioma, retinal pigment epithelial carcinoma, retinal vein occlusions and intraocular lymphoma.

[0005] Therefore, although advances in the understanding of the molecular events accompanying neovascularization have been made, there exists a need to utilize this understanding to develop improved methods for treating or preventing neovascular diseases disorders, including ocular neovascular diseases and disorders such as the neovascularization that occurs with AMD, diabetic retinopathy, and retinal vein occlusions.

SUMMARY OF THE INVENTION

[0006] The present invention relates to methods and compositions useful for the treatment or prevention of an ophthalmological disease or disorder.

[0007] The present invention provides a method for treating or preventing wet age-related macular degeneration (wet AMD), comprising administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein (a) and (b) are administered in an amount that is effective for treating or preventing wet AMD, and wherein the administering occurs once every month, ±about seven days, for a first administration period of at least 3 consecutive months, followed by administering (a) and (b) for a second administration period at a frequency of at least every other month±about seven days beginning at two months±about seven days after the day of the last month of the first administration period on which (a) and (b) are administered.

[0008] Also provided herein is a method for treating or preventing sub-retinal fibrosis, comprising administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof in an amount that is effective for treating or preventing sub-retinal fibrosis.

[0009] A method for treating or preventing von Hippel-Lindau (VHL) disease, comprising administering to a subject in need thereof. Antagonist A or another pharmaceutically acceptable salt thereof in an amount that is effective for treating or preventing VHL disease is also provided herein.

BRIEF DESCRIPTION OF THE DRAWINGS

[0010] Reference is made to the following detailed description, which sets forth illustrative embodiments and the accompanying drawings of which:

[0011] FIGS. **1**A-F show the chemical structure of Antagonist A, wherein the 5' end of its aptamer (SEQ ID NO: 1) is modified with $Me(OCH_2CH_2)_nOC(O)NH(CH_2)_4CH(NHC(O)O(CH_2CH_2O)_nMe)C(O)NH(CH_2)_6$—, where n is about 450. The designations B̂-F̂ indicate a continuation from a previous panel.

[0012] FIG. **2** shows a graph depicting the mean change in visual acuity in wet AMD patients in a phase 2b clinical trial, who were treated with 0.5 mg of Lucentis® alone or with 0.5 mg of Lucentis® and either 1.5 mg of Antagonist A or 0.3 mg of Antagonist A.

[0013] FIG. **3** shows a bar graph showing comparative visual-acuity benefit in wet AMD patients with treatment with 0.5 mg of Lucentis® and either 1.5 mg or 0.3 mg of Antagonist A as compared to treatment with Lucentis® monotherapy (0.5 mg).

[0014] FIG. **4** shows a graph depicting the early and sustained visual-acuity improvement over time in wet AMD patients treated with Lucentis® monotherapy (0.5 mg) or with 0.5 mg of Lucentis® and either 1.5 mg of Antagonist or 0.3 mg of Antagonist A.

[0015] FIGS. **5**A and **5**B provide bar graphs showing that the increased efficacy of treatment with 0.5 mg of Lucentis® and either 1.5 mg or 0.3 mg of Antagonist A as compared to treatment with Lucentis® monotherapy (0.5 mg) in patients with wet AMD is independent of baseline lesion size or baseline vision. FIG. **5**A shows the mean change in visual acuity for patients in each of the indicated baseline lesion quartiles, and FIG. **5**B shows the mean change in visual acuity for patients with the indicated baseline vision.

[0016] FIGS. **6**A and **6**B provide bar graphs showing that the cohort of patients treated with a combination of 0.5 mg of Lucentis® and 1.5 mg of Antagonist A included a greater proportion of patients with significant visual gain (FIG. **6**A) and fewer patients with visual loss (FIG. **6**B) as compared to the cohort of patients with treated Lucentis® monotherapy (0.5 mg).

[0017] FIGS. **7**A-C provide bar graphs showing that patients treated with 0.5 mg of Lucentis® and 1.5 mg of Antagonist A exhibited a greater mean improvement in final visual acuity as compared to patients treated with Lucentis® monotherapy (0.5 mg). FIG. **7**A shows the percentage of patients who demonstrated a visual acuity of 20/40 or better; FIG. **7**B shows the percentage of patients who demonstrated a visual acuity of 20/25 or better; and FIG. **7**C shows the percentage of patients who demonstrated a visual acuity of 20/200 or worse.

[0018] FIGS. **8**A and **8**B provide bar graphs showing increased reduction in choroidal neovascularization (CNV) lesion size in small and large baseline CNV lesions in wet AMD patients treated with both 0.5 mg of Lucentis® and 1.5 mg of Antagonist A as compared to patients treated with Lucentis® monotherapy (0.5 mg). FIG. **8**A shows the results in all patients, and FIG. **8**B shows the results in patients with a visual outcome >3-lines.

[0019] FIG. **9** shows a graph depicting the mean change in geographic atrophy (GA) lesion area in dry AMD patients measured at 24 weeks in patients treated with either a 0.3 mg or 1 mg dose of ARC1905 monthly from weeks 0 to 24 in a phase 2a trial.

[0020] FIG. **10** shows a graph depicting the mean change in GA lesion area in dry AMD patients measured at 24 weeks and 48 weeks in patients treated with either a 0.3 mg or 1 mg dose of ARC1905 monthly from weeks 0 to 48 in a phase 2a trial.

[0021] FIG. **11** shows Early Treatment for Diabetic Retinopathy Study ("ETDRS") Chart 1.

[0022] FIG. **12** shows Early Treatment for Diabetic Retinopathy Study ("ETDRS") Chart 2.

[0023] FIG. **13** shows Early Treatment for Diabetic Retinopathy Study ("ETDRS") Chart R.

DETAILED DESCRIPTION OF THE INVENTION

[0024] In certain aspects, the present invention provides new and improved methods and compositions for treating and preventing ophthalmological diseases and disorders, including, e.g., new uses, combination therapies, treatment and dosing regimens, and coformulations.

[0025] In one aspect, the invention provides methods for treating or preventing an ophthalmological disease or disorder, comprising administering to a subject in need thereof an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof. In particular embodiments, the subject is administered Antagonist A or another pharmaceutically acceptable salt thereof and not administered an anti-C5 agent. In some embodiments, the subject is administered Antagonist A or another pharmaceutically acceptable salt thereof and not administered a VEGF antagonist.

[0026] In particular embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof is administered in combination with a VEGF antagonist. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered in combination with ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008.

[0027] In particular embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof is administered in combination with a VEGF antagonist and an anti-C5 agent. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered in combination with a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008), and ARC1905.

[0028] The invention also provides treatment regimens, including treatment and dosing regimens, related to the coadministration of Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, optionally also in combination with an anti-C5 agent.

[0029] In further embodiments, another agent (e.g., an agent that is not Antagonist A, VEGF antagonist or an anti-C5 agent) that is useful for treating or preventing an ophthalmological disease or disorder is administered. In some embodiments, the methods comprise administering one or more (e.g., two) VEGF antagonists and/or one or more (e.g., two) anti-C5 agents to the subject in need thereof.

[0030] In another aspect, the invention provides methods for treating or preventing an ophthalmological disease or disorder, comprising administering to a subject in need thereof an effective amount of an anti-C5 agent (e.g., ARC1905). In particular embodiments, the subject is not administered Antagonist A or another pharmaceutically acceptable salt thereof. In some embodiments, the subject is not administered a VEGF antagonist.

[0031] In addition, the invention provides coformulations that comprise Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist. In certain embodiments, the coformulations further comprise an anti-C5 agent. In certain embodiments, the coformulations are pharmaceutically compositions comprising an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist, and a pharmaceutically acceptable carrier or vehicle. In certain embodiments, the coformulations are pharmaceutically compositions comprising an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist, and anti-C5 agent, and a pharmaceutically acceptable carrier or vehicle.

[0032] In one embodiment, the present invention provides methods for treating or preventing an ophthalmological disease or disorder, comprising administering to a subject in need thereof. Antagonist A or another pharmaceutically acceptable salt thereof and optionally a VEGF antagonist, wherein the methods further comprise performing a surgery to treat the ophthalmological disease or disorder and/or administration of an anti-C5 agent.

DEFINITIONS AND ABBREVIATIONS

[0033] As used herein, the following terms and phrases shall have the meanings set forth below. Unless defined otherwise, all technical and scientific terms used herein have the same meaning as commonly understood to one of skill in the art to which this invention belongs.

[0034] The term "about" when used in connection with a referenced numeric indication means the referenced numeric indication plus or minus up to 10% of that referenced numeric indication. For example, "about 100" means from 90 to 110 and "about six" means from 5.4 to 6.6.

[0035] The term "antagonist" refers to an agent that inhibits, either partially or fully, the activity or production of a

target molecule. In particular, the term "antagonist," as applied selectively herein, means an agent capable of decreasing levels of gene expression, mRNA levels, protein levels or protein activity of the target molecule. Illustrative forms of antagonists include, for example, proteins, polypeptides, peptides (such as cyclic peptides), antibodies or antibody fragments, peptide mimetics, nucleic acid molecules, antisense molecules, ribozymes, aptamers, RNAi molecules, and small organic molecules. Illustrative non-limiting mechanisms of antagonist inhibition include repression of ligand synthesis and/or stability (e.g., using, antisense, ribozymes or RNAi compositions targeting the ligand gene/nucleic acid), blocking of binding of the ligand to its cognate receptor (e.g., using anti-ligand aptamers, antibodies or a soluble, decoy cognate receptor), repression of receptor synthesis and/or stability (e.g., using, antisense, ribozymes or RNAi compositions targeting the ligand receptor gene/nucleic acid), blocking of the binding of the receptor to its cognate receptor (e.g., using receptor antibodies) and blocking of the activation of the receptor by its cognate ligand (e.g., using receptor tyrosine kinase inhibitors). In addition, the antagonist may directly or indirectly inhibit the target molecule.

[0036] The term "antibody fragment" includes a portion of an antibody that is an antigen binding fragment or single chains thereof. An antibody fragment can be a synthetically or genetically engineered polypeptide. 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_L, V_H, C_L and C_{H1} 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 C_{H1} domains; (iv) a Fv fragment consisting of the V_L and V_H 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). Furthermore, although the two domains of the Fv fragment, V_L and V_H, 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_L and V_H 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 fragment" of an antibody. These antibody fragments are obtained using conventional techniques known to those in the art, and the fragments can be screened for utility in the same manner as whole antibodies.

[0037] The term "aptamer" refers to a peptide or nucleic acid that has an inhibitory effect on a target. Inhibition of the target by the aptamer can occur by binding of the target, by catalytically altering the target, by reacting with the target in a way which modifies the target or the functional activity of the target, by ionically or covalently attaching to the target as in a suicide inhibitor or by facilitating the reaction between the target and another molecule. Aptamers can be peptides, ribonucleotides, deoxyribonucleotides, other nucleic acids or a mixture of the different types of nucleic acids. Aptamers can comprise one or more modified amino acid, bases, sugars, polyethylene glycol spacers or phosphate backbone units as described in further detail herein.

[0038] A nucleotide sequence is "complementary" to another nucleotide sequence if each of the bases of the two sequences matches, i.e., are capable of forming Watson Crick base pairs. The complement of a nucleic acid strand can be the complement of a coding strand or the complement of a non-coding strand.

[0039] The phrase "conserved residue" refers to an amino acid of a group of amino acids having particular common properties. A functional way to define common properties among individual amino acids is to analyze the normalized frequencies of amino acid changes among corresponding proteins of homologous organisms. According to such analyses, groups of amino acids may be characterized where amino acids within a group exchange preferentially with each other, and therefore resemble each other most in their impact on the overall protein structure (Schulz, G. E. and R. H. Schirmer, *Principles of Protein Structure*, Springer-Verlag). Examples of amino acid groups defined in this manner include:

[0040] (i) a charged group, consisting of Glu and Asp, Lys, Arg and His,

[0041] (ii) a positively-charged group, consisting of Lys, Arg and His,

[0042] (iii) a negatively-charged group, consisting of Glu and Asp,

[0043] (iv) an aromatic group, consisting of Phe, Tyr and Trp,

[0044] (v) a nitrogen ring group, consisting of His and Trp,

[0045] (vi) a large aliphatic nonpolar group, consisting of Val, Leu and Ile,

[0046] (vii) a slightly-polar group, consisting of Met and Cys,

[0047] (viii) a small-residue group, consisting of Ser, Thr, Asp, Asn, Gly, Ala, Glu, Gln and Pro,

[0048] (ix) an aliphatic group consisting of Val, Leu, Ile, Met and Cys, and

[0049] (x) a small hydroxyl group consisting of Ser and Thr.

[0050] Members of each of the above groups are conserved residues.

[0051] The term "label" includes, but is not limited to, a radioactive isotope, a fluorophore, a chemiluminescent moiety, an enzyme, an enzyme substrate, an enzyme cofactor, an enzyme inhibitor, a dye, a metal ion, a ligand (e.g., biotin or a hapten) and the like. Examples of fluorophore labels include fluorescein, rhodamine, dansyl, umbelliferone, Texas red, luminol, NADPH, alpha-beta-galactosidase and horseradish peroxidase.

[0052] The term "nucleic acid" refers to a polynucleotide such as deoxyribonucleic acid (DNA) or ribonucleic acid (RNA). The term also includes analogs of RNA or DNA made from nucleotide analogs, and, as applicable to the embodiment being described, single (sense or antisense) and double-stranded polynucleotides, ESTs, chromosomes, cDNAs, mRNAs, and rRNAs.

[0053] The terms "RNA interference," "RNAi," "miRNA," and "siRNA" refer to any method by which expression of a gene or gene product is decreased by introducing into a target cell one or more double-stranded RNAs, which are homologous to a gene of interest (particularly to the messenger RNA of the gene of interest, e.g., PDGF or VEGF).

[0054] The term "neovascularization" refers to new blood vessel formation in abnormal tissue or in abnormal positions.

[0055] The term "angiogenesis" refers to formation of new blood vessels in normal or in abnormal tissue or positions.

[0056] The term "ophthalmological disease" includes diseases of the eye and the ocular adnexa.

[0057] The term "ocular neovascular disorder" refers to an ocular disorder characterized by neovascularization. In one embodiment, the ocular neovascular disorder is a disorder other than cancer. Examples of ocular neovascular disorders include diabetic retinopathy and age-related macular degeneration.

[0058] The term "mammal" includes a human, monkey, cow, hog, sheep, horse, dog, cat, rabbit, rat and mouse. In certain embodiments, a subject is a mammal.

[0059] The term "PDGF" refers to a platelet-derived growth factor that regulates cell growth or division. As used herein, the term "PDGF" includes the various subtypes of PDGF including PDGF-B (see SEQ ID NOS: 2 (nucleic acid) and 3 (polypeptide)), PDGF-A (see SEQ ID NOS: 4 (nucleic acid) and 5 (polypeptide), PDGF-C (see SEQ ID NOS: 6 (nucleic acid) and 7 (polypeptide)), PDGF-D, variants 1 (see SEQ ID NOS: 8 (nucleic acid) and 9 (polypeptide)) and 2 (see SEQ ID NOS: 10 (nucleic acid) and 11 (polypeptide)), and dimerized forms thereof, including PDGF-AA, PDGF-AB, PDGF-BB, PDGF-CC, and PDGF-DD. Platelet derived growth factors includes homo- or heterodimers of A-chain (PDGF-A) and B-chain (PDGF-B) that exert their action via binding to and dimerization of two related receptor tyrosine kinase platelet-derived growth factor cell surface receptors (i.e., PDGFRs), PDGFR-α (see SEQ ID NOS: 12 (nucleic acid) and 13 (polypeptide)) and PDGFR-β (see SEQ ID NOS: 14 (nucleic acid) and 15 (polypeptide)). In addition, PDGF-C and PDGF-D, two additional protease-activated ligands for the PDGFR complexes, have been identified (Li et al., (2000) *Nat. Cell. Biol.* 2: 302-9; Bergsten et al., (2001) *Nat. Cell. Biol.* 3: 512-6; and Uutele et al., (2001) *Circulation* 103: 2242-47). Due to the different ligand binding specificities of the PDGFRs, it is known that PDGFR-α/α binds PDGF-AA, PDGF-BB, PDGF-AB, and PDGF-CC; PDGFR-β/β binds PDGF-BB and PDGF-DD; whereas PDGFR-α/β binds PDGF-AB, PDGF-BB, PDGF-CC, and PDGF-DD (Betsholtz et al., (2001) *BioEssays* 23: 494-507). As used herein, the term "PDGF" also refers to those members of the class of growth factors that induce DNA synthesis and mitogenesis through the binding and activation of a PDGFR on a responsive cell type. PDGFs can effect, for example: directed cell migration (chemotaxis) and cell activation; phospholipase activation; increased phosphatidylinositol turnover and prostaglandin metabolism; stimulation of both collagen and collagenase synthesis by responsive cells; alteration of cellular metabolic activities, including matrix synthesis, cytokine production, and lipoprotein uptake; induction, indirectly, of a proliferative response in cells lacking PDGF receptors; and potent vasoconstrictor activity. The term "PDGF" can be used to refer to a "PDGF" polypeptide, a "PDGF" encoding gene or nucleic acid, or a dimerized form thereof.

[0060] The term "PDGF-A" refers to an A chain polypeptide of PDGF or its corresponding encoding gene or nucleic acid.

[0061] The term "PDGF-B" refers to a B chain polypeptide of PDGF or its corresponding encoding gene or nucleic acid.

[0062] The term "PDGF-C" refers to a C chain polypeptide of PDGF or its corresponding encoding gene or nucleic acid.

[0063] The term "PDGF-D" refers to a D chain polypeptide of PDGF or its corresponding encoding gene or nucleic acid, including variants 1 and 2 of the D chain polypeptide of PDGF.

[0064] The term "PDGF-AA" refers to a dimer having two PDGF-A chain polypeptides.

[0065] The term "PDGF-AB" refers to a dimer having one PDGF-A chain polypeptide and one PDGF-B chain polypeptide.

[0066] The term "PDGF-BB" refers to a dimer having two PDGF-B chain polypeptides.

[0067] The term "PDGF-CC" refers to a dimer having two PDGF-C chain polypeptides.

[0068] The term "PDGF-DD" refers to a dimer having two PDGF-D chain polypeptides.

[0069] The term "VEGF" refers to a vascular endothelial growth factor that induces angiogenesis or an angiogenic process. As used herein, the term "VEGF" includes the various subtypes of VEGF (also known as vascular permeability factor (VPF) and VEGF-A) (see SEQ ID NOS: 16 (nucleic acid) and 17 (polypeptide)) that arise by, e.g., alternative splicing of the VEGF-A/VPF gene including $VEGF_{121}$, $VEGF_{165}$ and $VEGF_{189}$. Further, as used herein, the term "VEGF" includes VEGF-related angiogenic factors such as PIGF (placenta growth factor), VEGF-B, VEGF-C, VEGF-D and VEGF-E, which act through a cognate VEFG receptor (i.e., VEGFR) to induce angiogenesis or an angiogenic process. The term "VEGF" includes any member of the class of growth factors that binds to a VEGF receptor such as VEGFR-1 (Flt-1) (see SEQ ID NOS: 18 (nucleic acid) and 19 (polypeptide)), VEGFR-2 (KDR/Flk-1) (see SEQ ID NOS: 20 (nucleic acid) and 21 (polypeptide)), or VEGFR-3 (FLT-4). The term "VEGF" can be used to refer to a "VEGF" polypeptide or a "VEGF" encoding gene or nucleic acid.

[0070] The term "PDGF antagonist" refers to an agent that reduces, or inhibits, either partially or fully, the activity or production of a PDGF. In certain embodiments, the PDGF antagonist inhibits one or more of PDGF-A, PDGF-B, PDGF-C and PDGF-D. In certain embodiments, the PDGF antagonist inhibits one or more of PDGF-A, PDGF-B, and PDGF-C. In some embodiments, the PDGF antagonist inhibits a dimerized form of PDGF, such as PDGF-AA, PDGF-AB, PDGF-BB, PDGF-CC, and PDGF-DD. In certain embodiments, the PDGF antagonist inhibits PDGF-BB. In other embodiments, the PDGF antagonist inhibits PDGF-AB. A PDGF antagonist can directly or indirectly reduce or inhibit the activity or production of a specific PDGF such as PDGF-B. Furthermore, "PDGF antagonists" consistent with the above definition of "antagonist," include agents that act on a PDGF ligand or its cognate receptor so as to reduce or inhibit a PDGF-associated receptor signal. Examples of "PDGF antagonists" include antisense molecules, ribozymes or RNAi that target a PDGF nucleic acid; anti-PDGF aptamers, anti-PDGF antibodies to PDGF itself or its receptor, or soluble PDGF receptor decoys that prevent binding of a PDGF to its cognate receptor, antisense molecules, ribozymes or RNAi that target a cognate PDGF receptor (PDGFR) nucleic acid; anti-PDGFR aptamers or anti-PDGFR antibodies that bind to a cognate PDGFR receptor; and PDGFR tyrosine kinase inhibitors.

[0071] The term "VEGF antagonist" refers to an agent that reduces, or inhibits, either partially or fully, the activity or production of a VEGF. In certain embodiments, the VEGF antagonist inhibits one or more of VEGF-A, VEGF-B, VEGF-C and VEGF-D. A VEGF antagonist can directly or indirectly reduce or inhibit the activity or production of a specific VEGF such as $VEGF_{165}$. Furthermore, "VEGF antagonists" consistent with the above definition of "antago-

nist," include agents that act on either a VEGF ligand or its cognate receptor so as to reduce or inhibit a VEGF-associated receptor signal. Examples of "VEGF antagonists" include antisense molecules, ribozymes or RNAi that target a VEGF nucleic acid; anti-VEGF aptamers, anti-VEGF antibodies to VEGF itself or its receptor, or soluble VEGF receptor decoys that prevent binding of a VEGF to its cognate receptor; antisense molecules, ribozymes, or RNAi that target a cognate VEGF receptor (VEGFR) nucleic acid; anti-VEGFR aptamers or anti-VEGFR antibodies that bind to a cognate VEGFR receptor; and VEGFR tyrosine kinase inhibitors. In certain embodiments, the VEGF antagonist is a peptide, e.g., a peptide comprising three or more amino acid residues. In certain embodiments, the VEGF antagonist is a bicyclic peptide.

[0072] The term "effective amount" when used in connection with an active agent, refers to an amount of the active agent, e.g., a PDGF antagonist, a VEGF antagonist or an anti-C5 agent, alone or in combination with another active agent, that is useful to treat or prevent an ophthalmological disease or disorder. The "effective amount" can vary depending upon the mode of administration, specific locus of the ophthalmological disease or disorder, the age, body weight, and general health of the subject. The effective amount of two or more active agents is the combined amount of the active agents that is useful for treating or preventing an ophthalmological disease or disorder, even if the amount of one of the agents, in the absence of one or more of the other agents, is ineffective to treat or prevent the ophthalmological disease or disorder.

[0073] A "variant" of polypeptide X refers to a polypeptide having the amino acid sequence of polypeptide X in which is altered in one or more amino acid residues. The variant can have "conservative" changes, wherein a substituted amino acid has similar structural or chemical properties (e.g., replacement of leucine with isoleucine). More rarely, a variant can have "nonconservative" changes (e.g., replacement of glycine with tryptophan). Analogous minor variations may also include amino acid deletions or insertions, or both. Guidance in determining which amino acid residues may be substituted, inserted, or deleted without eliminating biological or immunological activity can be determined using computer programs well known in the art, for example, LASERGENE software (DNASTAR).

[0074] The term "variant," when used in the context of a polynucleotide sequence, can encompass a polynucleotide sequence related to that of gene or the coding sequence thereof. This definition also includes, for example, "allelic," "splice," "species," or "polymorphic" variants. A splice variant can have significant identity to a reference molecule, but will generally have a greater or lesser number of polynucleotides due to alternative splicing of exons during mRNA processing. The corresponding polypeptide can possess additional functional domains or an absence of domains. Species variants are polynucleotide sequences that vary from one species to another. The resulting polypeptides generally will have significant amino acid identity relative to each other. A polymorphic variant is a variation in the polynucleotide sequence of a particular gene between individuals of a given species.

[0075] The term "anti-C5 agent" refers to an agent that reduces, or inhibits, either partially or fully, the activity or production of a C5 complement protein or a variant thereof. An anti-C5 agent can directly or indirectly reduce or inhibit the activity or production of a C5 complement protein or variant thereof. An anti-C5 agent can reduce or inhibit the conversion of C5 complement protein into its component polypeptides C5a and C5b. Anti-C5 agents can also reduce or inhibit the activity or production of C5a and/or C5b. Examples of "anti-C5 agents" include antisense molecules, ribozymes or RNAi that target a C5 nucleic acid; anti-C5 aptamers including anti-C5a and anti-C5b aptamers, anti-C5 antibodies directed against C5, C5a, C5b, or C5b-9, or soluble C5 receptor decoys that prevent binding of a C5 complement protein or variant or fragment thereof (e.g., C5a or C5b) to a binding partner or receptor.

Agents Useful for Treatment or Prevention of an Opthalmological Disease or Disorder

[0076] Antagonist A

[0077] Antagonist A is a PEGylated, anti-PDGF aptamer having the sequence CAGGCUACGC GTAGAGCAUC ATGATCCUGT (SEQ ID NO: 1) (see Example 3 of US Patent Application Publication No. 20050096257, incorporated herein by reference in its entirety) having 2'-fluoro-2'-deoxyuridine at positions 6, 19 and 28; 2'-fluoro-2'-deoxycytidine at positions 8, 20, 26, and 27; 2'-O-Methyl-2'-deoxyguanosine at positions 9, 14, 16, and 29; 2'-O-Methyl-2'-deoxyadenosine at position 21; an inverted orientation T (i.e., 3'-3'-linked) at position 30; and two heaxethylene-glycol phosphoramidite linkages that join together the 9^{th} and 10^{th} nucleotides and 21^{st} and 22^{nd} nucleotides via phosphodiester linkages between the linker and the respective nucleotides.

[0078] The chemical name of Antagonist A is [(monomethoxy 20K polyethylene glycol carbamoyl-N2-)(monomethoxy 20K polyethylene glycol carbamoyl-N6-)]-lysine-amido-6-hexandilyl-(1-5')-2'-deoxycytidylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-2'-deoxyguanylyl-(3'-5')-2'-deoxyguanylyl-(3'-5')-2'-deoxycytidylyl-(3'-5')-2'-deoxy-2'-fluorouridylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-2'-deoxy-2'-fluorocytidylyl-(3'-5')-2'-deoxy-2'-methoxyguanylyl-(3'-1)-PO_3-hexa(ethyloxy)-(18-5')-2'-deoxycytidylyl-(3'-5')-2'-deoxyguanylyl-(3'-5')-thymidylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-2'-deoxy-2'-methoxyguanylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-2'-deoxy-2'-methoxyguanylyl-(3'-5')-2'-deoxycytidylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-2'-deoxy-2'-fluorouridylyl-(3'-5')-2'-deoxy-2'-fluorocytidylyl-(3'-5')-2'-deoxy-2'-methoxyadenylyl-(3'-1)-PO_3-hexa(ethyloxy)-(18-5')-thymidylyl-(3'-5')-2'-deoxyguanylyl-(3'-5')-2'-deoxyadenylyl-(3'-5')-thymidylyl-(3'-5')-2'-deoxy-2'-fluorocytidylyl-(3'-5')-2'-deoxy-2'-fluorocytidylyl-(3'-5')-2'-deoxy-2'-fluorouridylyl-(3'-5')-2'-deoxy-2'-methoxyguanylyl-(3'-3')-thymidine.

[0079] The structure of Antagonist A is shown in FIG. **1**.

[0080] The sequence of Antagonist A is:

[0081] 5'-[mPEG2 40 kD]-[HN—$(CH_2)_6$O]CAGGCU-$_f$AC$_f$G$_m$ [$PO_3(CH_2CH_2O)_6$]CGTAG$_m$AG$_m$CAU$_f$C$_f$Am [$PO_3(CH_2CH_2O)_6$]TGATC$_f$C$_f$U$_f$G$_m$-[3T]-3', whose aptamer sequence is set forth in (SEQ ID NO: 1),

[0082] where [3T] refers to an inverted thymidine nucleotide that is attached to the 3' end of the oligonucleotide at the 3' position on the ribose sugar, and [mPEG2 40 kD] represents two 20 kD polyethylene glycol (PEG) polymer chains, in one embodiment two about 20 kD PEG polymer chains, that are covalently attached to the two amino groups of a lysine residue via carbamate linkages. This moiety is in turn linked with the oligonucleotide via the amino linker described below.

[0083] [HN—$(CH_2)_6$O] represents a bifunctional α-hydroxy-ω-amino linker that is covalently attached to the PEG

polymer via an amide bond. The linker is attached to the oligonucleotide at the 5'-end of Antagonist A by a phosphodiester linkage.

[0084] $[PO_3(CH_2CH_2O)_6]$ represents the hexaethylene glycol (HEX) moieties that join segments of the oligonucleotide via phosphodiester linkages. Antagonist A has two HEX linkages that join together the 9^{th} and 10^{th} nucleotides and 21^{st} and 22^{nd} nucleotides via phosphodiester linkages between the linker and the respective nucleotides.

[0085] C, A, G, and T represent the single letter code for the 2'-deoxy derivatives of cytosine, adenosine, guanosine, and thymidine nucleic acids, respectively. Antagonist A has four 2'-deoxyribocytosine, six 2'-deoxyriboadenosine, four 2'-deoxyriboguanosine, and four 2'-deoxyribothymidine.

[0086] G_m and A_m represent 2'-methoxy substituted forms of guanosine and adenosine, respectively. Antagonist A has four 2'-methoxyguanosines and one 2'-methoxyadenosine. C_f and U_f represent the 2'-fluoro substituted forms of cytosine and uridine, respectively. Antagonist A has four 2'-fluorocytosines and three 2'-fluorouridines.

[0087] The phosphodiester linkages in the oligonucleotide, with the exception of the 3'-terminus, connect the 5'- and 3'-oxygens of the ribose ring with standard nucleoside phosphodiester linkages. The phosphodiester linkage between the 3'-terminal thymidine and the penultimate G_m links their respective 3'-oxygens, which is referred to as the 3',3'-cap.

[0088] Antagonist A has a molecular weight from 40,000 to 60,000 Daltons, in one embodiment from about 40,000 to about 60,000 Daltons, and can be colorless to slightly yellow in solution. Antagonist A can be present in a solution of monobasic sodium phosphate monohydrate and dibasic sodium phosphate heptahydrate as buffering agents and sodium chloride as a tonicity adjuster. Antagonist A is a hydrophilic polymer. The Antagonist A is soluble in water and in phosphate-buffered saline (PBS), as assessed by visual inspection, to at least 50 mg (based on oligonucleotide weight)/mL solution.

[0089] Antagonist A can be synthesized using an iterative chemical synthesis procedure to produce the oligonucleotide portion, which is then covalently bonded to a pegylation reagent, as further described in Example 4 of US Patent Publication NO. 2012/0100136.

[0090] Antagonist A is a persodium salt. Other pharmaceutically acceptable salts, however, of Antagonist are useful in the compositions and methods disclosed herein.

[0091] VEGF Antagonists

[0092] In some embodiments, the VEGF antagonist is ranibizumab (commercially available under the trademark Lucentis® (Genentech, San Francisco, Calif.); see FIG. 1 of U.S. Pat. No. 7,060,269 for the heavy chain and light chain variable region sequences), bevacizumab (commercially available under the trademark Avastin® (Genentech, San Francisco, Calif.); see FIG. 1 of U.S. Pat. No. 6,054,297 for the heavy chain and light chain variable region sequences), aflibercept (commercially available under the trademark Eylea® (Regeneron, Tarrytown, N.Y.), KH902 VEGF receptor-Fc fusion protein (see Zhang et al. (2008) Mol. Vis. 14:37-49), 2C3 antibody (see U.S. Pat. No. 6,342,221, Column 8, lines 48-67, Column 9, lines 1-21). ORA102 (available from Ora Bio, Ltd.), pegaptanib (e.g., pegaptanib sodium; commercially available under the trademark Macugenx (Valeant Pharmaceuticals, Bridgewater, N.J.; see FIG. 1 of U.S. Pat. No. 6,051,698)), bevasiranib (see Dejneka et al. (2008) Mol. Vis. 14:997-1005), SIRNA-027 (Shen et al. (2006) Gene Ther. 13:225-34), decursin (see U.S. Pat. No. 6,525,089 (Column 3, lines 5-16)), decursinol (see Ahn et al. (1997) Planta Med. 63:360-1), picropodophyllin (see Economou (2008) Investigative Ophthalmology & Visual Science. 49:2620-6), guggulsterone (see Kim et al. (2008) Oncol. Rep. 20:1321-7), PLG 101 (see Ahmadi and Lim (2008) Expert Opin Pharmacother. 9:3045-52), PLG201 (see Ahmadi and Lim (2008)), eicosanoid LXA4 (see Baker et al (2009) J. Immun. 182: 3819-26), PTK787 (commercially available under the trademark Vitalanib™; see Barakat and Kaiser (2009) Expert Opin Investig Drugs 18:637-46), pazopanib (see Takahashi et al. (2009) Arch Ophthalmol. 127:494-9), axitinib (see Hu-Lowe et al. (2008) Clin Cancer Res. 14:7272-83), CDDO-Me (see Sogno et al. (2009) Recent Results Cancer Res. 181:209-12), CDDO-Me (see Sogno et al. (2009)), shikonin (see Hisa et al. (1998) Anticancer Res. 18:783-90), beta-hydroxyisovalerylshikonin (see Hisa et al. (1998)), ganglioside GM3 (Chung et al. (2009) Glycobio. 19:229-39), DC101 antibody (see U.S. Pat. No. 6,448,077, Column 2, lines 61-65), Mab25 antibody (see U.S. Pat. No. 6,448,077, Column 2, lines 61-65), Mab73 antibody (see U.S. Pat. No. 6,448,077, Column 2, lines 61-65), 4A5 antibody (see U.S. Pat. No. 6,383,484, Column 12, lines 50-54), 4E10 antibody (see U.S. Pat. No. 6,383,484, Column 10, lines 66-67, Column 11, lines 1-2), 5F12 antibody (see U.S. Pat. No. 6,383,484, Column 10, lines 62-65), VA01 antibody (see U.S. Pat. No. 5,730,977, Column 6, lines 26-30), BL2 antibody (U.S. Pat. No. 5,730,977, Column 6, lines 30-32), VEGF-related protein (see U.S. Pat. No. 6,451, 764, FIG. 1), sFLT01 (see Pechan et al. (2009) Gene Ther. 16:10-6), sFLT02 (see Pechan et al. (2009)), Peptide B3 (see Lacal et al. (2008) Eur J Cancer 44:1914-21), TG 100801 (see Palanki et al. (2008) J Med. Chem. 51:1546-59), sorafenib (commercially available under the trademark Nexavar™; see Kernt et al. (2008) Acta Ophthalmol. 86:456-8), G6-31 antibody (see Crawford et al. (2009) Cancer Cell 15:21-34), ESBA1008 (see U.S. Pat. No. 8,349,322), tivozanib (see U.S. Pat. No. 6,821,987, incorporated by reference in its entirety; Campas et al. (2009) Drugs Fut 2009, 34(10): 793), or a pharmaceutically acceptable salt thereof.

[0093] In another embodiment, the VEGF antagonist is an antibody or an antibody fragment which binds to an epitope VEGF-A (SEQ ID NO: 22) or VEGF-B (SEQ ID NO: 23), or any portion of the epitopes. In one embodiment, the VEGF antagonist is an antibody or antibody fragment that binds to one or more of an epitope of VEGF (e.g., SEQ ID NOS: 22 and 23). In another embodiment, the VEGF antagonist is an antibody or an antibody fragment which binds to an epitope of VEGF, such as an epitope of VEGF-A, VEGF-B, VEGF-C, VEGF-D, or VEGF-E. In some embodiments, the VEGF antagonist binds to an epitope of VEGF such that binding of VEGF and VEGFR are inhibited. In one embodiment, the epitope encompasses a component of the three dimensional structure of VEGF that is displayed, such that the epitope is exposed on the surface of the folded VEGF molecule. In one embodiment, the epitope is a linear amino acid sequence from VEGF.

[0094] In some embodiments, an inhibitory antibody directed against VEGF is known in the art, e.g., those described in U.S. Pat. Nos. 6,524,583, 6,451,764 (VRP antibodies), U.S. Pat. Nos. 6,448,077, 6,416,758, 6,403,088 (to VEGF-C), U.S. Pat. No. 6,383,484 (to VEGF-D), U.S. Pat. No. 6,342,221 (anti-VEGF antibodies), U.S. Pat. Nos. 6,342, 219 6,331,301 (VEGF-B antibodies), and U.S. Pat. No. 5,730,977, and PCT publications WO96/30046, WO

97/44453, and WO 98/45331, the contents of which are incorporated by reference in their entirety.

[0095] Other non-antibody VEGF antagonists include antibody mimetics (e.g., Affibody® molecules, affilins, affitins, anticalins, avimers, Kunitz domain peptides, and monobodies) with VEGF antagonist activity. This includes recombinant binding proteins comprising an ankyrin repeat domain that binds VEGF-A and prevents it from binding to VEGFR-2. One example is MP0112, also known as AGN 150998 (DARPin®). The ankyrin binding domain may have an amino acid sequence of SEQ ID NO: 97.

[0096] Recombinant binding proteins comprising an ankyrin repeat domain that binds VEGF-A and prevents it from binding to VEGFR-2 are described in more detail in WO2010/060748 and WO2011/135067.

[0097] Further specific antibody mimetics with VEGF antagonist activity are the 40 kD pegylated anticalin PRS-050 and the monobody angiocept (CT-322).

[0098] The aforementioned non-antibody VEGF antagonist may be modified to further improve their pharmacokinetic properties or bioavailability. For example, a non-antibody VEGF antagonist may be chemically modified (e.g., pegylated) to extend its in vivo half-life. Alternatively or in addition, it may be modified by glycosylation or the addition of further glycosylation sites not present in the protein sequence of the natural protein from which the VEGF antagonist was derived.

[0099] Other non-antibody VEGF antagonist immunoadhesin currently in pre-clinical development is a recombinant human soluble VEGF receptor fusion protein similar to VEGF-trap containing extracellular ligand-binding domains 3 and 4 from VEGFR2KDR, and domain 2 from VEGFR1/Flt-1; these domains are fused to a human IgG Fc protein fragment (Li et al., 2011 *Molecular Vision* 17:797-803). This antagonist binds to isoforms VEGF-A. VEGF-B and VEGF-C. The molecule is prepared using two different production processes resulting in different glycosylation patterns on the final proteins. The two glycoforms are referred to as KH902 (conbercept) and KH906. The fusion protein can have the amino acid sequence of SEQ ID NO: 98 and, like VEGF-trap, can be present as a dimer. This fusion protein and related molecules are further characterized in EP 1767546.

[0100] Anti-C5 Agents

[0101] In certain embodiments, the anti-C5 agent modulates a function of a C5 complement protein or a variant thereof. In some embodiments, the anti-C5 agent inhibits a function of C5 complement protein or a variant thereof. In one embodiment, the function inhibited by the anti-C5 agent is C5 complement protein cleavage.

[0102] A C5 complement protein variant as used herein encompasses a variant that performs substantially the same function as a C5 complement protein function. A C5 complement protein variant in some embodiments comprises substantially the same structure and in some embodiments comprises at least 80% sequence identity, in some embodiments at least 90% sequence identity, and in some embodiments at least 95% sequence identity to the amino acid sequence of the C5 complement protein comprising the amino acid sequence SEQ ID NO: 24.

[0103] In some embodiments, the anti-C5 agent is selected from a nucleic acid molecule, an aptamer, an antisense molecule, an RNAi molecule, a protein, a peptide, a cyclic peptide, an antibody or antibody fragment, a sugar, a polymer, or a small molecule. In certain embodiments, the anti-C5 agent is an anti-C5 agent described in PCT Patent Application Publication No. WO 2007/103549.

[0104] In particular embodiments, the anti-C5 agent is an anti-C5 aptamer. Aptamers are nucleic acid molecules having specific binding affinity to molecules through interactions other than classic Watson-Crick base pairing. Aptamers, like peptides generated by phage display or monoclonal antibodies ("mAbs"), are capable of specifically binding to selected targets and modulating the target's activity, e.g., through binding aptamers may block their target's ability to function. The aptamers may be unpegylated or pegylated. In particular embodiments, the aptamers may contain one or more 2' sugar modifications, such as 2'-O-alkyl (e.g., 2'-O-methyl or 2'-O-methoxyethyl) or 2'-fluoro modifications.

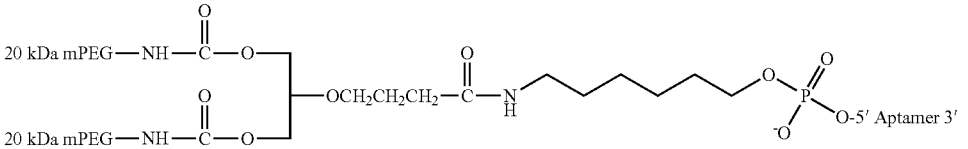
[0105] Illustrative C5 specific aptamers include the aptamers disclosed in PCT Publication No. WO 2007/103549, which is incorporated by reference in its entirety. Illustrative C5 specific aptamers include the aptamers ARC185 (SEQ ID NO: 25), ARC186 (SEQ ID NO: 26), ARC188 (SEQ ID NO: 27), ARC189 (SEQ ID NO: 28), ARC243 (SEQ ID NO: 29), ARC244 (SEQ ID NO: 30), ARC250 (SEQ ID NO: 31), ARC296 (SEQ ID NO: 32), ARC297 (SEQ ID NO: 33), ARC330 (SEQ ID NO: 34), ARC331(SEQ ID NO: 35), ARC332 (SEQ ID NO: 36), ARC333 (SEQ ID NO: 37), ARC334 (SEQ ID NO: 38), ARC411 (SEQ ID NO: 39), ARC412 (SEQ ID NO: 40), ARC413 (SEQ ID NO: 41), ARC414 (SEQ ID NO: 42), ARC415 (SEQ ID NO: 43), ARC416 (SEQ ID NO: 44), ARC417 (SEQ ID NO: 45), ARC418 (SEQ ID NO: 46), ARC419 (SEQ ID NO: 47), ARC420 (SEQ ID NO: 48), ARC421 (SEQ ID NO: 49), ARC422 (SEQ ID NO: 50), ARC423 (SEQ ID NO: 51), ARC424 (SEQ ID NO: 52), ARC425 (SEQ ID NO: 53), ARC426 (SEQ ID NO: 54), ARC427 (SEQ ID NO: 55), ARC428 (SEQ ID NO: 56), ARC429 (SEQ ID NO: 57), ARC430 (SEQ ID NO: 58), ARC431 (SEQ ID NO: 59), ARC432 (SEQ ID NO: 60), ARC433 (SEQ ID NO: 61), ARC434 (SEQ ID NO: 62), ARC435 (SEQ ID NO: 63), ARC436 (SEQ ID NO: 64), ARC437 (SEQ ID NO: 65), ARC438 (SEQ ID NO: 66). ARC439 (SEQ ID NO: 67), ARC440 (SEQ ID NO: 68), ARC457 (SEQ ID NO: 69), ARC458 (SEQ ID NO: 70), ARC459 (SEQ ID NO: 71). ARC473 (SEQ ID NO: 72), ARC522 (SEQ ID NO: 73), ARC523 (SEQ ID NO: 74), ARC524 (SEQ ID NO: 75), ARC525 (SEQ ID NO: 76), ARC532 (SEQ ID NO: 77), ARC543 (SEQ ID NO: 78), ARC544 (SEQ ID NO: 79), ARC550 (SEQ ID NO: 80), ARC551 (SEQ ID NO: 81), ARC552 (SEQ ID NO: 82), ARC553 (SEQ ID NO: 83), ARC554 (SEQ ID NO: 84), ARC657 (SEQ ID NO: 85), ARC658 (SEQ ID NO: 86), ARC672 (SEQ ID NO: 87), ARC706 (SEQ ID NO: 88), ARC913 (SEQ ID NO: 89), ARC874 (SEQ ID NO: 90), ARC954 (SEQ ID NO: 91), ARC1537 (SEQ ID NO: 92), ARC1730 (SEQ ID NO: 93), or a pharmaceutically acceptable salt thereof.

[0106] In some embodiments, the anti-C5 agent is an aptamer with SEQ ID NO: 94, 95, or 96.

[0107] In a particular embodiment, the anti-C5 agent is a C5 specific aptamer comprising the nucleotide sequence of SEQ ID NO: 26 conjugated to a polyethylene glycol moiety via a linker. In some embodiments, the polyethylene glycol moiety has a molecular weight greater than about 10 kDa, particularly a molecular weight of about 20 kDa, more particularly about 30 kDa and more particularly about 40 kDa. In some embodiments, the polyethylene glycol moiety is conjugated via a linker to the 5' end of the aptamer. In some

embodiments, the PEG conjugated to the 5' end of is a PEG of about 40 kDa molecular weight. In particular embodiments the about 40 kDa PEG is a branched PEG. In some embodiments the branched about 40 kDa PEG is 1,3-bis(mPEG-[about 20 kDa])-propyl-2-(4'-butamide). In other embodiments the branched about 40 kDa PEG is 2,3-bis(mPEG-[about 20 kDa])-propyl-1-carbamoyl.

[0108] In a particular embodiment, the C5 specific aptamer is a compound, ARC187, having the structure set forth below:

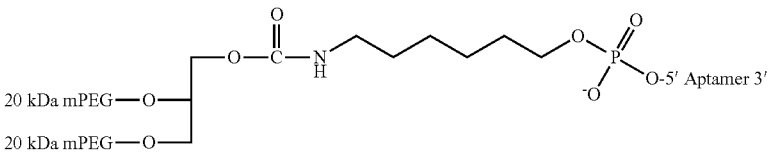


[0109] or a pharmaceutically acceptable salt thereof, where Aptamer=

[0110] fCmGfCfCGfCmGmGfUfCfUfCmAmG-mGfCGfCfUmGmAmGfUfCfUmGmAmGf UfUfUAfCf CfUmGfCmG-3T (SEQ ID NO: 26)

[0111] wherein fC and fU=2'-fluoro nucleotides, and mG and mA=2'-OMe nucleotides and all other nucleotides are 2'-OH and where 3T indicates an inverted deoxy thymidine. In some embodiments, each 20 kDa mPEG of the above structure has a molecular weight of about 20 kDa.

[0112] In another particular embodiment, the C5 specific aptamer is a compound, ARC1905, having the structure set forth below:



[0113] or a pharmaceutically acceptable salt thereof, where Aptamer=

[0114] fCmGfCfCGfCmGmGfUfCfUfCmAmG-mGfCGfCfUmGmAmGfUfCfUmGmAmGfUfUfUAfCf CfUmGfCmG-3T (SEQ ID NO: 26)

[0115] wherein fC and fU=2'-fluoro nucleotides, and mG and mA=2'-OMe nucleotides and all other nucleotides are 2'-OH and where 3T indicates and inverted deoxy thymidine. In some embodiments, each 20 kDa mPEG of the above structure has a molecular weight of about 20 kDa.

[0116] In other embodiments, the anti-C5 agent is an antisense oligonucleotide or ribozyme targeted to C5 that effects C5 inhibition by inhibiting protein translation from the messenger RNA or by targeting degradation of the corresponding C5 mRNA.

[0117] In still other embodiments, the anti-C5 agent is an anti-C5 RNA interference (RNAi) construct. Certain double stranded oligonucleotides useful to effect RNAi against C5 complement protein are less than 30 base pairs in length and may comprise about 25, 24, 23, 22, 21, 20, 19, 18 or 17 base pairs of ribonucleic acid and comprise a sequence with substantial sequence identity to the mRNA sequence of complement C5 protein, particularly human complement C5 protein. Optionally, the dsRNA oligonucleotides may include 3' overhang ends. Non-limiting illustrative 2-nucleotide 3' overhangs are composed of ribonucleotide residues of any type and may even be composed of 2'-deoxythymidine resides, which lowers the cost of RNA synthesis and may enhance nuclease resistance of siRNAs in the cell culture medium and within transfected cells (see Elbashi et al., (2001) Nature, 411: 494-8).

[0118] Other Agents for Treatment or Prevention of an Ophthalmological Disease or Disorder

[0119] In another embodiment, another agent useful for treating or preventing an ophthalmological disease or disorder is volociximab or a pharmaceutically acceptable salt thereof (Ramakrishnan et al. (2008) J Exp Ther Oncol. 5:273-86, which is hereby incorporated by reference in its entirety).

[0120] In some embodiments, a plurality of aptamers can be associated with a single Non-Immunogenic, High Molecular Weight Compound, such as Polyalkylene Glycol or PEG, or a Lipophilic Compound, such as a glycerolipid. The aptamers can all be to one target or to different targets. In embodiments where a compound comprises more than one PDGF aptamer, there can be an increase in avidity due to multiple binding interactions with a target, such as PDGF or VEGF. In yet further embodiments, a plurality of Polyalkylene Glycol, PEG, glycerol lipid molecules can be attached to each other. In these embodiments, one or more aptamers can be associated with each Polyalkylene Glycol, PEG, or glycerol lipid. This can result in an increase in avidity of each aptamer to its target. In addition, in embodiments where there are aptamers to PDGF or aptamers to PDGF and different Targets associated with Polyalkylene Glycol, PEG, or glycerol lipid, a drug can also be associated with, e.g., covalently bonded to, Polyalkylene Glycol, PEG, or glycerol lipid. Thus the compound would provide targeted delivery of the drug, with Polyalkylene Glycol, PEG, or glycerol lipid serving as a Linker, optionally, with one or more additional linkers.

[0121] Aptamers can be 5'-capped and/or 3'-capped with a 5'-5' inverted nucleoside cap structure at the 5' end and/or a 3'-3' inverted nucleoside cap structure at the 3' end. In several embodiments, Antagonist A, Antagonist B, Antagonist C. Antagonist D, pegaptanib, bevasiranib and Sirna-027 are 5' or 3' end-capped.

[0122] Methods for Treating or Preventing an Ophthalmological Disease or Disorder

[0123] The invention provides methods and compositions useful for treating or preventing ophthalmological diseases and disorders, including but not limited to any of the ophthalmological diseases and disorders described herein.

[0124] In some embodiments, the methods for treating or preventing an ophthalmological disease or disorder disclosed herein improve retinal attachment success, improve visual acuity, or stabilize vision. In some embodiments, the methods disclosed herein prevent or retard the rate of further vision loss in a subject.

[0125] In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof in combination with a VEGF antagonist or pharmaceutically acceptable salt thereof and/or an anti-C5 agent improves retinal attachment success, improves visual acuity, or stabilizes vision to a degree that is greater than administration of Antagonist A or another pharmaceutically acceptable salt thereof alone, the VEGF antagonist or pharmaceutically acceptable salt thereof alone, or the anti-C5 agent alone. In some embodiments, the administration of Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist or pharmaceutically acceptable salt thereof, and optionally, an anti-C5 agent, has a synergistic effect in treating or preventing an ophthalmological disease or disorder. For example, the administration of both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist or pharmaceutically acceptable salt thereof can improve retinal attachment success, improve visual acuity, or stabilize vision to a degree that is greater than an additive effect of administering both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist or pharmaceutically acceptable salt thereof. In some embodiments, administration of Antagonist A, alone or in combination with a VEGF antagonist and/or an anti-C5 agent, according to the methods described herein, e.g., treatment or dosing regimens, improves retinal attachment success, improves visual acuity, or stabilizes vision to a degree that is greater than administration of Antagonist A, alone or in combination with a VEGF antagonist and/or an anti-C5 agent, according to previously described methods.

[0126] In particular embodiments, any of the methods and compositions of the present invention are used to treat or prevent an ophthalmological disease or disorder in particular subjects. For example, in certain embodiments, subjects treated according to a method described herein are defined or identified based on their previous treatments for the disease or disorder, specific manifestations of their disease or disorder being treated, and/or other characteristics. In one embodiment, the subject has a defined phenotype or medical history.

[0127] Accordingly, any of the methods described herein may further comprise identifying the subject to be treated, such as by determining whether the subject was previously administered a VEGF antagonist for treating or preventing the disease or disorder or whether the subject had previously failed monotherapy with a VEGF antagonist, e.g., by inquiring of the subject or his health care provider, or by reviewing the subject's medical records.

[0128] In one embodiment, the subject was previously treated with a VEGF antagonist or anti-VEGF monotherapy for any ocular disease or disorder for which a VEGF antagonist is used, or for any of the ocular diseases or disorders described herein (e.g., wet-type AMD).

[0129] In particular embodiments, the methods and compositions described herein are useful for treating or preventing an ophthalmological disease or disorder in a subject who is anti-VEGF resistant, was previously administered or treated with anti-VEGF monotherapy, does not respond or had not responded favorably or adequately to anti-VEGF monotherapy, and/or failed monotherapy with a VEGF antagonist. In some embodiments, a subject who failed monotherapy is anti-VEGF resistant, has complement-mediated inflammation, and/or did not respond adequately to anti-VEGF monotherapy. In one embodiment, the subject who failed monotherapy with a VEGF antagonist is a subject who experienced a poor visual or anatomic outcome after treatment or administration with a VEGF antagonist. In one embodiment, the subject did not exhibit improved vision or exhibited reduced vision following anti-VEGF monotherapy.

[0130] In certain embodiments, the subject does not respond or had not responded favorably or adequately to anti-VEGF monotherapy, as determined by the subject's vision loss or by the subject's lack of significant vision gain following anti-VEGF monotherapy. In one embodiment, the subject's lack of significant vision gain following anti-VEGF monotherapy is determined by the subject's loss of ability to read one or more, in some embodiments three or more, and in some embodiments fifteen or more, letters of a standardized chart of vision testing, e.g., the Early Treatment for Diabetic Retinopathy Study Chart ("ETDRS chart"). In some embodiments, the vision testing is as described in Early Treatment Diabetic Retinopathy Study Research Group (ETDRS), Manual of Operations, Baltimore: ETDRS Coordinating Center, University of Maryland. Available from: National Technical Information Service, 5285 Port Royal Road, Springfield, Va. 22161; Accession No. PB85 223006/AS; Ferris et al., Am J Ophthalmol 94:91-96, 1982; or Example 4, as described herein. In some embodiments, the vision testing uses one or more charts available from http://www.nei.nih.gov/photo/keyword.asp?conditions=Eye+Charts&match=all, e.g., ETDRS visual acuity Chart 1, 2 and/or R.

[0131] In another embodiment, the subject's vision loss following anti-VEGF monotherapy is determined by the subject's loss of ability to read one or more, in some embodiments three or more, letters or lines of a standardized chart of vision testing, e.g., the ETDRS chart, from baseline. In one embodiment, the subject's lack of significant vision gain following anti-VEGF monotherapy is determined by the subject's inability to read an additional one or more, in some embodiment three or more, and in some embodiments fifteen or more, letters of a standardized chart of vision testing, e.g., the ETDRS chart, from baseline. In another embodiment, the subject's lack of significant vision gain following anti-VEGF monotherapy is determined by the subject's inability to read an additional one or more, in some embodiments three or more, lines of a standardized chart of visual testing, e.g., the ETDRS chart, from baseline. In some embodiments, a subject's vision loss or lack of significant vision gain is determined by the subject's visual loss or anatomic signs of poor treatment response, for example, persistent leakage, increased hemorrhage, persistent or increased retinal pigment epithelium (RPE) detachment, signs of neovascular activity, or growth of neovascularization or increased deposition of abnormal matrix or fibrosis. In particular embodiments, a subject's vision loss or lack of significant vision gain is determined at 12 weeks or at 24 weeks following the initiation of treatment.

[0132] In certain embodiments, the subject is anti-VEGF-resistant to a VEGF antagonist, e.g., anti-VEGF monotherapy. In one embodiment, a subject is anti-VEGF resistant if the subject was previously administered with a VEGF antagonist, e.g., anti-VEGF monotherapy, that did not result in the treatment or prevention of the ophthalmological disease or disorder; resulted in only a temporary treatment or prevention of the ophthalmological disease or disorder and rendered the subject in further need of treatment or prevention of the ophthalmological disease or disorder; or that resulted in the subject's visual decline and rendered the subject in further need of treatment or prevention of the ophthalmological disease or disorder.

[0133] In another embodiment, a subject is anti-VEGF resistant if the subject was previously treated or administered with an anti-VEGF treatment, e.g., anti-VEGF monotherapy, and failed to achieve any visual gain or experienced visual decline. In some embodiments, the subject did not respond adequately to anti-VEGF treatment. In one embodiment, the subject was administered the anti-VEGF treatment for one year or longer. In some such embodiments, the subject is in need of treatment for wet AMD.

[0134] Accordingly, the present invention provides methods for treating, preventing, or stabilizing wet AMD in a subject, such as a subject who has failed monotherapy with a VEGF antagonist (e.g., is anti-VEGF resistant, has complement-mediated inflammation, and/or did not respond adequately to anti-VEGF monotherapy). In particular embodiments, the methods comprise determining whether the subject was previously administered or treated with anti-VEGF monotherapy. In certain embodiments, anti-VEGF monotherapy means administration of only one or more VEGF antagonists. In certain embodiments, anti-VEGF monotherapy includes the optional administration of other drugs that are not agents specifically adapted for treating an ophthalmological disease or disorder, e.g, wet AMD.

[0135] In certain embodiments, the methods and compositions described herein are useful for treating or preventing an ophthalmological disease or disorder in a subject that is treatment-naïve. In some embodiments, the subject is treatment-naïve if the subject was not previously treated for the ophthalmological disease or disorder. In some embodiments, the subject is treatment-naïve if the subject was not previously administered or treated with a VEGF antagonist or anti-VEGF monotherapy ("anti-VEGF-treatment-naïve"). In particular embodiments, the methods further comprise determining whether the subject was previously treated for the ophthalmological disease or disorder or administered a VEGF antagonist or anti-VEGF monotherapy, e.g., by inquiring of the subject or his or her health care provider, or by reviewing the subject's medical records. In certain embodiments, anti-VEGF monotherapy means administration of only one or more VEGF antagonists. In certain embodiments, anti-VEGF monotherapy includes the optional administration of other drugs that are not agents specifically adapted for treating an ophthalmological disease or disorder, e.g, wet AMD. In some embodiments, the subject is treatment-naïve if the subject was not previously treated for AMD (e.g., wet AMD). In some embodiments, the subject is treatment-naïve if the subject was not previously treated, or has underwent no previous treatment for AMD (e.g., wet AMD) in either eye. In yet other embodiments, the subject is treatment-naïve if the subject was not previously treated, or has underwent no previous treatment, for AMD (e.g., wet AMD; e.g., in either eye) except for one or more oral supplements of vitamins and minerals. In some embodiments, the subject is treatment-naïve if the subject was not previously administered a therapeutic agent used for the treatment of AMD (e.g., wet AMD).

[0136] In certain embodiments, the subject has complement-mediated inflammation. In certain embodiments, the subject is anti-VEGF resistant and has complement-mediated inflammation. In certain embodiments, the complement-mediated inflammation is present in an eye of the subject. In certain embodiments, the complement-mediated inflammation results from previous administration with anti-VEGF monotherapy. In other embodiments, the subject has or has been diagnosed with complement-mediated inflammation. In still other embodiments, the subject did not respond adequately to anti-VEGF monotherapy and has or has been diagnosed with complement-mediated inflammation. In certain embodiments, complement-mediated inflammation is diagnosed in the subject using a genetic screening method. Such genetic screening methods are known to those of skill in the art and include, but are not limited to, screening for mutations in complement genes, such as complement factor H(CFH), CFI, CFHR5, and MCP, BF, and C2 genes.

[0137] In certain embodiments, the methods and compositions described herein are useful for treating or preventing an ophthalmological disease or disorder in a subject who is newly diagnosed with the ophthalmological disease or disorder. In some embodiments, the subject is newly diagnosed if the subject was not previously diagnosed for the ophthalmological disease or disorder. In some embodiments, the subject is newly diagnosed with age-related macular degeneration. In some embodiments, the subject is newly diagnosed with dry age-related macular degeneration. In some embodiments, the subject is newly diagnosed with wet-type AMD. In particular embodiments, the methods further comprise determining whether the subject was previously diagnosed for the ophthalmological disease or disorder, e.g., by inquiring of the subject or his or her health care provider, or by reviewing the subject's medical records.

[0138] In some embodiments of the invention, the methods and compositions described herein are useful for treating or preventing an ophthalmological disease or disorder that is a neovascular disorder. In other embodiments of the invention, the ophthalmological disease or disorder results in retinal edema. Illustrative ophthalmological diseases or disorders that can be treated or prevented are described herein.

[0139] Treatment or Prevention of Age-Related Macular Degeneration

[0140] In one embodiment, the ophthalmological disease or disorder treated or prevented by any of the methods or compositions described herein is age-related macular degeneration. Vision changes that can be associated with macular degeneration include distortions and/or blind spots (scotoma) detected using an Amsler grid, changes in dark adaptation (diagnostic of rod cell health), changes in color interpretation (diagnostic of cone cell health), or a decrease in visual acuity. Examples of age-related macular degeneration are normeovascular (also known as "dry") and neovascular (also known as "wet" or "exudative") macular degeneration.

[0141] In one embodiment, the dry age-related macular degeneration is associated with the formation of drusen. In one embodiment, treating or preventing dry macular degeneration encompasses treating or preventing an abnormality of the retinal pigment epithelium and/or underlying vasculature, known as choriocapilaries. Examples of abnormalities of the

retinal pigment epithelium include geographic atrophy, non-geographic atrophy, focal hypopigmentation, and focal hyperpigmentation. In another embodiment, treating or preventing wet age-related macular degeneration encompasses treating or preventing choroidal neovascularization or pigment epithelial detachment.

[0142] In one embodiment, the invention provides methods for treating or preventing wet age-related macular degeneration. Another aspect of the present invention is methods for treating, preventing, or inhibiting a choroidal neovascular complex in a subject, e.g., inhibiting the formation or growth of a choroidal neovascular complex.

[0143] In another aspect of the invention, the invention provides methods for treating or preventing choroidal neovascularization in a subject. In some embodiments, the choroidal neovascularization is subfoveal choroidal neovascularization. In some embodiments, the subfoveal choroidal neovascularization is due to age-related macular degeneration. In one embodiment, the subfoveal choroidal neovascularization is secondary to exudative type AMD. In other embodiments, the subfoveal choroidal neovascularization is present in subjects who have exudative type AMD, and in other embodiments, subfoveal choroidal neovascularization is present in subjects who do not have exudative type AMD. In some embodiments, the subfoveal choroidal neovascularization is secondary to inflammatory, traumatic, myopic, idiopathic or neoplastic afflictions of the macula.

[0144] In some embodiments, wet age-related macular degeneration is classified according to the appearance of its choroidal neovascularization (CNV), into classic, occult or mixed (classic and occult) CNV types, as determined by an angiography, known as fluorescence angiography. Classic, occult or mixed (classic and occult) CNV classification can be based on the time, intensity and level of definition of dye appearance, and leakage from the CNV, as assessed by the fluorescein angiography. In some embodiments, the subject has classic CNV (e.g., pure classic) or mixed CNV (predominantly or minimally classic CNV). In some embodiments, the subject has occult CNV (e.g., pure occult CNV).

[0145] The administration of Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist and/or anti-C5 agent can have a synergistic effect in treating or preventing classic CNV or occult CNV. For example, administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can improve visual acuity or stabilize vision to a degree that is greater than an additive effect of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist. In another example, administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can reduce CNV or inhibit the growth of CNV to a greater degree than administration of Antagonist A or another pharmaceutically acceptable salt thereof or the VEGF antagonist. In some embodiments, administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can reduce CNV in a shorter timeframe or with a lower dosage amount or frequency, as compared to the timeframe or dosage amount with administration of Antagonist A or another pharmaceutically acceptable salt thereof or the VEGF antagonist. In some embodiments, administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can reduce CNV or inhibit the growth of CNV to a greater degree than an additive effect of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist. In some embodiments, administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can reduce CNV in a shorter timeframe or with a lower dosage amount or frequency, as compared to an additive timeframe, dosage amount or frequency with administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist.

[0146] In one embodiment, the present invention provides methods for treating, preventing, or stabilizing non-exudative type ("dry type") AMD. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof, an anti-C5 agent, the combination of Antagonist A or another pharmaceutically acceptable salt thereof and an anti-C5 agent, or the combination of an anti-C5 agent and a VEGF antagonist is administered in an amount effective to maintain about the same level of drusen or reduce the level of drusen (e.g., amount, size, number, area and/or morphology) (e.g., size, number, area and/or morphology) as compared to the subject's drusen level prior to administration of Antagonist A or another pharmaceutically acceptable salt thereof, the anti-C5 agent, the combination of Antagonist A or another pharmaceutically acceptable salt thereof and the anti-C5 agent, or the combination of an anti-C5 agent and a VEGF antagonist, respectively. In a particular embodiment, the level of drusen is reduced by at least or about 5%, at least or about 10%, at least or about 20%, at least or about 30%, at least or about 40%, or at least or about 50%.

[0147] In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, an anti-C5 agent, the combination of Antagonist A or another pharmaceutically acceptable salt thereof and the anti-C5 agent, or the combination of the anti-C5 agent and a VEGF antagonist is administered in an amount effective to inhibit, slow, or prevent the progression of non-exudative type AMD to geographic atrophy (GA). GA is an advanced form of non-exudative type AMD. In other embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof and/or the anti-C5 agent or a pharmaceutically acceptable salt thereof is administered in an amount effective to reduce the growth or area of a GA lesion over time as compared to that in a subject not receiving Antagonist A or another pharmaceutically acceptable salt thereof and/or the anti-C5 agent. In other embodiments, the anti-C5 agent or a pharmaceutically acceptable salt thereof and a VEGF antagonist is administered in an amount effective to reduce the growth or area of a GA lesion over time as compared to that in a subject not receiving the anti-C5 agent and/or the VEGF antagonist. In a particular embodiment, the change in area or growth of the geographic atrophy lesion over time is reduced by at least or about 5%, at least or about 10%, at least or about 20%, at least or about 30%, at least or about 40%, or at least or about 50%. Methods of identifying and assessing the size of geographic lesions are known to those of skill in the art and include autofluorescence imaging and optical coherence tomography.

[0148] In particular embodiments, a subject in whom non-exudative AMD converts to exudative AMD, e.g., when new blood vessels invade the overlying retina, is treated. The present invention further provides methods for treating, preventing, or stabilizing drusen retinopathy secondary to complement-mediated immune disorders, including drusen retinopathy secondary to membranoproliferative glomerulonephritis type II disease. In some embodiments, Antagonist A

or another pharmaceutically acceptable salt thereof and/or an anti-C5 agent and/or a VEGF antagonist is administered in an amount effective to reduce retinal drusen in subjects having or having been diagnosed with membranoproliferative glomerulonephritis type II disease or exudative-type AMD as compared to the level of retinal drusen prior to administration of Antagonist A or another pharmaceutically acceptable salt thereof and/or an anti-C5 agent and/or a VEGF antagonist. In certain embodiments, the level of drusen is reduced by at least or about 5%, at least or about 10%, at least or about 20%, at least or about 30%, at least or about 40%, or at least or about 50%.

[0149] In one embodiment, the ophthalmological disease or disorder is polypoidal choroidal vasculopathy (PCV), a variant of wet AMD.

[0150] Treatment or Prevention of a Condition Associated with Choroidal Neovascularization

[0151] In one embodiment, the ophthalmological disease or disorder is a condition associated with choroidal neovascularization. Examples of conditions associated with choroidal neovascularization include a degenerative, inflammatory, traumatic or idiopathic condition. Treating or preventing a degenerative disorder associated with choroidal neovascularization also encompasses treating or preventing a heredodegerative disorder. Examples of heredodegenerative disorders include vitelliform macular dystrophy, fundus flavimaculatus and optic nerve head drusen. Examples of degenerative conditions associated with choroidal neovascularization include myopic degeneration or angioid streaks. In some embodiments, treating or preventing an inflammatory disorder associated with choroidal neovascularization encompasses treating or preventing ocular histoplasmosis syndrome, multifocal choroiditis, serpininous choroiditis, toxoplasmosis, toxocariasis, rubella, Vogt-Koyanagi-Harada syndrome, Behcet syndrome or sympathetic ophthalmia. In some embodiments, treating or preventing a traumatic disorder associated with choroidal neovascularization encompasses treating or preventing choroidal rupture or a traumatic condition caused by intense photocoagulation.

[0152] Treatment or Prevention of Proliferative Retinopathy

[0153] One particular aspect of the invention provides methods and compositions for treating or preventing proliferative vitreoretinopathy (PVR). In some embodiments, the PVR is a moderate form. In other embodiments, the PVR is a severe form. In some embodiments, the PVR is a recurrent form. In one embodiment, the subject with PVR also has or had retinal detachment, or the subject has PVR associated with retinal detachment, or PVR related scarring (e.g., scarring resulting from PVR, e.g., retinal scarring). In some embodiments, the PVR is characterized based on the configuration of the retina and the location of the scar tissue, such as in shown in Table 2 (See Lean J, et al. *Classification of proliferative vitreoretinopathy used in the silicone study. The Silicone study group. Ophthalmology* 1989; 96:765-771). Any of these categories or types of PVR can be treated or prevented according to the present invention.

TABLE 2

Classification of PVR

Type no.	Type of contraction	Location of PVR	Summary of Clinical Signs
1	Focal	Posterior	Starfold
2	Diffuse	Posterior	Confluent irregular retinal folds in posterior retina; remainder of retina drawn posteriorly; optic disc may not be visible
3	Sub-retinal	Posterior	"Napkin ring" around disc or "clothesline" elevation of retina
4	Circumferential	Anterior	Irregular retinal folds in the anterior retina; series of radial folds more posteriorly; peripheral retina within vitreous base stretched inward
5	Perpendicular	Anterior	Smooth circumferential fold of retina, at insertion of posterior hyaloid
6	Anterior	Anterior	Circumferential fold of retina at insertion of posterior hyaloid pulled forward; trough of peripheral retina anteriorly; ciliary processes stretched with possible hypotony; iris retracted

[0154] The present methods for treating PVR can further comprise administering another agent useful for treating PVR, such as a corticosteriod; antineoplastic drug, such as 5-fluorouracil; colchicine; retinoid; heparin; epidermal growth factor receptor (EGFR) inhibitor, such as gefitinib or erlotinib.

[0155] Another aspect of the invention is methods for treating or preventing a proliferative retinopathy, such as one related to PVR (e.g., treating or preventing an ocular manifestation of a proliferative retinopathy), such as proliferative diabetic retinopathy, sickle cell retinopathy, post traumatic retinopathy, hyperviscosity syndromes, Aortic arch syndromes, ocular ischemic syndromes, carotid-cavernous fistula, multiple sclerosis, retinal vasculitis, systemic lupus erythematosus, arteriolitis with SS-A autoantibody, acute multifocal hemorrhagic vasculitis, vasculitis resulting from infection, vasculitis resulting from Behçet's disease, sarcoidosis, coagulopathies, sickling hemoglobinopathies, AC and C-B thalassemia, small vessel hyalinosis, incontinentia pigmenti, Eales' disease, branch retinal artery or vein occlusion, frosted branch angiitis, idiopathic retinal vasculitis, aneurysms, neuroretinitis, retinal embolization, retinopathy of prematurity, Uveitis, pars planitis, acute retinal necrosis, birdshot retinochoroidopathy, long-standing retinal detachment, choroidal melanoma, radiation retinopathy, familial exudative vitreoretinopathy, inherited retinal venous beading, retinoschisis, retinitis pigmentosa, or autosomal dominant vitreoretinochoroidopathy.

[0156] Another aspect of the invention is methods for treating or preventing a disease or condition that is a cause that results in proliferative retinopathy or PVR. In one embodiment, post-retinal detachment (e.g., that causes or results in PVR) is treated or prevented. In another embodiment, proliferative diabetic retinopathy (e.g., that causes or results in PVR) or sickle-cell retinopathy (e.g., that causes or results in PVR), as well as scarring caused by one or more of these disorders is treated or prevented.

[0157] Treatment or Prevention of Glaucoma

[0158] In one embodiment, the opthalmological disease or disorder is glaucoma. In one embodiment the glaucoma is open angle glaucoma, primary open angle glaucoma, secondary open angle glaucoma, closed angle glaucoma, glaucoma

that is associated with diabetes, glaucoma that is associated with diabetic retinopathy, angle closure glaucoma, narrow angle glaucoma or acute glaucoma.

[0159] Treatment or Prevention of a Neoplasm

[0160] In one embodiment, the ophthalmological disease or disorder is a neoplasm. Examples of neoplams include an eyelid tumor, a conjunctival tumor, a choroidal tumor, an iris tumor, an optic nerve tumor, a retinal tumor, an infiltrative intraocular tumor or an orbital tumor. Examples of an eyelid tumor include basal cell carcinoma, squamous carcinoma, sebaceous carcinoma, malignant melanoma, capillary hemangioma, hydrocystoma, nevus or seborrheic keratosis. Examples of a conjunctival tumor include conjunctival Kaposi's sarcoma, squamous carcinoma, intraepithelial neoplasia of the conjunctiva, epibular dermoid, lymphoma of the conjunctiva, melanoma, pingueculum, or pterygium. Examples of a choroidal tumor include choroidal nevus, choroidal hemangioma, metastatic choroidal tumor, choroidal osteoma, choroidal melanoma, ciliary body melanoma or nevus of Ota. Examples of an iris tumor include anterior uveal metastasis, iris cyst, iris melanocytoma, iris melanoma, or pearl cyst of the iris. Examples of an optic nerve tumor include optic nerve melanocytoma, optic nerve sheath meningioma, choroidal melanoma affecting the optic nerve, or circumpapillary metastasis with optic neuropathy. Examples of a retinal tumor include retinal pigment epithelial (RPE) hypertrophy, RPE adenoma, RPE carcinoma, retinoblastoma, or hamartoma of the RPE. In some embodiments, the present invention provides methods for inhibiting retinal pigment epithelium (RPE) or glial cells, such as inhibiting the migration of RPE or glial cells. Examples of an infiltrative intraocular tumor include chronic lymphocytic leukemia, infiltrative choroidopathy, or intraocular lymphoma. Examples of an orbital tumor include adenoid cystic carcinoma of the lacrimal gland, cavernous hemangioma of the orbit, lymphangioma of the orbit, orbital mucocele, orbital pseudotumor, orbital rhabdomyosarcoma, periocular hemangioma of childhood, or sclerosing orbital psuedotumor.

[0161] Another aspect of the invention is methods for treating or preventing von Hippel-Lindau (VHL) disease (e.g., treating or preventing visual loss associated VHL disease). In some embodiments, VHL disease is characterized by tumors. The tumors may be malignant or benign. In another embodiment, a benign or malignant tumor in the eye (e.g., ocular tumor) or a cyst (e.g., an ocular cyst), associated with VHL is treated or prevented. In some embodiments, the tumors are hemangioblastomas. In some embodiments, the tumors are von Hippel angioma or retinal capillary hemangiomas (e.g., juxtapapillary hemangioma).

[0162] In some embodiments, the subject with VHL disease has a deficiency of the protein "pVHL."

[0163] In some embodiments, the VHL disease is severe (e.g., a subject with severe VHL disease has a lesion that cannot be effectively treated with a non-pharmacologic modality (e.g., laser or or cryotherapy), for example, as the lesion resides over or adjacent to a significant neural structure (e.g., optic nerve, macula, papillomacular bundle) that can be damaged with laser or cryotherapy).

[0164] In some embodiments, the methods for treating or preventing VHL disease comprise treating an ocular or non-ocular manifestation (e.g., benign or malignant neoplasm or cyst of the kidney, adrenal gland, pancreas, brain, spinal cord, inner ear, epididymis, or broad ligament) of VHL.

[0165] In some embodiments, the subjected being treated has a family history of VHL disease or one or more of retinal capillary hemangioma (RCH), spinal or cerebellar hemangioblastoma, pheochromocytoma, multiple pancreatic cysts, epididymal or broad ligament cystadenoma, multiple renal cysts, and renal cell carcinoma. In some embodiments, the subject has one or more RCH, spinal and cerebellar hemangioblastoma, pheochromocytoma, multiple pancreatic cysts, epididymal or broad ligament cystadenomas, multiple renal cysts, or renal cell carcinoma before the age of 60 years. In some embodiments, the subject has two or more hemangioblastomas of the retina or brain or a single hemangioblastoma in association with a visceral manifestation, such as kidney or pancreatic cysts; renal cell carcinoma; adrenal or extra-adrenal pheochromocytomas; endolymphatic sac tumors; papillary cystadenomas of the epididymis or broad ligament; or neuroendocrine tumors of the pancreas. In some embodiments, the subject has a disease-causing germline mutation in the VHL gene.

[0166] In some embodiments, the subject has RCH that exhibit activity, such as associated intra- or sub-retinal exudation or lipid deposition (which may reflect ongoing vascular incompetence and is not reflective of residual changes following previous treatment or secondary to coexistent retinal traction); increased size of the tumor compared to a previous time point as assessed by fundus photography or fluorescein angiography (FA); associated intra-, sub-, or pre-retinal hemorrhage not secondary to previous treatment, as assessed by fundus photography or FA; appearance of new feeder vessels or greater dilation or tortuosity of existing feeder vessels compared to a previous time point; and/or vitreous cell or haze indicative of vitreous exudation, in the absence of other ocular features potentially responsible for such findings. In some embodiments, the subject has RCH that is not readily treatable using cryotherapy or thermal laser because of its size, posterior location, poor previous response to conventional therapy, or other factors.

[0167] In some embodiments, methods or compositions of the invention are used to treat or prevent a complication of VHL, visual dysfunction (e.g., from VHL), or a fibrous complication of VHL (e.g., fibrous meningioma). In certain embodiments, the methods or compositions of the present invention are used to treat a manifestation of VHL as vascular proliferation that comprises fine, superficial, juxtapapillary vessels that are often associated with fibrovascular proliferation and epiretinal membrane formation.

[0168] Treatment or Prevention of Scarring or Fibrosis

[0169] Another aspect the invention provides methods for treating, inhibiting or preventing scarring or fibrosis (e.g., scarring or fibrosis is under the macular region of the retina). In some embodiments, the scarring is a fibrovascular scar (e.g., in the retina). In some embodiments, the fibrosis is hepatic, pulmonary or renal fibrosis. In some embodiments, the fibrosis is ocular fibrosis. In some embodiments, the fibrosis is sub-retinal fibrosis (e.g., associated with neovascular AMD). In some embodiments, the sub-retinal fibrosis is not associated with neovascular AMD. In some embodiments, the fibrosis is subfoveal fibrosis. In some embodiments, the subfoveal fibrosis is with retinal atrophy. In some embodiments, subfoveal fibrosis or sub-retinal fibrosis develops after administration of a VEGF antagonist, e.g., anti-VEGF monotherapy.

[0170] In some embodiments, the scarring results from glaucoma surgery, or follows glaucoma surgery, such as tra-

beculectomy, filtering surgery (such as partial thickness filtering surgery), glaucoma filtering procedures, minimally invasive glaucoma surgery, glaucoma valve implant surgery, glaucoma seton surgery, glaucoma tube shunt placement, glaucoma stent placement, or combined cataract and glaucoma surgery. In some embodiments, the methods of the present invention are useful to treat or prevent scarring relating to or resulting from glaucoma surgery (e.g., that can result in scar related proliferation). In some embodiments, the scarring is sub-retinal scarring. In some embodiments, the scarring is sub-retinal scarring that occurs following choroidal neovascular regression.

[0171] In particular embodiments, methods for treating, inhibiting or preventing sub-retinal fibrosis (e.g., reducing the formation of sub-retinal fibrosis) comprise administering to a subject in need thereof an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist. In some embodiments, the subject has or is diagnosed with AMD (e.g., wet AMD). In some embodiments, the subject has or is diagnosed with advanced wet AMD.

[0172] Treatment or Prevention of Other Ophthalmological Diseases and Disorders

[0173] In certain embodiments, the ophthalmological disease or disorder is a cataract (e.g., age-related cataract), diabetic macula edema, macular telangiectasia (e.g., type 1 or 2 macular telangiectasia), atrophic macular degeneration, chorioretinopathy (e.g., central serous chorioretinopathy), retinal inflammatory vasculopathy, pathological retinal angiogenesis, age-related maculopathy, retinoblastoma, Pseudoxanthoma elasticum, a vitreoretinal disease, choroidal sub-retinal neovascularization, central serous chorioretinopathy, ischemic retinopathy, hypertensive retinopathy or diabetic retinopathy (e.g., nonproliferative or proliferative diabetic retinopathy, such as macular edema or macular ischemia), retinopathy of prematurity (e.g., associated with abnormal growth of blood vessels in the vascular bed supporting the developing retina), venous occlusive disease (e.g., a retinal vein occlusion, branch retinal vein occlusion or central retinal vein occlusion), arterial occlusive disease (e.g., branch retinal artery occlusion (BRAO), central retinal artery occlusion or ocular ischemic syndrome), central serous chorioretinopathy (CSC), cystoid macular edema (CME) (e.g., affecting the central retina or macula, or after cataract surgery), retinal telangiectasia (e.g., characterized by dilation and tortuosity of retinal vessels and formation of multiple aneurysms, idiopathic JXT, Leber's miliary aneurysms, or Coats' disease), arterial macroaneurysm, retinal angiomatosis, radiation-induced retinopathy (RIRP), or rubeosis iridis (e.g., associated with the formation of neovascular glaucoma, diabetic retinopathy, central retinal vein occlusion, ocular ischemic syndrome, or chronic retinal detachment).

[0174] In other embodiments, the ophthalmological disease or disorder is sickle cell disease (SCD), anemia, or sickle cell retinopathy (e.g., non-neovascular or non-proliferative ocular manifestations). In some embodiments, vaso-occlusive phenomena or hemolysis associated with SCD is treated or prevented. In some embodiments, ocular manifestations of SCD include vascular occlusions in the conjunctiva, iris, retina, or choroid. Non-neovascular or non-proliferative ocular manifestations can include conjunctival vascular occlusions which transform smooth vessels into comma-shaped fragments, iris atrophy, retinal "salmon patch" hemorrhages, retinal pigmentary changes and other abnormalities of the retinal vasculature, macula, choroid, and optic disc. In some embodiments, neovascularization or the proliferative ocular manifestation involves the growth of abnormal vascular fronds which can lead to vitreous hemorrhage, retinal detachment, epiretinal membranes, resulting in vision loss. In some embodiments, the methods further comprise performing another treatment, such as diathermy, cryotherapy, laser photocoagulation or surgery (e.g., vitrectomy).

[0175] In one embodiment, the ophthalmological disease or disorder is a condition associated with peripheral retinal neovascularization. Examples of conditions associated with peripheral retinal neovascularization include ischemic vascular disease, inflammatory disease with possible ischemia, incontinentia pigmenti, retinitis pigmentosa, retinoschisis or chronic retinal detachment.

[0176] Examples of ischemic vascular disease include proliferative diabetic retinopathy, branch retinal vein occlusion, branch retinal arteriolar occlusion, carotid cavernous fistula, sickling hemoglobinopathy, non-sickling hemoglobinopathy, IRVAN syndrome (retinal vasculitic disorder characterized by idiopathic retinal vasculitis, an aneurysm, and neuroretinitis), retinal embolization, retinopathy of prematurity, familial exudative vitreoretinopathy, hyperviscosity syndrome, aortic arch syndrome or Eales disease. Examples of sickling hemoglobinopathy include SS hemoglobinopathy and SC hemoglobinopathy. Examples of non-sickling hemoglobinopathy include AC hemoglobinopathy and AS hemoglobinopathy. Examples of hyperviscosity syndrome include leukemia, Waldenstrom macroglobulinemia, multiple myeloma, polycythemia or myeloproliferative disorder.

[0177] In some embodiments, treating or preventing an inflammatory disease with possible ischemia encompasses treating or preventing retinal vasculitis associated with systemic disease, retinal vasculitis associated with an infectious agent, uveitis or birdshot retinopathy. Examples of systemic diseases include systemic lupus erythematosis, Behcet's disease, inflammatory bowel disease, sarcoidosis, multiple sclerosis, Wegener's granulomatosis and polyarteritis nodosa. Examples of infectious agents include a bacterial agent that is the causative agent for syphilis, tuberculosis, Lyme disease or cat-scratch disease, a virus such as herpesvirus, or a parasite such as *Toxocara canis* or *Toxoplasma gondii*. Examples of uveitis include pars planitis or Fuchs uveitis syndrome.

[0178] Compositions for Therapeutic or Prophylactic Administration

[0179] Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonists, or anti-C5 agents can be administered as a component of a composition that further comprises a pharmaceutically acceptable carrier or vehicle, e.g., a pharmaceutical composition. In certain embodiments, each therapeutic agent is administered to the subject in a separate composition. However, in other embodiments, two or more therapeutic agents may be administered to the subject in the same composition. In one embodiment, a composition of the invention comprises an effective amount of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist, and/or an anti-C5 agent and a pharmaceutically acceptable carrier or vehicle. In another embodiment, a composition comprising Antagonist A or another pharmaceutically acceptable salt thereof and another composition comprising a VEGF antagonist are administered. In some embodiments, another composition comprising an anti-C5 agent is administered. In some embodiments, a composition comprising Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist is administered. In

some embodiments, another composition comprising an anti-C5 agent is also administered.

[0180] Administration of each antagonist may be by any suitable means that results in an amount of Antagonist A or another pharmaceutically acceptable salt thereof, VEG F antagonist, and/or anti-C5 agent that is effective for the treatment or prevention of an ophthalmological disease or disorder. Each antagonist, for example, can be admixed with a suitable carrier substance, and is generally present in an amount of 1-95% by weight of the total weight of the composition. The composition may be provided in a dosage form that is suitable for ophthalmic, oral, parenteral (e.g., intravenous, intramuscular, subcutaneous), rectal, transdermal, nasal, or inhalant administration. In one embodiment, the composition is in a form that is suitable for injection directly in the eye. The composition may be in form of, e.g., tablets, capsules, pills, powders, granulates, suspensions, emulsions, solutions, gels including hydrogels, pastes, ointments, creams, plasters, delivery devices, suppositories, enemas, injectables, implants, sprays, drops or aerosols. The compositions comprising one or more antagonists can be formulated according to conventional pharmaceutical practice (see, e.g., Remington: *The Science and Practice of Pharmacy*, (20th ed.) ed. A. R. Gennaro, 2000, Lippincott Williams & Wilkins, Philadelphia, Pa. and *Encyclopedia of Pharmaceutical Technology*, eds., J. Swarbrick and J. C. Boylan, 1988-2002, Marcel Dekker, New York).

[0181] The compositions are, in one useful aspect, administered parenterally (e.g., by intramuscular, intraperitoneal, intravenous, intraocular, intravitreal, retro-bulbar, subconjunctival, subtenon or subcutaneous injection or implant) or systemically. Formulations for parenteral or systemic administration include sterile aqueous or non-aqueous solutions, suspensions, or emulsions. A variety of aqueous carriers can be used, e.g., water, buffered water, saline, and the like. Examples of other suitable vehicles include polypropylene glycol, polyethylene glycol, vegetable oils, gelatin, hydrogels, hydrogenated naphalenes, and injectable organic esters, such as ethyl oleate. Such formulations may also contain auxiliary substances, such as preserving, wetting, buffering, emulsifying, and/or dispersing agents. Biocompatible, biodegradable lactide polymer, lactide/glycolide copolymer, or polyoxyethylene-polyoxypropylene copolymers may be used to control the release of the active ingredients.

[0182] Alternatively, the compositions can be administered by oral ingestion. Compositions intended for oral use can be prepared in solid or liquid forms, according to any method known to the art for the manufacture of pharmaceutical compositions.

[0183] Solid dosage forms for oral administration include capsules, tablets, pills, powders, and granules. Generally, these pharmaceutical preparations contain active ingredients admixed with non-toxic pharmaceutically acceptable excipients. These include, for example, inert diluents, such as calcium carbonate, sodium carbonate, lactose, sucrose, glucose, mannitol, cellulose, starch, calcium phosphate, sodium phosphate, kaolin and the like. Binding agents, buffering agents, and/or lubricating agents (e.g., magnesium stearate) may also be used. Tablets and pills can additionally be prepared with enteric coatings. The compositions may optionally contain sweetening, flavoring, coloring, perfuming, and preserving agents in order to provide a more palatable preparation.

[0184] Compositions useful for ophthalmic use include tablets comprising one or more antagonists in admixture with a pharmaceutically acceptable excipient. These excipients may be, for example, inert diluents or fillers (e.g., sucrose and sorbitol), lubricating agents, glidants, and antiadhesives (e.g., magnesium stearate, zinc stearate, stearic acid, silicas, hydrogenated vegetable oils, or talc).

[0185] The antagonists of the present invention may be admixed in a tablet or other vehicle, or may be partitioned. In one example, one antagonist is contained on the inside of the tablet, and the other antagonist is on the outside, such that a substantial portion of the other antagonist is released prior to the release of the contained antagonist. If desired, antagonists in a tablet form may be administered using a drug delivery device (see below).

[0186] For example, compositions of the present invention may be administered intraocularly by intravitreal injection into the eye as well as by subconjunctival and subtenon injections. Other routes of administration include transcleral, retrobulbar, intraperitoneal, intramuscular, and intravenous. Alternatively, compositions can be administered using a drug delivery device or an intraocular implant (see below).

[0187] In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof or VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008) is administered intravitreally with a 30-gauge or 27-gauge needle. In some embodiments, a 0.5 inch needle is used. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally with a 30-gauge 0.5 inch needle and a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008) is administered intravitreally with a 27-gauge needle. In some embodiments, 50 μL (1.5 mg in 0.05 mL) of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally with a 30-gauge 0.5 inch needle and 50 μL of a VEGF antagonist (e.g., 0.5 mg of ranibizumab, 1.25 mg of bevacizuamb, or 2.0 mg of aflibercept) is administered intravitreally with a 27-gauge needle.

[0188] Liquid dosage forms for oral administration can include pharmaceutically acceptable emulsions, solutions, suspensions, syrups, and soft gelatin capsules. These forms can contain inert diluents commonly used in the art, such as water or an oil medium, and can also include adjuvants, such as wetting agents, emulsifying agents, and suspending agents.

[0189] In some instances, the compositions can also be administered topically, for example, by patch or by direct application to a region, such as the epidermis or the eye, susceptible to or affected by a neovascular disorder, or by iontophoresis.

[0190] In one embodiment, the compositions can comprise one or more pharmaceutically acceptable excipients. In one embodiment, excipients for compositions that comprise an antagonist include, but are not limited to, buffering agents, nonionic surfactants, preservatives, tonicity agents, sugars, amino acids, and pH-adjusting agents. Suitable buffering agents include, but are not limited to, monobasic sodium phosphate, dibasic sodium phosphate, and sodium acetate. Suitable nonionic surfactants include, but are not limited to, polyoxyethylene sorbitan fatty acid esters such as polysorbate 20 and polysorbate 80. Suitable preservatives include, but are not limited to, benzyl alcohol. Suitable tonicity agents include, but are not limited to sodium chloride, mannitol, and sorbitol. Suitable sugars include, but are not limited to, α,α-trehalose. Suitable amino acids include, but are not limited to

glycine and histidine. Suitable pH-adjusting agents include, but are not limited to, hydrochloric acid, acetic acid, and sodium hydroxide. In one embodiment, the pH-adjusting agent or agents are present in an amount effective to provide a pH of about 3 to about 8, about 4 to about 7, about 5 to about 6, about 6 to about 7, or about 7 to about 7.5. In one embodiment, the compositions do not comprise a preservative. In another embodiment, the composition does not comprise an antimicrobial agent. In another embodiment, the composition does not comprise a bacteriostat. Suitable excipients for a VEGF antagonist also include those described in U.S. Pat. No. 7,365,166, the contents of which are herein incorporated by reference in their entirety.

[0191] In one embodiment, the composition is in the form of an aqueous solution that is suitable for injection. In one embodiment, a composition is in the form of an aqueous solution that is suitable for injection. In one embodiment, a composition comprises Antagonist A or another pharmaceutically acceptable salt thereof, a buffering agent, a pH-adjusting agent, and water for injection. In another embodiment, a composition comprises Antagonist A or another pharmaceutically acceptable salt thereof, monobasic sodium phosphate, dibasic sodium phosphate, sodium chloride, hydrochloride acid, and sodium hydroxide.

[0192] In one embodiment, the composition comprises a VEGF antagonist, a buffering agent, a sugar, a nonionic surfactant, and water for injection. In another embodiment, the composition comprises a VEGF antagonist, monobasic sodium phosphate, dibasic sodium phosphate, α,α-trehalose dehydrate, and polysorbate 20. In one embodiment, the composition comprises a VEGF antagonist, a buffering agent, a pH-adjusting agent, a tonicity agent, and water that is suitable for injection. In another embodiment, the composition comprises a VEGF antagonist, monobasic sodium phosphate, dibasic sodium phosphate, sodium chloride, hydrochloric acid, and sodium hydroxide. In one embodiment, the VEGF antagonist is a pegylated anti-VEGF aptamer, e.g., pegaptanib sodium

[0193] In another embodiment, the VEGF antagonist is ranibizumab, bevacizumab, aflibercept or ESBA1008. This invention provides the pharmaceutically acceptable salts of the antagonists. An antagonist of the present invention can possess a sufficiently basic functional group, which can react with any of a number of inorganic and organic acids, to form a pharmaceutically acceptable salt. A pharmaceutically-acceptable acid addition salt is formed from a pharmaceutically-acceptable acid, as is well known in the art. Such salts include the pharmaceutically acceptable salts listed in Journal of Pharmaceutical Science, 66, 2-19 (1977) and The Handbook of Pharmaceutical Salts; Properties, Selection, and Use. P. H. Stahl and C. G. Wermuth (ED.s), Verlag, Zurich (Switzerland) 2002, which are hereby incorporated by reference in their entirety.

[0194] Examples of a pharmaceutically acceptable salts include sulfate, citrate, acetate, oxalate, chloride, bromide, iodide, nitrate, bisulfate, phosphate, acid phosphate, isonicotinate, lactate, salicylate, acid citrate, tartrate, oleate, tannate, pantothenate, bitartrate, ascorbate, succinate, maleate, gentisinate, fumarate, gluconate, glucaronate, saccharate, formate, benzoate, glutamate, methanesulfonate, ethanesulfonate, benzenesulfonate, p-toluenesulfonate, camphorsulfonate, pamoate, phenylacetate, trifluoroacetate, acrylate, chlorobenzoate, dinitrobenzoate, hydroxybenzoate, methoxybenzoate, methylbenzoate, o-acetoxybenzoate, naphthalene-2-benzoate, isobutyrate, phenylbutyrate, α-hydroxybutyrate, butyne-1,4-dicarboxylate, hexyne-1,4-dicarboxylate, caprate, caprylate, cinnamate, glycollate, heptanoate, hippurate, malate, hydroxymaleate, malonate, mandelate, mesylate, nicotinate, phthalate, teraphthalate, propiolate, propionate, phenylpropionate, sebacate, suberate, p-bromobenzenesulfonate, chlorobenzenesulfonate, ethylsulfonate, 2-hydroxyethylsulfonate, methylsulfonate, naphthalene-1-sulfonate, naphthalene-2-sulfonate, naphthalene-1,5-sulfonate, xylenesulfonate, and tartarate salts. The term "pharmaceutically acceptable salt" includes a hydrate of a compound of the invention and also refers to a salt of an antagonist of the present invention having an acidic functional group, such as a carboxylic acid functional group or a hydrogen phosphate functional group, and a base. Suitable bases include, but are not limited to, hydroxides of alkali metals such as sodium, potassium, and lithium; hydroxides of alkaline earth metal such as calcium and magnesium; hydroxides of other metals, such as aluminum and zinc; ammonia, and organic amines, such as unsubstituted or hydroxy-substituted mono-, di-, or tri-alkylamines, dicyclohexylamine; tributyl amine; pyridine; N-methyl, N-ethylamine; diethylamine; triethylamine; mono-, bis-, or tris-(2-OH-lower alkylamines), such as mono-; bis-, or tris-(2-hydroxyethyl)amine, 2-hydroxy-tert-butylamine, or tris-(hydroxymethyl)methylamine, N,N-di-lower alkyl-N-(hydroxyl-lower alkyl)-amines, such as N,N-dimethyl-N-(2-hydroxyethyl)amine or tri-(2-hydroxyethyl)amine; N-methyl-D-glucamine; and amino acids such as arginine, lysine, and the like. In one embodiment, the pharmaceutically acceptable salt is a sodium salt. In another embodiment, the pharmaceutically acceptable salt is a persodium salt.

[0195] The present invention further provides comprising Antagonist A or another pharmaceutically acceptable salt thereof. In one embodiment, the present compositions comprise about 30.0 mg of Antagonist A or another pharmaceutically acceptable salt thereof, about 0.3 mg of monobasic sodium phosphate monohydrate, about 2.1 mg of dibasic sodium phosphate heptahydrate and about 9.0 mg of sodium chloride per about 1 mL. In some embodiments, hydrochloric acid and/or sodium hydroxide are present as needed to adjust the pH of the composition. In some embodiments, the pH is about pH 5.5 to about pH 7.5 or about pH 6.0.

[0196] In some embodiments, the compositions comprise about 3% (w/v) of Antagonist A or another pharmaceutically acceptable salt thereof, about 0.03% (w/v) of monobasic sodium phosphate monohydrate, about 0.2% (w/v) of dibasic sodium phosphate heptahydrate, about 0.9% (w/v) of sodium chloride and about 95.9% (w/v) of water. In some embodiments, hydrochloric acid and/or sodium hydroxide are present as needed to adjust the pH of the composition. In some embodiments, the pH is about pH 5.5 to about pH 7.5 or about pH 6.0.

[0197] In certain embodiments, the concentration of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and/or an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof) in a composition is about 0.002 mg/mL to about 50 mg/mL. In some embodiments, the concentration of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and/or an anti-C5 agent (e.g., ARC1905 or a pharmaceutically accept-

able salt thereof) in a composition is less than or about 100 mg/mL, less than about 50 mg/mL, less than about 40 mg/mL, less than about 30 mg/mL, less than about 25 mg/mL, less than about 20 mg/mL, less than about 15 mg/mL, less than about 10 mg/mL, or less than about 5 mg/mL. In certain embodiments, the concentration of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and/or an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof) in a composition is about 0.3 mg/mL to about 100 mg/mL, about 0.3 mg/mL to about 50 mg/mL, about 0.3 mg/mL to about 40 mg/mL, about 0.3 mg/mL to about 30 mg/mL, about 0.3 to about 25 mg/mL, about 0.3 mg/mL to about 20 mg/mL, about 0.3 mg/mL to about 15 mg/mL, about 0.3 mg/mL to about 10 mg/mL, about 1 mg/mL to about 100 mg/mL, about 1 mg/mL to about 50 mg/mL, about 1 mg/mL to about 40 mg/mL, about 1 mg/mL to about 30 mg/mL, about 1 mg/mL to about 25 mg/mL, about 1 mg/mL to about 20 mg/mL, about 1 mg/mL to about 15 mg/mL, about 1 mg/mL to about 10 mg/mL, about 1 mg/mL to about 5 mg/mL, about 5 mg/mL to about 100 mg/mL, or about 5 mg/mL to about 50 mg/mL.

[0198] In certain embodiments, methods of the invention comprise administering Antagonist A and optionally one or both of a VEGF antagonist and an anti-C5 agent as a component of a pharmaceutical composition. In one embodiment, the present invention provides compositions comprising an effective amount of: (a) Antagonist A or another pharmaceutically acceptable salt thereof; and (b) a VEGF antagonist or a pharmaceutically acceptable salt thereof. In certain embodiments, the compositions further comprise an effective amount of an anti-C5 agent or a pharmaceutically acceptable salt thereof. In some embodiments, the compositions stabilize one or more of the Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist, and the anti-C5 agent. In certain embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist and/or the anti-C5 agent does not adversely affect the activity of the other active agent(s) present in the composition. In particular embodiments, at least about 90% of one or more of the active agents in the composition, e.g., Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist, or anti-C5 agent, is chemically stable when the composition is stored at a temperature of from about 2.0° C. to about 8.0° C. for at least about twelve weeks.

[0199] In particular embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist or the anti-C5 agent is chemically stable when it shows no sign of decomposition or modification resulting in formation of a new chemical entity. In particular embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist or the anti-C5 agent is chemically stable when at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, a least about 95%, or at least about 99% of Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist or the anti-C5 agent shows no sign of decomposition or modification resulting in formation of a new chemical entity, e.g., when stored at a temperature of from about 2.0° C. to about 8.0° C. for at least about twelve weeks.

[0200] In certain embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof does not adversely affect the activity of the VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008) or the ARC1905 or a pharmaceutically acceptable salt thereof. In certain embodiments, the VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008) does not adversely affect the activity of the Antagonist A or another pharmaceutically acceptable salt thereof, or ARC1905 or a pharmaceutically acceptable salt thereof. In certain embodiments, ARC1905 or a pharmaceutically acceptable salt thereof does not adversely affect the activity of the Antagonist A or another pharmaceutically acceptable salt thereof, or the VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008).

[0201] In particular embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof; and ranibizumab, bevacizumab, aflibercept, pegaptanib sodium or ESBA1008, or a pharmaceutically acceptable salt thereof, and the compositions are physically or chemically stable with respect to both active agents at a particular pH or suitable for parenteral administration. In particular embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof; ranibizumab, bevacizumab, aflibercept, pegaptanib sodium or ESBA1008 or a pharmaceutically acceptable salt thereof; and ARC1905 or a pharmaceutically acceptable salt thereof, and the compositions are physically or chemically stable with respect to all active agents at a particular pH or suitable for parenteral administration. In particular embodiments, a composition is physically stable if at least about 50%, at least about 60%, at least about 70%, at least about 80%, at least about 90%, at least about 95%, or at least about 99% of all active agents, i.e., the Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist, and the anti-C5 agent (when present) present in the composition show no sign of aggregation, precipitation or denaturation upon visual examination of color or clarity, or as measured by UV light scattering or by size exclusion chromatography (SEC) or differential scanning calorimetry (DSC).

[0202] In particular embodiments, the compositions of the invention are considered physically stable if after storage the average number of particles detected does not exceed about 50 particles/mL, where the particles have a diameter >about 10 μm and does not exceed 5 particles/mL, where the particles have a diameter >25 μm, as measured by the Light Obscuration Particle Count Test described in (788) *Particulate Matter in Injections*, Revised Bulletin, Official Oct. 1, 2011, The United States Pharmacopeial Convention.

[0203] In particular embodiments, the compositions are considered physically stable if after storage the average number of particles detected does not exceed 50 particles/mL, where the particles have a diameter >10 μm; does not exceed 5 particles/mL, where the particles have a diameter >25 μm; and does not exceed 2 particles/mL, where the particles have a diameter >50 μm, as measured by the microscopic method particle count test described in (788) *Particulate Matter in Injections*, Revised Bulletin, Official Oct. 1, 2011, The United States Pharmacopeial Convention.

[0204] In particular embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium) and, optionally, an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof) and are chemically stable for at least eight weeks or at least twelve weeks at 25° C. or for at least twelve weeks or at least sixteen weeks or at least 24

weeks at 4° C. In particular embodiments, at least 80% of each of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist, and anti-C5 agent (if present) show no sign of decomposition or modification resulting in formation of a new chemical entity under at least one of these conditions.

[0205] In particular embodiments, compositions comprise the following: (1) Antagonist A or another pharmaceutically acceptable salt thereof; (2) a VEGF antagonist; optionally, (3) an anti-C5 agent; (4) a buffer, optionally, (5) a tonicity modifier; and, optionally, (6) a surfactant. In specific embodiments of such compositions, the buffer is an acetate, phosphate, Tris or histidine buffer, or a mixture thereof; the tonicity modifier is sodium chloride, mannitol, sorbitol, or trehalose, or a mixture thereof; and the surfactant is polysorbate 20. In various embodiments, Antagonist A or another pharmaceutically acceptable salt thereof is present in compositions of the invention at a concentration of about 0.1 mg/mL to about 200 mg/mL; and the VEGF antagonist is present at a concentration of about 0.1 mg/mL to about 200 mg/mL. When present, the anti-C5 agent is present at a concentration of about 0.1 mg/mL to about 200 mg/mL. The buffer is present at a concentration of about 1 mM to about 200 mM; the tonicity modifier is present at a concentration of about 10 mM to about 200 mM (sodium chloride), about 1% to about 10% (w/v) (sorbitol), or about 1% to about 20% (w/v) (trehalose); and the surfactant, when present, is present at a concentration of about 0.005% to about 0.05% or a concentration of about 0.001% to about 0.05%.

[0206] In particular embodiments, the ratio of the concentration (mass of Antagonist A or another pharmaceutically acceptable salt thereof less that of its —R group/volume of composition) of Antagonist A or another pharmaceutically acceptable salt thereof to the concentration (mass/volume of composition) of the VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008), ARC1905, or a pharmaceutically acceptable salt thereof, present in the composition is less than, or less than or equal to, 25.0, less than, or less than or equal to, 10.0, less than, or less than or equal to, 9.0, less than, or less than or equal to, 8.0, less than, or less than or equal to, 7.0, less than, or less than or equal to, 6.0, less than, or less than or equal to, 5.0, less than, or less than or equal to, 4.0, less than, or less than or equal to, 3.0, less than, or less than or equal to, 2.0 or less than, or less than or equal to, 1.0. Antagonist A's —R group is depicted in FIG. **1**. In particular embodiments, the ratio of the concentration (mass of Antagonist A or another pharmaceutically acceptable salt thereof less that of its —R group/volume of composition) of Antagonist A or another pharmaceutically acceptable salt thereof to the concentration (mass/volume of composition) of the VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008), ARC1905, or a pharmaceutically acceptable salt thereof, present in the composition is in the range of about 1 to about 10, about 2 to about 5, about 3 about 4, or about 5. In certain embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, pegaptanib sodium, or ESBA1008), and ARC1905 or a pharmaceutically acceptable salt thereof.

[0207] In one particular embodiment, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008, or pegaptanib sodium), and, optionally, an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof), wherein the ratio of the concentration of PDGF antagonist to the concentration of VEGF antagonist (and/or anti-C5 agent) is less than 2; and the compositions further comprise sodium chloride at a concentration of about 10 mM to about 200 mM, histidine at a concentration of about 1 mM to about 100 mM, and polysorbate (e.g., polysorbate 20) at a concentration of about 0.005% to about 0.05%, where the pH of the composition is about 5.5 to about 7.0.

[0208] In certain embodiments, the compositions comprise one or more of a tonicity modifier, a surfactant, and a buffer suitable to achieve or maintain the particular pH or be suitable for parenteral administration. Appropriate buffers include those described herein as well as others known in the art, such as, e.g., Good's buffers, e.g., MES.

[0209] In certain embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and a tonicity modifier that is sorbitol or sodium chloride, or mixtures thereof. In certain embodiments, the compositions further comprise an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In particular embodiments, the tonicity modifier is sorbitol, and the pH of the composition is about 5.0 to about 8.0, about 5.0 to about 7.0, about 6.0 or about 7.0. In particular embodiments, the tonicity modifier is sodium chloride, and the pH of the composition is about 5.0 to about 8.0, about 5.0 to about 7.0, about 5.5 to about 7.5, about 6.0 to about 8.0, about 8.0, about 7.0, or about 6.0. In certain embodiments, the tonicity modifier is sorbitol at about 1% to about 10% (w/v), or about 1% (w/v), about 2% (w/v), about 3% (w/v), about 4% (w/v), about 5% (w/v), about 6% (w/v), about 7% (w/v), about 8% (w/v), about 9% (w/v), or about 10% (w/v). In particular embodiments, the tonicity modifier is sodium chloride at a concentration of about 10 mM to about 200 mM, about 50 mM to 200 mM, about 75 mM to about 200 mM, about 50 mM to about 150 mM, about 100 mM, about 110 mM, about 120 mM, about 130 mM about 140 mM or about 150 mM. In one embodiment, the tonicity modifier is sodium chloride at a concentration of about 130 mM. In other embodiments, the tonicity modifier is sodium chloride at a concentration of about 75 mM or about 120 mM. With respect to tonicity modifier concentration, "mM" refers to milimoles of the tonicity modifier per liter of composition.

[0210] In certain embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and a buffer capable of achieving or maintaining the pH of the composition within a desired range. In certain embodiments, the compositions further comprise an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In certain embodiments, the compositions comprise histidine (e.g., L-histidine or a pharmaceutically acceptable salt thereof) or phosphate as a buffer, e.g., sodium phosphate, potassium phosphate, or both. In certain embodiments, the buffer is present at a concentration of about 1 mM to about 200 mM, about 1 mM to about 150 mM, about 1 mM to about 20 mM, about 1 mM to about 10 mM, about 2 mM to about 100 mM, about 2 mM to about 20 mM, about 5 mM to about 20 mM, or about 10 mM. In particular embodiments, the pH of the buffered composition is about 5.0 to about 8.0, about 5.0 to about 7.0, about 5.5 to about 7.5, about 5.5 to about 7.0, or about 6.0.

In one embodiment, the buffered composition has a pH of about 5.5 to about 7.0. In certain embodiments, the buffer comprises histidine at a concentration of about 1 mM to about 200 mM, about 1 mM to about 150 mM, about 2 mM to about 100 mM, about 5 mM to about 20 mM, or about 10 mM, and the buffered composition has a pH of about 5.5 to about 7.0, or about 6.0. In one particular embodiment, the buffer comprises histidine at a concentration of about 10 mM and the pH of the histidine-buffered composition is about 6.0. With respect to buffer concentration, "mM" refers to millimoles of buffer (e.g., histidine) per liter of composition.

[0211] In certain embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and a buffer that comprises phosphate, alone or in combination with histidine. In certain embodiments, the compositions further comprise an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). The phosphate buffer may be, e.g., a sodium phosphate or potassium phosphate buffer. In certain embodiments, the buffer comprises phosphate at a concentration of about 1 mM to about 200 mM, about 1 mM to about 50 mM, about 2 mM to about 200 mM, about 2 mM to about 50 mM, about 5 mM to about 200 mM, about 5 mM to about 100 mM, about 5 mM to about 50 mM, about 10 mM to about 150 mM, about 10 mM to about 100 mM, about 5 mM, about 10 mM, about 25 mM, or about 50 mM. In particular embodiments, the pH of the buffered composition is about 5.0 to about 8.0, about 6.0 to about 8.0, about 5.5 to about 7.5, about 5.5 to about 7.0, about 6.0, about 7.0, or about 8.0. In one embodiment, the buffer comprises phosphate, and the buffered composition has a pH of about 6.0 to about 8.0. In certain embodiments, the buffer comprises phosphate at a concentration of about 5 mM to about 200 mM, about 5 mM to about 150 mM, about 5 mM to about 100 mM, about 5 mM, about 8 mM, about 10 mM, about 25 mM, or about 50 mM, and the buffered composition has a pH of about 5.5 to about 7.5, about 5.5 to about 7.0, or about 6.0. In one particular embodiment, the buffer comprises phosphate at a concentration of about 10 mM, and the buffered composition has a pH of about 6.2.

[0212] In certain embodiments, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof), a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), and a surfactant. In certain embodiments, the compositions further comprise an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In particular embodiments, the surfactant is polysorbate 20 at a concentration of about 0.001% (w/v) to about 0.05% (w/v), about 0.002% (w/v) to about 0.05% (w/v), about 0.005% (w/v) to about 0.05% (w/v), about 0.01% (w/v) to about 0.05% (w/v), or about 0.02% (w/v).

[0213] In one embodiment, the compositions comprise Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium), histidine, and NaCl. In certain embodiments, the compositions further comprise an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). The composition may further comprise polysorbate.

[0214] In certain embodiments, the compositions comprise an effective amount of: (a) about 0.3 mg/mL to about 30 mg/mL of Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 0.5 mg/mL to about 20 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium); and one or both of: (c) a buffer capable of achieving or maintaining the pH of the compositions at about pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, the compositions further comprise (e) about 0.3 mg/mL to about 30 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In certain embodiments, the buffer is about 1 mM to about 20 mM L-histidine or about 1 mM to about 20 mM sodium phosphate, and the tonicity modifier is about 10 mM to about 200 mM NaCl, about 1% to about 20% (w/v) sorbitol, or about 1% to about 20% (w/v) trehalose. In particular embodiments, the compositions further comprise: (f) about 0.001% (w/v) to about 0.05% (w/v) surfactant.

[0215] In certain embodiments, the compositions comprise: (a) about 0.3 mg/mL to about 30 mg/mL of Antagonist A or another pharmaceutically acceptable salt thereof; and (b) about 0.5 mg/mL to about 20 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium). In certain embodiments, the compositions further comprise (c) about 0.3 mg/mL to about 30 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In certain embodiments, any of these the compositions further comprise one or both of: (d) about 1 mM to about 20 mM L-histidine; and (e) about 10 mM to about 200 mM NaCl. In further embodiments, the compositions further comprise: (f) about 0.001% (w/v) to about 0.05% (w/v) surfactant, which is optionally polysorbate. In a particular embodiment, the compositions comprise: (a) about 0.3 mg/mL to about 30 mg/mL of Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 0.5 mg/mL to about 20 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium); (c) about 1 mM to about 20 mM L-histidine; and (d) about 10 mM to about 200 mM NaCl, wherein the pH of the compositions is about pH 5.0 to about pH 7.0. In certain embodiments, the compositions further comprise (e) about 0.3 mg/mL to about 30 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In certain embodiments, the compositions further comprise: (f) about 0.01% (w/v) polysorbate 20.

[0216] In certain embodiments, compositions comprise: (a) about 1.0 mg/mL to about 100 mg/mL, or about 5.0 mg/mL to about 50 mg/mL of Antagonist A or another pharmaceutically acceptable salt thereof); and (b) about 1.0 mg/mL to about 50 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium). In certain embodiments, the compositions further comprise (c) about 1.0 mg/mL to about 100 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In other embodiments, any of the compositions further comprise one or both of (d) about 1 mM to about 20 mM L-histidine; and (e) about 10 mM to about 200 mM NaCl. In further embodiments, any of the compositions further comprise: (f) about 0.001% (w/v) to about 0.05% (w/v) surfactant, which is optionally polysorbate.

[0217] In certain embodiments, compositions comprise: (a) about 0.3 mg/mL to about 30 mg/mL of Antagonist A or another pharmaceutically acceptable salt thereof); (b) about 0.5 mg/mL to about 20 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium); and one or both of (c) a buffer capable of achieving or maintaining the pH of the composition to about

pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, the compositions further comprise about 0.3 mg/mL to about 30 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In particular embodiments, the buffer, where present, is about 1 mM to about 20 mM L-histidine or about 1 mM to about 20 mM sodium phosphate; and the tonicity modifier, where present, is about 10 mM to about 200 mM NaCl, about 1% to about 20% (w/v) sorbitol, or about 1% to about 20% (w/v) trehalose. In certain embodiments, the buffer is about 1 mM to about 20 mM L-histidine; and the tonicity modifier is about 10 mM to about 200 mM NaCl, wherein the pH of the compositions is about pH 5.0 to about pH 7.0.

[0218] Any of the compositions can also comprise a surfactant, e.g., about 0.001% (w/v) to about 0.05% (w/v) surfactant.

[0219] In certain embodiments the compositions comprise: (a) about 3 mg/mL to about 90 mg/mL Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 1.0 mg/mL to about 30 mg/mL of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium); and one or both of (c) a buffer capable of achieving or maintaining the pH of the compositions to about pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, any of the compositions further comprises (e) about 3 mg/mL to about 90 mg/mL of an anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof). In particular embodiments, the buffer, where present, comprises about 1 mM to about 100 mM sodium phosphate or about 1.0 mM to about 10 mM histidine.HCl; and the tonicity modifier, where present, is about 0.5% (w/v) to about 10% (w/v) trehalose.

[0220] In certain embodiments, a composition of the invention comprises: (a) about 0.3 mg/mL to about 30 mg/mL Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 0.5 mg/mL to about 20 mg/mL ranibizumab or a pharmaceutically acceptable salt thereof; and one or both of: (c) a buffer capable of achieving or maintaining the pH of the composition at about pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, the buffer is about 1 mM to about 20 mM L-histidine or about 1 mM to about 20 mM sodium phosphate, and the tonicity modifier is about 10 mM to about 200 mM NaCl, about 1% to about 20% (w/v) sorbitol, or about 1% to about 20% (w/v) trehalose. In particular embodiments, the composition of the invention further comprises: (e) about 0.001% (w/v) to about 0.05% (w/v) surfactant. In particular embodiments, the composition further comprises: (f) an anti-C5 agent, another PDGF antagonist, or another VEGF antagonist. In particular embodiments, the anti-C5 agent is ARC 186, ARC 187, or ARC1905, and the other VEGF antagonist is bevacizumab or aflibercept.

[0221] In certain embodiments, a composition of the invention comprises: (a) about 0.3 mg/mL to about 30 mg/mL Antagonist A or another pharmaceutically acceptable salt thereof; and (b) about 0.5 mg/mL to about 25 mg/mL bevacizumab or a pharmaceutically acceptable salt thereof; and one or both of: (c) a buffer capable of achieving or maintaining the pH of the composition at about pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, the buffer is about 5 mM to about 200 mM sodium phosphate or about 5 mM to about 200 mM Tris.HCl, and the tonicity modifier is about 10 mM to about 200 mM NaCl, about 1% to about 20% (w/v) sorbitol, or about 1% to about 20% (w/v) trehalose. In particular embodiments, the composition of the invention further comprises: (e) about 0.001% (w/v) to about 0.05% (w/v) surfactant. In particular embodiments, the composition further comprises: (f) an anti-C5 agent, another PDGF antagonist, and/or another VEGF antagonist. In particular embodiments, the anti-C5 agent is ARC 186, ARC 187, or ARC1905, and the other VEGF antagonist is ranibizumab or aflibercept.

[0222] In certain embodiments, a composition of the invention comprises: (a) about 0.3 mg/mL to about 30 mg/mL Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 5 mg/mL to about 40 mg/mL aflibercept or a pharmaceutically acceptable salt thereof; and one or more of: (c) a buffer capable of achieving or maintaining the pH of the composition at about pH 5.0 to about pH 8.0; (d) a tonicity modifier; and (e) 0 to about 10% (w/v) sucrose. In certain embodiments, the buffer is about 5 mM to about 50 mM phosphate, and the tonicity modifier is about 10 mM to about 200 mM NaCl. In particular embodiments, the composition of the invention further comprises: (f) about 0.001% (w/v) to about 0.05% (w/v) surfactant. In particular embodiments, the composition further comprises: (g) an anti-C5 agent, another PDGF antagonist, and/or another VEGF antagonist. In particular embodiments, the anti-C5 agent is ARC 186, ARC 187, or ARC1905, and the other VEGF antagonist is ranibizumab or bevacizumab.

[0223] In certain embodiments, a composition of the invention comprises: (a) about 3 mg/mL to about 90 mg/mL Antagonist A or another pharmaceutically acceptable salt thereof; (b) about 1.0 mg/mL to about 30 mg/mL ranibizumab or a pharmaceutically acceptable salt thereof; and one or both of: (c) a buffer capable of achieving or maintaining the pH of the composition at about pH 5.0 to about pH 8.0; and (d) a tonicity modifier. In certain embodiments, the buffer comprises about 1 mM to about 100 mM sodium phosphate or about 1.0 mM to about 10 mM histidine.HCl, and the tonicity modifier is about 0.5% (w/v) to about 10% (w/v) trehalose. In particular embodiments, the composition further comprises: (e) an anti-C5 agent, another PDGF antagonist, and/or another VEGF antagonist. In particular embodiments, the anti-C5 agent is ARC186, ARC187, or ARC1905, and the other VEGF antagonist is bevacizumab or aflibercept.

[0224] Illustrative compositions include F1-F31, as described in Tables 3 and 4. Illustrative compositions are also described in PCT Application Publication No. WO2013/181495. Any of these compositions may further comprise an anti-C5 agent, such as ARC1905 or a pharmaceutically acceptable salt thereof.

TABLE 3

Composition Matrix for Illustrative Antagonist A: Ranibizumab Compositions

Comp.	Buffer	pH	Tonicity Modifier	[Ant. A] (mg/mL)	[ran.] (mg/mL)	Polysorbate 20 (% v/v)
F1	10 mM Sodium Phosphate	7.3	150 mM NaCl	3	0	0%
F2	10 mM Sodium Acetate	5.0	5% (w/v) Sorbitol	3	5	0.01%

TABLE 3-continued

Composition Matrix for Illustrative Antagonist A: Ranibizumab Compositions

Comp.	Buffer	pH	Tonicity Modifier	[Ant. A] (mg/mL)	[ran.] (mg/mL)	Polysorbate 20 (% v/v)
F3	10 mM Sodium Acetate	5.0	130 mM NaCl	3	5	0.01%
F4	10 mM Histidine•HCl	5.5	10% (w/v) Trehalose	0	5	0.01%
F5	10 mM Histidine•HCl	6.0	5% (w/v) Sorbitol	3	5	0.01%
F6	10 mM Histidine•HCl	6.0	130 mM NaCl	3	5	0.01%
F7	10 mM Sodium Phosphate	7.0	5% (w/v) Sorbitol	3	5	0.01%
F8	10 mM Sodium Phosphate	7.0	130 mM NaCl	3	5	0.01%
F9	10 mM Tris•HCl	8.0	5% (w/v) Sorbitol	3	5	0.01%
F10	10 mM Tris•HCl	8.0	130 mM NaCl	3	5	0.01%
F11	5 mM Sodium Phosphate + 5 mM Histidine	6.5	75 mM NaCl + 5% (w/v) Trehalose	3	5	0.005%
F27	10 mM Sodium Phosphate	7.3	150 mM NaCl	30	0	0%
F28	10 mM Histidine•HCl	5.5	10% (w/v) Trehalose	0	10	0.01%
F29	10 mM Histidine•HCl	5.5	10% (w/v) Trehalose	0	40	0.01%
F30	5 mM Sodium Phosphate + 5 mM Histidine•HCl		75 mM NaCl + 5% (w/v) Trehalose	15	5	0.005%
F31	8 mM Sodium Phosphate + 2 mM Histidine•HCl		120 mM NaCl + 2% (w/v) Trehalose	24	8	0.002%

"Ant. A" is Antagonist A;
"ran." is ranibizumab

TABLE 4

Composition Matrix for Illustrative Antagonist A: Bevacizumab Compositions

Comp.	Buffer	pH	Tonicity Modifier	Antagonist A Concentration (mg/mL, oligo wt.)	Bevacizumab Concentration (mg/mL)	Surfactant
F12	10 mM Phosphate	7.3	150 mM Sodium Chloride	30	0.0	0%
F13	50 mM Acetate	4	5% (w/v) Sorbitol	3	12.5	0.02% Polysorbate 20
F14	50 mM Acetate	4	130 mM Sodium Chloride	3	12.5	0.02% Polysorbate 20
F15	50 mM Acetate	5	5% (w/v) Sorbitol	3	12.5	0.02% Polysorbate 20
F16	50 mM Acetate	5	130 mM Sodium Chloride	3	12.5	0.02% Polysorbate 20
F17	50 mM Phosphate	6	5% (w/v) Sorbitol	3	12.5	0.02% Polysorbate 20
F18	50 mM Phosphate	6.2	6% (w/v) Trehalose	0	12.5	0.02% Polysorbate 20
F19	50 mM Phosphate	6	130 mM Sodium Chloride	3	12.5	0.02% Polysorbate 20
F20	50 mM Phosphate	7	5% (w/v) Sorbitol	3	12.5	0.02% Polysorbate 20
F21	50 mM Phosphate	7	130 mM Sodium Chloride	3	12.5	0.02% Polysorbate 20
F22	50 mM Tris	8	5% (w/v) Sorbitol	3	12.5	0.02% Polysorbate 20
F23	30 mM Tris	8	130 mM Sodium Chloride	3	12.5	0.02% Polysorbate 20
F24	30 mM Phosphate	6.3	75 mM sodium Chloride + 3% (w/v) Trehalose	15	12.5	0.02% Polysorbate 20
F25	10 mM Phosphate	7.3	150 mM Sodium Chloride	3	0.0	0%
F26	30 mM Phosphate	6.3	75 mM sodium Chloride + 3% (w/v) Trehalose	3	12.5	0.02% Polysorbate 20

[0225] The methods or compositions according to the invention can be administered alone or in conjunction with another therapy and can be provided at home, a doctor's office, a clinic, a hospital's outpatient department, or a hospital. Treatment can begin at a hospital so that the doctor can observe the therapy's effects closely and make any adjustments that are needed. The duration of the administration can depend on the type of ophthalmological disease or disorder being treated or prevented, the age and condition of the subject, the stage and type of the subject's disease or disorder, and how the subject responds to the treatment. Additionally, a subject having a greater risk of developing an ophthalmologi-

cal disease or disorder (e.g., a diabetic patient) can receive treatment to inhibit or delay the onset of symptoms. In one embodiment, the present methods or compositions allow for the administration of a relatively lower dose of each antagonist.

[0226] The dosage and frequency of administration of each antagonist can be controlled independently. For example, one antagonist can be administered three times per day, while the other antagonist can be administered once per day. Administration can be performed in on-and-off cycles that include rest periods so that the subject's body has a chance to recover from a side effect, if any. The antagonists can also be present in the same composition.

[0227] In other embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and optionally, a VEGF antagonist and/or anti-C5 agent are administered prior to, during, and/or after another treatment. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist and/or anti-C5 agent are administered concurrently, such as in a co-formulation, prior to, during, and/or after the other treatment. In other embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist are administered sequentially, prior to, during, and/or after the other treatment. In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof is administered prior to the administration of the VEGF antagonist. In other embodiments, Antagonist A or another pharmaceutically acceptable salt thereof is administered subsequent to the administration of the VEGF antagonist. In some embodiments, the other treatment is performing surgery. Examples of other treatment include pneumatic retinopexy, laser retinopexy, scleral buckling, and pars plana vitrectomy (PPV), laser photocoagulation, or cryotherapy.

[0228] Administration of a composition disclosed herein with performing another treatment can improve retinal attachment success, improve visual acuity, reduce choroidal neovascularization or stabilize vision to a degree that is greater than performing the other treatment alone. For example, in some embodiments, the administration of both Antagonist A or another pharmaceutically acceptable salt thereof with performing another treatment can improve retinal attachment success, improve visual acuity, or stabilize vision to a degree that is greater than an additive effect of both Antagonist A or another pharmaceutically acceptable salt thereof with performing the other treatment. In some embodiments, the synergistic effect is in reducing the size or growth of a tumor (e.g., in treating or preventing VHL disease, retinal capillary hemangioma, or von Hippel angioma). In some embodiments, the synergistic effect is reducing or inhibiting scarring or fibrosis (e.g., ocular scarring of fibrosis, such as subretinal fibrosis).

[0229] Administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can improve retinal attachment success, improve visual acuity, or stabilize vision to a degree that is greater than administration of Antagonist A or another pharmaceutically acceptable salt thereof or the VEGF antagonist. In some embodiments, the administration of Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can have a synergistic effect in treating or preventing an ophthalmological disease or disorder. For example, the administration of both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can improve retinal attachment success, improve visual acuity, or stabilize vision to a degree that is greater than an additive effect of administering both Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist. In some embodiments, the synergistic effect is in reducing the size or growth of a tumor (e.g., in treating or preventing VHL disease, retinal capillary hemangioma, or von Hippel angioma). In some embodiments, the synergistic effect is reducing or inhibiting scarring or fibrosis (e.g., ocular scarring of fibrosis, such as subretinal fibrosis).

[0230] In some embodiments, the methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent, in which two or more of Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist and the anti-C5 agent are present in the same composition. In certain embodiments, the PDGF antagonist and the VEGF antagonist are present in the same composition; in certain embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and the anti-C5 agent are present in the same composition; and in certain embodiments, the VEGF antagonist and the anti-C5 agent are present in the same composition. In some embodiments, all three of Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist and the anti-C5 agent are present in the same composition.

[0231] In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist and the anti-C5 agent are administered sequentially. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered prior to the VEGF antagonist or the anti-C5 agent. In one embodiment, the VEGF antagonist is administered prior to Antagonist A or another pharmaceutically acceptable salt thereof or the anti-C5 agent. In one embodiment, the anti-C5 agent is administered prior to the VEGF antagonist or Antagonist A or another pharmaceutically acceptable salt thereof. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered prior to the VEGF antagonist and anti-C5 agent. In one embodiment, the VEGF antagonist is administered prior to Antagonist A or another pharmaceutically acceptable salt thereof and he—C5 agent. In one embodiment, the anti-C5 agent is administered prior to the VEGF antagonist and PDGF antagonist.

[0232] In certain embodiments, the subject is administered two or more active agents (e.g., Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist) in a staggered dosing regimen, wherein one or more of the two or more active agents is administered before another one or more of the two or more active agents is administered to the subject.

[0233] In certain embodiments, the one or more active agent(s) is administered at least one day before the other one or more active agent(s). Accordingly, in some embodiments the present methods comprise administering on one or more days Antagonist A or another pharmaceutically acceptable salt thereof, one or more VEGF antagonists or one or more anti-C5 agents.

[0234] In one embodiment, the order of administration is: Antagonist A or another pharmaceutically acceptable salt thereof, followed by VEGF antagonist, followed by anti-C5 agent. In another embodiment, the order of administration is: Antagonist A or another pharmaceutically acceptable salt thereof, followed by anti-C5 agent, followed by VEGF

antagonist. In another embodiment, the order of administration is: VEGF antagonist, followed by anti-C5 agent, followed by Antagonist A or another pharmaceutically acceptable salt thereof. In another embodiment, the order of administration is: VEGF antagonist, followed by Antagonist A or another pharmaceutically acceptable salt thereof, followed by anti-C5 agent. In yet another embodiment the order of administration is: anti-C5 agent, followed by Antagonist A or another pharmaceutically acceptable salt thereof, followed by VEGF antagonist. In another embodiment the order of administration is: anti-C5 agent, followed by VEGF antagonist, followed by PDGF antagonist.

[0235] In some embodiments, the Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist are administered concurrently, and the anti-C5 agent is administered prior to or subsequent to administration of the PDGF antagonist and VEGF antagonist. In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and the anti-C5 agent are administered concurrently, and the VEGF antagonist is administered prior to or subsequent to administration of Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist. In some embodiments, the VEGF antagonist and anti-C5 agent are administered concurrently, and Antagonist A or another pharmaceutically acceptable salt thereof is administered prior to or subsequent to administration of the anti-C5 agent and VEGF antagonist.

[0236] In other embodiments, the order of administration is: Antagonist A or another pharmaceutically acceptable salt thereof, followed by VEGF antagonist and anti-C5 agent, wherein the VEGF antagonist and anti-C5 agent are present in the same composition. In another embodiment, the order of administration is: VEGF antagonist, followed by anti-C5 agent and Antagonist A or another pharmaceutically acceptable salt thereof, wherein the anti-C5 agent and PDGF antagonist are present in the same composition. In yet another embodiment the order of administration is: anti-C5 agent, followed by Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist, wherein the PDGF antagonist and VEGF antagonist are present in the same composition.

[0237] In still other embodiments, the order of administration is: Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist, wherein Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist are present in the same composition, followed by anti-C5 agent. In another embodiment, the order of administration is: Antagonist A or another pharmaceutically acceptable salt thereof and anti-C5 agent, wherein Antagonist A or another pharmaceutically acceptable salt thereof and the anti-C5 agent are present in the same composition, followed by VEGF antagonist. In another embodiment, the order of administration is: VEGF antagonist and anti-C5 agent, wherein the VEGF antagonist and anti-C5 agent are present in the same composition, followed by Antagonist A or another pharmaceutically acceptable salt thereof.

[0238] For example, Antagonist A or another pharmaceutically acceptable salt thereof can be administered prior to or subsequent to administration of a VEGF antagonist and/or an anti-C5 agent; a VEGF antagonist can be administered prior to or subsequent to administration of Antagonist A or another pharmaceutically acceptable salt thereof and/or anti-C5 agent; or an anti-C5 agent can be administered prior to or subsequent to administration of Antagonist A or another pharmaceutically acceptable salt thereof and/or a VEGF antagonist.

[0239] In some embodiments, the present methods comprise administering a first agent prior to administering a second agent. In some embodiments, the present methods comprise administering a first agent prior to administering a second agent and administering the second agent prior to administering a third agent.

[0240] In some embodiments, the present methods comprise concurrently administering a first agent and a second agent. In some embodiments, the present methods comprise concurrently administering a first agent and a second agent prior to administering a third agent.

[0241] In some embodiments, the present methods comprise administering a first agent prior to concurrently administering a second agent and third agent.

[0242] In some embodiments, the present methods comprise concurrently administering a first agent, a second agent and a third agent.

[0243] Illustrative groups of first agent, second agent and third agent are set forth below in Tables 5 and 6.

TABLE 5

Group	First Agent	Second Agent	Third Agent
A	Antagonist A or another pharmaceutically acceptable salt thereof	VEGF antagonist	Anti-C5 Agent
B	Antagonist A or another pharmaceutically acceptable salt thereof	Anti-C5 Agent	VEGF antagonist
C	VEGF antagonist	Antagonist A or another pharmaceutically acceptable salt thereof	Anti-C5 Agent
D	VEGF antagonist	Anti-C5 Agent	Antagonist A or another pharmaceutically acceptable salt thereof
E	Anti-C5 Agent	Antagonist A or another pharmaceutically acceptable salt thereof	VEGF antagonist
F	Anti-C5 Agent	VEGF antagonist	Antagonist A or another pharmaceutically acceptable salt thereof

TABLE 6

Group	First Agent	Second Agent	Third Agent
A	Antagonist A	ranibizumab	ARC1905
B	Antagonist A	bevacizumab	ARC1905
C	Antagonist A	aflibercept	ARC1905
D	Antagonist A	pegaptanib sodium	ARC1905
E	Antagonist A	ESBA1008	ARC1905
F	Antagonist A	ARC1905	ranibizumab
G	Antagonist A	ARC1905	bevacizumab
H	Antagonist A	ARC1905	aflibercept
I	Antagonist A	ARC1905	pegaptanib sodium
J	Antagonist A	ARC1905	ESBA1008
K	ranibizumab	Antagonist A	ARC1905
L	bevacizumab	Antagonist A	ARC1905
M	aflibercept	Antagonist A	ARC1905

TABLE 6-continued

Group	First Agent	Second Agent	Third Agent
N	pegaptanib sodium	Antagonist A	ARC1905
O	ESBA1008	Antagonist A	ARC1905
P	ranibizumab	ARC1905	Antagonist A
Q	bevacizumab	ARC1905	Antagonist A
R	aflibercept	ARC1905	Antagonist A
S	pegaptanib sodium	ARC1905	Antagonist A
T	ESBA1008	ARC1905	Antagonist A
U	ARC1905	Antagonist A	ranibizumab
V	ARC1905	Antagonist A	bevacizumab
W	ARC1905	Antagonist A	aflibercept
X	ARC1905	Antagonist A	pegaptanib sodium
Y	ARC1905	Antagonist A	ESBA1008
Z	ARC1905	ranibizumab	Antagonist A
AA	ARC1905	bevacizumab	Antagonist A
AB	ARC1905	aflibercept	Antagonist A
AC	ARC1905	pegaptanib sodium	Antagonist A
AD	ARC1905	ESBA1008	Antagonist A

[0244] In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof and two or more VEGF antagonists. In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof and two or more anti-C5 agents. In some embodiments, the present methods comprise administering a VEGF antagonist and two or more anti-C5 agents.

[0245] In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to administering two or more VEGF antagonists. In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to administering a first VEGF antagonist and administering the first VEGF antagonist prior to administering a second VEGF antagonist.

[0246] In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist. In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof and a first VEGF antagonist prior to administering a second VEGF antagonist.

[0247] In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to concurrently administering a first VEGF antagonist and a second VEGF antagonist.

[0248] In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof, a first VEGF antagonist and a second VEGF antagonist.

[0249] In some embodiments, the present methods comprise administering a VEGF antagonist prior to administering two PDGF antagonists (e.g., Antagonist A or another pharmaceutically acceptable salt thereof and another PDGF antagonist). In some embodiments, the present methods comprise administering a VEGF antagonist prior to administering a first PDGF antagonist and administering the first PDGF antagonist prior to administering a second PDGF antagonist.

[0250] In some embodiments, the present methods comprise concurrently administering a VEGF antagonist and Antagonist A or another pharmaceutically acceptable salt thereof. In some embodiments, the present methods comprise concurrently administering a VEGF antagonist and a first PDGF antagonist prior to administering a second PDGF antagonist.

[0251] In some embodiments, the present methods comprise administering a VEGF antagonist prior to concurrently administering a first PDGF antagonist and a second PDGF antagonist.

[0252] In some embodiments, the present methods comprise concurrently administering a VEGF antagonist, a first PDGF antagonist and a second PDGF antagonist.

[0253] In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to administering two or more anti-C5 agents. In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to administering a first anti-C5 agent and administering the first anti-C5 agent prior to administering a second anti-C5 agent.

[0254] In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof and an anti-C5 agent. In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof and a first anti-C5 agent prior to administering a second anti-C5 agent.

[0255] In some embodiments, the present methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof prior to concurrently administering a first anti-C5 agent and a second anti-C5 agent.

[0256] In some embodiments, the present methods comprise concurrently administering Antagonist A or another pharmaceutically acceptable salt thereof, a first anti-C5 agent and a second anti-C5 agent.

[0257] In some embodiments, the present methods comprise administering an anti-C5 agent prior to administering two or more PDGF antagonists. In some embodiments, the present methods comprise administering an anti-C5 agent prior to administering a first PDGF antagonist and administering the first PDGF antagonist prior to administering a second PDGF antagonist.

[0258] In some embodiments, the present methods comprise concurrently administering an anti-C5 agent and Antagonist A or another pharmaceutically acceptable salt thereof. In some embodiments, the present methods comprise concurrently administering an anti-C5 agent and a first PDGF antagonist prior to administering a second PDGF antagonist.

[0259] In some embodiments, the present methods comprise administering an anti-C5 agent prior to concurrently administering a first PDGF antagonist and a second PDGF antagonist.

[0260] In some embodiments, the present methods comprise concurrently administering an anti-C5 agent, a first PDGF antagonist and a second PDGF antagonist.

[0261] In some embodiments, the present methods comprise administering a VEGF antagonist prior to administering two or more anti-C5 agents. In some embodiments, the present methods comprise administering a VEGF antagonist prior to administering a first anti-C5 agent and administering the first anti-C5 agent prior to administering a second anti-C5 agent.

[0262] In some embodiments, the present methods comprise concurrently administering a VEGF antagonist and an anti-C5 agent. In some embodiments, the present methods comprise concurrently administering a VEGF antagonist and a first anti-C5 agent prior to administering a second anti-C5 agent.

[0263] In some embodiments, the present methods comprise administering a VEGF antagonist prior to concurrently administering a first anti-C5 agent and a second anti-C5 agent.

[0264] In some embodiments, the present methods comprise concurrently administering a VEGF antagonist, a first anti-C5 agent and a second anti-C5 agent.

[0265] In some embodiments, the present methods comprise administering an anti-C5 agent prior to administering two or more VEGF antagonists. In some embodiments, the present methods comprise administering an anti-C5 agent prior to administering a first VEGF antagonist and administering the first VEGF antagonist prior to administering a second VEGF antagonist.

[0266] In some embodiments, the present methods comprise concurrently administering an anti-C5 agent and a VEGF antagonist. In some embodiments, the present methods comprise concurrently administering an anti-C5 agent and a first VEGF antagonist prior to administering a second VEGF antagonist.

[0267] In some embodiments, the present methods comprise administering an anti-C5 agent prior to concurrently administering a first VEGF antagonist and a second VEGF antagonist.

[0268] In some embodiments, the present methods comprise concurrently administering an anti-C5 agent, a first VEGF antagonist and a second VEGF antagonist.

[0269] In some embodiments, the first agent and second agent are PDGF antagonists, which can be the same or different. In some embodiment, the first agent and second agent are VEGF antagonists, which can be the same or different. In some embodiments, the first agent and second agent are anti-C5 agents, which can be the same or different.

[0270] In some embodiments, the first agent and third agent are PDGF antagonists, which can be the same or different. In some embodiment, the first agent and third agent are VEGF antagonists, which can be the same or different. In some embodiments, the first agent and third agent are anti-C5 agents, which can be the same or different.

[0271] In some embodiments, the second agent and third agent are PDGF antagonists, which can be the same or different. In some embodiment, the second agent and third agent are VEGF antagonists, which can be the same or different. In some embodiments, the second agent and third agent are anti-C5 agents, which can be the same or different.

[0272] Illustrative groups of first agent, second agent and third agent are set forth below in Tables 7, 8, 9 and 10.

TABLE 7

Group	First Agent	Second Agent	Third Agent
A	PDGF Antagonist	VEGF antagonist	VEGF antagonist
B	VEGF antagonist	PDGF Antagonist	VEGF antagonist
C	VEGF antagonist	VEGF antagonist	PDGF Antagonist
D	PDGF Antagonist	Anti-C5 Agent	Anti-C5 Agent
E	Anti-C5 Agent	PDGF Antagonist	Anti-C5 Agent
F	Anti-C5 Agent	Anti-C5 Agent	PDGF Antagonist
G	PDGF Antagonist	PDGF Antagonist	VEGF antagonist
H	PDGF Antagonist	VEGF antagonist	PDGF Antagonist
I	VEGF antagonist	PDGF Antagonist	PDGF Antagonist
J	PDGF Antagonist	PDGF Antagonist	Anti-C5 Agent
K	PDGF Antagonist	Anti-C5 Agent	PDGF Antagonist
L	Anti-C5 Agent	PDGF Antagonist	PDGF Antagonist

TABLE 8

Group	First Agent	Second Agent	Third Agent
A	PDGF Antagonist	First VEGF antagonist	Second VEGF antagonist
B	First VEGF antagonist	PDGF Antagonist	Second VEGF antagonist
C	First VEGF antagonist	Second VEGF antagonist	PDGF Antagonist
D	PDGF Antagonist	First Anti-C5 Agent	Second Anti-C5 Agent
E	First Anti-C5 Agent	PDGF Antagonist	Second Anti-C5 Agent
F	First Anti-C5 Agent	Second Anti-C5 Agent	PDGF Antagonist
G	First PDGF Antagonist	Second PDGF Antagonist	VEGF antagonist
H	First PDGF Antagonist	VEGF antagonist	Second PDGF Antagonist
I	VEGF antagonist	First PDGF Antagonist	Second PDGF Antagonist
J	First PDGF Antagonist	Second PDGF Antagonist	Anti-C5 Agent
K	First PDGF Antagonist	Anti-C5 Agent	Second PDGF Antagonist
L	Anti-C5 Agent	First PDGF Antagonist	Second PDGF Antagonist

TABLE 9

Group	First Agent	Second Agent	Third Agent
A	Antagonist A	ranibizumab	Antagonist A
B	Antagonist A	ranibizumab	ranibizumab
C	Antagonist A	bevacizumab	Antagonist A
D	Antagonist A	bevacizumab	bevacizumab
E	Antagonist A	aflibercept	Antagonist A
F	Antagonist A	aflibercept	aflibercept
G	Antagonist A	pegaptanib sodium	Antagonist A
H	Antagonist A	pegaptanib sodium	pegaptanib sodium
I	Antagonist A	ESBA1008	Antagonist A
J	Antagonist A	ESBA1008	ESBA1008
K	Antagonist A	ARC1905	Antagonist A
L	Antagonist A	ARC1905	ARC1905
M	ranibizumab	Antagonist A	ranibizumab
N	ranibizumab	Antagonist A	Antagonist A
O	bevacizumab	Antagonist A	bevacizumab
P	bevacizumab	Antagonist A	Antagonist A
Q	aflibercept	Antagonist A	aflibercept
R	aflibercept	Antagonist A	Antagonist A
S	pegaptanib sodium	Antagonist A	pegaptanib sodium
T	pegaptanib sodium	Antagonist A	Antagonist A
U	ESBA1008	Antagonist A	ESBA1008
V	ESBA1008	Antagonist A	Antagonist A
W	ARC1905	Antagonist A	ARC1905
X	ARC1905	Antagonist A	Antagonist A
Y	ranibizumab	ranibizumab	Antagonist A
Z	bevacizumab	bevacizumab	Antagonist A
AA	aflibercept	aflibercept	Antagonist A
AB	pegaptanib sodium	pegaptanib sodium	Antagonist A
AC	ESBA1008	ESBA1008	Antagonist A
AD	ARC1905	ARC1905	Antagonist A
AE	ranibizumab	ranibizumab	bevacizumab
AF	ranibizumab	bevacizumab	ranibizumab
AG	ranibizumab	ranibizumab	aflibercept
AH	ranibizumab	aflibercept	ranibizumab

TABLE 9-continued

Group	First Agent	Second Agent	Third Agent
AI	ranibizumab	ranibizumab	pegaptanib sodium
AJ	ranibizumab	pegaptanib sodium	ranibizumab
AK	ranibizumab	ranibizumab	ESBA1008
AL	ranibizumab	ESBA1008	ranibizumab
AM	ranibizumab	ranibizumab	ARC1905
AN	ranibizumab	ARC1905	ranibizumab
AO	bevacizumab	bevacizumab	ranibizumab
AP	bevacizumab	ranibizumab	bevacizumab
AQ	bevacizumab	bevacizumab	aflibercept
AR	bevacizumab	aflibercept	bevacizumab
AS	bevacizumab	bevacizumab	pegaptanib sodium
AT	bevacizumab	pegaptanib sodium	bevacizumab
AU	bevacizumab	bevacizumab	ESBA1008
AV	bevacizumab	ESBA1008	bevacizumab
AW	bevacizumab	bevacizumab	ARC1905
AX	bevacizumab	ARC1905	bevacizumab
AY	aflibercept	aflibercept	ranibizumab
AZ	aflibercept	ranibizumab	aflibercept
BA	aflibercept	aflibercept	bevacizumab
BB	aflibercept	bevacizumab	aflibercept
BC	aflibercept	aflibercept	pegaptanib sodium
BD	aflibercept	pegaptanib sodium	aflibercept
BE	aflibercept	aflibercept	ESBA1008
BF	aflibercept	ESBA1008	aflibercept
BG	aflibercept	aflibercept	ARC1905
BH	aflibercept	ARC1905	aflibercept
BI	pegaptanib sodium	pegaptanib sodium	ranibizumab
BJ	pegaptanib sodium	ranibizumab	pegaptanib sodium
BK	pegaptanib sodium	pegaptanib sodium	bevacizumab
BL	pegaptanib sodium	bevacizumab	pegaptanib sodium
BM	pegaptanib sodium	pegaptanib sodium	aflibercept
BN	pegaptanib sodium	aflibercept	pegaptanib sodium
BO	pegaptanib sodium	pegaptanib sodium	ESBA1008
BP	pegaptanib sodium	ESBA1008	pegaptanib sodium
BQ	pegaptanib sodium	pegaptanib sodium	ARC1905
BR	pegaptanib sodium	ARC1905	pegaptanib sodium
BS	ESBA1008	ESBA1008	ranibizumab
BT	ESBA1008	ranibizumab	ESBA1008
BU	ESBA1008	ESBA1008	bevacizumab
BV	ESBA1008	bevacizumab	ESBA1008
BW	ESBA1008	ESBA1008	aflibercept
BX	ESBA1008	aflibercept	ESBA1008
BY	ESBA1008	ESBA1008	pegaptanib sodium
BZ	ESBA1008	pegaptanib sodium	ESBA1008
CA	ESBA1008	ESBA1008	ARC1905
CB	ESBA1008	ARC1905	ESBA1008
CC	ARC1905	ARC1905	ranibizumab
CD	ARC1905	ranibizumab	ARC1905
CE	ARC1905	ARC1905	bevacizumab
CF	ARC1905	bevacizumab	ARC1905
CO	ARC1905	ARC1905	aflibercept
CH	ARC1905	aflibercept	ARC1905
CI	ARC1905	ARC1905	pegaptanib sodium
CJ	ARC1905	pegaptanib sodium	ARC1905
CK	ARC1905	ARC1905	ESBA1008
CL	ARC1905	ESBA1008	ESBA1008

TABLE 10

Group	First Agent	Second Agent	Third Agent
A	Antagonist A	ranibizumab	bevacizumab
B	Antagonist A	ranibizumab	aflibercept
C	Antagonist A	ranibizumab	pegaptanib sodium
D	Antagonist A	bevacizumab	aflibercept
E	Antagonist A	bevacizutnab	pegaptanib sodium
F	Antagonist A	aflibercept	pegaptanib sodium
G	ranibizumab	bevacizumab	Antagonist A
H	ranibizumab	aflibercept	Antagonist A
I	ranibizumab	pegaptanib sodium	Antagonist A
J	bevacizumab	aflibercert	Antagonist A
K	bevacizumab	pegaptanib sodium	Antagonist A

TABLE 10-continued

Group	First Agent	Second Agent	Third Agent
L	aflibercept	pegaptanib sodium	Antagonist A
M	ranibizumab	Antagonist A	bevacizumab
N	ranibizumab	Antagonist A	aflibercept
O	ranibizumab	Antagonist A	pegaptanib sodium
P	bevacizumab	Antagonist A	aflibercept
Q	bevacizumab	Antagonist A	pegaptanib sodium
R	aflibercept	Antagonist A	pegaptanib sodium
S	bevacizumab	ranibizumab	Antagonist A
T	aflibercept	ranibizumab	Antagonist A
U	pegaptanib sodium	ranibizumab	Antagonist A
V	aflibercept	bevacizumab	Antagonist A
W	pegaptanib sodium	bevacizumab	Antagonist A
X	pegaptanib sodium	aflibercept	Antagonist A
Y	bevacizumab	Antagonist A	ranibizumab
Z	aflibercept	Antagonist A	ranibizumab
AA	pegaptanib sodium	Antagonist A	ranibizumab
AB	aflibercept	Antagonist A	bevacizumab
AC	pegaptanib sodium	Antagonist A	bevacizumab
AD	pegaptanib sodium	Antagonist A	aflibercept
AE	Antagonist A	ARC187	ARC1905
AF	Antagonist A	ARC1905	ARC187
AG	ARC187	ARC1905	Antagonist A
AH	ARC1905	ARC187	Antagonist A
AI	ARC187	Antagonist A	ARC1905
AJ	ARC1905	Antagonist A	ARC187

[0273] In one embodiment, two or more agents are administered concurrently. In one embodiment, the two or more agents administered concurrently are present in the same composition. In another embodiment, the two or more agents administered concurrently are each present in a separate composition.

[0274] In certain embodiments, the time period from administration of a first agent to administration of a second agent is at least 1 min, at least 5 min, at least 10 min, at least 15 min, at least 30 min, or at least one hour. In certain embodiments, the time period from administration of a first agent to administration of a second agent is between 1 min and 2 hours, between 5 min and 2 hours, between 10 min and 2 hours, between 15 min and 2 hours, between 30 min and 2 hours, between 45 min and 2 hours, between 1 hour and 2 hours, or between 30 min and 1 hour. In certain embodiments, the time period from administration of a first agent to administration of a second agent is about 1 min, about 2 min, about 3 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 35 min, about 40 min, about 45 min, about 50 min, about 55 min, about 60 min, about 90 min, or about 120 min. In certain embodiments, a second agent is administered within 90 days, 30 days, 10 days, 5 days, 2 days, 1 day, 24 hours, 1 hour, 30 minutes, 10 minutes, 5 minutes or one minute after administration of a second agent.

[0275] In certain embodiments, the time period from administration of a second agent to administration of a third agent is at least 1 min, at least 5 min, at least 10 min, at least 15 min, at least 30 min, or at least one hour. In certain embodiments, the time period between administration of a second agent and administration of a third agent is between 1 min and 2 hours, between 5 min and 2 hours, between 10 min and 2 hours, between 15 min and 2 hours, between 30 min and 2 hours, between 45 min and 2 hours, between 1 hour and 2 hours, or between 30 min and 1 hour. In certain embodiments, the time period between administration of a second agent and administration of a third agent is about 1 min, about 2 min, about 3 min, about 5 min, about 10 min, about 15 min, about

20 min, about 25 min, about 30 min, about 35 min, about 40 min, about 45 min, about 50 min, about 55 min, about 60 min, about 90 min, or about 120 min. In certain embodiments, a third agent is administered within 90 days, 30 days, 10 days, 5 days, 2 days, 1 day, 24 hours, 1 hour, 30 minutes, 10 minutes, 5 minutes or one minute after administration of a second agent.

[0276] In certain embodiments, the time period between concurrent administration of a first agent and a second agent and administration of a third agent is at least 1 min, at least 5 min, at least 10 min, at least 15 min, at least 30 min, or at least one hour. In certain embodiments, the time period between concurrent administration of a first agent and a second agent and administration of a third agent is between 1 min and 2 hours, between 5 min and 2 hours, between 10 min and 2 hours, between 15 min and 2 hours, between 30 min and 2 hours, between 45 min and 2 hours, between 1 hour and 2 hours, or between 30 min and 1 hour. In certain embodiments, the time period from concurrent administration of a first agent and a second agent to administration of a third agent is about 1 min, about 2 min, about 3 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 35 min, about 40 min, about 45 min, about 50 min, about 55 min, about 60 min, about 90 min, or about 120 min. In certain embodiments, administration of a third agent is within 90 days, 30 days, 10 days, 5 days, 2 days, 1 day, 24 hours, 1 hour, 30 minutes, 10 minutes, 5 minutes or one minute of concurrent administration of a first agent and a second agent.

[0277] In certain embodiments, the time period from administration of a first agent to concurrent administration a second agent and a third agent is at least 1 min, at least 5 min, at least 10 min, at least 15 min, at least 30 min, or at least one hour. In certain embodiments, the time period from administration of a first agent to concurrent administration of a second agent and a third agent is between 1 min and 2 hours, between 5 min and 2 hours, between 10 min and 2 hours, between 15 min and 2 hours, between 30 min and 2 hours, between 45 min and 2 hours, between 1 hour and 2 hours, or between 30 min and 1 hour. In certain embodiments, the time period from administration of a first agent to concurrent administration of a second agent and a third agent is about 1 min, about 2 min, about 3 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 35 min, about 40 min, about 45 min, about 50 min, about 55 min, about 60 min, about 90 min, or about 120 min. In certain embodiments, concurrent administration of a second agent and a third agent is within 90 days, 30 days, 10 days, 5 days, 2 days, 1 day, 24 hours, 1 hour, 30 minutes, 10 minutes, 5 minutes or one minute of administration of a first agent.

[0278] The administration of two or more, such as three or more, active agents (e.g., Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist and an anti-C5 agent) can have a synergistic effect in treating or preventing a disease or disorder, e.g., an ophthalmological disease or disorder. For example, administration of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent (or any two of these active agents) can improve retinal attachment success, improve visual acuity, reduce choroidal neovascularization or stabilize vision to a degree that is greater than an additive effect of the active agents.

[0279] In certain embodiments, the invention provides methods for treating or preventing an ophthalmological disease or disorder, comprising administering to a subject in need thereof one or more, in some embodiments two or more or three or more, active agents via an apparatus. In other embodiments, the methods further comprise performing surgery on the subject. In other embodiments, the methods further comprise administering another active agent, such as an antineoplastic drug, including but not limited to any of those described herein. In particular embodiments, the methods further comprise administering another active agent and performing surgery on the subject.

[0280] In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent to a subject results in improved vision, such as increased visual acuity. In some embodiments, the subject experienced moderate vision loss, defined as losing 15 letters or more from baseline on ETDRS visual acuity testing, measured at week 24, prior to treatment with Antagonist A or another pharmaceutically acceptable salt thereof.

[0281] In some embodiments, visual acuity testing is as described in Early Treatment Diabetic Retinopathy Study Research Group (ETDRS), Manual of Operations, Baltimore: ETDRS Coordinating Center, University of Maryland. Available from: National Technical Information Service, 5285 Port Royal Road, Springfield, Va. 22161; Accession No. PB85 223006/AS; Ferris et al., Am J Ophthalmol 94:91-96, 1982; or Example 4, as described herein. In some embodiments, the visual acuity testing uses one or more charts available from http://www.nei.nih.gov/photo/keyword.asp?conditions=Eye+Charts&match=all, e.g., ETDRS visual acuity Chart 1, 2 and/or R.

[0282] In other embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist results in fewer ocular adverse events, a decrease in size of RCH (e.g., measured by fundus photography and FA), a decrease in exudation (measured by fundus photography, OCT, and FA), or a decrease in epiretinal proliferation or retinal traction (assessed by fundus photography), compared to those experienced by a subject who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof. In some embodiments, the subject does not require, and the methods do not comprise, ablative treatment of RCH or ocular surgery.

[0283] In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in improved vision independent of baseline lesion size or baseline vision, compared to vision of a subject who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof, or compared to a subject administered anti-VEGF monotherapy. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in the subject having a visual acuity of 20/40 or better, or 20/25 or better vision. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent to a subject results in an increased reduction in CNV size in the subject, compared to CNV size in a patient who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof, or compared to a subject administered anti-VEGF monotherapy. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable

salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in a reduction in CNV size (e.g., reduction in disc area (DA) size). In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent to a subject result in an increased reduction in DA in the subject, compared to DA in a patient who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof, or compared to a subject administered anti-VEGF monotherapy. In some embodiments, the increased reduction in CNV size is in subjects with small baseline CNV, e.g., less than or equal to 1.62 DA (disc area). In some embodiments, the increased reduction in CNV size (e.g., in disc area) is in subjects with large baseline CNV, e.g., greater than 1.62 DA. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in neovascular regression. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in reduced neovascular growth, compared to that occurring in a subject who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof, or compared to a subject administered anti-VEGF monotherapy. In some embodiments, the reduced neovascular growth is anti-fibrosis. In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in a decrease in or absence of hyper-reflective material, e.g., sub-retinal hyper-reflective material, such as a decrease in the size of sub-retinal hyper-reflective material (SHRM) as evidenced by spectral domain optical coherence tomography (SD-OCT). In some embodiments, administration of Antagonist A or another pharmaceutically acceptable salt thereof, and optionally a VEGF antagonist and/or an anti-C5 agent, to a subject results in an increase in resolution of hyper-reflective material, e.g., sub-retinal hyper-reflective material, such as compared to a subject who was not administered with Antagonist A or another pharmaceutically acceptable salt thereof, or compared to a subject administered a VEGF antagonist, anti-VEGF monotherapy, and/or an anti-C5 agent.

[0284] In some embodiments, a subject with improved vision has a greater than 3-line, 4-line or 5-line gain in visual acuity. In one embodiment, a subject's visual acuity is determined using a protocol such as the Early Treatment for Diabetic Retinopathy Study ("ETDRS") or the Age-Related Eye Disease Study ("AREDS") protocol. In some embodiments, visual acuity is measured using a modified ETDRS and/or AREDS protocol, such as the measurement of visual acuity described in Ferris et al., Am J Ophthalmol 94:91-96, 1982. In some embodiments, visual acuity is measured as described in Early Treatment Diabetic Retinopathy Study Research Group (ETDRS), Manual of Operations, Baltimore: ETDRS Coordinating Center, University of Maryland. Available from: National Technical Information Service, 5285 Port Royal Road, Springfield, Va. 22161; Accession No. PB85 223006/AS. In other embodiments, visual acuity testing is measured as described in Example 4 below. In some embodiments, the visual acuity testing uses one or more charts available from http://www.nei.nih.gov/photo/keyword.asp?conditions=Eye+Charts&match=all, e.g., ETDRS visual acuity Chart 1, 2 and/or R.

[0285] In one embodiment, a subject's visual acuity is determined by one or more of the following procedures: (1) measurement of best-corrected visual acuity (BCVA) with required manifest refraction; (2) measurement of corrected visual acuity with conditional manifest refraction; or (3) measurement of corrected visual acuity without manifest refraction.

[0286] In one embodiment, each of the PDGF and VEGF antagonists is administered in an amount effective to treat or prevent an ophthalmological disease or disorder. The amount of antagonist that is admixed with the carrier materials to produce a single dosage can vary depending upon the subject being treated and the particular mode of administration.

[0287] The dosage of each antagonist can depend on several factors including the severity of the condition, whether the condition is to be treated or prevented, and the age, weight, and health of the person to be treated. Additionally, pharmacogenomic (the effect of genotype on the pharmacokinetic, pharmacodynamic or efficacy profile of a therapeutic) information about a particular patient may affect dosage used. Furthermore, the exact individual dosages can be adjusted somewhat depending on a variety of factors, including the specific combination of antagonists being administered, the time of administration, the route of administration, the nature of the formulation, the rate of excretion, the particular ophthalmological disease or disorder being treated, the severity of the disorder, and the anatomical location of the neovascular disorder. Some variations in the dosage can be expected.

[0288] Generally, when orally administered to a subject, the dosage of an antagonist of the present invention is normally 0.001 mg/kg/day to 100 mg/kg/day, 0.01 mg/kg/day to 50 mg/kg/day, or 0.1 mg/kg/day to 10 mg/kg/day. Generally, when orally administered to a human, the dosage of an antagonist of the present invention is normally 0.001 mg to 300 mg per day, 1 mg to 200 mg per day, or 5 mg to 50 mg per day. Dosages up to 200 mg per day may be necessary. For administration of an antagonist of the present invention by parenteral injection, the dosage is normally 0.1 mg to 250 mg per day, 1 mg to 20 mg per day, or 3 mg to 5 mg per day. Injections may be given up to four times daily. In some embodiments, the dosage of a PDGF or VEGF antagonist for use in the present invention is normally 0.1 mg to 1500 mg per day, or 0.5 mg to 10 mg per day, or 0.5 mg to 5 mg per day. A dosage of up to 3000 mg per day can be administered.

[0289] In some embodiments, for administration by parenteral injection of a three active agents (e.g., Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and an anti-C5 agent or other combination disclosed herein), the dosage of each of the PDGF antagonist, VEGF antagonist and anti-C5 agent, is typically 0.1 mg to 250 mg per day, 1 mg to 20 mg per day, or 3 mg to 5 mg per day. Injections may be given up to four times daily. Generally, when parenterally administered, the dosage of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist, or anti-C5 agent is typically 0.1 mg to 1500 mg per day, or 0.5 mg to 10 mg per day, or 0.5 mg to 5 mg per day. A dosage of at least up to 3000 mg per day can be administered.

[0290] In some embodiments, in which Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and/or anti-C5 agent are ophthalmologically administered to a human, for example intravitreally, the dosage of each of Antagonist A or another pharmaceutically

acceptable salt thereof. VEGF antagonist and anti-C5 agent is typically 0.003 mg to 5.0 mg per eye per administration, or 0.03 mg to 3.0 mg per eye per administration, or 0.1 mg to 1.0 mg per eye per administration. In one embodiment, the dosage of each of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent is about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.25 mg, about 1.5 mg, about 2.0 mg or about 3.0 mg per eye. In one embodiment, the dosage Antagonist A or another pharmaceutically acceptable salt thereof is about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.25 mg, about 1.5 mg, about 2.0 mg, about 3.0 mg, or about 4.0 mg per eye. In another embodiment, the dosage of a VEGF antagonist (e.g., ranibizumab, bevacizumab, aflibercept, ESBA1008 or pegaptanib sodium) is about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.25 mg, about 1.5 mg, about 1.65 mg, about 2.0 mg, about 3.0 mg, or about 4.0 mg per eye. In another embodiment, the dosage of the anti-C5 agent (e.g., ARC1905 or a pharmaceutically acceptable salt thereof) is about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.25 mg, about 1.5 mg, about 1.65 mg, about 2.0 mg, about 3.0 mg, or about 4.0 per eye.

[0291] In certain embodiments where a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, and optionally an anti-C5 agent, the dosage of Antagonist A or another pharmaceutically acceptable salt thereof) is about 1.5 mg, and the dosage of the VEGF antagonist (e.g., ranibizumab) is about 0.5 mg. In certain embodiments where a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 3.0 mg, and the dosage of the VEGF antagonist (e.g., ranibizumab) is about 0.5 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof) is about 1.5 mg, and the dosage of the VEGF antagonist (e.g., bevacizumab) is about 1.25 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 3.0 mg, and the dosage of the VEGF antagonist (e.g., bevacizumab) is about 1.25 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 1.5 mg, and the dosage of the VEGF antagonist (e.g., aflibercept) is about 2.0 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 3.0 mg, and the dosage of the VEGF antagonist (e.g., aflibercept) is about 2.0 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 1.5 mg, and the dosage of the VEGF antagonist, e.g., pegaptanib sodium, is about 1.65 mg. In certain embodiments, a subject is administered both Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist, wherein the dosage of Antagonist A or another pharmaceutically acceptable salt thereof is about 3.0 mg, and the dosage of the VEGF antagonist, e.g., pegaptanib sodium, is about 1.65 mg.

[0292] The dosage can range from about 0.01 mL to about 0.2 mL administered per eye, or about 0.03 mL to about 0.15 mL administered per eye, or about 0.05 mL to about 0.10 mL administered per eye.

[0293] Antagonist A or a pharmaceutically acceptable salt thereof can be delivered intravitreally at up to about 30 mg/ml with injection volumes up to 100 μL.

[0294] Illustrative Antagonist A/VEGF antagonist combination pairs and their dosages are set forth in Table 11:

TABLE 11

Combination No.	PDGF Antagonist	VEGF Antaponist
1	Antagonist A (about 1.5 mg)	ranibizumab (about 0.5 mg)
2	Antagonist A (about 3.0 mg)	ranibizumab (about 0.5 mg)
3	Antagonist A (about 1.5 mg)	bevacizumab (about 1.25 mg)
4	Antagonist A (about 3.0 mg)	bevacizumab (about 1.25 mg)
5	Antagonist A (about 1.5 mg)	aflibercept (about 2.0 mg)
6	Antagonist A (about 3.0 mg)	aflibercept (about 2.0 mg)
7	Antagonist A (about 3.0 mg)	pegaptanib sodium (about 1.65 mg)
8	Antagonist A (about 3.0 mg)	pegaptanib sodium (about 1.65 mg)

[0295] In particular embodiments wherein the subject is administered an anti-C5 agent in combination with Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist, the anti-C5 agent may be administered at a dosage of about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.25 mg, about 1.5 mg, about 2.0 mg or about 3.0 mg per eye.

[0296] In certain embodiments, ocular dosages of compositions comprising anti-C5 aptamers, such as ARC1905 and ARC187, or a pharmaceutically acceptable salt thereof, can range from about 0.01 mg to about 5 mg/eye or from about 0.1 mg to about 3 mg/eye. For instance, ocular dosages of compositions comprising ARC1905, ARC187, or a pharmaceutically acceptable salt thereof may be about 0.01 mg, about 0.03 mg, about 0.05 mg, about 0.1 mg, about 0.3 mg, about 0.5 mg, about 1 mg, about 1.5 mg, about 2 mg, about 2.5 mg, about 3 mg, about 3.5 mg, about 4 mg, about 4.5 mg, or about 5 mg. Such dosages may be administered ocularly, for example by intravitreal injection, weekly, biweekly, monthly, or quarterly, optionally by a sustained release device or formulation. In some embodiments, the anti-C5 aptamers (e.g., ARC1905, ARC187, or a pharmaceutically acceptable salt thereof) can be administered in multiple injections (e.g., intravitreal injections) over a period of months separated by varying time intervals. In certain such embodiments, initial injections received early in the treatment regimen are separated by a shorter interval than injections received later in the treatment regimen. For instance, one dosage regimen, particularly useful in methods for treating, preventing, or stabilizing AMD (e.g., non-exudative type AMD or geographic atrophy), comprises administering initial injections at the start of treatment (e.g., first two, three, four, or five injections) of anti-C5 aptamer (e.g., ARC1905, ARC187, or a pharmaceutically acceptable salt thereof) on a monthly basis and administering subsequent injections at longer intervals (e.g., every three, four, five, or six months). By way of example, the

first three injections of anti-C5 aptamer are administered to a subject every month, whereas the fourth and fifth injections are administered three or four months after the previous injection. Intervals between injections of anti-C5 aptamer may be adjusted based on the subject's response to treatment as measured, for example, by change in geographic atrophy lesion size or improvement or stabilization of visual acuity.

[0297] In some embodiments, an anti-C5 aptamer is administered to a subject with a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 0.03 mg, and the dosage of the VEGF antagonist, e.g., ranibizumab, is about 0.5 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 1.0 mg, and the dosage of the VEGF antagonist, e.g., ranibizumab, is about 0.5 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 2.0 mg, and the dosage of the VEGF antagonist, e.g., ranibizumab, is about 0.5 mg.

[0298] In some embodiments, an anti-C5 aptamer is administered to a subject with a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 0.03 mg, and the dosage of the VEGF antagonist, e.g., bevacizumab, is about 1.25 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 1.0 mg, and the dosage of the VEGF antagonist, e.g., bevacizumab, is about 1.25 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 2.0 mg, and the dosage of the VEGF antagonist, e.g., bevacizumab, is about 1.25 mg.

[0299] In some embodiments, an anti-C5 aptamer is administered to a subject with a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 0.03 mg, and the dosage of the VEGF antagonist, e.g., aflibercept, is about 2.0 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 1.0 mg, and the dosage of the VEGF antagonist, e.g., aflibercept, is about 2.0 mg. In certain embodiments, a subject is administered both an anti-C5 aptamer and a VEGF antagonist, wherein the dosage of the anti-C5 aptamer is about 2.0 mg, and the dosage of the VEGF antagonist, e.g., aflibercept, is about 2.0 mg.

[0300] Administration of each antagonist can, independently, be one to four times daily or one to four times per month or one to six times per year or once every two, three, four or five years. Administration can be for the duration of one day or one month, two months, three months, six months, one year, two years, three years, and may even be for the life of the patient. In one embodiment, the administration is performed once a month for three months. Chronic, long-term administration will be indicated in many cases. The dosage may be administered as a single dose or divided into multiple doses. In general, the desired dosage should be administered at set intervals for a prolonged period, usually at least over several weeks or months, although longer periods of administration of several months or years or more may be needed.

[0301] In addition to treating pre-existing ophthalmological diseases and disorders, the compositions can be administered prophylactically in order to prevent or slow the onset of these disease and disorders. The term "prevent" encompasses inhibiting or delaying the onset or progression of a disease or disorder. In prophylactic applications, the composition can be administered to a patient susceptible to or otherwise at risk of a particular ophthalmological disease or disorder.

[0302] In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist are administered to a subject in need of treatment therewith, typically in the form of an injectable pharmaceutical composition. Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist can be administered either in separate compositions or in a pharmaceutical composition comprising both the PDGF antagonist and VEGF antagonist. The administration can be by injection, for example by intraocular injection, or by using a drug delivery device. Parenteral, systemic, or transdermal administration is also within the scope of the invention. The administration of Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist can be sequential in time or concurrent. When administered sequentially, the administration of each can be by the same or different route. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered within 90 days, 30 days, 10 days, 5 days, 24 hours, 1 hour, 30 minutes, 10 minutes, 5 minutes or one minute of administration of a VEGF antagonist. Where Antagonist A or another pharmaceutically acceptable salt thereof is administered prior to the VEGF antagonist, the VEGF antagonist is administered within a time and in an amount such that the total amount of Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist is effective to treat or prevent an ophthalmological disease or disorder. Where the VEGF antagonist is administered prior to Antagonist A or another pharmaceutically acceptable salt thereof, Antagonist A or another pharmaceutically acceptable salt thereof is administered within a time and in an amount such that the total amount of Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist is effective to treat or prevent an ophthalmological disease or disorder.

[0303] In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof or VEGF antagonist (e.g., ranibizumab, bevacizumab, pegaptanib sodium, ESBA1008 or aflibercept) is administered intravitreally with a 30-gauge or 27-gauge needle. In some embodiments, a 0.5 inch needle is used. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally with a 30-gauge 0.5 inch needle and a VEGF antagonist (e.g., ranibizumab, bevacizumab, pegaptanib sodium, ESBA1008 or aflibercept) is administered intravitreally with a 27-gauge needle. In some embodiments, 50 μL (1.5 mg in 0.05 mL) of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally with a 30-gauge 0.5 inch needle and 50 μL (0.5 mg in 0.05 mL) of a VEGF antagonist (e.g., ranibizumab, bevacizumab, pegaptanib sodium or aflibercept) is administered intravitreally with a 27-gauge needle.

[0304] In certain embodiments where Antagonist A or another pharmaceutically acceptable salt thereof such as Antagonist A or another pharmaceutically acceptable salt thereof is used in combination with a VEGF antagonist, such as ranibizumab, bevacizumab, ESBA1008, pegaptanib sodium or aflibercept, one of these two agents is first administered to the subject, and then the other agent is administered to the subject. In particular embodiments, the two agents are both administered to the same eye of the subject. In particular embodiments, the two agents are both administered to both eyes of the subject. The two agents may be administered to an

eye in either order, i.e., Antagonist A or another pharmaceutically acceptable salt thereof may be administered first, and then the VEGF antagonist administered, or the VEGF antagonist may be administered first, and then Antagonist A or another pharmaceutically acceptable salt thereof administered. The agent administered second may be administered immediately following administration of the agent administered first, or the agent administered second may be administered after a time period following administration of the agent administered first.

[0305] In certain embodiments, the time period from administration of the first agent to administration of the second agent is at least 1 min, at least 5 min, at least 10 min, at least 15 min, at least 30 min, or at least one hour. In certain embodiments, the time period from administration of the first agent to administration of the second agent is between 1 min and 2 hours, between 5 min and 2 hours, between 10 min and 2 hours, between 15 min and 2 hours, between 30 min and 2 hours, between 45 min and 2 hours, between 1 hour and 2 hours, or between 30 min and 1 hour. In certain embodiments, the time period from administration of the first agent to administration of the second agent is about 1 min, about 2 min, about 3 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 35 min, about 40 min, about 45 min, about 50 min, about 55 min, about 60 min, about 90 min, or about 120 min.

[0306] In certain embodiments, the present invention provides methods for treating or preventing any of the ophthalmological diseases described herein, comprising providing to a subject in need thereof. Antagonist A or another pharmaceutically acceptable salt thereof at a first time point, and providing to the subject a VEGF antagonist, e.g., aflibercept, bevacizumab, ranibizumab, ESBA1008, or pegaptanib sodium, at a second time point, wherein the amount of time between the first time point and the second time point is about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days.

[0307] In certain embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and the VEGF antagonist are administered intravitreally. In certain embodiments, about 1.5 mg or 3.0 mg of Antagonist A or another pharmaceutically acceptable salt thereof to an eye, and about 0.5 mg, about 1.25 mg, about 1.65 mg, or about 2.0 mg of the VEGF antagonist is administered to an eye. In some embodiments, the VEGF antagonist is administered intravitreally about 30 minutes after Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally. In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally about 30 minutes after the VEGF antagonist is administered intravitreally.

[0308] In one embodiment, a VEGF antagonist is administered to at least one eye of the subject, about 1 hour is allowed to elapse following administration of the VEGF antagonist, and then Antagonist A or another pharmaceutically acceptable salt thereof is administered to the same eye. In one embodiment, Antagonist A or another pharmaceutically acceptable salt thereof is administered to at least one eye of the subject, about 1 hour is allowed to lapse following administration of the PDGF antagonist, and then a VEGF antagonist is administered to the same eye.

[0309] In certain embodiments, the PDGF antagonist and the VEGF antagonist are administered to each eye in a total combined volume of less than or about 50 μL, less than or about 60 μL, less than or about 70 μL, less than or about 80 μL, less than or about 90 μL, less than or about 100 μL, less than or about 120 μL, less than or about 150 μL, or less than or about 200 μL.

[0310] In certain embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are administered intraocularly, e.g., intravitreally. In particular embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are administered to the mammal via a single injection, e.g., a single intraocular or intravitreal injection. In particular embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are administered sequentially. In certain embodiments, two or more of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist and an anti-C5 agent are administered at the same time, e.g., in the same composition. In particular embodiments, one of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist and an anti-C5 agent is administered, and within about 30 seconds, one or two of others are subsequently administered. In particular embodiments, all three of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist and an anti-C5 agent are administered within about 30 seconds or one minute of each other. In other embodiments, one of Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist and an anti-C5 agent is administered, and one or both of the others are administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days later. In other embodiments, one or two of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are administered, and the other is administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days later. In certain embodiments, one of the PDGF antagonist, VEGF antagonist and anti-C5 agent is administered; and another is administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days later; and the remaining one is administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about

four days, about five days, about six days, or about seven days later. In certain embodiments wherein two of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are present in the same composition, the composition is administered and the PDGF antagonist, VEGF antagonist or anti-C5 agent that is not present in the composition is administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days later. In other embodiments wherein two of Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist and anti-C5 agent are present in the same composition, Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist or anti-C5 agent that is not present in the composition is administered, and the composition is administered about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours, about 24 hours, about 36 hours, about 48 hours, about three days, about four days, about five days, about six days, or about seven days later.

[0311] In certain embodiments, Antagonist A or another pharmaceutically acceptable salt thereof, e.g., Antagonist A or another pharmaceutically acceptable salt thereof, is administered about every 24 hours for two or more, three or more, four or more, five or more, six or more, or seven or more days, and a VEGF antagonist, e.g., aflibercept, bevacizumab, ESBA1008, pegaptanib sodium or ranimizumab, is administered about 48 hours following the first administration of Antagonist A or another pharmaceutically acceptable salt thereof. In certain embodiments, Antagonist A or another pharmaceutically acceptable salt thereof is administered on each of four successive days, i.e., day 1, day 2, day 3 and day 4, and the VEGF antagonist (e.g., bevacizumab, ranicizumab, ESBA1008, pegaptanib sodium or aflibercept) is administered on the third day, i.e., day 3. In particular embodiments, a composition comprising Antagonist A or another pharmaceutically acceptable salt thereof, e.g., Antagonist A or another pharmaceutically acceptable salt thereof, is administered to a subject, and a composition comprising a VEGF antagonist is administered to the subject about forty-eight hours later.

[0312] In one embodiment, about 50 mg/kg of Antagonist A or another pharmaceutically acceptable salt thereof (e.g., Antagonist A or another pharmaceutically acceptable salt thereof) is administered, e.g., intraperitoneally, on day 1, day 2, day 3 and day 4, and about 1 mg/kg of a VEGF antagonist (e.g., bevacizumab, ranibizumab, ESBA1008, pegaptanib sodium, or aflibercept) is administered on day 3. In one embodiment, about 50 mg/kg of Antagonist A or another pharmaceutically acceptable salt thereof (e.g., Antagonist A or another pharmaceutically acceptable salt thereof) is administered on day 1, day 2, day 3 and day 4, and about 5 mg/kg of a VEGF antagonist (e.g., bevacizumab, ranibizumab, ESBA1008, pegaptanib sodium, or aflibercept) is administered on day 3.

[0313] In one embodiment, about 50 mg/kg of Antagonist A or another pharmaceutically acceptable salt thereof is administered on day 1, day 2, day 3 and day 4, and about 1 mg/kg of aflibercept is administered on day 3. In one embodiment, about 50 mg/kg of Antagonist A or another pharmaceutically acceptable salt thereof is administered on day 1, day 2, day 3 and day 4, and about 5 mg/kg of aflibercept is administered on day 3.

[0314] In one embodiment, about 0.03 mg, about 0.3 mg, about 0.5 mg, about 1.0 mg, about 1.5 mg or about 3.0 mg of Antagonist A or another pharmaceutically acceptable salt thereof (e.g., Antagonist A or another pharmaceutically acceptable salt thereof) is administered intravitreally on day 1, day 2, day 3 and day 4, and about 0.5 mg, about 1.0 mg, about 1.5 mg, about 1.65 mg, about 3.0 mg, or about 4.0 mg of a VEGF antagonist (e.g., bevacizumab, ranibizumab, ESBA1008, pegaptanib sodium, or aflibercept) is administered intravitreally on day 3. In one embodiment, about 0.3 mg or about 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally on day 1, day 2, day 3 and day 4, and about 0.5 mg of ranibizumab is administered intravitreally on day 3. In one embodiment, about 0.3 mg or about 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally on day 1, day 2, day 3 and day 4, and about 1.25 mg of bevacizumab is administered intravitreally on day 3. In one embodiment, about 0.3 mg or about 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally on day 1, day 2, day 3 and day 4, and about 2.0 mg of aflibercept is administered intravitreally on day 3. In one embodiment, about 0.3 mg or about 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally on day 1, day 2, day 3 and day 4, and about 1.65 mg of pegaptanib sodium is administered intravitreally on day 3.

[0315] In some embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and VEGF antagonist are administered every four weeks or every 30 days, for six treatments. In some embodiments, the VEGF antagonist is ranibizumab. In some embodiments, 0.3 mg of Antagonist A or another pharmaceutically acceptable salt thereof and 0.5 mg of ranibizumab are administered every four weeks or every 30 days, for six treatments. In some embodiments, 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof and 0.5 mg of ranibizumab are administered every four weeks or every 30 days, for six treatments.

[0316] In some embodiments, 0.3 mg of Antagonist A or another pharmaceutically acceptable salt thereof and 1.25 mg of bevacizumab, 2.0 mg of aflibercept, or 1.65 mg of pegaptanib sodium are administered every four weeks or every 30 days, for six treatments. In some embodiments, 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof and 1.25 mg of bevacizumab, 2.0 mg of aflibercept, or 1.65 mg of pegaptanib sodium are administered every four weeks or every 30 days, for six treatments.

[0317] In some embodiments, the methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof, bevacizumab and aflibercept. In some embodiments, the methods comprise administering Antagonist A or another pharmaceutically acceptable salt thereof, bevacizumab and aflibercept every four weeks or every 30 days, for six treatments. In some embodiments, the methods comprise administering 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof, 1.25 mg of bevacizumab, and 2 mg of aflibercept. In some embodiments, the methods comprise administering 1.5 mg of Antagonist A or another pharmaceutically acceptable salt thereof, 1.25 mg of

bevacizumab, and 2 mg of aflibercept every four weeks or every 30 days, for six treatments.

[0318] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein (a) and (b) are administered in an amount that is effective for treating or preventing an ocular condition (e.g., wet AMD), and wherein the administering occurs once every month, ±about seven days, for 12 consecutive months.

[0319] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein: (a) and (b) are administered in an amount that is effective for treating or preventing an ocular condition (e.g., wet AMD); and the administering occurs once every month, ±about seven days, for a first 12 consecutive months, and immediately thereafter once every two months, ±about seven days, for a second 12 consecutive months, commencing on the second month of the second 12 consecutive months.

[0320] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein: (a) and (b) are administered in an amount that is effective for treating or preventing an ocular condition (e.g., wet AMD); and the administering occurs once every month, ±about seven days, for 24 consecutive months is also provided herein.

[0321] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein: (a) and (b) are administered in an amount that is effective for treating or preventing an ocular condition (e.g., wet AMD); and the administering occurs once every month, ±about seven days, for three consecutive months, and immediately thereafter once every two months, ±about seven days, for 12 consecutive months, commencing on the second month of the 12 consecutive months.

[0322] In some embodiments, the methods comprise continuous treatment, continuous and discontinuous treatments, and/or retreatments, e.g., for the treatment or preventing of wet-type AMD or subfoveal neovascular AMD. In some embodiments, continuous treatment comprises administering to Antagonist A or another pharmaceutically acceptable salt thereof and an anti-VEGF agent monthly (±7 days) for at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, or 12 consecutive months. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered within about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours of administration of the VEGF antagonist. In some embodiments, the VEGF antagonist is administered prior to administration of Antagonist A or a pharmaceutically acceptable salt thereof. In other embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered prior to administration of the VEGF antagonist. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist are administered as a co-formulation. In some embodiments, the amount of Antagonist A or a pharmaceutically acceptable salt thereof administered is about 1.5 mg/eye and the amount of VEGF antagonist administered is about 0.5 mg/eye (e.g., ranibizumab), about 1.25 mg/eye (e.g., bevacizumab), about 1.65 mg/eye (e.g., pegaptanib sodium), or about 2.0 mg/eye (e.g., aflibercept).

[0323] In some embodiments, the methods further comprise measuring the subject's visual acuity. In some embodiments, the subject's visual acuity is measured once every month, ±about seven days. In some embodiments, visual acuity is stable when it is stable for three consecutive months. In some embodiments, visual acuity is stable when at each of the last two of the three consecutive months, visual acuity is within 5 ETDRS letters (better or worse) of the subject's visual acuity at the first of the three consecutive months (i.e., the month immediately preceding the first of the two consecutive following months).

[0324] In some embodiments, a subject is administered in accordance with the present methods until the subject's visual acuity is stable. In some embodiments, a subject is administered in accordance with the present methods until the subject's visual acuity is stable for three consecutive months. In some embodiments, a subject is administered in accordance with the present methods until the subject's visual acuity at each of the last two of the three consecutive months is ≤a five-ETDRS-letter difference from the subject's visual acuity of the first of the three consecutive months. In some embodiments, a subject is administered in accordance with the present methods until the subject experiences no new or significant intraretinal or sub-retinal hemorrhage, or no increase of ≥50 μm in foveal intraretinal fluid. In some embodiments, a subject is administered in accordance with the present methods until the subject's visual acuity measured at each of the last two of the three consecutive months is 5 a five-ETDRS-letter difference from the subject's visual acuity of the first of the three consecutive months, and the subject experiences no new or significant intraretinal or sub-retinal hemorrhage, and no increase of ≥50 μm in foveal intraretinal fluid.

[0325] In some embodiments, discontinuous treatment is administered after continuous treatment, in which discontinuous treatment is based on a physician's discretion, and the subject has stabilized vision as determined by 5 a five-ETDRS-letter difference in the subject's visual acuity after continuous and discontinuous treatment.

[0326] In some embodiments, subjects with a loss of visual acuity of >5 ETDRS letters from the previous monthly assessment, new and significant intraretinal or sub-retinal hemorrhage, and/or an increase of ≥50 μm in foveal intraretinal fluid are retreated.

[0327] In some embodiments, the continuous method comprises administering Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist in an amount that is effective for treating or preventing wet AMD, wherein the administering occurs once every month, ±about seven days, for 12 consecutive months. In some embodiments, the methods further comprise measuring the subject's visual acuity at one month, ±about seven days, immediately following the 12 consecutive months, wherein the subject's visual acuity measured on the twelfth of the 12 consecutive months and the one month immediately following the 12 consecutive months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on the eleventh of the 12 consecutive months.

[0328] In some embodiments, the methods further comprise measuring the subject's visual acuity once every month, ±about seven days, on each of an additional 11 consecutive months. In some embodiments, the subject's visual acuity

measured on any two consecutive months of the additional 11 consecutive months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the two consecutive months.

[0329] In some embodiments, the subject's visual acuity measured on the twelfth of the 12 consecutive months and the one month immediately following the 12 consecutive months is not ≤a five-ETDRS-letter difference in the subject's visual acuity measured on the eleventh of the 12 consecutive months and the subject is retreated. In some embodiments, retreatment comprises administering to the patient on the one month immediately following the 12 consecutive months Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist in an amount that is effective for treating or preventing wet AMD, measuring the patient's visual acuity on a month, ±about seven days, immediately following the one month immediately following the 12 consecutive months, and administering to the subject on each immediately following month Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity on any two consecutive following months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive following months. In some embodiments, the total number of months does not exceed 24.

[0330] In some embodiments, wherein the subject's visual acuity measured on the one month immediately following the 12 consecutive months is not ≤5 a five-ETDRS-letter difference in the subject's visual acuity measured on the twelfth of the 12 consecutive months and is not solely attributable to newly diagnosed foveal atrophy or worsening ocular media opacity, the method further comprises administering to the subject on the one month immediately following the 12 consecutive months Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist in an amount that is effective for treating or preventing wet AMD; and administering to the subject on each immediately following month (a) and (b) in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity measured on any two consecutive following months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive following months. In some embodiments, the total number of months does not exceed 24.

[0331] In some embodiments, wherein the subject presents intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid at one month, ±about seven days, immediately following the 12 consecutive months, the method further comprises administering to the subject on the one month immediately following the 12 consecutive months Antagonist A or another pharmaceutically acceptable salt thereof an a VEGF antagonist in an amount that is effective for treating or preventing wet AMD; and administering to the subject on each immediately following month (a) and (b) in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity measured on any two consecutive following months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive following months. In some embodiments, the total number of months does not exceed 24.

[0332] Also provided herein is a method comprising administering Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist intravitreally once every month, ±about seven days, for a first 12 consecutive months, and immediately thereafter once every two months, ±about seven days, for a second 12 consecutive months, commencing on the second month of the second 12 consecutive months. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered within about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours of administration of the VEGF antagonist. In some embodiments, the VEGF antagonist is administered prior to administration of Antagonist A or a pharmaceutically acceptable salt thereof. In other embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered prior to administration of the VEGF antagonist. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist are administered as a co-formulation. In some embodiments, the amount of Antagonist A or a pharmaceutically acceptable salt thereof administered is about 1.5 mg/eye and the amount of VEGF antagonist administered is about 0.5 mg/eye (e.g., ranibizumab), about 1.25 mg/eye (e.g., bevacizumab), about 1.65 mg/eye (e.g., pegaptanib sodium), or about 2.0 mg/eye (e.g., aflibercept).

[0333] In some embodiments, the method further comprises measuring the subject's visual acuity once every month, ±about seven days, during the first 12 consecutive months and second 12 consecutive months. In some embodiments, the subject's visual acuity measured on any one of the first, third, fifth, seven, ninth and eleventh months of the second consecutive 12 months decreased at least five ETDRS letters relative to the patient's visual acuity measured on the month immediately preceding the first, third, fifth, seven, ninth or eleventh month of the second consecutive 12 months.

[0334] In some embodiments, the methods further comprises administering to the subject an amount of Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist effective for treating or preventing wet AMD on the month in which the subject's visual acuity measured the decrease of at least five ETDRS letters relative to the patient's visual acuity measured on the immediately preceding month.

[0335] In some embodiments, the method further comprises administering Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist on any one of the first, third, fifth, seven, ninth and eleventh months of the second consecutive 12 months.

[0336] In some embodiments, the decrease in visual acuity is attributed to solely newly diagnosed foveal atrophy or opacified ocular media.

[0337] In some embodiments, the subject presents intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid on any one of the first, third, fifth, seven, ninth and eleventh months of the second consecutive 12 months.

[0338] In some embodiments, the method further comprises administering Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist on month in which the subject presents intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid.

[0339] Also provided herein is a method comprising administering Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist intravitreally

once every month, ±about seven days, for 24 consecutive months. In other embodiments, Antagonist A or another pharmaceutically acceptable salt thereof and a VEGF antagonist are administered intravitreally once a month for three months and then every other month for the next 21 months. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered within about 1 min, about 2 min, about 5 min, about 10 min, about 15 min, about 20 min, about 25 min, about 30 min, about 40 min, about 50 min, about 60 min, about 90 min, about 2 hours, about 4 hours, about 6 hours, about 8 hours, about 12 hours of administration of the VEGF antagonist. In some embodiments, the VEGF antagonist is administered prior to administration of Antagonist A or a pharmaceutically acceptable salt thereof. In other embodiments, Antagonist A or a pharmaceutically acceptable salt thereof is administered prior to administration of the VEGF antagonist. In some embodiments, Antagonist A or a pharmaceutically acceptable salt thereof and a VEGF antagonist are administered as a co-formulation. In some embodiments, the amount of Antagonist A or a pharmaceutically acceptable salt thereof administered is about 1.5 mg/eye and the amount of VEGF antagonist administered is about 0.5 mg/eye (e.g., ranibizumab), about 1.25 mg/eye (e.g., bevacizumab), about 1.65 mg/eye (e.g., pegaptanib sodium), or about 2.0 mg/eye (e.g., aflibercept).

[0340] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein (a) and (b) are administered in an amount that is effective for treating or preventing an ophthalmological disease or disorder (e.g., wet AMD), and wherein the administering occurs once every month, ±about seven days, for a first administration period of at least 3 consecutive months, followed by administering (a) and (b) for a second administration period at a frequency of at least every other month±about seven days beginning at two months±about seven days after the day of the last month of the first administration period on which (a) and (b) are administered. In some embodiments, the first administration period is for at least 6 consecutive months. In some embodiments, the VEGF antagonist is ranibizumab or bevacizumab, wherein (a) and (b) are administered at a frequency of once every month±about seven days during the second administration period and wherein the second administration period is at least about nine months.

[0341] In some embodiments, the methods further comprise measuring the subject's visual acuity on a day that is prior to and within about one month of administration of (a) and (b). In some embodiments, the methods further comprise administering to the subject (a) and (b) in an amount that is effective for treating or preventing an an ophthalmological disease or disorder (e.g., wet AMD), until the subject's visual acuity on any two consecutive following months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive following months.

[0342] In some embodiments, the method further comprise administering to the subject (a) and (b) every other month in an amount that is effective for treating or preventing an ophthalmological disease or disorder (e.g., wet AMD), until the subject's visual acuity on any two consecutive visual acuity assessments is not ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a visual acuity assessment immediately preceding the first of the two consecutive visual acuity assessments.

[0343] In other embodiments, the methods further comprise administering to the subject (a) and (b) every month in an amount that is effective for treating or preventing an ophthalmological disease or disorder (e.g., wet AMD), until the subject's visual acuity on any two consecutive following months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive following months.

[0344] In some embodiments, the methods comprise administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) aflibercept, wherein (a) and (b) are administered in an amount that is effective for treating or preventing an ophthalmological disease or disorder (e.g., wet AMD), and wherein the administering occurs once every month, ±about seven days, for a first administration period of at least 3 consecutive months, followed by administering (a) and (b) for a second administration period at a frequency of at least every other month±about seven days beginning at two months±about seven days after the day of the last month of the first administration period on which (a) and (b) are administered.

[0345] In some embodiments, the subject has intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid at one month, ±about seven days, immediately following the second administration period. In some embodiments, the methods further comprise administering to the subject on each month±about seven days, beginning on the month that immediately follows the second administration period (a) and (b) in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity measured on any two consecutive months that follow the 12 consecutive months is ≤a five-ETDRS-letter difference in the subject's visual acuity measured on a month immediately preceding the first of the two consecutive months.

[0346] In some embodiments, the total number of months of treatment does not exceed 24.

[0347] Pharmaceutical compositions according to the invention may be formulated to release Antagonist A or another pharmaceutically acceptable salt thereof, a VEGF antagonist, or an anti-C5 agent, substantially immediately upon administration or at any predetermined time period after administration, using controlled release formulations. For example, a pharmaceutical composition can be provided in sustained-release form. The use of immediate or sustained release compositions depends on the nature of the condition being treated. If the condition consists of an acute disorder, treatment with an immediate release form can be utilized over a prolonged release composition. For certain preventative or long-term treatments, a sustained released composition can also be appropriate.

[0348] Administration of one or both of the antagonists of, or an anti-C5 agent, in controlled release formulations can be useful where the antagonist, either alone or in combination, has (i) a narrow therapeutic index (e.g., the difference between the plasma concentration leading to harmful side effects or toxic reactions and the plasma concentration leading to a therapeutic effect is small; generally, the therapeutic index, TI, is defined as the ratio of median lethal dose (LD_{50}) to median effective dose (ED_{50})); (ii) a narrow absorption window in the gastro-intestinal tract; or (iii) a short biological

half-life, so that frequent dosing during a day is required in order to sustain the plasma level at a therapeutic level.

[0349] Many strategies can be pursued to obtain controlled release in which the rate of release outweighs the rate of degradation or metabolism of the therapeutic antagonist. For example, controlled release can be obtained by the appropriate selection of formulation parameters and ingredients, including, e.g., appropriate controlled release compositions and coatings. Examples include single or multiple unit tablet or capsule compositions, oil solutions, suspensions, emulsions, microcapsules, microspheres, nanoparticles, patches, and liposomes. Methods for preparing such sustained or controlled release formulations are well known in the art.

[0350] Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist, or the anti-C5 agent can also be delivered using a drug-delivery device such as an implant. Such implants can be biodegradable and/or biocompatible, or can be non-biodegradable. The implants can be permeable to Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist, or the anti-C5 agent. Ophthalmic drug delivery devices can be inserted into a chamber of the eye, such as the anterior or posterior chamber or can be implanted in or on the sclera, choroidal space, or an avascularized region exterior to the vitreous. In one embodiment, the implant can be positioned over an avascular region, such as on the sclera, so as to allow for transcleral diffusion of Antagonist A or another pharmaceutically acceptable salt thereof, the VEGF antagonist, or the anti-C5 agent to the desired site of treatment, e.g., the intraocular space and macula of the eye. Furthermore, the site of transcleral diffusion can be proximal to a site of neovascularization such as a site proximal to the macula. Suitable drug delivery devices are described, for example, in U.S. Publication Nos. 2008/0286334; 2008/0145406; 2007/0184089; 2006/0233860; 2005/0244500; 2005/0244471; and 2005/0244462, and U.S. Pat. Nos. 6,808,719 and 5,322,691, the contents of each of which is herein incorporated by reference in its entirety.

[0351] In one embodiment, the implant comprises Antagonist A or another pharmaceutically acceptable salt thereof and/or VEGF antagonist dispersed in a biodegradable polymer matrix. The matrix can comprise PLGA (polylactic acid-polyglycolic acid copolymer), an ester-end capped polymer, an acid end-capped polymer, or a mixture thereof. In another embodiment, the implant comprises Antagonist A or another pharmaceutically acceptable salt thereof and/or a VEGF antagonist, a surfactant, and lipophilic compound. The lipophilic compound can be present in an amount of about 80-99% by weight of the implant. Suitable lipophilic compounds include, but are not limited to, glyceryl palmitostearate, diethylene glycol monostearate, propylene glycol monostearate, glyceryl monostearate, glyceryl monolinoleate, glyceryl monooleate, glyceryl monopalmitate, glyceryl monolaurate, glyceryl dilaurate, glyceryl monomyristate, glyceryl dimyristate, glyceryl monopalmitate, glyceryl dipalmitate, glyceryl monostearate, glyceryl distearate, glyceryl monooleate, glyceryl dioleate, glyceryl monolinoleate, glyceryl dilinoleate, glyceryl monoarachidate, glyceryl diarachidate, glyceryl monobehenate, glyceryl dibehenate, and mixtures thereof. In another embodiment, the implant comprises Antagonist A or another pharmaceutically acceptable salt thereof and/or a VEGF antagonist housed within a hollow sleeve. The PDGF antagonist or VEGF antagonist, or both, are delivered to the eye by inserting the sleeve into the eye, releasing the implant from the sleeve into the eye, and then removing the sleeve from the eye. An example of this delivery device is described in U.S. Publication No. 2005/0244462, which is hereby incorporated by reference in its entirety.

[0352] In one embodiment, the implant is a flexible ocular insert device adapted for the controlled sustained release of Antagonist A or another pharmaceutically acceptable salt thereof and/or a VEGF antagonist into the eye. In one embodiment, the device includes an elongated body of a polymeric material in the form of a rod or tube containing Antagonist A or another pharmaceutically acceptable salt thereof, VEGF antagonist or both, and with at least two anchoring protrusions extending radially outwardly from the body. The device may have a length of at least 8 mm and the diameter of its body portion including the protrusions does not exceed 1.9 mm. The sustained release mechanism can, for example, be by diffusion or by osmosis or bioerosion. The insert device can be inserted into the upper or lower formix of the eye so as to be independent of movement of the eye by virtue of the formix anatomy. The protrusions can be of various shapes such as, for example, ribs, screw threads, dimples or bumps, truncated cone-shaped segments or winding braid segments. In a further embodiment, the polymeric material for the body is selected as one which swells in a liquid environment. Thus a device of smaller initial size can be employed. The insert device can be of a size and configuration such that, upon insertion into the upper or lower formix, the device remains out of the field of vision so as to be well retained in place and imperceptible by a recipient over a prolonged period of use. The device can be retained in the upper or lower formix for 7 to 14 days or longer. An example of this device is described in U.S. Pat. No. 5,322,691, which is hereby incorporated by reference in its entirety.

[0353] Kits

[0354] The invention relates to kits comprising one or more pharmaceutical compositions and instructions for use. At least two antagonists can be formulated together or in separate compositions and in individual dosage amounts. The antagonists are also useful when formulated as pharmaceutically acceptable salts. In one embodiment, the kits comprise a composition comprising Antagonist A or another pharmaceutically acceptable salt thereof and a pharmaceutically acceptable carrier or vehicle and another composition comprising a VEGF antagonist and a pharmaceutically acceptable carrier or vehicle. In another embodiment, the kits comprise a composition comprising a VEGF antagonist, Antagonist A or another pharmaceutically acceptable salt thereof and a pharmaceutically acceptable carrier or vehicle. Each of the kits' compositions can be contained in a container. In some embodiments, the kits comprise an anti-C5 agent.

[0355] The kits can comprise (1) an amount of Antagonist A or another pharmaceutically acceptable salt thereof and a pharmaceutically acceptable carrier, vehicle, or diluent in a first unit dosage form; (2) an amount of a VEGF antagonist and a pharmaceutically acceptable carrier, vehicle, or diluent in a second unit dosage form; and (3) a container. The container can be used to separate components and include, for example, a divided bottle or a divided foil packet. The separate antagonist compositions may also, if desired, be contained within a single, undivided container. In some embodiments, the kits comprise an anti-C5 agent.

[0356] The kits can also comprise directions for the administration of the antagonists. The kits are particularly advanta-

geous when the separate components are administered in different dosage forms, are administered at different dosage levels, or when titration of the individual antagonists is desired.

EXAMPLES

Example 1

Antagonist a and Ranibizumab Combination Therapy for Treating Subfoveal Neovascular Lesions Secondary to Neovascular Age-Related Macular Degeneration (NVAMD)

[0357] In this study, 449 subjects with subfoveal neovascular lesions secondary to NVAMD received six monthly intravitreous injections of Antagonist A given in combination with ranibizumab (administered as Lucentis®, commercially available from Genentech, South San Francisco, Calif.). Antagonist A was injected as the formulation shown in Table 12. The primary efficacy endpoint in the study was the mean change in visual acuity from baseline at the week 24 visit. As pre-specified in the analysis plan, the Hochberg procedure (Hochberg, Y. (1988). A sharper Bonferroni procedure for multiple tests of significance. *Biometrika.* 75, 800-802) was employed to account for multiple dose comparisons.

[0358] The subjects were randomized in a 1:1:1 ratio to the groups shown in Table 13.

TABLE 12

Antagonist A formulation

			30 mg/mL	
Name of Ingredient	Reference to Standards	Function	Solution Composition	Percent (w/v)
Antagonist A	In-house standard	Drug substance	30.0 mg	3%
Monobasic Sodium Phosphate Monohydrate	USP/Ph. Eur	pH buffering agent	0.3 mg	0.03%
Dibasic Sodium Phosphate Heptahydrate	USP/Ph. Eur	pH buffering agent	2.1 mg	0.2%
Sodium Chloride	USP/Ph. Eur	Tonicity adjuster	9.0 mg	0.9%
Hydrochloric Acid	NF/Ph. Eur	pH adjuster	As needed	
Sodium Hydroxide	NF/Ph. Eur	pH adjuster	As needed	
Water for Injection	USP/Ph. Eur	Diluent	q.s.	95.9%
Nitrogen	NF/Ph. Eur	Inert gas overlay	—	—
Total Volume			1 ml	
Volume in Final Drug Product Presentation			230 microliters	

TABLE 13

Antagonist A and Ranibizumab Combination Therapy for Subfoveal Neovascular Lesions Secondary to NVAMD Treatment Groups

Group No.	Group Name	Treatment Regimen
1	Combination Therapy (0.3 mg)	Subjects were administered 0.3 mg/eye of Antagonist A and 0.5 mg/eye of Lucentis ®
2	Combination Therapy (1.5 mg)	Subjects were administered 1.5 mg/eye of Antagonist A and 0.5 mg/eye of Lucentis ®
3	Ranibizumab Monotherapy	Subjects were administered Antagonist A Sham and 0.5 mg/eye of Lucentis ®

[0359] Combination therapy proved superior in terms of mean visual gain when compared to eyes that were treated with anti-VEGF monotherapy. Subjects treated with Lucentis® and either 1.5 mg/eye or 0.3 mg/eye Antagonist A showed an increase in visual acuity compared with those treated with Lucentis® alone (FIG. **2**). The combination of 1.5 mg/eye of Antagonist A and 0.5 mg of Lucentis' met the pre-specified, alpha protected primary endpoint of superiority in mean change of visual acuity gain compared to ranibizumab monotherapy from baseline to 24 weeks (10.6 ETDRS letters at week 24, compared to 6.5 letters, p=0.019, representing a 62% additional benefit). (FIG. **3**) Subjects treated with Lucentis® and either 1.5 mg or 0.3 mg Antagonist A showed a 62% comparative benefit from baseline compared to treatment with Lucentis® alone.

[0360] In addition, the mean change in vision over time demonstrated the benefit of combination therapy at each measured time point over 24 weeks. (FIG. **4**) That benefit was sustained during the study and demonstrated increasing differentiation of the curves at study closure.

[0361] Treatment with 0.5 mg of Lucentis® and either 1.5 mg or 0.3 mg Antagonist A in wet AMD patients also had increased efficacy as compared to patients treated with Lucentis® alone, independent of baseline lesion size or vision. (FIGS. **5**A and **5**B)

[0362] A greater percentage of subjects in the Combination Therapy (1.5 mg) group achieved enhanced visual outcomes compared to those in the Ranibizumab Monotherapy group with respect to multiple treatment endpoints at week 24, as shown in FIG. **6**A, and Table 14.

TABLE 14

Percentage of Subjects in the Combination Therapy (1.5 mg) Group and Ranibizumab Monotherapy Group with Visual Acuity Improvement

	Percentage of Patients	
Treatment Endpoint	Combination Therapy (1.5 mg)	Ranibizumab Monotherapy
>3-lines of visual acuity improvement	36.4%	28.6%
>4-lines of visual acuity improvement	19.9%	11.6%

TABLE 14-continued

Percentage of Subjects in the Combination Therapy (1.5 mg) Group and Ranibizumab Monotherapy Group with Visual Acuity Improvement

	Percentage of Patients	
Treatment Endpoint	Combination Therapy (1.5 mg)	Ranibizumab Monotherapy
>5-lines of visual acuity improvement	11.9%	4.1%
≥20/40 vision after treatment	37.0%	31.9%
≥20/25 vision after treatment	12.3%	5.6%

[0363] Moreover, fewer subjects in the Combination Therapy (1.5 mg) group demonstrated a loss of visual acuity as compared to the number of subjects in the Ranibizumab Monotherapy group at week 24, as shown in FIG. **6**B and Table 15.

TABLE 15

Percentage of Subjects in the Combination Therapy (1.5 mg) Group and Ranibizumab Monotherapy Group with Visual Acuity Loss

	Percentage of Patients	
Treatment Endpoint	Combination Therapy (1.5 mg)	Ranibizumab Monotherapy
≥1-lines of visual acuity loss	8.3%	21.5%
≥2-lines of visual acuity loss	3.4%	12.5%
≤20/125 vision after treatment	19.2%	27.8%
≤20/200 vision after treatment	10.3%	13.9%

[0364] Subjects treated with Lucentis® and 1.5 mg Antagonist A showed improved final visual acuity compared to patients treated with Lucentis® monotherapy. (FIG. **7**) Subjects in the Combination Therapy (1.5 mg) group also showed increased reduction in CNV size in small and large baseline CNV as compared to subjects in the Ranibizumab Monotherapy group (FIGS. **8**A and **8**B).

[0365] Combination therapy was well tolerated. There were no events of endophthalmitis, retinal detachment, retinal tear or iatrogenic traumatic cataract after a total of 4431 intravitreal injections (1776 administrations of Antagonist A and 2655 administrations of Lucentis®). As expected, mean intraocular pressure (IOP) increased after each intravitreal injection consistent with a volume effect. However, mean IOP in all arms returned to pre-injection levels at the next visit, including at the end of the study. The systemic safety profile of combination therapy was similar to that of ranibizumab monotherapy.

[0366] The results of the trial show statistically significant superior efficacy of the combination treatment with Antagonist A and ranibizumab over Lucentis® (ranibizumab) monotherapy for the treatment of wet AMD.

Example 2

ARC1905 for the Treatment of Wet AMD

[0367] Forty-three patients with subfoveal neovascular AMD received six monthly administrations of ARC1905 (0.3 mg/eye, 1 mg/eye or 2 mg/eye) in combination with Lucentis. The mean change in visual acuity at week 24 was an increase of +13.6, +11.7 and +15.3 letters at the doses of 0.3 mg, 1 mg and 2 mg, respectively. Furthermore, 46%, 47% and 60% of patients gained 3 or more lines of visual acuity at the doses of 0.3 mg, 1 mg, and 2 mg, respectively.

Example 3

ARC1905 for the Treatment and Prevention of Dry AMD

[0368] Forty-seven patients with dry AMD were enrolled to receive five intravitreal injections of either 0.3 mg/eye or 1.0 mg/eye of ARC1905 over a 36-week treatment period. FIG. **9** shows the mean change in geographic atrophy (GA) lesion area in dry AMD patients measured at week 24 in patients treated with either 0.3 mg or 1.0 mg doses of ARC1905 at weeks 0, 4, and 8. FIG. **10** shows the mean change in GA lesion in dry AMD patients measured at week 24 and week 48 in patients treated with either 0.3 mg or 1.0 mg doses of ARC1905 at weeks 0, 4, 8, 24, and 36. The results show a dose-dependent reduction in growth of the GA lesion, indicating ARC1905 can slow the progression of GA in non-exudative type AMD patients

Example 4

Visual Acuity Testing Using ETDRS Chart

[0369] Best-corrected visual acuity is measured using standard charts, lighting, and procedures. Best correction is determined by careful refraction at that visit.

[0370] Chart 1 (FIG. **11**) is used for testing the visual acuity of the right eye. Chart 2 (FIG. **12**) is used for testing the left eye. Chart R (FIG. **13**) is used for testing refraction. Subjects do not see any of the charts before the examination.

[0371] A distance of 4 meters is between the subject's eyes and the visual acuity chart. With the box light off, not more than 15 foot-candles of light (161.4 Lux) fall on the center of the chart. To measure the amount of light, the room is set up for visual acuity testing, but with the box light off. The light meter is placed at the fourth line from the top of the chart, with its back against the chart and the reading is taken. If more than one lane is available for testing visual acuity, the visual acuity of an individual subject should be measured in the same lane at each visit. If different lanes are used to test visual acuity, they each meet the same standards.

[0372] Retroilluminated ETDRS charts are used. The illuminator box is either wall-mounted or mounted on a stand (available from Lighthouse Low Vision Services). The light box is mounted at a height such that the top of the third row letter is 49±2 inches from the floor.

[0373] The visual acuity light box is equipped with two 20-watt fluorescent tubes (available from General Electric Cool Daylight) and a ballast which partially covers the tubes. Because the illumination of fluorescent tubes generally diminishes by 5 percent during the first 100 hours and by another 5 percent during the next 2000 hours, new tubes are kept on for 4 days (96 hours) continuously, and replaced once a year.

[0374] A sticker is placed on the back of the light box, indicating the date on which the present tubes were installed. A spare set of burned in bulbs is available.

[0375] Each tube is partly covered by a 14-inch fenestrated sleeve, which is open in the back. This serves as a baffle to reduce illumination. Each sleeve is centered on the tube with the opening towards the back.

[0376] All eyes are tested at 4 meters first, even if the refraction was performed at 1 meter. The subject is seated

comfortably directly in front of the chart so that the eyes remain at the 4 meter distance. Testing begins with the right eye. The subject's left eye is occluded. A folded tissue or eye pad lightly taped over the eye behind the trial frame serves as an effective occluder that allows eccentric fixation without inadvertent use of the covered eye. After testing the right eye, occlusion of the right eye is done before Chart 2 is put up for testing the left eye.

[0377] The lens correction from the subjective refraction is in the trial frame worn by the subject.

[0378] The subject is asked to read the letters slowly, approximately one letter per second. The subject is told that only one chance is given to read each letter on the chart. If the subject is unsure about the identity of the letter, then the subject is encouraged to guess.

[0379] The subject begins by reading the top line of the chart and continue reading every letter on each smaller line, from left to right on each line. The examiner circles every correct letter read and totals each line and the whole column (0 if no letters are correct) on the data collection form. An X is put through letters read incorrectly. Letters, for which no guess was attempted, are not circled. When a subject reaches a level where he/she cannot guess, the examiner may stop the test provided that the subject has made errors on previous guesses, which is a clear indication that the best visual acuity has been obtained.

[0380] When a subject cannot read at least 20 letters on the chart at 4.0 meters, the subject is tested at 1.0 meter. The distance from the subject to the chart should be measured again using the rigid one meter stick. The distance is measured from the outer canthus to the center of the fourth letter (right eye) or the second letter (left eye) of the third line of the chart. The spherical correction in the trial frame should be changed by adding +0.75 to correct for the closer test distance. The subject may fixate eccentrically or turn or shake his/her head to improve visual acuity. If this is done, the examiner ensures that the fellow eye remains occluded both centrally and peripherally and that the subject does not move forward in the chair. Particular care should be taken to ensure the subject does not move forward when testing at 1 meter. The subject is reminded to blink.

[0381] The examiner does not tell the subject if a letter was identified correctly. The subject may be encouraged by neutral comments, such as "good", "next", and "OK".

[0382] The examiner does not stand close to the chart during testing. The examiner's attention is focused on the subject and the data collection form. If the subject has difficulty locating the next line to read, the examiner may go up to the chart and point to the next line to be read, and then moves away from the chart.

[0383] When it is possible to measure the visual acuity of the eye at 4.0 meters (i.e., 20 or more letters read at 4 meters), the visual acuity score for that eye is recorded as the number of letters correct plus 30. The subject gets credit for the 30 1M letters even though they did not have to read them. Otherwise, the visual acuity score is the number of letters read correctly at 1.0 meter plus the number, if any, read at 4M. If no letters are read correctly at either 4.0 meters or 1 meter, then the visual acuity score is recorded as 0.

INCORPORATION BY REFERENCE

[0384] All publications and patent applications disclosed in this specification are herein incorporated by reference to the same extent as if each individual publication or patent application was specifically and individually indicated to be incorporated by reference.

SEQUENCE LISTING

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<221> NAME/KEY: misc_feature
<222> LOCATION: (8)..(8)
<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (9)..(9)

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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (9)..(10)
<223> OTHER INFORMATION: May be linked via hexaethylene glycol
moieties via phosphodiester
linkages
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (14)..(14)
<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (16)..(16)
<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (19)..(19)
<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxyuridine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (20)..(20)
<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (21)..(21)
<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyadenosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (21)..(22)
<223> OTHER INFORMATION: May be linked via hexaethylene glycol
moieties via phosphodiester
linkages
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (26)..(27)
<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (28)..(28)
<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxyuridine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (29)..(29)
<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (30)..(30)
<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 1

caggcuacgc gtagagcauc atgatccugt 30

<210> SEQ ID NO 2
<211> LENGTH: 2137
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 2

ccctgcctgc ctccctgcgc acccgcagcc tcccccgctg cctccctagg gctcccctcc 60

ggccgccagc gcccattttt cattccctag atagagatac tttgcgcgca cacacataca 120

tacgcgcgca aaaaggaaaa aaaaaaaaaa aagcccaccc tccagcctcg ctgcaaagag 180

aaaaccggag cagccgcagc tcgcagctcg cagcccgcag cccgcagagg acgcccagag 240

cggcgagcgg gcgggcagac ggaccgacgg actcgcgccg cgtccacctg tcggccgggc 300

ccagccgagc gcgcagcggg cacgccgcgc gcgcggagca gccgtgcccg ccgcccgggc 360

ccgccgccag ggcgcacacg ctcccgcccc cctacccggc ccgggcggga gtttgcacct 420

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```
ctccctgccc gggtgctcga gctgccgttg caaagccaac tttggaaaaa gtttttggg    480

ggagacttgg gccttgaggt gcccagctcc gcgctttccg attttggggg cctttccaga    540

aaatgttgca aaaaagctaa gccggcgggc agaggaaaac gcctgtagcc ggcgagtgaa    600

gacgaaccat cgactgccgt gttccttttc ctcttggagg ttggagtccc ctgggcgccc    660

ccacacggct agacgcctcg gctggttcgc gacgcagccc cccggccgtg gatgctgcac    720

tcgggctcgg gatccgccca ggtagcggcc tcggacccag gtcctgcgcc caggtcctcc    780

cctgcccccc agcgacggag ccggggccgg gggcggcggc gccgggggca tgcgggtgag    840

ccgcggctgc agaggcctga gcgcctgatc gccgcggacc cgagccgagc ccacccccct    900

ccccagcccc ccaccctggc cgcgggggcg gcgcgctcga tctacgcgtt cggggccccg    960

cggggccggg cccggagtcg gcatgaatcg ctgctgggcg ctcttcctgt ctctctgctg   1020

ctacctgcgt ctggtcagcg ccgaggggga ccccattccc gaggagcttt atgagatgct   1080

gagtgaccac tcgatccgct cctttgatga tctccaacgc ctgctgcacg gagaccccgg   1140

agaggaagat ggggccgagt tggacctgaa catgacccgc tcccactctg gaggcgagct   1200

ggagagcttg gctcgtggaa gaaggagcct gggttccctg accattgctg agccggccat   1260

gatcgccgag tgcaagacgc gcaccgaggt gttcgagatc tcccggcgcc tcatagaccg   1320

caccaacgcc aacttcctgg tgtggccgcc ctgtgtggag gtgcagcgct gctccggctg   1380

ctgcaacaac cgcaacgtgc agtgccgccc cacccaggtg cagctgcgac ctgtccaggt   1440

gagaaagatc gagattgtgc ggaagaagcc aatctttaag aaggccacgg tgacgctgga   1500

agaccacctg gcatgcaagt gtgagacagt ggcagctgca cggcctgtga cccgaagccc   1560

gggggggttcc caggagcagc gagccaaaac gccccaaact cgggtgacca ttcggacggt   1620

gcgagtccgc cggcccccca agggcaagca ccggaaattc aagcacacgc atgacaagac   1680

ggcactgaag gagacccttg gagcctaggg gcatcggcag gagagtgtgt gggcagggtt   1740

atttaatatg gtatttgctg tattgccccc atggggcctt ggagtagata atattgtttc   1800

cctcgtccgt ctgtctcgat gcctgattcg gacggccaat ggtgcctccc ccacccctcc   1860

acgtgtccgt ccacccttcc atcagcgggt ctcctcccag cggcctccgg ctcttgccca   1920

gcagctcaag aagaaaaaga aggactgaac tccatcgcca tcttcttccc ttaactccaa   1980

gaacttggga taagagtgtg agagagactg atggggtcgc tctttggggg aaacgggttc   2040

cttcccctgc acctggcctg ggccacacct gagcgctgtg gactgtcctg aggagccctg   2100

aggacctctc agcatagcct gcctgatccc tgaaccc                            2137
```

<210> SEQ ID NO 3
<211> LENGTH: 241
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 3

```
Met Asn Arg Cys Trp Ala Leu Phe Leu Ser Leu Cys Cys Tyr Leu Arg
1               5                   10                  15

Leu Val Ser Ala Glu Gly Asp Pro Ile Pro Glu Glu Leu Tyr Glu Met
            20                  25                  30

Leu Ser Asp His Ser Ile Arg Ser Phe Asp Asp Leu Gln Arg Leu Leu
        35                  40                  45

His Gly Asp Pro Gly Glu Glu Asp Gly Ala Glu Leu Asp Leu Asn Met
```

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    50                  55                  60

Thr Arg Ser His Ser Gly Gly Glu Leu Glu Ser Leu Ala Arg Gly Arg
65                  70                  75                  80

Arg Ser Leu Gly Ser Leu Thr Ile Ala Glu Pro Ala Met Ile Ala Glu
                85                  90                  95

Cys Lys Thr Arg Thr Glu Val Phe Glu Ile Ser Arg Arg Leu Ile Asp
            100                 105                 110

Arg Thr Asn Ala Asn Phe Leu Val Trp Pro Pro Cys Val Glu Val Gln
        115                 120                 125

Arg Cys Ser Gly Cys Cys Asn Asn Arg Asn Val Gln Cys Arg Pro Thr
    130                 135                 140

Gln Val Gln Leu Arg Pro Val Gln Val Arg Lys Ile Glu Ile Val Arg
145                 150                 155                 160

Lys Lys Pro Ile Phe Lys Lys Ala Thr Val Thr Leu Glu Asp His Leu
                165                 170                 175

Ala Cys Lys Cys Glu Thr Val Ala Ala Ala Arg Pro Val Thr Arg Ser
            180                 185                 190

Pro Gly Gly Ser Gln Glu Gln Arg Ala Lys Thr Pro Gln Thr Arg Val
        195                 200                 205

Thr Ile Arg Thr Val Arg Val Arg Arg Pro Pro Lys Gly Lys His Arg
    210                 215                 220

Lys Phe Lys His Thr His Asp Lys Thr Ala Leu Lys Glu Thr Leu Gly
225                 230                 235                 240

Ala
```

<210> SEQ ID NO 4
<211> LENGTH: 2305
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 4

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ttcttggggc tgatgtccgc aaatatgcag aattaccggc cgggtcgctc ctgaagccag     60

cgcggggagc gagcgcggcg gcggccagca ccgggaacgc accgaggaag aagcccagcc    120

cccgccctcc gccccttccg tccccacccc ctacccggcg gcccaggagg ctccccggct    180

gcggcgcgca ctccctgttt ctcctcctcc tggctggcgc tgcctgcctc tccgcactca    240

ctgctcgccg ggcgccgtcc gccagctccg tgctccccgc gccaccctcc tccgggccgc    300

gctccctaag ggatggtact gaatttcgcc gccacaggag accggctgga gcgcccgccc    360

cgcgcctcgc ctctcctccg agcagccagc gcctcgggac gcgatgagga ccttggcttg    420

cctgctgctc ctcggctgcg gatacctcgc ccatgttctg gccgaggaag ccgagatccc    480

ccgcgaggtg atcgagaggc tggcccgcag tcagatccac agcatccggg acctccagcg    540

actcctggag atagactccg tagggagtga ggattctttg gacaccagcc tgagagctca    600

cggggtccac gccactaagc atgtgcccga gaagcggccc ctgcccattc ggaggaagag    660

aagcatcgag gaagctgtcc ccgctgtctg caagaccagg acggtcattt acgagattcc    720

tcggagtcag gtcgacccca cgtccgccaa cttcctgatc tggcccccgt gcgtggaggt    780

gaaacgctgc accggctgct gcaacacgag cagtgtcaag tgccagccct cccgcgtcca    840

ccaccgcagc gtcaaggtgg ccaaggtgga atacgtcagg aagaagccaa aattaaaaga    900

agtccaggtg aggttagagg agcatttgga gtgcgcctgc gcgaccacaa gcctgaatcc    960
```

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```
ggattatcgg gaagaggaca cggatgtgag gtgaggatga gccgcagccc tttcctggga   1020

catggatgta catggcgtgt tacattcctg aacctactat gtacggtgct ttattgccag   1080

tgtgcggtct ttgttctcct ccgtgaaaaa ctgtgtccga gaacactcgg gagaacaaag   1140

agacagtgca catttgttta atgtgacatc aaagcaagta ttgtagcact cggtgaagca   1200

gtaagaagct tccttgtcaa aaagagagag agagagagag agagagaaaa caaaaccaca   1260

aatgacaaaa acaaaacgga ctcacaaaaa tatctaaact cgatgagatg gagggtcgcc   1320

ccgtgggatg gaagtgcaga ggtctcagca gactggattt ctgtccgggt ggtcacaggt   1380

gctttttttgc cgaggatgca gagcctgctt tgggaacgac tccagagggg tgctggtggg   1440

ctctgcaggg cccgcaggaa gcaggaatgt cttggaaacc gccacgcgaa ctttagaaac   1500

cacacctcct cgctgtagta tttaagccca tacagaaacc ttcctgagag ccttaagtgg   1560

ttttttttttt tgtttttgtt ttgttttttt ttttttgtt ttttttttt ttttttttt   1620

ttacaccata aagtgattat taagcttcct tttactcttt ggctagcttt ttttttttt   1680

ttttttttttt tttttttaa ttatctcttg gatgacattt acaccgataa cacacaggct   1740

gctgtaactg tcaggacagt gcgacggtat ttttcctagc aagatgcaaa ctaatgagat   1800

gtattaaaat aaacatggta tacctaccta tgcatcattt cctaaatgtt tctggctttg   1860

tgtttctccc ttaccctgct ttatttgtta atttaagcca ttttgaaaga actatgcgtc   1920

aaccaatcgt acgccgtccc tgcggcacct gccccagagc ccgtttgtgg ctgagtgaca   1980

acttgttccc cgcagtgcac acctagaatg ctgtgttccc acgcggcacg tgagatgcat   2040

tgccgcttct gtctgtgttg ttggtgtgcc ctggtgccgt ggtggcggtc actccctctg   2100

ctgccagtgt ttggacagaa cccaaattct ttatttttgg taagatattg tgctttacct   2160

gtattaacag aaatgtgtgt gtgtggtttg tttttttgta aaggtgaagt ttgtatgttt   2220

acctaatatt acctgttttg tatacctgag agcctgctat gttcttcttt tgttgatcca   2280

aaattaaaaa aaaaatacca ccaac                                        2305
```

```
<210> SEQ ID NO 5
<211> LENGTH: 196
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 5

Met Arg Thr Leu Ala Cys Leu Leu Leu Leu Gly Cys Gly Tyr Leu Ala
1               5                   10                  15

His Val Leu Ala Glu Glu Ala Glu Ile Pro Arg Glu Val Ile Glu Arg
            20                  25                  30

Leu Ala Arg Ser Gln Ile His Ser Ile Arg Asp Leu Gln Arg Leu Leu
        35                  40                  45

Glu Ile Asp Ser Val Gly Ser Glu Asp Ser Leu Asp Thr Ser Leu Arg
    50                  55                  60

Ala His Gly Val His Ala Thr Lys His Val Pro Glu Lys Arg Pro Leu
65                  70                  75                  80

Pro Ile Arg Arg Lys Arg Ser Ile Glu Glu Ala Val Pro Ala Val Cys
                85                  90                  95

Lys Thr Arg Thr Val Ile Tyr Glu Ile Pro Arg Ser Gln Val Asp Pro
            100                 105                 110
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```
Thr Ser Ala Asn Phe Leu Ile Trp Pro Pro Cys Val Glu Val Lys Arg
        115                 120                 125

Cys Thr Gly Cys Cys Asn Thr Ser Ser Val Lys Cys Gln Pro Ser Arg
    130                 135                 140

Val His His Arg Ser Val Lys Val Ala Lys Val Glu Tyr Val Arg Lys
145                 150                 155                 160

Lys Pro Lys Leu Lys Glu Val Gln Val Arg Leu Glu Glu His Leu Glu
                165                 170                 175

Cys Ala Cys Ala Thr Thr Ser Leu Asn Pro Asp Tyr Arg Glu Glu Asp
            180                 185                 190

Thr Asp Val Arg
        195
```

<210> SEQ ID NO 6
<211> LENGTH: 3018
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 6

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ttggcttcca gtctggctgc gggcaaccct tgagttttcg cctctgtcct gtcccccgaa    120

ctgacaggtg ctcccagcaa cttgctgggg acttctcgcc gctcccccgc gtccccaccc    180

cctcattcct ccctcgcctt caccccccacc cccaccactt cgccacagct caggatttgt    240

ttaaaccttg ggaaactggt tcaggtccag gttttgcttt gatccttttc aaaaactgga    300

gacacagaag agggctctag gaaaaagttt tggatgggat tatgtggaaa ctaccctgcg    360

attctctgct gccagagcag gctcggcgct tccaccccag tgcagccttc ccctggcggt    420

ggtgaaagag actcgggagt cgctgcttcc aaagtgcccg ccgtgagtga gctctcaccc    480

cagtcagcca aatgagcctc ttcgggcttc tcctgctgac atctgccctg gccggccaga    540

gacaggggac tcaggcggaa tccaacctga gtagtaaatt ccagttttcc agcaacaagg    600

aacagaacgg agtacaagat cctcagcatg agagaattat tactgtgtct actaatggaa    660

gtattcacag cccaaggttt cctcatactt atccaagaaa tacggtcttg gtatggagat    720

tagtagcagt agaggaaaat gtatggatac aacttacgtt tgatgaaaga tttgggcttg    780

aagacccaga agatgacata tgcaagtatg attttgtaga agttgaggaa cccagtgatg    840

gaactatatt agggcgctgg tgtggttctg gtactgtacc aggaaaacag atttctaaag    900

gaaatcaaat taggataaga tttgtatctg atgaatattt tccttctgaa ccagggttct    960

gcatccacta caacattgtc atgccacaat tcacagaagc tgtgagtcct tcagtgctac   1020

ccccttcagc tttgccactg gacctgctta ataatgctat aactgccttt agtaccttgg   1080

aagaccttat tcgatatctt gaaccagaga gatggcagtt ggacttagaa gatctatata   1140

ggccaacttg gcaacttctt ggcaaggctt ttgtttttgg aagaaaatcc agagtggtgg   1200

atctgaacct tctaacagag gaggtaagat tatacagctg cacacctcgt aacttctcag   1260

tgtccataag ggaagaacta aagagaaccg ataccatttt ctggccaggt tgtctcctgg   1320

ttaaacgctg tggtgggaac tgtgcctgtt gtctccacaa ttgcaatgaa tgtcaatgtg   1380

tcccaagcaa agttactaaa aaataccacg aggtccttca gttgagacca aagaccggtg   1440

tcaggggatt gcacaaatca ctcaccgacg tggccctgga gcaccatgag gagtgtgact   1500
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gtgtgtgcag agggagcaca ggaggatagc cgcatcacca ccagcagctc ttgcccagag 1560

ctgtgcagtg cagtggctga ttctattaga gaacgtatgc gttatctcca tccttaatct 1620

cagttgtttg cttcaaggac ctttcatctt caggatttac agtgcattct gaaagaggag 1680

acatcaaaca gaattaggag ttgtgcaaca gctcttttga gaggaggcct aaaggacagg 1740

agaaaaggtc ttcaatcgtg gaaagaaaat taaatgttgt attaaataga tcaccagcta 1800

gtttcagagt taccatgtac gtattccact agctgggttc tgtatttcag ttctttcgat 1860

acggcttagg gtaatgtcag tacaggaaaa aaactgtgca agtgagcacc tgattccgtt 1920

gccttgctta actctaaagc tccatgtcct gggcctaaaa tcgtataaaa tctggatttt 1980

ttttttttt tttgctcata ttcacatatg taaaccagaa cattctatgt actacaaacc 2040

tggtttttaa aaaggaacta tgttgctatg aattaaactt gtgtcgtgct gataggacag 2100

actggatttt tcatatttct tattaaaatt tctgccattt agaagaagag aactacattc 2160

atggtttgga agagataaac ctgaaaagaa gagtggcctt atcttcactt tatcgataag 2220

tcagtttatt tgtttcattg tgtacatttt tatattctcc ttttgacatt ataactgttg 2280

gctttttctaa tcttgttaaa tatatctatt tttaccaaag gtatttaata ttctttttta 2340

tgacaactta gatcaactat ttttagcttg gtaaatttttt ctaaacacaa ttgttatagc 2400

cagaggaaca aagatgatat aaaatattgt tgctctgaca aaaatacatg tatttcattc 2460

tcgtatggtg ctagagttag attaatctgc attttaaaaa actgaattgg aatagaattg 2520

gtaagttgca aagacttttt gaaaataatt aaattatcat atcttccatt cctgttattg 2580

gagatgaaaa taaaaagcaa cttatgaaag tagacattca gatccagcca ttactaacct 2640

attccttttt tggggaaatc tgagcctagc tcagaaaaac ataaagcacc ttgaaaaaga 2700

cttggcagct tcctgataaa gcgtgctgtg ctgtgcagta ggaacacatc ctatttattg 2760

tgatgttgtg gttttattat cttaaactct gttccataca cttgtataaa tacatggata 2820

tttttatgta cagaagtatg tctcttaacc agttcactta ttgtactctg gcaatttaaa 2880

agaaaatcag taaaatattt tgcttgtaaa atgcttaata tcgtgcctag gttatgtggt 2940

gactatttga atcaaaaatg tattgaatca tcaaataaaa gaatgtggct attttgggga 3000

gaaaattaaa aaaaaaaa 3018

<210> SEQ ID NO 7
<211> LENGTH: 345
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 7

Met Ser Leu Phe Gly Leu Leu Leu Leu Thr Ser Ala Leu Ala Gly Gln
1 5 10 15

Arg Gln Gly Thr Gln Ala Glu Ser Asn Leu Ser Ser Lys Phe Gln Phe
20 25 30

Ser Ser Asn Lys Glu Gln Asn Gly Val Gln Asp Pro Gln His Glu Arg
35 40 45

Ile Ile Thr Val Ser Thr Asn Gly Ser Ile His Ser Pro Arg Phe Pro
50 55 60

His Thr Tyr Pro Arg Asn Thr Val Leu Val Trp Arg Leu Val Ala Val
65 70 75 80

Glu Glu Asn Val Trp Ile Gln Leu Thr Phe Asp Glu Arg Phe Gly Leu

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                85                  90                  95

Glu Asp Pro Glu Asp Asp Ile Cys Lys Tyr Asp Phe Val Glu Val Glu
            100                 105                 110

Glu Pro Ser Asp Gly Thr Ile Leu Gly Arg Trp Cys Gly Ser Gly Thr
        115                 120                 125

Val Pro Gly Lys Gln Ile Ser Lys Gly Asn Gln Ile Arg Ile Arg Phe
    130                 135                 140

Val Ser Asp Glu Tyr Phe Pro Ser Glu Pro Gly Phe Cys Ile His Tyr
145                 150                 155                 160

Asn Ile Val Met Pro Gln Phe Thr Glu Ala Val Ser Pro Ser Val Leu
                165                 170                 175

Pro Pro Ser Ala Leu Pro Leu Asp Leu Leu Asn Asn Ala Ile Thr Ala
            180                 185                 190

Phe Ser Thr Leu Glu Asp Leu Ile Arg Tyr Leu Glu Pro Glu Arg Trp
        195                 200                 205

Gln Leu Asp Leu Glu Asp Leu Tyr Arg Pro Thr Trp Gln Leu Leu Gly
    210                 215                 220

Lys Ala Phe Val Phe Gly Arg Lys Ser Arg Val Val Asp Leu Asn Leu
225                 230                 235                 240

Leu Thr Glu Glu Val Arg Leu Tyr Ser Cys Thr Pro Arg Asn Phe Ser
                245                 250                 255

Val Ser Ile Arg Glu Glu Leu Lys Arg Thr Asp Thr Ile Phe Trp Pro
            260                 265                 270

Gly Cys Leu Leu Val Lys Arg Cys Gly Gly Asn Cys Ala Cys Cys Leu
        275                 280                 285

His Asn Cys Asn Glu Cys Gln Cys Val Pro Ser Lys Val Thr Lys Lys
    290                 295                 300

Tyr His Glu Val Leu Gln Leu Arg Pro Lys Thr Gly Val Arg Gly Leu
305                 310                 315                 320

His Lys Ser Leu Thr Asp Val Ala Leu Glu His His Glu Glu Cys Asp
                325                 330                 335

Cys Val Cys Arg Gly Ser Thr Gly Gly
            340                 345


<210> SEQ ID NO 8
<211> LENGTH: 3997
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 8

tctcaggggc cgcggccggg gctggagaac gctgctgctc cgctcgcctg ccccgctaga     60

ttcggcgctg cccgcccccc gcagcctgtg ctgcagctgc cggccaccgg agggggcgaa    120

caaacaaacg tcaacctgtt gtttgtcccg tcaccattta tcagctcagc accacaagga    180

agtgcggcac ccacacgcgc tcggaaagtt cagcatgcag gaagtttggg gagagctcgg    240

cgattagcac agcgacccgg gccagcgcag ggcgagcgca ggcggcgaga gcgcagggcg    300

gcgcggcgtc ggtcccggga gcagaacccg gctttttctt ggagcgacgc tgtctctagt    360

cgctgatccc aaatgcaccg gctcatcttt gtctacactc taatctgcgc aaacttttgc    420

agctgtcggg acacttctgc aaccccgcag agcgcatcca tcaaagcttt gcgcaacgcc    480

aacctcaggc gagatgagag caatcacctc acagacttgt accgaagaga tgagaccatc    540
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caggtgaaag gaaacggcta cgtgcagagt cctagattcc cgaacagcta ccccaggaac 600

ctgctcctga catggcggct tcactctcag gagaatacac ggatacagct agtgtttgac 660

aatcagtttg gattagagga agcagaaaat gatatctgta ggtatgattt tgtggaagtt 720

gaagatatat ccgaaaccag taccattatt agaggacgat ggtgtggaca caaggaagtt 780

cctccaagga taaaatcaag aacgaaccaa attaaaatca cattcaagtc cgatgactac 840

tttgtggcta aacctggatt caagatttat tattctttgc tggaagattt ccaacccgca 900

gcagcttcag agaccaactg ggaatctgtc acaagctcta tttcaggggt atcctataac 960

tctccatcag taacggatcc cactctgatt gcggatgctc tggacaaaaa aattgcagaa 1020

tttgatacag tggaagatct gctcaagtac ttcaatccag agtcatggca agaagatctt 1080

gagaatatgt atctggacac ccctcggtat cgaggcaggt cataccatga ccggaagtca 1140

aaagttgacc tggataggct caatgatgat gccaagcgtt acagttgcac tcccaggaat 1200

tactcggtca atataagaga agagctgaag ttggccaatg tggtcttctt tccacgttgc 1260

ctcctcgtgc agcgctgtgg aggaaattgt ggctgtggaa ctgtcaactg gaggtcctgc 1320

acatgcaatt cagggaaaac cgtgaaaaag tatcatgagg tattacagtt tgagcctggc 1380

cacatcaaga ggaggggtag agctaagacc atggctctag ttgacatcca gttggatcac 1440

catgaacgat gtgattgtat ctgcagctca agaccacctc gataagagaa tgtgcacatc 1500

cttacattaa gcctgaaaga acctttagtt taaggagggt gagataagag acccttttcc 1560

taccagcaac caaacttact actagcctgc aatgcaatga acacaagtgg ttgctgagtc 1620

tcagccttgc tttgttaatg ccatggcaag tagaaaggta tatcatcaac ttctatacct 1680

aagaatatag gattgcattt aataatagtg tttgaggtta tatatgcaca aacacacaca 1740

gaaatatatt catgtctatg tgtatataga tcaaatgttt tttttggtat atataaccag 1800

gtacaccaga gcttacatat gtttgagtta gactcttaaa atcctttgcc aaaataaggg 1860

atggtcaaat atatgaaaca tgtctttaga aaatttagga gataaattta tttttaaatt 1920

ttgaaacaca aaacaatttt gaatcttgct ctcttaaaga aagcatcttg tatattaaaa 1980

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tggtttccag agaaaaggcc aatacctaag catttttttcc atgagaagca ctgcatactt 2100

acctatgtgg actataataa cctgtctcca aaaccatgcc ataataatat aagtgcttta 2160

gaaattaaat cattgtgttt tttatgcatt ttgctgaggc atgcttattc atttaacacc 2220

tatctcaaaa acttacttag aaggtttttt attatagtcc tacaaaagac aatgtataag 2280

ctgtaacaga attttgaatt gtttttcttt gcaaaacccc tccacaaaag caaatccttt 2340

caagaatggc atgggcattc tgtatgaacc tttccagatg gtgttcagtg aaagatgtgg 2400

gtagttgaga acttaaaaag tgaacattga aacatcgacg taactggaaa ttaggtggga 2460

tatttgatag gatccatatc taataatgga ttcgaactct ccaaactaca ccaattaatt 2520

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aataaatcca catttccgca tggctcattc acctggaata atgcctttta ttgaatatgt 3840

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<210> SEQ ID NO 9
<211> LENGTH: 370
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 9

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1 5 10 15

Ser Cys Arg Asp Thr Ser Ala Thr Pro Gln Ser Ala Ser Ile Lys Ala
20 25 30

Leu Arg Asn Ala Asn Leu Arg Arg Asp Glu Ser Asn His Leu Thr Asp
35 40 45

Leu Tyr Arg Arg Asp Glu Thr Ile Gln Val Lys Gly Asn Gly Tyr Val
50 55 60

Gln Ser Pro Arg Phe Pro Asn Ser Tyr Pro Arg Asn Leu Leu Leu Thr
65 70 75 80

Trp Arg Leu His Ser Gln Glu Asn Thr Arg Ile Gln Leu Val Phe Asp
85 90 95

Asn Gln Phe Gly Leu Glu Glu Ala Glu Asn Asp Ile Cys Arg Tyr Asp
100 105 110

Phe Val Glu Val Glu Asp Ile Ser Glu Thr Ser Thr Ile Ile Arg Gly
115 120 125

Arg Trp Cys Gly His Lys Glu Val Pro Pro Arg Ile Lys Ser Arg Thr
130 135 140

Asn Gln Ile Lys Ile Thr Phe Lys Ser Asp Asp Tyr Phe Val Ala Lys

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145                 150                 155                 160

Pro Gly Phe Lys Ile Tyr Tyr Ser Leu Leu Glu Asp Phe Gln Pro Ala
                165                 170                 175

Ala Ala Ser Glu Thr Asn Trp Glu Ser Val Thr Ser Ser Ile Ser Gly
            180                 185                 190

Val Ser Tyr Asn Ser Pro Ser Val Thr Asp Pro Thr Leu Ile Ala Asp
        195                 200                 205

Ala Leu Asp Lys Lys Ile Ala Glu Phe Asp Thr Val Glu Asp Leu Leu
    210                 215                 220

Lys Tyr Phe Asn Pro Glu Ser Trp Gln Glu Asp Leu Glu Asn Met Tyr
225                 230                 235                 240

Leu Asp Thr Pro Arg Tyr Arg Gly Arg Ser Tyr His Asp Arg Lys Ser
                245                 250                 255

Lys Val Asp Leu Asp Arg Leu Asn Asp Asp Ala Lys Arg Tyr Ser Cys
            260                 265                 270

Thr Pro Arg Asn Tyr Ser Val Asn Ile Arg Glu Glu Leu Lys Leu Ala
        275                 280                 285

Asn Val Val Phe Phe Pro Arg Cys Leu Leu Val Gln Arg Cys Gly Gly
    290                 295                 300

Asn Cys Gly Cys Gly Thr Val Asn Trp Arg Ser Cys Thr Cys Asn Ser
305                 310                 315                 320

Gly Lys Thr Val Lys Lys Tyr His Glu Val Leu Gln Phe Glu Pro Gly
                325                 330                 335

His Ile Lys Arg Arg Gly Arg Ala Lys Thr Met Ala Leu Val Asp Ile
            340                 345                 350

Gln Leu Asp His His Glu Arg Cys Asp Cys Ile Cys Ser Ser Arg Pro
        355                 360                 365

Pro Arg
    370
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<210> SEQ ID NO 10
<211> LENGTH: 3979
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 10

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agtgcggcac ccacacgcgc tcggaaagtt cagcatgcag gaagtttggg gagagctcgg    240

cgattagcac agcgacccgg gccagcgcag ggcgagcgca ggcggcgaga gcgcagggcg    300

gcgcggcgtc ggtcccggga gcagaacccg gctttttctt ggagcgacgc tgtctctagt    360

cgctgatccc aaatgcaccg gctcatcttt gtctacactc taatctgcgc aaacttttgc    420

agctgtcggg acacttctgc aaccccgcag agcgcatcca tcaaagcttt gcgcaacgcc    480

aacctcaggc gagatgactt gtaccgaaga gatgagacca tccaggtgaa aggaaacggc    540

tacgtgcaga gtcctagatt cccgaacagc taccccagga acctgctcct gacatggcgg    600

cttcactctc aggagaatac acggatacag ctagtgtttg acaatcagtt tggattagag    660

gaagcagaaa atgatatctg taggtatgat tttgtggaag ttgaagatat atccgaaacc    720
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ttcaagattt attattcttt gctggaagat ttccaacccg cagcagcttc agagaccaac 900

tgggaatctg tcacaagctc tatttcaggg gtatcctata actctccatc agtaacggat 960

cccactctga ttgcggatgc tctggacaaa aaaattgcag aatttgatac agtggaagat 1020

ctgctcaagt acttcaatcc agagtcatgg caagaagatc ttgagaatat gtatctggac 1080

acccctcggt atcgaggcag gtcataccat gaccggaagt caaaagttga cctggatagg 1140

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gaagagctga agttggccaa tgtggtcttc tttccacgtt gcctcctcgt gcagcgctgt 1260

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aatcagtcct agaggctatg gttcttaatc tatggagctt gctttaagaa gccagttgtc 2760

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aaagtcaaaa tgaacgttac atcactctaa cctgaacagc tcacaatgta gctgtaaata   3480

taaaaaatga gagtgttcta cccagttttc aataaacctt ccaggctgca ataaccagca   3540

aggttttcag ttaaagccct atctgcactt tttatttatt agctgaaatg taagcaggca   3600

tattcactca cttttctttg cctttcctga gagttttatt aaaacttctc ccttggttac   3660

ctgttatctt ttgcacttct aacatgtagc caataaatct atttgatagc catcaaagga   3720

ataaaaagct ggccgtacaa attacatttc aaaacaaacc ctaataaatc cacatttccg   3780

catggctcat tcacctggaa taatgccttt tattgaatat gttcttatag ggcaaaacac   3840

tttcataagt agagtttttt atgtttttg tcatatcggt aacatgcagc tttttcctct   3900

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ttttgaacaa aactaaaaa                                               3979
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<210> SEQ ID NO 11
<211> LENGTH: 364
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 11

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Met His Arg Leu Ile Phe Val Tyr Thr Leu Ile Cys Ala Asn Phe Cys
1               5                   10                  15

Ser Cys Arg Asp Thr Ser Ala Thr Pro Gln Ser Ala Ser Ile Lys Ala
            20                  25                  30

Leu Arg Asn Ala Asn Leu Arg Arg Asp Asp Leu Tyr Arg Arg Asp Glu
        35                  40                  45

Thr Ile Gln Val Lys Gly Asn Gly Tyr Val Gln Ser Pro Arg Phe Pro
    50                  55                  60

Asn Ser Tyr Pro Arg Asn Leu Leu Leu Thr Trp Arg Leu His Ser Gln
65                  70                  75                  80

Glu Asn Thr Arg Ile Gln Leu Val Phe Asp Asn Gln Phe Gly Leu Glu
                85                  90                  95

Glu Ala Glu Asn Asp Ile Cys Arg Tyr Asp Phe Val Glu Val Glu Asp
            100                 105                 110

Ile Ser Glu Thr Ser Thr Ile Ile Arg Gly Arg Trp Cys Gly His Lys
        115                 120                 125

Glu Val Pro Pro Arg Ile Lys Ser Arg Thr Asn Gln Ile Lys Ile Thr
    130                 135                 140

Phe Lys Ser Asp Asp Tyr Phe Val Ala Lys Pro Gly Phe Lys Ile Tyr
145                 150                 155                 160

Tyr Ser Leu Leu Glu Asp Phe Gln Pro Ala Ala Ala Ser Glu Thr Asn
                165                 170                 175

Trp Glu Ser Val Thr Ser Ser Ile Ser Gly Val Ser Tyr Asn Ser Pro
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            180                 185                 190

Ser Val Thr Asp Pro Thr Leu Ile Ala Asp Ala Leu Asp Lys Lys Ile
        195                 200                 205

Ala Glu Phe Asp Thr Val Glu Asp Leu Leu Lys Tyr Phe Asn Pro Glu
    210                 215                 220

Ser Trp Gln Glu Asp Leu Glu Asn Met Tyr Leu Asp Thr Pro Arg Tyr
225                 230                 235                 240

Arg Gly Arg Ser Tyr His Asp Arg Lys Ser Lys Val Asp Leu Asp Arg
                245                 250                 255

Leu Asn Asp Asp Ala Lys Arg Tyr Ser Cys Thr Pro Arg Asn Tyr Ser
            260                 265                 270

Val Asn Ile Arg Glu Glu Leu Lys Leu Ala Asn Val Val Phe Phe Pro
        275                 280                 285

Arg Cys Leu Leu Val Gln Arg Cys Gly Gly Asn Cys Gly Cys Gly Thr
    290                 295                 300

Val Asn Trp Arg Ser Cys Thr Cys Asn Ser Gly Lys Thr Val Lys Lys
305                 310                 315                 320

Tyr His Glu Val Leu Gln Phe Glu Pro Gly His Ile Lys Arg Arg Gly
                325                 330                 335

Arg Ala Lys Thr Met Ala Leu Val Asp Ile Gln Leu Asp His His Glu
            340                 345                 350

Arg Cys Asp Cys Ile Cys Ser Ser Arg Pro Pro Arg
        355                 360


<210> SEQ ID NO 12
<211> LENGTH: 6574
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 12

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tacttagagc aaatgattag ttttagaagg atggactata acattgaatc aattacaaaa    180

cgcggttttt gagcccatta ctgttggagc tacagggaga gaaacagagg aggagactgc    240

aagagatcat tggaggccgt gggcacgctc tttactccat gtgtgggaca ttcattgcgg    300

aataacatcg gaggagaagt ttcccagagc tatggggact tcccatccgg cgttcctggt    360

cttaggctgt cttctcacag ggctgagcct aatcctctgc cagctttcat taccctctat    420

ccttccaaat gaaaatgaaa aggttgtgca gctgaattca tccttttctc tgagatgctt    480

tggggagagt gaagtgagct ggcagtaccc catgtctgaa gaagagagct ccgatgtgga    540

aatcagaaat gaagaaaaca acagcggcct ttttgtgacg gtcttggaag tgagcagtgc    600

ctcggcggcc cacacagggt tgtacacttg ctattacaac cacactcaga cagaagagaa    660

tgagcttgaa ggcaggcaca tttacatcta tgtgccagac ccagatgtag cctttgtacc    720

tctaggaatg acggattatt tagtcatcgt ggaggatgat gattctgcca ttataccttg    780

tcgcacaact gatcccgaga ctcctgtaac cttacacaac agtgaggggg tggtacctgc    840

ctcctacgac agcagacagg gctttaatgg gaccttcact gtagggccct atatctgtga    900

ggccaccgtc aaaggaaaga agttccagac catcccattt aatgtttatg ctttaaaagc    960

aacatcagag ctggatctag aaatggaagc tcttaaaacc gtgtataagt caggggaaac   1020
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aactcaagtt ccttcatcca ttctggactt ggtcgatgat caccatggct caactggggg 1620

acagacggtg aggtgcacag ctgaaggcac gccgcttcct gatattgagt ggatgatatg 1680

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gaaccgagag ctgaagctgg tggctcccac cctgcgttct gaactcacgg tggctgctgc 1920

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<210> SEQ ID NO 13
<211> LENGTH: 1089
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 13

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Gly Leu Ser Leu Ile Leu Cys Gln Leu Ser Leu Pro Ser Ile Leu Pro
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Asn Glu Asn Glu Lys Val Val Gln Leu Asn Ser Ser Phe Ser Leu Arg
35 40 45

Cys Phe Gly Glu Ser Glu Val Ser Trp Gln Tyr Pro Met Ser Glu Glu
50 55 60

Glu Ser Ser Asp Val Glu Ile Arg Asn Glu Glu Asn Asn Ser Gly Leu
65 70 75 80

Phe Val Thr Val Leu Glu Val Ser Ser Ala Ser Ala Ala His Thr Gly
85 90 95

Leu Tyr Thr Cys Tyr Tyr Asn His Thr Gln Thr Glu Glu Asn Glu Leu
100 105 110

Glu Gly Arg His Ile Tyr Ile Tyr Val Pro Asp Pro Asp Val Ala Phe
115 120 125

Val Pro Leu Gly Met Thr Asp Tyr Leu Val Ile Val Glu Asp Asp Asp
130 135 140

Ser Ala Ile Ile Pro Cys Arg Thr Thr Asp Pro Glu Thr Pro Val Thr
145 150 155 160

Leu His Asn Ser Glu Gly Val Val Pro Ala Ser Tyr Asp Ser Arg Gln
165 170 175

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Gly Phe Asn Gly Thr Phe Thr Val Gly Pro Tyr Ile Cys Glu Ala Thr
            180                 185                 190

Val Lys Gly Lys Lys Phe Gln Thr Ile Pro Phe Asn Val Tyr Ala Leu
        195                 200                 205

Lys Ala Thr Ser Glu Leu Asp Leu Glu Met Glu Ala Leu Lys Thr Val
    210                 215                 220

Tyr Lys Ser Gly Glu Thr Ile Val Val Thr Cys Ala Val Phe Asn Asn
225                 230                 235                 240

Glu Val Val Asp Leu Gln Trp Thr Tyr Pro Gly Glu Val Lys Gly Lys
                245                 250                 255

Gly Ile Thr Met Leu Glu Glu Ile Lys Val Pro Ser Ile Lys Leu Val
            260                 265                 270

Tyr Thr Leu Thr Val Pro Glu Ala Thr Val Lys Asp Ser Gly Asp Tyr
        275                 280                 285

Glu Cys Ala Ala Arg Gln Ala Thr Arg Glu Val Lys Glu Met Lys Lys
    290                 295                 300

Val Thr Ile Ser Val His Glu Lys Gly Phe Ile Glu Ile Lys Pro Thr
305                 310                 315                 320

Phe Ser Gln Leu Glu Ala Val Asn Leu His Glu Val Lys His Phe Val
                325                 330                 335

Val Glu Val Arg Ala Tyr Pro Pro Pro Arg Ile Ser Trp Leu Lys Asn
            340                 345                 350

Asn Leu Thr Leu Ile Glu Asn Leu Thr Glu Ile Thr Thr Asp Val Glu
        355                 360                 365

Lys Ile Gln Glu Ile Arg Tyr Arg Ser Lys Leu Lys Leu Ile Arg Ala
    370                 375                 380

Lys Glu Glu Asp Ser Gly His Tyr Thr Ile Val Ala Gln Asn Glu Asp
385                 390                 395                 400

Ala Val Lys Ser Tyr Thr Phe Glu Leu Leu Thr Gln Val Pro Ser Ser
                405                 410                 415

Ile Leu Asp Leu Val Asp Asp His His Gly Ser Thr Gly Gly Gln Thr
            420                 425                 430

Val Arg Cys Thr Ala Glu Gly Thr Pro Leu Pro Asp Ile Glu Trp Met
        435                 440                 445

Ile Cys Lys Asp Ile Lys Lys Cys Asn Asn Glu Thr Ser Trp Thr Ile
    450                 455                 460

Leu Ala Asn Asn Val Ser Asn Ile Ile Thr Glu Ile His Ser Arg Asp
465                 470                 475                 480

Arg Ser Thr Val Glu Gly Arg Val Thr Phe Ala Lys Val Glu Glu Thr
                485                 490                 495

Ile Ala Val Arg Cys Leu Ala Lys Asn Leu Leu Gly Ala Glu Asn Arg
            500                 505                 510

Glu Leu Lys Leu Val Ala Pro Thr Leu Arg Ser Glu Leu Thr Val Ala
        515                 520                 525

Ala Ala Val Leu Val Leu Leu Val Ile Val Ile Ile Ser Leu Ile Val
    530                 535                 540

Leu Val Val Ile Trp Lys Gln Lys Pro Arg Tyr Glu Ile Arg Trp Arg
545                 550                 555                 560

Val Ile Glu Ser Ile Ser Pro Asp Gly His Glu Tyr Ile Tyr Val Asp
                565                 570                 575

Pro Met Gln Leu Pro Tyr Asp Ser Arg Trp Glu Phe Pro Arg Asp Gly
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Leu Val Leu Gly Arg Val Leu Gly Ser Gly Ala Phe Gly Lys Val Val
        595                 600                 605

Glu Gly Thr Ala Tyr Gly Leu Ser Arg Ser Gln Pro Val Met Lys Val
    610                 615                 620

Ala Val Lys Met Leu Lys Pro Thr Ala Arg Ser Ser Glu Lys Gln Ala
625                 630                 635                 640

Leu Met Ser Glu Leu Lys Ile Met Thr His Leu Gly Pro His Leu Asn
                645                 650                 655

Ile Val Asn Leu Leu Gly Ala Cys Thr Lys Ser Gly Pro Ile Tyr Ile
            660                 665                 670

Ile Thr Glu Tyr Cys Phe Tyr Gly Asp Leu Val Asn Tyr Leu His Lys
        675                 680                 685

Asn Arg Asp Ser Phe Leu Ser His His Pro Glu Lys Pro Lys Lys Glu
    690                 695                 700

Leu Asp Ile Phe Gly Leu Asn Pro Ala Asp Glu Ser Thr Arg Ser Tyr
705                 710                 715                 720

Val Ile Leu Ser Phe Glu Asn Asn Gly Asp Tyr Met Asp Met Lys Gln
                725                 730                 735

Ala Asp Thr Thr Gln Tyr Val Pro Met Leu Glu Arg Lys Glu Val Ser
            740                 745                 750

Lys Tyr Ser Asp Ile Gln Arg Ser Leu Tyr Asp Arg Pro Ala Ser Tyr
        755                 760                 765

Lys Lys Lys Ser Met Leu Asp Ser Glu Val Lys Asn Leu Leu Ser Asp
    770                 775                 780

Asp Asn Ser Glu Gly Leu Thr Leu Leu Asp Leu Leu Ser Phe Thr Tyr
785                 790                 795                 800

Gln Val Ala Arg Gly Met Glu Phe Leu Ala Ser Lys Asn Cys Val His
                805                 810                 815

Arg Asp Leu Ala Ala Arg Asn Val Leu Leu Ala Gln Gly Lys Ile Val
            820                 825                 830

Lys Ile Cys Asp Phe Gly Leu Ala Arg Asp Ile Met His Asp Ser Asn
        835                 840                 845

Tyr Val Ser Lys Gly Ser Thr Phe Leu Pro Val Lys Trp Met Ala Pro
    850                 855                 860

Glu Ser Ile Phe Asp Asn Leu Tyr Thr Thr Leu Ser Asp Val Trp Ser
865                 870                 875                 880

Tyr Gly Ile Leu Leu Trp Glu Ile Phe Ser Leu Gly Gly Thr Pro Tyr
                885                 890                 895

Pro Gly Met Met Val Asp Ser Thr Phe Tyr Asn Lys Ile Lys Ser Gly
            900                 905                 910

Tyr Arg Met Ala Lys Pro Asp His Ala Thr Ser Glu Val Tyr Glu Ile
        915                 920                 925

Met Val Lys Cys Trp Asn Ser Glu Pro Glu Lys Arg Pro Ser Phe Tyr
    930                 935                 940

His Leu Ser Glu Ile Val Glu Asn Leu Leu Pro Gly Gln Tyr Lys Lys
945                 950                 955                 960

Ser Tyr Glu Lys Ile His Leu Asp Phe Leu Lys Ser Asp His Pro Ala
                965                 970                 975

Val Ala Arg Met Arg Val Asp Ser Asp Asn Ala Tyr Ile Gly Val Thr
            980                 985                 990
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Tyr Lys Asn Glu Glu Asp Lys Leu  Lys Asp Trp Glu Gly  Gly Leu Asp
        995                1000                1005

Glu Gln  Arg Leu Ser Ala Asp  Ser Gly Tyr Ile Ile  Pro Leu Pro
    1010                1015                1020

Asp Ile  Asp Pro Val Pro Glu  Glu Glu Asp Leu Gly  Lys Arg Asn
    1025                1030                1035

Arg His  Ser Ser Gln Thr Ser  Glu Glu Ser Ala Ile  Glu Thr Gly
    1040                1045                1050

Ser Ser  Ser Ser Thr Phe Ile  Lys Arg Glu Asp Glu  Thr Ile Glu
    1055                1060                1065

Asp Ile  Asp Met Met Asp Asp  Ile Gly Ile Asp Ser  Ser Asp Leu
    1070                1075                1080

Val Glu  Asp Ser Phe Leu
    1085


<210> SEQ ID NO 14
<211> LENGTH: 5718
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 14

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agccctgctg cccagcagca gcctgtgctc gccctgccca acgcagacag ccagacccag    180

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caatgtctcc agcaccttcg ttctgacctg ctcgggttca gctccggtgg tgtgggaacg    660

gatgtcccag gagcccccac aggaaatggc caaggcccag gatggcacct tctccagcgt    720

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<210> SEQ ID NO 15
<211> LENGTH: 1106
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

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<400> SEQUENCE: 15

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Leu Leu Leu Ser Leu Leu Leu Leu Leu Glu Pro Gln Ile Ser Gln Gly
            20                  25                  30

Leu Val Val Thr Pro Pro Gly Pro Glu Leu Val Leu Asn Val Ser Ser
        35                  40                  45

Thr Phe Val Leu Thr Cys Ser Gly Ser Ala Pro Val Val Trp Glu Arg
    50                  55                  60

Met Ser Gln Glu Pro Pro Gln Glu Met Ala Lys Ala Gln Asp Gly Thr
65                  70                  75                  80

Phe Ser Ser Val Leu Thr Leu Thr Asn Leu Thr Gly Leu Asp Thr Gly
                85                  90                  95

Glu Tyr Phe Cys Thr His Asn Asp Ser Arg Gly Leu Glu Thr Asp Glu
            100                 105                 110

Arg Lys Arg Leu Tyr Ile Phe Val Pro Asp Pro Thr Val Gly Phe Leu
        115                 120                 125

Pro Asn Asp Ala Glu Glu Leu Phe Ile Phe Leu Thr Glu Ile Thr Glu
    130                 135                 140

Ile Thr Ile Pro Cys Arg Val Thr Asp Pro Gln Leu Val Val Thr Leu
145                 150                 155                 160

His Glu Lys Lys Gly Asp Val Ala Leu Pro Val Pro Tyr Asp His Gln
                165                 170                 175

Arg Gly Phe Ser Gly Ile Phe Glu Asp Arg Ser Tyr Ile Cys Lys Thr
            180                 185                 190

Thr Ile Gly Asp Arg Glu Val Asp Ser Asp Ala Tyr Tyr Val Tyr Arg
        195                 200                 205

Leu Gln Val Ser Ser Ile Asn Val Ser Val Asn Ala Val Gln Thr Val
    210                 215                 220

Val Arg Gln Gly Glu Asn Ile Thr Leu Met Cys Ile Val Ile Gly Asn
225                 230                 235                 240

Glu Val Val Asn Phe Glu Trp Thr Tyr Pro Arg Lys Glu Ser Gly Arg
                245                 250                 255

Leu Val Glu Pro Val Thr Asp Phe Leu Leu Asp Met Pro Tyr His Ile
            260                 265                 270

Arg Ser Ile Leu His Ile Pro Ser Ala Glu Leu Glu Asp Ser Gly Thr
        275                 280                 285

Tyr Thr Cys Asn Val Thr Glu Ser Val Asn Asp His Gln Asp Glu Lys
    290                 295                 300

Ala Ile Asn Ile Thr Val Val Glu Ser Gly Tyr Val Arg Leu Leu Gly
305                 310                 315                 320

Glu Val Gly Thr Leu Gln Phe Ala Glu Leu His Arg Ser Arg Thr Leu
                325                 330                 335

Gln Val Val Phe Glu Ala Tyr Pro Pro Pro Thr Val Leu Trp Phe Lys
            340                 345                 350

Asp Asn Arg Thr Leu Gly Asp Ser Ser Ala Gly Glu Ile Ala Leu Ser
        355                 360                 365

Thr Arg Asn Val Ser Glu Thr Arg Tyr Val Ser Glu Leu Thr Leu Val
    370                 375                 380

Arg Val Lys Val Ala Glu Ala Gly His Tyr Thr Met Arg Ala Phe His
385                 390                 395                 400
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Glu Asp Ala Glu Val Gln Leu Ser Phe Gln Leu Gln Ile Asn Val Pro
                405                 410                 415

Val Arg Val Leu Glu Leu Ser Glu Ser His Pro Asp Ser Gly Glu Gln
            420                 425                 430

Thr Val Arg Cys Arg Gly Arg Gly Met Pro Gln Pro Asn Ile Ile Trp
        435                 440                 445

Ser Ala Cys Arg Asp Leu Lys Arg Cys Pro Arg Glu Leu Pro Pro Thr
    450                 455                 460

Leu Leu Gly Asn Ser Ser Glu Glu Glu Ser Gln Leu Glu Thr Asn Val
465                 470                 475                 480

Thr Tyr Trp Glu Glu Glu Gln Glu Phe Glu Val Val Ser Thr Leu Arg
                485                 490                 495

Leu Gln His Val Asp Arg Pro Leu Ser Val Arg Cys Thr Leu Arg Asn
            500                 505                 510

Ala Val Gly Gln Asp Thr Gln Glu Val Ile Val Val Pro His Ser Leu
        515                 520                 525

Pro Phe Lys Val Val Val Ile Ser Ala Ile Leu Ala Leu Val Val Leu
    530                 535                 540

Thr Ile Ile Ser Leu Ile Ile Leu Ile Met Leu Trp Gln Lys Lys Pro
545                 550                 555                 560

Arg Tyr Glu Ile Arg Trp Lys Val Ile Glu Ser Val Ser Ser Asp Gly
                565                 570                 575

His Glu Tyr Ile Tyr Val Asp Pro Met Gln Leu Pro Tyr Asp Ser Thr
            580                 585                 590

Trp Glu Leu Pro Arg Asp Gln Leu Val Leu Gly Arg Thr Leu Gly Ser
        595                 600                 605

Gly Ala Phe Gly Gln Val Val Glu Ala Thr Ala His Gly Leu Ser His
    610                 615                 620

Ser Gln Ala Thr Met Lys Val Ala Val Lys Met Leu Lys Ser Thr Ala
625                 630                 635                 640

Arg Ser Ser Glu Lys Gln Ala Leu Met Ser Glu Leu Lys Ile Met Ser
                645                 650                 655

His Leu Gly Pro His Leu Asn Val Val Asn Leu Leu Gly Ala Cys Thr
            660                 665                 670

Lys Gly Gly Pro Ile Tyr Ile Ile Thr Glu Tyr Cys Arg Tyr Gly Asp
        675                 680                 685

Leu Val Asp Tyr Leu His Arg Asn Lys His Thr Phe Leu Gln His His
    690                 695                 700

Ser Asp Lys Arg Arg Pro Pro Ser Ala Glu Leu Tyr Ser Asn Ala Leu
705                 710                 715                 720

Pro Val Gly Leu Pro Leu Pro Ser His Val Ser Leu Thr Gly Glu Ser
                725                 730                 735

Asp Gly Gly Tyr Met Asp Met Ser Lys Asp Glu Ser Val Asp Tyr Val
            740                 745                 750

Pro Met Leu Asp Met Lys Gly Asp Val Lys Tyr Ala Asp Ile Glu Ser
        755                 760                 765

Ser Asn Tyr Met Ala Pro Tyr Asp Asn Tyr Val Pro Ser Ala Pro Glu
    770                 775                 780

Arg Thr Cys Arg Ala Thr Leu Ile Asn Glu Ser Pro Val Leu Ser Tyr
785                 790                 795                 800
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Met Asp Leu Val Gly Phe Ser Tyr Gln Val Ala Asn Gly Met Glu Phe
                805                 810                 815

Leu Ala Ser Lys Asn Cys Val His Arg Asp Leu Ala Ala Arg Asn Val
            820                 825                 830

Leu Ile Cys Glu Gly Lys Leu Val Lys Ile Cys Asp Phe Gly Leu Ala
        835                 840                 845

Arg Asp Ile Met Arg Asp Ser Asn Tyr Ile Ser Lys Gly Ser Thr Phe
    850                 855                 860

Leu Pro Leu Lys Trp Met Ala Pro Glu Ser Ile Phe Asn Ser Leu Tyr
865                 870                 875                 880

Thr Thr Leu Ser Asp Val Trp Ser Phe Gly Ile Leu Leu Trp Glu Ile
                885                 890                 895

Phe Thr Leu Gly Gly Thr Pro Tyr Pro Glu Leu Pro Met Asn Glu Gln
            900                 905                 910

Phe Tyr Asn Ala Ile Lys Arg Gly Tyr Arg Met Ala Gln Pro Ala His
        915                 920                 925

Ala Ser Asp Glu Ile Tyr Glu Ile Met Gln Lys Cys Trp Glu Glu Lys
    930                 935                 940

Phe Glu Ile Arg Pro Pro Phe Ser Gln Leu Val Leu Leu Leu Glu Arg
945                 950                 955                 960

Leu Leu Gly Glu Gly Tyr Lys Lys Lys Tyr Gln Gln Val Asp Glu Glu
                965                 970                 975

Phe Leu Arg Ser Asp His Pro Ala Ile Leu Arg Ser Gln Ala Arg Leu
            980                 985                 990

Pro Gly Phe His Gly Leu Arg Ser  Pro Leu Asp Thr Ser  Ser Val Leu
        995                 1000                1005

Tyr Thr  Ala Val Gln Pro Asn  Glu Gly Asp Asn Asp  Tyr Ile Ile
    1010                1015                1020

Pro Leu  Pro Asp Pro Lys Pro  Glu Val Ala Asp Glu  Gly Pro Leu
    1025                1030                1035

Glu Gly  Ser Pro Ser Leu Ala  Ser Ser Thr Leu Asn  Glu Val Asn
    1040                1045                1050

Thr Ser  Ser Thr Ile Ser Cys  Asp Ser Pro Leu Glu  Pro Gln Asp
    1055                1060                1065

Glu Pro  Glu Pro Glu Pro Gln  Leu Glu Leu Gln Val  Glu Pro Glu
    1070                1075                1080

Pro Glu  Leu Glu Gln Leu Pro  Asp Ser Gly Cys Pro  Ala Pro Arg
    1085                1090                1095

Ala Glu  Ala Glu Asp Ser Phe  Leu
    1100                1105


<210> SEQ ID NO 16
<211> LENGTH: 3626
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 16

tcgcggaggc ttggggcagc cgggtagctc ggaggtcgtg gcgctggggg ctagcaccag     60

cgctctgtcg ggaggcgcag cggttaggtg gaccggtcag cggactcacc ggccagggcg    120

ctcggtgctg gaatttgata ttcattgatc cgggttttat ccctcttctt ttttcttaaa    180

catttttttt taaaactgta ttgtttctcg ttttaattta tttttgcttg ccattcccca    240
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cttgaatcgg gccgacggct tggggagatt gctctacttc cccaaatcac tgtggatttt 300

ggaaaccagc agaaagagga aagaggtagc aagagctcca gagagaagtc gaggaagaga 360

gagacggggt cagagagagc gcgcgggcgt gcgagcagcg aaagcgacag gggcaaagtg 420

agtgacctgc ttttgggggt gaccgccgga gcgcggcgtg agccctcccc cttgggatcc 480

cgcagctgac cagtcgcgct gacggacaga cagacagaca ccgcccccag ccccagctac 540

cacctcctcc ccggccggcg gcggacagtg gacgcggcgg cgagccgcgg gcaggggccg 600

gagcccgcgc ccggaggcgg ggtggagggg gtcggggctc gcggcgtcgc actgaaactt 660

ttcgtccaac ttctgggctg ttctcgcttc ggaggagccg tggtccgcgc gggggaagcc 720

gagccgagcg gagccgcgag aagtgctagc tcgggccggg aggagccgca gccggaggag 780

ggggaggagg aagaagagaa ggaagaggag agggggccgc agtggcgact cggcgctcgg 840

aagccgggct catggacggg tgaggcggcg gtgtgcgcag acagtgctcc agccgcgcgc 900

gctccccagg ccctggcccg ggcctcgggc cggggaggaa gagtagctcg ccgaggcgcc 960

gaggagagcg ggccgcccca cagcccgagc cggagaggga gcgcgagccg cgccggcccc 1020

ggtcgggcct ccgaaaccat gaactttctg ctgtcttggg tgcattggag ccttgccttg 1080

ctgctctacc tccaccatgc caagtggtcc caggctgcac ccatggcaga aggaggaggg 1140

cagaatcatc acgaagtggt gaagttcatg gatgtctatc agcgcagcta ctgccatcca 1200

atcgagaccc tggtggacat cttccaggag taccctgatg agatcgagta catcttcaag 1260

ccatcctgtg tgcccctgat gcgatgcggg ggctgctgca atgacgaggg cctggagtgt 1320

gtgcccactg aggagtccaa catcaccatg cagattatgc ggatcaaacc tcaccaaggc 1380

cagcacatag gagagatgag cttcctacag cacaacaaat gtgaatgcag accaaagaaa 1440

gatagagcaa gacaagaaaa aaaatcagtt cgaggaaagg gaaaggggca aaaacgaaag 1500

cgcaagaaat cccggtataa gtcctggagc gttccctgtg ggccttgctc agagcggaga 1560

aagcatttgt ttgtacaaga tccgcagacg tgtaaatgtt cctgcaaaaa cacagactcg 1620

cgttgcaagg cgaggcagct tgagttaaac gaacgtactt gcagatgtga caagccgagg 1680

cggtgagccg ggcaggagga aggagcctcc ctcagggttt cgggaaccag atctctcacc 1740

aggaaagact gatacagaac gatcgataca gaaaccacgc tgccgccacc acaccatcac 1800

catcgacaga acagtcctta atccagaaac ctgaaatgaa ggaagaggag actctgcgca 1860

gagcactttg ggtccggagg gcgagactcc ggcggaagca ttcccgggcg ggtgacccag 1920

cacggtccct cttggaattg gattcgccat tttatttttc ttgctgctaa atcaccgagc 1980

ccggaagatt agagagtttt atttctggga ttcctgtaga cacacccacc cacatacata 2040

catttatata tatatatatt atatatatat aaaaataaat atctctattt tatatatata 2100

aaatatatat attctttttt taaattaaca gtgctaatgt tattggtgtc ttcactggat 2160

gtatttgact gctgtggact tgagttggga ggggaatgtt cccactcaga tcctgacagg 2220

gaagaggagg agatgagaga ctctggcatg atctttttttt tgtcccactt ggtggggcca 2280

gggtcctctc ccctgcccag gaatgtgcaa ggccagggca tgggggcaaa tatgacccag 2340

ttttgggaac accgacaaac ccagccctgg cgctgagcct ctctacccca ggtcagacgg 2400

acagaaagac agatcacagg tacagggatg aggacaccgg ctctgaccag gagtttgggg 2460

agcttcagga cattgctgtg ctttggggat tccctccaca tgctgcacgc gcatctcgcc 2520

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cccaggggca ctgcctggaa gattcaggag cctgggcggc cttcgcttac tctcacctgc   2580

ttctgagttg cccaggagac cactggcaga tgtcccggcg aagagaagag acacattgtt   2640

ggaagaagca gcccatgaca gctccccttc ctgggactcg ccctcatcct cttcctgctc   2700

cccttcctgg ggtgcagcct aaaaggacct atgtcctcac accattgaaa ccactagttc   2760

tgtcccccca ggagacctgg ttgtgtgtgt gtgagtggtt gaccttcctc catcccctgg   2820

tccttccctt cccttcccga ggcacagaga gacagggcag gatccacgtg cccattgtgg   2880

aggcagagaa aagagaaagt gttttatata cggtacttat ttaatatccc tttttaatta   2940

gaaattaaaa cagttaattt aattaaagag tagggttttt tttcagtatt cttggttaat   3000

atttaatttc aactatttat gagatgtatc ttttgctctc tcttgctctc ttatttgtac   3060

cggtttttgt atataaaatt catgtttcca atctctctct ccctgatcgg tgacagtcac   3120

tagcttatct tgaacagata tttaattttg ctaacactca gctctgccct ccccgatccc   3180

ctggctcccc agcacacatt cctttgaaat aaggtttcaa tatacatcta catactatat   3240

atatatttgg caacttgtat ttgtgtgtat atatatatat atatgtttat gtatatatgt   3300

gattctgata aaatagacat tgctattctg ttttttatat gtaaaaacaa aacaagaaaa   3360

aatagagaat tctacatact aaatctctct ccttttttaa ttttaatatt tgttatcatt   3420

tatttattgg tgctactgtt tatccgtaat aattgtgggg aaaagatatt aacatcacgt   3480

ctttgtctct agtgcagttt ttcgagatat tccgtagtac atatttattt ttaaacaacg   3540

acaaagaaat acagatatat cttaaaaaaa aaaaagcatt ttgtattaaa gaatttaatt   3600

ctgatctcaa aaaaaaaaaa aaaaaa                                       3626


<210> SEQ ID NO 17
<211> LENGTH: 395
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 17

Met Thr Asp Arg Gln Thr Asp Thr Ala Pro Ser Pro Ser Tyr His Leu
1               5                   10                  15

Leu Pro Gly Arg Arg Arg Thr Val Asp Ala Ala Ala Ser Arg Gly Gln
            20                  25                  30

Gly Pro Glu Pro Ala Pro Gly Gly Gly Val Glu Gly Val Gly Ala Arg
        35                  40                  45

Gly Val Ala Leu Lys Leu Phe Val Gln Leu Leu Gly Cys Ser Arg Phe
    50                  55                  60

Gly Gly Ala Val Val Arg Ala Gly Glu Ala Glu Pro Ser Gly Ala Ala
65                  70                  75                  80

Arg Ser Ala Ser Ser Gly Arg Glu Glu Pro Gln Pro Glu Glu Gly Glu
                85                  90                  95

Glu Glu Glu Glu Lys Glu Glu Glu Arg Gly Pro Gln Trp Arg Leu Gly
            100                 105                 110

Ala Arg Lys Pro Gly Ser Trp Thr Gly Glu Ala Ala Val Cys Ala Asp
        115                 120                 125

Ser Ala Pro Ala Ala Arg Ala Pro Gln Ala Leu Ala Arg Ala Ser Gly
    130                 135                 140

Arg Gly Gly Arg Val Ala Arg Arg Gly Ala Glu Glu Ser Gly Pro Pro
145                 150                 155                 160
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His Ser Pro Ser Arg Arg Gly Ser Ala Ser Arg Ala Gly Pro Gly Arg
                165                 170                 175

Ala Ser Glu Thr Met Asn Phe Leu Leu Ser Trp Val His Trp Ser Leu
            180                 185                 190

Ala Leu Leu Leu Tyr Leu His His Ala Lys Trp Ser Gln Ala Ala Pro
        195                 200                 205

Met Ala Glu Gly Gly Gly Gln Asn His His Glu Val Val Lys Phe Met
    210                 215                 220

Asp Val Tyr Gln Arg Ser Tyr Cys His Pro Ile Glu Thr Leu Val Asp
225                 230                 235                 240

Ile Phe Gln Glu Tyr Pro Asp Glu Ile Glu Tyr Ile Phe Lys Pro Ser
                245                 250                 255

Cys Val Pro Leu Met Arg Cys Gly Gly Cys Cys Asn Asp Glu Gly Leu
            260                 265                 270

Glu Cys Val Pro Thr Glu Glu Ser Asn Ile Thr Met Gln Ile Met Arg
        275                 280                 285

Ile Lys Pro His Gln Gly Gln His Ile Gly Glu Met Ser Phe Leu Gln
    290                 295                 300

His Asn Lys Cys Glu Cys Arg Pro Lys Lys Asp Arg Ala Arg Gln Glu
305                 310                 315                 320

Lys Lys Ser Val Arg Gly Lys Gly Lys Gly Gln Lys Arg Lys Arg Lys
                325                 330                 335

Lys Ser Arg Tyr Lys Ser Trp Ser Val Pro Cys Gly Pro Cys Ser Glu
            340                 345                 350

Arg Arg Lys His Leu Phe Val Gln Asp Pro Gln Thr Cys Lys Cys Ser
        355                 360                 365

Cys Lys Asn Thr Asp Ser Arg Cys Lys Ala Arg Gln Leu Glu Leu Asn
    370                 375                 380

Glu Arg Thr Cys Arg Cys Asp Lys Pro Arg Arg
385                 390                 395


<210> SEQ ID NO 18
<211> LENGTH: 4017
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 18

atggtcagct actgggacac cggggtcctg ctgtgcgcgc tgctcagctg tctgcttctc     60

acaggatcta gttcaggttc aaaattaaaa gatcctgaac tgagtttaaa aggcacccag    120

cacatcatgc aagcaggcca gacactgcat ctccaatgca ggggggaagc agcccataaa    180

tggtctttgc ctgaaatggt gagtaaggaa agcgaaaggc tgagcataac taaatctgcc    240

tgtggaagaa atggcaaaca attctgcagt actttaacct tgaacacagc tcaagcaaac    300

cacactggct tctacagctg caaatatcta gctgtaccta cttcaaagaa gaaggaaaca    360

gaatctgcaa tctatatatt tattagtgat acaggtagac ctttcgtaga gatgtacagt    420

gaaatccccg aaattataca catgactgaa ggaagggagc tcgtcattcc ctgccgggtt    480

acgtcaccta acatcactgt tactttaaaa aagtttccac ttgacacttt gatccctgat    540

ggaaaacgca taatctggga cagtagaaag ggcttcatca tatcaaatgc aacgtacaaa    600

gaaatagggc ttctgacctg tgaagcaaca gtcaatgggc atttgtataa gacaaactat    660

ctcacacatc gacaaaccaa tacaatcata gatgtccaaa taagcacacc acgcccagtc    720
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aaattactta gaggccatac tcttgtcctc aattgtactg ctaccactcc cttgaacacg 780

agagttcaaa tgacctggag ttaccctgat gaaaaaaata agagagcttc cgtaaggcga 840

cgaattgacc aaagcaattc ccatgccaac atattctaca gtgttcttac tattgacaaa 900

atgcagaaca aagacaaagg actttatact tgtcgtgtaa ggagtggacc atcattcaaa 960

tctgttaaca cctcagtgca tatatatgat aaagcattca tcactgtgaa acatcgaaaa 1020

cagcaggtgc ttgaaaccgt agctggcaag cggtcttacc ggctctctat gaaagtgaag 1080

gcatttccct cgccggaagt tgtatggtta aaagatgggt tacctgcgac tgagaaatct 1140

gctcgctatt tgactcgtgg ctactcgtta attatcaagg acgtaactga agaggatgca 1200

gggaattata caatcttgct gagcataaaa cagtcaaatg tgtttaaaaa cctcactgcc 1260

actctaattg tcaatgtgaa accccagatt tacgaaaagg ccgtgtcatc gtttccagac 1320

ccggctctct acccactggg cagcagacaa atcctgactt gtaccgcata tggtatccct 1380

caacctacaa tcaagtggtt ctggcacccc tgtaaccata atcattccga agcaaggtgt 1440

gacttttgtt ccaataatga agagtcctct atcctggatg ctgacagcaa catgggaaac 1500

agaattgaga gcatcactca gcgcatggca ataatagaag gaaagaataa gatggctagc 1560

accttggttg tggctgactc tagaatttct ggaatctaca tttgcatagc ttccaataaa 1620

gttgggactg tgggaagaaa cataagcttt tatatcacag atgtgccaaa tgggtttcat 1680

gttaacttgg aaaaaatgcc gacggaagga gaggacctga aactgtcttg cacagttaac 1740

aagttcttat acagagacgt tacttggatt ttactgcgga cagttaataa cagaacaatg 1800

cactacagta ttagcaagca aaaaatggcc atcactaagg agcactccat cactcttaat 1860

cttaccatca tgaatgtttc cctgcaagat tcaggcacct atgcctgcag agccaggaat 1920

gtatacacag gggaagaaat cctccagaag aaagaaatta caatcagaga tcaggaagca 1980

ccatacctcc tgcgaaacct cagtgatcac acagtggcca tcagcagttc caccacttta 2040

gactgtcatg ctaatggtgt ccccgagcct cagatcactt ggtttaaaaa caaccacaaa 2100

atacaacaag agcctggaat tattttagga ccaggaagca gcacgctgtt tattgaaaga 2160

gtcacagaag aggatgaagg tgtctatcac tgcaaagcca ccaaccagaa gggctctgtg 2220

gaaagttcag catacctcac tgttcaagga acctcggaca agtctaatct ggagctgatc 2280

actctaacat gcacctgtgt ggctgcgact ctcttctggc tcctattaac cctctttatc 2340

cgaaaaatga aaaggtcttc ttctgaaata aagactgact acctatcaat tataatggac 2400

ccagatgaag ttcctttgga tgagcagtgt gagcggctcc cttatgatgc cagcaagtgg 2460

gagtttgccc gggagagact taaactgggc aaatcacttg gaagaggggc ttttggaaaa 2520

gtggttcaag catcagcatt tggcattaag aaatcaccta cgtgccggac tgtggctgtg 2580

aaaatgctga aagaggggc cacggccagc gagtacaaag ctctgatgac tgagctaaaa 2640

atcttgaccc acattggcca ccatctgaac gtggttaacc tgctgggagc ctgcaccaag 2700

caaggagggc ctctgatggt gattgttgaa tactgcaaat atggaaatct ctccaactac 2760

ctcaagagca aacgtgactt atttttttctc aacaaggatg cagcactaca catggagcct 2820

aagaaagaaa aaatggagcc aggcctggaa caaggcaaga aaccaagact agatagcgtc 2880

accagcagcg aaagctttgc gagctccggc tttcaggaag ataaaagtct gagtgatgtt 2940

gaggaagagg aggattctga cggtttctac aaggagccca tcactatgga agatctgatt 3000

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tcttacagtt ttcaagtggc cagaggcatg gagttcctgt cttccagaaa gtgcattcat 3060

cgggacctgg cagcgagaaa cattctttta tctgagaaca acgtggtgaa gatttgtgat 3120

tttggccttg cccgggatat ttataagaac cccgattatg tgagaaaagg agatactcga 3180

cttcctctga aatggatggc tcctgaatct atctttgaca aaatctacag caccaagagc 3240

gacgtgtggt cttacggagt attgctgtgg gaaatcttct ccttaggtgg gtctccatac 3300

ccaggagtac aaatggatga ggacttttgc agtcgcctga gggaaggcat gaggatgaga 3360

gctcctgagt actctactcc tgaaatctat cagatcatgc tggactgctg gcacagagac 3420

ccaaaagaaa ggccaagatt tgcagaactt gtggaaaaac taggtgattt gcttcaagca 3480

aatgtacaac aggatggtaa agactacatc ccaatcaatg ccatactgac aggaaatagt 3540

gggtttacat actcaactcc tgccttctct gaggacttct tcaaggaaag tatttcagct 3600

ccgaagttta attcaggaag ctctgatgat gtcagatatg taaatgcttt caagttcatg 3660

agcctggaaa gaatcaaaac ctttgaagaa cttttaccga atgccacctc catgtttgat 3720

gactaccagg gcgacagcag cactctgttg gcctctccca tgctgaagcg cttcacctgg 3780

actgacagca aacccaaggc ctcgctcaag attgacttga gagtaaccag taaaagtaag 3840

gagtcggggc tgtctgatgt cagcaggccc agtttctgcc attccagctg tgggcacgtc 3900

agcgaaggca agcgcaggtt cacctacgac cacgctgagc tggaaaggaa aatcgcgtgc 3960

tgctccccgc ccccagacta caactcggtg gtcctgtact ccaccccacc catctag 4017

<210> SEQ ID NO 19
<211> LENGTH: 1338
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 19

Met Val Ser Tyr Trp Asp Thr Gly Val Leu Leu Cys Ala Leu Leu Ser
1 5 10 15

Cys Leu Leu Leu Thr Gly Ser Ser Ser Gly Ser Lys Leu Lys Asp Pro
20 25 30

Glu Leu Ser Leu Lys Gly Thr Gln His Ile Met Gln Ala Gly Gln Thr
35 40 45

Leu His Leu Gln Cys Arg Gly Glu Ala Ala His Lys Trp Ser Leu Pro
50 55 60

Glu Met Val Ser Lys Glu Ser Glu Arg Leu Ser Ile Thr Lys Ser Ala
65 70 75 80

Cys Gly Arg Asn Gly Lys Gln Phe Cys Ser Thr Leu Thr Leu Asn Thr
85 90 95

Ala Gln Ala Asn His Thr Gly Phe Tyr Ser Cys Lys Tyr Leu Ala Val
100 105 110

Pro Thr Ser Lys Lys Lys Glu Thr Glu Ser Ala Ile Tyr Ile Phe Ile
115 120 125

Ser Asp Thr Gly Arg Pro Phe Val Glu Met Tyr Ser Glu Ile Pro Glu
130 135 140

Ile Ile His Met Thr Glu Gly Arg Glu Leu Val Ile Pro Cys Arg Val
145 150 155 160

Thr Ser Pro Asn Ile Thr Val Thr Leu Lys Lys Phe Pro Leu Asp Thr
165 170 175

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Leu Ile Pro Asp Gly Lys Arg Ile Ile Trp Asp Ser Arg Lys Gly Phe
            180                 185                 190

Ile Ile Ser Asn Ala Thr Tyr Lys Glu Ile Gly Leu Leu Thr Cys Glu
        195                 200                 205

Ala Thr Val Asn Gly His Leu Tyr Lys Thr Asn Tyr Leu Thr His Arg
    210                 215                 220

Gln Thr Asn Thr Ile Ile Asp Val Gln Ile Ser Thr Pro Arg Pro Val
225                 230                 235                 240

Lys Leu Leu Arg Gly His Thr Leu Val Leu Asn Cys Thr Ala Thr Thr
                245                 250                 255

Pro Leu Asn Thr Arg Val Gln Met Thr Trp Ser Tyr Pro Asp Glu Lys
            260                 265                 270

Asn Lys Arg Ala Ser Val Arg Arg Arg Ile Asp Gln Ser Asn Ser His
        275                 280                 285

Ala Asn Ile Phe Tyr Ser Val Leu Thr Ile Asp Lys Met Gln Asn Lys
    290                 295                 300

Asp Lys Gly Leu Tyr Thr Cys Arg Val Arg Ser Gly Pro Ser Phe Lys
305                 310                 315                 320

Ser Val Asn Thr Ser Val His Ile Tyr Asp Lys Ala Phe Ile Thr Val
                325                 330                 335

Lys His Arg Lys Gln Gln Val Leu Glu Thr Val Ala Gly Lys Arg Ser
            340                 345                 350

Tyr Arg Leu Ser Met Lys Val Lys Ala Phe Pro Ser Pro Glu Val Val
        355                 360                 365

Trp Leu Lys Asp Gly Leu Pro Ala Thr Glu Lys Ser Ala Arg Tyr Leu
    370                 375                 380

Thr Arg Gly Tyr Ser Leu Ile Ile Lys Asp Val Thr Glu Glu Asp Ala
385                 390                 395                 400

Gly Asn Tyr Thr Ile Leu Leu Ser Ile Lys Gln Ser Asn Val Phe Lys
                405                 410                 415

Asn Leu Thr Ala Thr Leu Ile Val Asn Val Lys Pro Gln Ile Tyr Glu
            420                 425                 430

Lys Ala Val Ser Ser Phe Pro Asp Pro Ala Leu Tyr Pro Leu Gly Ser
        435                 440                 445

Arg Gln Ile Leu Thr Cys Thr Ala Tyr Gly Ile Pro Gln Pro Thr Ile
    450                 455                 460

Lys Trp Phe Trp His Pro Cys Asn His Asn His Ser Glu Ala Arg Cys
465                 470                 475                 480

Asp Phe Cys Ser Asn Asn Glu Glu Ser Ser Ile Leu Asp Ala Asp Ser
                485                 490                 495

Asn Met Gly Asn Arg Ile Glu Ser Ile Thr Gln Arg Met Ala Ile Ile
            500                 505                 510

Glu Gly Lys Asn Lys Met Ala Ser Thr Leu Val Val Ala Asp Ser Arg
        515                 520                 525

Ile Ser Gly Ile Tyr Ile Cys Ile Ala Ser Asn Lys Val Gly Thr Val
    530                 535                 540

Gly Arg Asn Ile Ser Phe Tyr Ile Thr Asp Val Pro Asn Gly Phe His
545                 550                 555                 560

Val Asn Leu Glu Lys Met Pro Thr Glu Gly Glu Asp Leu Lys Leu Ser
                565                 570                 575

Cys Thr Val Asn Lys Phe Leu Tyr Arg Asp Val Thr Trp Ile Leu Leu
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            580                 585                 590

Arg Thr Val Asn Asn Arg Thr Met His Tyr Ser Ile Ser Lys Gln Lys
        595                 600                 605

Met Ala Ile Thr Lys Glu His Ser Ile Thr Leu Asn Leu Thr Ile Met
    610                 615                 620

Asn Val Ser Leu Gln Asp Ser Gly Thr Tyr Ala Cys Arg Ala Arg Asn
625                 630                 635                 640

Val Tyr Thr Gly Glu Glu Ile Leu Gln Lys Lys Glu Ile Thr Ile Arg
                645                 650                 655

Asp Gln Glu Ala Pro Tyr Leu Leu Arg Asn Leu Ser Asp His Thr Val
            660                 665                 670

Ala Ile Ser Ser Ser Thr Thr Leu Asp Cys His Ala Asn Gly Val Pro
        675                 680                 685

Glu Pro Gln Ile Thr Trp Phe Lys Asn Asn His Lys Ile Gln Gln Glu
    690                 695                 700

Pro Gly Ile Ile Leu Gly Pro Gly Ser Ser Thr Leu Phe Ile Glu Arg
705                 710                 715                 720

Val Thr Glu Glu Asp Glu Gly Val Tyr His Cys Lys Ala Thr Asn Gln
                725                 730                 735

Lys Gly Ser Val Glu Ser Ser Ala Tyr Leu Thr Val Gln Gly Thr Ser
            740                 745                 750

Asp Lys Ser Asn Leu Glu Leu Ile Thr Leu Thr Cys Thr Cys Val Ala
        755                 760                 765

Ala Thr Leu Phe Trp Leu Leu Leu Thr Leu Phe Ile Arg Lys Met Lys
    770                 775                 780

Arg Ser Ser Ser Glu Ile Lys Thr Asp Tyr Leu Ser Ile Ile Met Asp
785                 790                 795                 800

Pro Asp Glu Val Pro Leu Asp Glu Gln Cys Glu Arg Leu Pro Tyr Asp
                805                 810                 815

Ala Ser Lys Trp Glu Phe Ala Arg Glu Arg Leu Lys Leu Gly Lys Ser
            820                 825                 830

Leu Gly Arg Gly Ala Phe Gly Lys Val Val Gln Ala Ser Ala Phe Gly
        835                 840                 845

Ile Lys Lys Ser Pro Thr Cys Arg Thr Val Ala Val Lys Met Leu Lys
    850                 855                 860

Glu Gly Ala Thr Ala Ser Glu Tyr Lys Ala Leu Met Thr Glu Leu Lys
865                 870                 875                 880

Ile Leu Thr His Ile Gly His His Leu Asn Val Val Asn Leu Leu Gly
                885                 890                 895

Ala Cys Thr Lys Gln Gly Gly Pro Leu Met Val Ile Val Glu Tyr Cys
            900                 905                 910

Lys Tyr Gly Asn Leu Ser Asn Tyr Leu Lys Ser Lys Arg Asp Leu Phe
        915                 920                 925

Phe Leu Asn Lys Asp Ala Ala Leu His Met Glu Pro Lys Lys Glu Lys
    930                 935                 940

Met Glu Pro Gly Leu Glu Gln Gly Lys Lys Pro Arg Leu Asp Ser Val
945                 950                 955                 960

Thr Ser Ser Glu Ser Phe Ala Ser Ser Gly Phe Gln Glu Asp Lys Ser
                965                 970                 975

Leu Ser Asp Val Glu Glu Glu Glu Asp Ser Asp Gly Phe Tyr Lys Glu
            980                 985                 990
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Pro Ile Thr Met Glu Asp Leu Ile  Ser Tyr Ser Phe Gln  Val Ala Arg
        995                1000                1005

Gly Met  Glu Phe Leu Ser Ser  Arg Lys Cys Ile His  Arg Asp Leu
    1010                1015                1020

Ala Ala  Arg Asn Ile Leu Leu  Ser Glu Asn Asn Val  Val Lys Ile
    1025                1030                1035

Cys Asp  Phe Gly Leu Ala Arg  Asp Ile Tyr Lys Asn  Pro Asp Tyr
    1040                1045                1050

Val Arg  Lys Gly Asp Thr Arg  Leu Pro Leu Lys Trp  Met Ala Pro
    1055                1060                1065

Glu Ser  Ile Phe Asp Lys Ile  Tyr Ser Thr Lys Ser  Asp Val Trp
    1070                1075                1080

Ser Tyr  Gly Val Leu Leu Trp  Glu Ile Phe Ser Leu  Gly Gly Ser
    1085                1090                1095

Pro Tyr  Pro Gly Val Gln Met  Asp Glu Asp Phe Cys  Ser Arg Leu
    1100                1105                1110

Arg Glu  Gly Met Arg Met Arg  Ala Pro Glu Tyr Ser  Thr Pro Glu
    1115                1120                1125

Ile Tyr  Gln Ile Met Leu Asp  Cys Trp His Arg Asp  Pro Lys Glu
    1130                1135                1140

Arg Pro  Arg Phe Ala Glu Leu  Val Glu Lys Leu Gly  Asp Leu Leu
    1145                1150                1155

Gln Ala  Asn Val Gln Gln Asp  Gly Lys Asp Tyr Ile  Pro Ile Asn
    1160                1165                1170

Ala Ile  Leu Thr Gly Asn Ser  Gly Phe Thr Tyr Ser  Thr Pro Ala
    1175                1180                1185

Phe Ser  Glu Asp Phe Phe Lys  Glu Ser Ile Ser Ala  Pro Lys Phe
    1190                1195                1200

Asn Ser  Gly Ser Ser Asp Asp  Val Arg Tyr Val Asn  Ala Phe Lys
    1205                1210                1215

Phe Met  Ser Leu Glu Arg Ile  Lys Thr Phe Glu Glu  Leu Leu Pro
    1220                1225                1230

Asn Ala  Thr Ser Met Phe Asp  Asp Tyr Gln Gly Asp  Ser Ser Thr
    1235                1240                1245

Leu Leu  Ala Ser Pro Met Leu  Lys Arg Phe Thr Trp  Thr Asp Ser
    1250                1255                1260

Lys Pro  Lys Ala Ser Leu Lys  Ile Asp Leu Arg Val  Thr Ser Lys
    1265                1270                1275

Ser Lys  Glu Ser Gly Leu Ser  Asp Val Ser Arg Pro  Ser Phe Cys
    1280                1285                1290

His Ser  Ser Cys Gly His Val  Ser Glu Gly Lys Arg  Arg Phe Thr
    1295                1300                1305

Tyr Asp  His Ala Glu Leu Glu  Arg Lys Ile Ala Cys  Cys Ser Pro
    1310                1315                1320

Pro Pro  Asp Tyr Asn Ser Val  Val Leu Tyr Ser Thr  Pro Pro Ile
    1325                1330                1335


<210> SEQ ID NO 20
<211> LENGTH: 5830
<212> TYPE: DNA
<213> ORGANISM: Homo sapiens
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<400> SEQUENCE: 20

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cgccgggcat cacttgcgcg ccgcagaaag tccgtctggc agcctggata tcctctccta 120

ccggcacccg cagacgcccc tgcagccgcc ggtcggcgcc cgggctccct agccctgtgc 180

gctcaactgt cctgcgctgc ggggtgccgc gagttccacc tccgcgcctc cttctctaga 240

caggcgctgg gagaaagaac cggctcccga gttctgggca tttcgcccgg ctcgaggtgc 300

aggatgcaga gcaaggtgct gctggccgtc gccctgtggc tctgcgtgga gacccgggcc 360

gcctctgtgg gtttgcctag tgtttctctt gatctgccca ggctcagcat acaaaaagac 420

atacttacaa ttaaggctaa tacaactctt caaattactt gcaggggaca gagggacttg 480

gactggcttt ggcccaataa tcagagtggc agtgagcaaa gggtggaggt gactgagtgc 540

agcgatggcc tcttctgtaa gacactcaca attccaaaag tgatcggaaa tgacactgga 600

gcctacaagt gcttctaccg ggaaactgac ttggcctcgg tcatttatgt ctatgttcaa 660

gattacagat ctccatttat tgcttctgtt agtgaccaac atggagtcgt gtacattact 720

gagaacaaaa acaaaactgt ggtgattcca tgtctcgggt ccatttcaaa tctcaacgtg 780

tcactttgtg caagataccc agaaaagaga tttgttcctg atggtaacag aatttcctgg 840

gacagcaaga agggctttac tattcccagc tacatgatca gctatgctgg catggtcttc 900

tgtgaagcaa aaattaatga tgaaagttac cagtctatta tgtacatagt tgtcgttgta 960

gggtatagga tttatgatgt ggttctgagt ccgtctcatg gaattgaact atctgttgga 1020

gaaaagcttg tcttaaattg tacagcaaga actgaactaa atgtggggat tgacttcaac 1080

tgggaatacc cttcttcgaa gcatcagcat aagaaacttg taaaccgaga cctaaaaacc 1140

cagtctggga gtgagatgaa gaaatttttg agcaccttaa ctatagatgg tgtaacccgg 1200

agtgaccaag gattgtacac ctgtgcagca tccagtgggc tgatgaccaa gaagaacagc 1260

acatttgtca gggtccatga aaaacctttt gttgcttttg gaagtggcat ggaatctctg 1320

gtggaagcca cggtgggggа gcgtgtcaga atccctgcga agtaccttgg ttacccaccc 1380

ccagaaataa aatggtataa aaatggaata ccccttgagt ccaatcacac aattaaagcg 1440

gggcatgtac tgacgattat ggaagtgagt gaaagagaca caggaaatta cactgtcatc 1500

cttaccaatc ccatttcaaa ggagaagcag agccatgtgg tctctctggt tgtgtatgtc 1560

ccaccccaga ttggtgagaa atctctaatc tctcctgtgg attcctacca gtacggcacc 1620

actcaaacgc tgacatgtac ggtctatgcc attcctcccc cgcatcacat ccactggtat 1680

tggcagttgg aggaagagtg cgccaacgag cccagccaag ctgtctcagt gacaaaccca 1740

taccettgtg aagaatggag aagtgtggag gacttccagg gaggaaataa aattgaagtt 1800

aataaaaatc aatttgctct aattgaagga aaaaacaaaa ctgtaagtac ccttgttatc 1860

caagcggcaa atgtgtcagc tttgtacaaa tgtgaagcgg tcaacaaagt cgggagagga 1920

gagagggtga tctccttcca cgtgaccagg ggtcctgaaa ttactttgca acctgacatg 1980

cagcccactg agcaggagag cgtgtctttg tggtgcactg cagacagatc tacgtttgag 2040

aacctcacat ggtacaagct tggcccacag cctctgccaa tccatgtggg agagttgccc 2100

acacctgttt gcaagaactt ggatactctt tggaaattga atgccaccat gttctctaat 2160

agcacaaatg acattttgat catggagctt aagaatgcat ccttgcagga ccaaggagac 2220

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tatgtctgcc ttgctcaaga caggaagacc aagaaaagac attgcgtggt caggcagctc 2280

acagtcctag agcgtgtggc acccacgatc acaggaaacc tggagaatca gacgacaagt 2340

attggggaaa gcatcgaagt ctcatgcacg gcatctggga atccccctcc acagatcatg 2400

tggtttaaag ataatgagac ccttgtagaa gactcaggca ttgtattgaa ggatgggaac 2460

cggaacctca ctatccgcag agtgaggaag gaggacgaag gcctctacac ctgccaggca 2520

tgcagtgttc ttggctgtgc aaaagtggag gcatttttca taatagaagg tgcccaggaa 2580

aagacgaact tggaaatcat tattctagta ggcacggcgg tgattgccat gttcttctgg 2640

ctacttcttg tcatcatcct acggaccgtt aagcgggcca atggagggga actgaagaca 2700

ggctacttgt ccatcgtcat ggatccagat gaactcccat tggatgaaca ttgtgaacga 2760

ctgccttatg atgccagcaa atgggaattc cccagagacc ggctgaagct aggtaagcct 2820

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gagaagtccc tcagtgatgt agaagaagag gaagctcctg aagatctgta taaggacttc 3300

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gcatcgcgaa agtgtatcca cagggacctg gcggcacgaa atatcctctt atcggagaag 3420

aacgtggtta aaatctgtga ctttggcttg gcccgggata tttataaaga tccagattat 3480

gtcagaaaag gagatgctcg cctccctttg aaatggatgg ccccagaaac aatttttgac 3540

agagtgtaca caatccagag tgacgtctgg tcttttggtg ttttgctgtg ggaaatattt 3600

tccttaggtg cttctccata tcctggggta aagattgatg aagaattttg taggcgattg 3660

aaagaaggaa ctagaatgag ggcccctgat tatactacac cagaaatgta ccagaccatg 3720

ctggactgct ggcacgggga gcccagtcag agacccacgt tttcagagtt ggtggaacat 3780

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atatcagaga ctttgagcat ggaagaggat tctggactct ctctgcctac ctcacctgtt 3900

tcctgtatgg aggaggagga agtatgtgac cccaaattcc attatgacaa cacagcagga 3960

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gaagatatcc cgttagaaga accagaagta aaagtaatcc cagatgacaa ccagacggac 4080

agtggtatgg ttcttgcctc agaagagctg aaaactttgg aagacagaac caaattatct 4140

ccatcttttg gtggaatggt gcccagcaaa agcagggagt ctgtggcatc tgaaggctca 4200

aaccagacaa gcggctacca gtccggatat cactccgatg acacagacac caccgtgtac 4260

tccagtgagg aagcagaact tttaaagctg atagagattg gagtgcaaac cggtagcaca 4320

gcccagattc tccagcctga ctcggggacc acactgagct ctcctcctgt ttaaaaggaa 4380

gcatccacac cccaactccc ggacatcaca tgagaggtct gctcagattt tgaagtgttg 4440

ttctttccac cagcaggaag tagccgcatt tgattttcat ttcgacaaca gaaaaaggac 4500

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ccccatcctc aaagaagtag cagtacctgg ggagctgaca cttctgtaaa actagaagat   4740

aaaccaggca acgtaagtgt tcgaggtgtt gaagatggga aggatttgca gggctgagtc   4800

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cggggtttct ggttttagaa ggttgcgtgt tcttcgagtt gggctaaagt agagttcgtt   5040

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ccccaggaag aaaatgatgc agctctggct ccttgtctcc caggctgatc ctttattcag   5160

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atatattaaa gccttatata taatgaactt tgtactattc acattttgta tcagtattat   5760

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aaaaacttga                                                          5830
```

<210> SEQ ID NO 21
<211> LENGTH: 1356
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 21

```
Met Gln Ser Lys Val Leu Leu Ala Val Ala Leu Trp Leu Cys Val Glu
1               5                   10                  15

Thr Arg Ala Ala Ser Val Gly Leu Pro Ser Val Ser Leu Asp Leu Pro
            20                  25                  30

Arg Leu Ser Ile Gln Lys Asp Ile Leu Thr Ile Lys Ala Asn Thr Thr
        35                  40                  45

Leu Gln Ile Thr Cys Arg Gly Gln Arg Asp Leu Asp Trp Leu Trp Pro
    50                  55                  60

Asn Asn Gln Ser Gly Ser Glu Gln Arg Val Glu Val Thr Glu Cys Ser
65                  70                  75                  80

Asp Gly Leu Phe Cys Lys Thr Leu Thr Ile Pro Lys Val Ile Gly Asn
                85                  90                  95

Asp Thr Gly Ala Tyr Lys Cys Phe Tyr Arg Glu Thr Asp Leu Ala Ser
            100                 105                 110

Val Ile Tyr Val Tyr Val Gln Asp Tyr Arg Ser Pro Phe Ile Ala Ser
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        115                 120                 125

Val Ser Asp Gln His Gly Val Val Tyr Ile Thr Glu Asn Lys Asn Lys
    130                 135                 140

Thr Val Val Ile Pro Cys Leu Gly Ser Ile Ser Asn Leu Asn Val Ser
145                 150                 155                 160

Leu Cys Ala Arg Tyr Pro Glu Lys Arg Phe Val Pro Asp Gly Asn Arg
                165                 170                 175

Ile Ser Trp Asp Ser Lys Lys Gly Phe Thr Ile Pro Ser Tyr Met Ile
            180                 185                 190

Ser Tyr Ala Gly Met Val Phe Cys Glu Ala Lys Ile Asn Asp Glu Ser
        195                 200                 205

Tyr Gln Ser Ile Met Tyr Ile Val Val Val Val Gly Tyr Arg Ile Tyr
    210                 215                 220

Asp Val Val Leu Ser Pro Ser His Gly Ile Glu Leu Ser Val Gly Glu
225                 230                 235                 240

Lys Leu Val Leu Asn Cys Thr Ala Arg Thr Glu Leu Asn Val Gly Ile
                245                 250                 255

Asp Phe Asn Trp Glu Tyr Pro Ser Ser Lys His Gln His Lys Lys Leu
            260                 265                 270

Val Asn Arg Asp Leu Lys Thr Gln Ser Gly Ser Glu Met Lys Lys Phe
        275                 280                 285

Leu Ser Thr Leu Thr Ile Asp Gly Val Thr Arg Ser Asp Gln Gly Leu
    290                 295                 300

Tyr Thr Cys Ala Ala Ser Ser Gly Leu Met Thr Lys Lys Asn Ser Thr
305                 310                 315                 320

Phe Val Arg Val His Glu Lys Pro Phe Val Ala Phe Gly Ser Gly Met
                325                 330                 335

Glu Ser Leu Val Glu Ala Thr Val Gly Glu Arg Val Arg Ile Pro Ala
            340                 345                 350

Lys Tyr Leu Gly Tyr Pro Pro Pro Glu Ile Lys Trp Tyr Lys Asn Gly
        355                 360                 365

Ile Pro Leu Glu Ser Asn His Thr Ile Lys Ala Gly His Val Leu Thr
    370                 375                 380

Ile Met Glu Val Ser Glu Arg Asp Thr Gly Asn Tyr Thr Val Ile Leu
385                 390                 395                 400

Thr Asn Pro Ile Ser Lys Glu Lys Gln Ser His Val Val Ser Leu Val
                405                 410                 415

Val Tyr Val Pro Pro Gln Ile Gly Glu Lys Ser Leu Ile Ser Pro Val
            420                 425                 430

Asp Ser Tyr Gln Tyr Gly Thr Thr Gln Thr Leu Thr Cys Thr Val Tyr
        435                 440                 445

Ala Ile Pro Pro Pro His His Ile His Trp Tyr Trp Gln Leu Glu Glu
    450                 455                 460

Glu Cys Ala Asn Glu Pro Ser Gln Ala Val Ser Val Thr Asn Pro Tyr
465                 470                 475                 480

Pro Cys Glu Glu Trp Arg Ser Val Glu Asp Phe Gln Gly Gly Asn Lys
                485                 490                 495

Ile Glu Val Asn Lys Asn Gln Phe Ala Leu Ile Glu Gly Lys Asn Lys
            500                 505                 510

Thr Val Ser Thr Leu Val Ile Gln Ala Ala Asn Val Ser Ala Leu Tyr
        515                 520                 525
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Lys Cys Glu Ala Val Asn Lys Val Gly Arg Gly Glu Arg Val Ile Ser
    530                 535                 540

Phe His Val Thr Arg Gly Pro Glu Ile Thr Leu Gln Pro Asp Met Gln
545                 550                 555                 560

Pro Thr Glu Gln Glu Ser Val Ser Leu Trp Cys Thr Ala Asp Arg Ser
                565                 570                 575

Thr Phe Glu Asn Leu Thr Trp Tyr Lys Leu Gly Pro Gln Pro Leu Pro
            580                 585                 590

Ile His Val Gly Glu Leu Pro Thr Pro Val Cys Lys Asn Leu Asp Thr
        595                 600                 605

Leu Trp Lys Leu Asn Ala Thr Met Phe Ser Asn Ser Thr Asn Asp Ile
    610                 615                 620

Leu Ile Met Glu Leu Lys Asn Ala Ser Leu Gln Asp Gln Gly Asp Tyr
625                 630                 635                 640

Val Cys Leu Ala Gln Asp Arg Lys Thr Lys Lys Arg His Cys Val Val
                645                 650                 655

Arg Gln Leu Thr Val Leu Glu Arg Val Ala Pro Thr Ile Thr Gly Asn
            660                 665                 670

Leu Glu Asn Gln Thr Thr Ser Ile Gly Glu Ser Ile Glu Val Ser Cys
        675                 680                 685

Thr Ala Ser Gly Asn Pro Pro Pro Gln Ile Met Trp Phe Lys Asp Asn
    690                 695                 700

Glu Thr Leu Val Glu Asp Ser Gly Ile Val Leu Lys Asp Gly Asn Arg
705                 710                 715                 720

Asn Leu Thr Ile Arg Arg Val Arg Lys Glu Asp Glu Gly Leu Tyr Thr
                725                 730                 735

Cys Gln Ala Cys Ser Val Leu Gly Cys Ala Lys Val Glu Ala Phe Phe
            740                 745                 750

Ile Ile Glu Gly Ala Gln Glu Lys Thr Asn Leu Glu Ile Ile Ile Leu
        755                 760                 765

Val Gly Thr Ala Val Ile Ala Met Phe Phe Trp Leu Leu Leu Val Ile
    770                 775                 780

Ile Leu Arg Thr Val Lys Arg Ala Asn Gly Gly Glu Leu Lys Thr Gly
785                 790                 795                 800

Tyr Leu Ser Ile Val Met Asp Pro Asp Glu Leu Pro Leu Asp Glu His
                805                 810                 815

Cys Glu Arg Leu Pro Tyr Asp Ala Ser Lys Trp Glu Phe Pro Arg Asp
            820                 825                 830

Arg Leu Lys Leu Gly Lys Pro Leu Gly Arg Gly Ala Phe Gly Gln Val
        835                 840                 845

Ile Glu Ala Asp Ala Phe Gly Ile Asp Lys Thr Ala Thr Cys Arg Thr
    850                 855                 860

Val Ala Val Lys Met Leu Lys Glu Gly Ala Thr His Ser Glu His Arg
865                 870                 875                 880

Ala Leu Met Ser Glu Leu Lys Ile Leu Ile His Ile Gly His His Leu
                885                 890                 895

Asn Val Val Asn Leu Leu Gly Ala Cys Thr Lys Pro Gly Gly Pro Leu
            900                 905                 910

Met Val Ile Val Glu Phe Cys Lys Phe Gly Asn Leu Ser Thr Tyr Leu
        915                 920                 925
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Arg Ser Lys Arg Asn Glu Phe Val Pro Tyr Lys Thr Lys Gly Ala Arg
    930                 935                 940

Phe Arg Gln Gly Lys Asp Tyr Val Gly Ala Ile Pro Val Asp Leu Lys
945                 950                 955                 960

Arg Arg Leu Asp Ser Ile Thr Ser Ser Gln Ser Ser Ala Ser Ser Gly
                965                 970                 975

Phe Val Glu Glu Lys Ser Leu Ser Asp Val Glu Glu Glu Glu Ala Pro
            980                 985                 990

Glu Asp Leu Tyr Lys Asp Phe Leu  Thr Leu Glu His Leu  Ile Cys Tyr
        995                 1000                1005

Ser Phe  Gln Val Ala Lys Gly  Met Glu Phe Leu Ala  Ser Arg Lys
    1010                1015                1020

Cys Ile  His Arg Asp Leu Ala  Ala Arg Asn Ile Leu  Leu Ser Glu
    1025                1030                1035

Lys Asn  Val Val Lys Ile Cys  Asp Phe Gly Leu Ala  Arg Asp Ile
    1040                1045                1050

Tyr Lys  Asp Pro Asp Tyr Val  Arg Lys Gly Asp Ala  Arg Leu Pro
    1055                1060                1065

Leu Lys  Trp Met Ala Pro Glu  Thr Ile Phe Asp Arg  Val Tyr Thr
    1070                1075                1080

Ile Gln  Ser Asp Val Trp Ser  Phe Gly Val Leu Leu  Trp Glu Ile
    1085                1090                1095

Phe Ser  Leu Gly Ala Ser Pro  Tyr Pro Gly Val Lys  Ile Asp Glu
    1100                1105                1110

Glu Phe  Cys Arg Arg Leu Lys  Glu Gly Thr Arg Met  Arg Ala Pro
    1115                1120                1125

Asp Tyr  Thr Thr Pro Glu Met  Tyr Gln Thr Met Leu  Asp Cys Trp
    1130                1135                1140

His Gly  Glu Pro Ser Gln Arg  Pro Thr Phe Ser Glu  Leu Val Glu
    1145                1150                1155

His Leu  Gly Asn Leu Leu Gln  Ala Asn Ala Gln Gln  Asp Gly Lys
    1160                1165                1170

Asp Tyr  Ile Val Leu Pro Ile  Ser Glu Thr Leu Ser  Met Glu Glu
    1175                1180                1185

Asp Ser  Gly Leu Ser Leu Pro  Thr Ser Pro Val Ser  Cys Met Glu
    1190                1195                1200

Glu Glu  Glu Val Cys Asp Pro  Lys Phe His Tyr Asp  Asn Thr Ala
    1205                1210                1215

Gly Ile  Ser Gln Tyr Leu Gln  Asn Ser Lys Arg Lys  Ser Arg Pro
    1220                1225                1230

Val Ser  Val Lys Thr Phe Glu  Asp Ile Pro Leu Glu  Glu Pro Glu
    1235                1240                1245

Val Lys  Val Ile Pro Asp Asp  Asn Gln Thr Asp Ser  Gly Met Val
    1250                1255                1260

Leu Ala  Ser Glu Glu Leu Lys  Thr Leu Glu Asp Arg  Thr Lys Leu
    1265                1270                1275

Ser Pro  Ser Phe Gly Gly Met  Val Pro Ser Lys Ser  Arg Glu Ser
    1280                1285                1290

Val Ala  Ser Glu Gly Ser Asn  Gln Thr Ser Gly Tyr  Gln Ser Gly
    1295                1300                1305

Tyr His  Ser Asp Asp Thr Asp  Thr Thr Val Tyr Ser  Ser Glu Glu
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    1310                1315                1320

Ala Glu  Leu Leu Lys Leu Ile  Glu Ile Gly Val Gln  Thr Gly Ser
    1325                1330                1335

Thr Ala  Gln Ile Leu Gln Pro  Asp Ser Gly Thr Thr  Leu Ser Ser
    1340                1345                1350

Pro Pro  Val
    1355


<210> SEQ ID NO 22
<211> LENGTH: 16
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 22

Cys Asn Asp Glu Gly Leu Glu Cys Val Pro Thr Glu Glu Ser Asn Ile
1               5                   10                  15


<210> SEQ ID NO 23
<211> LENGTH: 16
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 23

Cys Pro Asp Asp Gly Leu Glu Cys Val Pro Thr Gly Gln His Gln Val
1               5                   10                  15


<210> SEQ ID NO 24
<211> LENGTH: 1676
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens

<400> SEQUENCE: 24

Met Gly Leu Leu Gly Ile Leu Cys Phe Leu Ile Phe Leu Gly Lys Thr
1               5                   10                  15

Trp Gly Gln Glu Gln Thr Tyr Val Ile Ser Ala Pro Lys Ile Phe Arg
            20                  25                  30

Val Gly Ala Ser Glu Asn Ile Val Ile Gln Val Tyr Gly Tyr Thr Glu
        35                  40                  45

Ala Phe Asp Ala Thr Ile Ser Ile Lys Ser Tyr Pro Asp Lys Lys Phe
    50                  55                  60

Ser Tyr Ser Ser Gly His Val His Leu Ser Ser Glu Asn Lys Phe Gln
65                  70                  75                  80

Asn Ser Ala Ile Leu Thr Ile Gln Pro Lys Gln Leu Pro Gly Gly Gln
                85                  90                  95

Asn Pro Val Ser Tyr Val Tyr Leu Glu Val Val Ser Lys His Phe Ser
            100                 105                 110

Lys Ser Lys Arg Met Pro Ile Thr Tyr Asp Asn Gly Phe Leu Phe Ile
        115                 120                 125

His Thr Asp Lys Pro Val Tyr Thr Pro Asp Gln Ser Val Lys Val Arg
    130                 135                 140

Val Tyr Ser Leu Asn Asp Asp Leu Lys Pro Ala Lys Arg Glu Thr Val
145                 150                 155                 160

Leu Thr Phe Ile Asp Pro Glu Gly Ser Glu Val Asp Met Val Glu Glu
                165                 170                 175

Ile Asp His Ile Gly Ile Ile Ser Phe Pro Asp Phe Lys Ile Pro Ser
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Asn Pro Arg Tyr Gly Met Trp Thr Ile Lys Ala Lys Tyr Lys Glu Asp
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Phe Ser Thr Thr Gly Thr Ala Tyr Phe Glu Val Lys Glu Tyr Val Leu
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Pro His Phe Ser Val Ser Ile Glu Pro Glu Tyr Asn Phe Ile Gly Tyr
225                 230                 235                 240

Lys Asn Phe Lys Asn Phe Glu Ile Thr Ile Lys Ala Arg Tyr Phe Tyr
                245                 250                 255

Asn Lys Val Val Thr Glu Ala Asp Val Tyr Ile Thr Phe Gly Ile Arg
            260                 265                 270

Glu Asp Leu Lys Asp Asp Gln Lys Glu Met Met Gln Thr Ala Met Gln
        275                 280                 285

Asn Thr Met Leu Ile Asn Gly Ile Ala Gln Val Thr Phe Asp Ser Glu
    290                 295                 300

Thr Ala Val Lys Glu Leu Ser Tyr Tyr Ser Leu Glu Asp Leu Asn Asn
305                 310                 315                 320

Lys Tyr Leu Tyr Ile Ala Val Thr Val Ile Glu Ser Thr Gly Gly Phe
                325                 330                 335

Ser Glu Glu Ala Glu Ile Pro Gly Ile Lys Tyr Val Leu Ser Pro Tyr
            340                 345                 350

Lys Leu Asn Leu Val Ala Thr Pro Leu Phe Leu Lys Pro Gly Ile Pro
        355                 360                 365

Tyr Pro Ile Lys Val Gln Val Lys Asp Ser Leu Asp Gln Leu Val Gly
    370                 375                 380

Gly Val Pro Val Thr Leu Asn Ala Gln Thr Ile Asp Val Asn Gln Glu
385                 390                 395                 400

Thr Ser Asp Leu Asp Pro Ser Lys Ser Val Thr Arg Val Asp Asp Gly
                405                 410                 415

Val Ala Ser Phe Val Leu Asn Leu Pro Ser Gly Val Thr Val Leu Glu
            420                 425                 430

Phe Asn Val Lys Thr Asp Ala Pro Asp Leu Pro Glu Glu Asn Gln Ala
        435                 440                 445

Arg Glu Gly Tyr Arg Ala Ile Ala Tyr Ser Ser Leu Ser Gln Ser Tyr
    450                 455                 460

Leu Tyr Ile Asp Trp Thr Asp Asn His Lys Ala Leu Leu Val Gly Glu
465                 470                 475                 480

His Leu Asn Ile Ile Val Thr Pro Lys Ser Pro Tyr Ile Asp Lys Ile
                485                 490                 495

Thr His Tyr Asn Tyr Leu Ile Leu Ser Lys Gly Lys Ile Ile His Phe
            500                 505                 510

Gly Thr Arg Glu Lys Phe Ser Asp Ala Ser Tyr Gln Ser Ile Asn Ile
        515                 520                 525

Pro Val Thr Gln Asn Met Val Pro Ser Ser Arg Leu Leu Val Tyr Tyr
    530                 535                 540

Ile Val Thr Gly Glu Gln Thr Ala Glu Leu Val Ser Asp Ser Val Trp
545                 550                 555                 560

Leu Asn Ile Glu Glu Lys Cys Gly Asn Gln Leu Gln Val His Leu Ser
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Pro Asp Ala Asp Ala Tyr Ser Pro Gly Gln Thr Val Ser Leu Asn Met
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Val Tyr Gly Val Gln Arg Gly Ala Lys Lys Pro Leu Glu Arg Val Phe
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Gln Phe Leu Glu Lys Ser Asp Leu Gly Cys Gly Ala Gly Gly Gly Leu
625                 630                 635                 640

Asn Asn Ala Asn Val Phe His Leu Ala Gly Leu Thr Phe Leu Thr Asn
                645                 650                 655

Ala Asn Ala Asp Asp Ser Gln Glu Asn Asp Glu Pro Cys Lys Glu Ile
            660                 665                 670

Leu Arg Pro Arg Arg Thr Leu Gln Lys Lys Ile Glu Glu Ile Ala Ala
        675                 680                 685

Lys Tyr Lys His Ser Val Val Lys Lys Cys Cys Tyr Asp Gly Ala Cys
    690                 695                 700

Val Asn Asn Asp Glu Thr Cys Glu Gln Arg Ala Ala Arg Ile Ser Leu
705                 710                 715                 720

Gly Pro Arg Cys Ile Lys Ala Phe Thr Glu Cys Cys Val Val Ala Ser
                725                 730                 735

Gln Leu Arg Ala Asn Ile Ser His Lys Asp Met Gln Leu Gly Arg Leu
            740                 745                 750

His Met Lys Thr Leu Leu Pro Val Ser Lys Pro Glu Ile Arg Ser Tyr
        755                 760                 765

Phe Pro Glu Ser Trp Leu Trp Glu Val His Leu Val Pro Arg Arg Lys
    770                 775                 780

Gln Leu Gln Phe Ala Leu Pro Asp Ser Leu Thr Thr Trp Glu Ile Gln
785                 790                 795                 800

Gly Val Gly Ile Ser Asn Thr Gly Ile Cys Val Ala Asp Thr Val Lys
                805                 810                 815

Ala Lys Val Phe Lys Asp Val Phe Leu Glu Met Asn Ile Pro Tyr Ser
            820                 825                 830

Val Val Arg Gly Glu Gln Ile Gln Leu Lys Gly Thr Val Tyr Asn Tyr
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Arg Thr Ser Gly Met Gln Phe Cys Val Lys Met Ser Ala Val Glu Gly
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Ile Cys Thr Ser Glu Ser Pro Val Ile Asp His Gln Gly Thr Lys Ser
865                 870                 875                 880

Ser Lys Cys Val Arg Gln Lys Val Glu Gly Ser Ser Ser His Leu Val
                885                 890                 895

Thr Phe Thr Val Leu Pro Leu Glu Ile Gly Leu His Asn Ile Asn Phe
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Ser Leu Glu Thr Trp Phe Gly Lys Glu Ile Leu Val Lys Thr Leu Arg
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Val Val Pro Glu Gly Val Lys Arg Glu Ser Tyr Ser Gly Val Thr Leu
    930                 935                 940

Asp Pro Arg Gly Ile Tyr Gly Thr Ile Ser Arg Arg Lys Glu Phe Pro
945                 950                 955                 960

Tyr Arg Ile Pro Leu Asp Leu Val Pro Lys Thr Glu Ile Lys Arg Ile
                965                 970                 975

Leu Ser Val Lys Gly Leu Leu Val Gly Glu Ile Leu Ser Ala Val Leu
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Ser Gln Glu Gly Ile Asn Ile Leu  Thr His Leu Pro Lys  Gly Ser Ala
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Leu Ile  Glu Lys Gln Lys Leu  Lys Lys Lys Leu Lys  Glu Gly Met
    1040                1045                1050

Leu Ser  Ile Met Ser Tyr Arg  Asn Ala Asp Tyr Ser  Tyr Ser Val
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Trp Lys  Gly Gly Ser Ala Ser  Thr Trp Leu Thr Ala  Phe Ala Leu
    1070                1075                1080

Arg Val  Leu Gly Gln Val Asn  Lys Tyr Val Glu Gln  Asn Gln Asn
    1085                1090                1095

Ser Ile  Cys Asn Ser Leu Leu  Trp Leu Val Glu Asn  Tyr Gln Leu
    1100                1105                1110

Asp Asn  Gly Ser Phe Lys Glu  Asn Ser Gln Tyr Gln  Pro Ile Lys
    1115                1120                1125

Leu Gln  Gly Thr Leu Pro Val  Glu Ala Arg Glu Asn  Ser Leu Tyr
    1130                1135                1140

Leu Thr  Ala Phe Thr Val Ile  Gly Ile Arg Lys Ala  Phe Asp Ile
    1145                1150                1155

Cys Pro  Leu Val Lys Ile Asp  Thr Ala Leu Ile Lys  Ala Asp Asn
    1160                1165                1170

Phe Leu  Leu Glu Asn Thr Leu  Pro Ala Gln Ser Thr  Phe Thr Leu
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Ala Ile  Ser Ala Tyr Ala Leu  Ser Leu Gly Asp Lys  Thr His Pro
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Gln Phe  Arg Ser Ile Val Ser  Ala Leu Lys Arg Glu  Ala Leu Val
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Lys Gly  Asn Pro Pro Ile Tyr  Arg Phe Trp Lys Asp  Asn Leu Gln
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His Lys  Asp Ser Ser Val Pro  Asn Thr Gly Thr Ala  Arg Met Val
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Glu Thr  Thr Ala Tyr Ala Leu  Leu Thr Ser Leu Asn  Leu Lys Asp
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Ile Asn  Tyr Val Asn Pro Val  Ile Lys Trp Leu Ser  Glu Glu Gln
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Arg Tyr  Gly Gly Gly Phe Tyr  Ser Thr Gln Asp Thr  Ile Asn Ala
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Ile Glu  Gly Leu Thr Glu Tyr  Ser Leu Leu Val Lys  Gln Leu Arg
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Leu Ser  Met Asp Ile Asp Val  Ser Tyr Lys His Lys  Gly Ala Leu
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His Asn  Tyr Lys Met Thr Asp  Lys Asn Phe Leu Gly  Arg Pro Val
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Glu Val  Leu Leu Asn Asp Asp  Leu Ile Val Ser Thr  Gly Phe Gly
    1340                1345                1350

Ser Gly  Leu Ala Thr Val His  Val Thr Thr Val Val  His Lys Thr
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Ser Thr  Ser Glu Glu Val Cys  Ser Phe Tyr Leu Lys  Ile Asp Thr
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Gln Asp  Ile Glu Ala Ser His  Tyr Arg Gly Tyr Gly  Asn Ser Asp
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Tyr Lys  Arg Ile Val Ala Cys  Ala Ser Tyr Lys Pro  Ser Arg Glu
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Glu Ser  Ser Ser Gly Ser Ser  His Ala Val Met Asp  Ile Ser Leu
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Pro Thr  Gly Ile Ser Ala Asn  Glu Glu Asp Leu Lys  Ala Leu Val
    1430                1435                1440

Glu Gly  Val Asp Gln Leu Phe  Thr Asp Tyr Gln Ile  Lys Asp Gly
    1445                1450                1455

His Val  Ile Leu Gln Leu Asn  Ser Ile Pro Ser Ser  Asp Phe Leu
    1460                1465                1470

Cys Val  Arg Phe Arg Ile Phe  Glu Leu Phe Glu Val  Gly Phe Leu
    1475                1480                1485

Ser Pro  Ala Thr Phe Thr Val  Tyr Glu Tyr His Arg  Pro Asp Lys
    1490                1495                1500

Gln Cys  Thr Met Phe Tyr Ser  Thr Ser Asn Ile Lys  Ile Gln Lys
    1505                1510                1515

Val Cys  Glu Gly Ala Ala Cys  Lys Cys Val Glu Ala  Asp Cys Gly
    1520                1525                1530

Gln Met  Gln Glu Glu Leu Asp  Leu Thr Ile Ser Ala  Glu Thr Arg
    1535                1540                1545

Lys Gln  Thr Ala Cys Lys Pro  Glu Ile Ala Tyr Ala  Tyr Lys Val
    1550                1555                1560

Ser Ile  Thr Ser Ile Thr Val  Glu Asn Val Phe Val  Lys Tyr Lys
    1565                1570                1575

Ala Thr  Leu Leu Asp Ile Tyr  Lys Thr Gly Glu Ala  Val Ala Glu
    1580                1585                1590

Lys Asp  Ser Glu Ile Thr Phe  Ile Lys Lys Val Thr  Cys Thr Asn
    1595                1600                1605

Ala Glu  Leu Val Lys Gly Arg  Gln Tyr Leu Ile Met  Gly Lys Glu
    1610                1615                1620

Ala Leu  Gln Ile Lys Tyr Asn  Phe Ser Phe Arg Tyr  Ile Tyr Pro
    1625                1630                1635

Leu Asp  Ser Leu Thr Trp Ile  Glu Tyr Trp Pro Arg  Asp Thr Thr
    1640                1645                1650

Cys Ser  Ser Cys Gln Ala Phe  Leu Ala Asn Leu Asp  Glu Phe Ala
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<400> SEQUENCE: 43

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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine

<400> SEQUENCE: 44

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<210> SEQ ID NO 45
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<400> SEQUENCE: 50

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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine

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<400> SEQUENCE: 59

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<210> SEQ ID NO 60
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<400> SEQUENCE: 63

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<400> SEQUENCE: 64

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<210> SEQ ID NO 65
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine

<400> SEQUENCE: 65

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<210> SEQ ID NO 66
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<223> OTHER INFORMATION: May be deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine

<400> SEQUENCE: 66

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<210> SEQ ID NO 67
<211> LENGTH: 38
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'OH-guanosine

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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 84

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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 85

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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 86

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<210> SEQ ID NO 87
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxyuridine
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<223> OTHER INFORMATION: May be 2'OH-adenosine
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<222> LOCATION: (35)..(35)
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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 87

cgccgcgguc ucaggcgcug agucugaguu uaccugcgt 39

<210> SEQ ID NO 88
<211> LENGTH: 39
<212> TYPE: DNA
<213> ORGANISM: Artificial Sequence
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attached via a hexylamine linker
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<221> NAME/KEY: misc_feature
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<220> FEATURE:

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<223> OTHER INFORMATION: May be 2'OH-adenosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 88

cgccgcgguc ucaggcgcug agucugaguu uaccugcgt 39

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<213> ORGANISM: Artificial Sequence
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<400> SEQUENCE: 89

gggagaggag agaacguucu accuugguuu ggcacaggca uacauacgca ggggucgauc 60

gaucgaucau cgaug 75

<210> SEQ ID NO 90
<211> LENGTH: 32
<212> TYPE: RNA
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Synthetic C5 specific aptamer

<400> SEQUENCE: 90

ccuugguuug gcacaggcau acauacgcag gg 32

<210> SEQ ID NO 91
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<223> OTHER INFORMATION: Synthetic C5 specific aptamer

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cguucuaccu ugguuuggca caggcauaca uacgcagggg ucgaucg 47

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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyadenosine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxyuridine
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<223> OTHER INFORMATION: May be 2'OH-adenosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be an inverted orientation T (3'-3'-linked)

<400> SEQUENCE: 92

cgccgcgguc ucaggcgcug agucugaguu uaccugcgt 39

<210> SEQ ID NO 93
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attached via a hexylamine linker
<220> FEATURE:
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxyuridine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
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<221> NAME/KEY: misc_feature
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<223> OTHER INFORMATION: May be 2'OH-adenosine
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<223> OTHER INFORMATION: May be 2'-fluoro-2'-deoxycytidine
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<223> OTHER INFORMATION: May be 2'-O-Methyl-2'-deoxyguanosine
<220> FEATURE:
<221> NAME/KEY: misc_feature
<222> LOCATION: (38)..(38)
<223> OTHER INFORMATION: May have a 20 kDa polyethylene glycol group attached via a hexylamine linker

<400> SEQUENCE: 93

cgccgcgguc ucaggcgcug agucugaguu uaccugcg 38

<210> SEQ ID NO 94
<211> LENGTH: 80
<212> TYPE: RNA
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
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<400> SEQUENCE: 94

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cguuacgacu agcaucgaug 80

<210> SEQ ID NO 95
<211> LENGTH: 79
<212> TYPE: RNA
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Synthetic C5 specific aptamer

<400> SEQUENCE: 95

gggagaggag agaacguucu accuuagguu cgcacuguca uacauacaca cgggcaaucg 60

guuacgacua gcaucgaug 79

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<210> SEQ ID NO 96
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<212> TYPE: RNA
<213> ORGANISM: Artificial Sequence
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<223> OTHER INFORMATION: n is a, c, g, or u
<220> FEATURE:
<221> NAME/KEY: misc_feature
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<223> OTHER INFORMATION: n is a, c, g, or u

<400> SEQUENCE: 96

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gguuacgacu agcau 75

<210> SEQ ID NO 97
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<212> TYPE: PRT
<213> ORGANISM: Unknown
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<400> SEQUENCE: 97

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Asp Asp Glu Val Arg Ile Leu Met Ala Asn Gly Ala Asp Val Asn Thr
20 25 30

Ala Asp Ser Thr Gly Trp Thr Pro Leu His Leu Ala Val Pro Trp Gly
35 40 45

His Leu Glu Ile Val Glu Val Leu Leu Lys Tyr Gly Ala Asp Val Asn
50 55 60

Ala Lys Asp Phe Gln Gly Trp Thr Pro Leu His Leu Ala Ala Ala Ile
65 70 75 80

Gly His Gln Glu Ile Val Glu Val Leu Leu Lys Asn Gly Ala Asp Val
85 90 95

Asn Ala Gln Asp Lys Phe Gly Lys Thr Ala Phe Asp Ile Ser Ile Asp
100 105 110

Asn Gly Asn Glu Asp Leu Ala Glu Ile Leu Gln Lys Ala Ala
115 120 125

<210> SEQ ID NO 98
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: recombinant human soluble VEGF receptor fusion protein

<400> SEQUENCE: 98

Met Val Ser Tyr Trp Asp Thr Gly Val Leu Leu Cys Ala Leu Leu Ser
1 5 10 15

Cys Leu Leu Leu Thr Gly Ser Ser Ser Gly Gly Arg Pro Phe Val Glu
20 25 30

Met Tyr Ser Glu Ile Pro Glu Ile Ile His Met Thr Glu Gly Arg Glu
35 40 45

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Leu Val Ile Pro Cys Arg Val Thr Ser Pro Asn Ile Thr Val Thr Leu
    50                  55                  60

Lys Lys Phe Pro Leu Asp Thr Leu Ile Pro Asp Gly Lys Arg Ile Ile
65                  70                  75                  80

Trp Asp Ser Arg Lys Gly Phe Ile Ile Ser Asn Ala Thr Tyr Lys Glu
                85                  90                  95

Ile Gly Leu Leu Thr Cys Glu Ala Thr Val Asn Gly His Leu Tyr Lys
            100                 105                 110

Thr Asn Tyr Leu Thr His Arg Gln Thr Asn Thr Ile Ile Asp Val Val
        115                 120                 125

Leu Ser Pro Ser His Gly Ile Glu Leu Ser Val Gly Glu Lys Leu Val
    130                 135                 140

Leu Asn Cys Thr Ala Arg Thr Glu Leu Asn Val Gly Ile Asp Phe Asn
145                 150                 155                 160

Trp Glu Tyr Pro Ser Ser Lys His Gln His Lys Lys Leu Val Asn Arg
                165                 170                 175

Asp Leu Lys Thr Gln Ser Gly Ser Glu Met Lys Lys Phe Leu Ser Thr
            180                 185                 190

Leu Thr Ile Asp Gly Val Thr Arg Ser Asp Gln Gly Leu Tyr Thr Cys
        195                 200                 205

Ala Ala Ser Ser Gly Leu Met Thr Lys Lys Asn Ser Thr Phe Val Arg
    210                 215                 220

Val His Glu Lys Pro Phe Val Ala Phe Gly Ser Gly Met Glu Ser Leu
225                 230                 235                 240

Val Glu Ala Thr Val Gly Glu Arg Val Arg Leu Pro Ala Lys Tyr Leu
                245                 250                 255

Gly Tyr Pro Pro Pro Glu Ile Lys Trp Tyr Lys Asn Gly Ile Pro Leu
            260                 265                 270

Glu Ser Asn His Thr Ile Lys Ala Gly His Val Leu Thr Ile Met Glu
        275                 280                 285

Val Ser Glu Arg Asp Thr Gly Asn Tyr Thr Val Ile Leu Thr Asn Pro
    290                 295                 300

Ile Ser Lys Glu Lys Gln Ser His Val Val Ser Leu Val Val Tyr Val
305                 310                 315                 320

Pro Pro Gly Pro Gly Asp Lys Thr His Thr Cys Pro Leu Cys Pro Ala
                325                 330                 335

Pro Glu Leu Leu Gly Gly Pro Ser Val Phe Leu Phe Pro Pro Lys Pro
            340                 345                 350

Lys Asp Thr Leu Met Ile Ser Arg Thr Pro Glu Val Thr Cys Val Val
        355                 360                 365

Val Asp Val Ser His Glu Asp Pro Glu Val Lys Phe Asn Trp Tyr Val
    370                 375                 380

Asp Gly Val Glu Val His Asn Ala Lys Thr Lys Pro Arg Glu Glu Gln
385                 390                 395                 400

Tyr Asn Ser Thr Tyr Arg Val Val Ser Val Leu Thr Val Leu His Gln
                405                 410                 415

Asp Trp Leu Asn Gly Lys Glu Tyr Lys Cys Lys Val Ser Asn Lys Ala
            420                 425                 430

Leu Pro Ala Pro Ile Glu Lys Thr Ile Ser Lys Ala Lys Gly Gln Pro
        435                 440                 445
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Arg Glu Pro Gln Val Tyr Thr Leu Pro Pro Ser Arg Asp Glu Leu Thr
    450                 455                 460

Lys Asn Gln Val Ser Leu Thr Cys Leu Val Lys Gly Phe Tyr Pro Ser
465                 470                 475                 480

Asp Ile Ala Val Glu Trp Glu Ser Asn Gly Gln Pro Glu Asn Asn Tyr
                485                 490                 495

Lys Ala Thr Pro Pro Val Leu Asp Ser Asp Gly Ser Phe Phe Leu Tyr
            500                 505                 510

Ser Lys Leu Thr Val Asp Lys Ser Arg Trp Gln Gln Gly Asn Val Phe
        515                 520                 525

Ser Cys Ser Val Met His Glu Ala Leu His Asn His Tyr Thr Gln Lys
    530                 535                 540

Ser Leu Ser Leu Ser Pro Gly Lys
545                 550
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What is claimed is:

1. A method for treating or preventing wet age-related macular degeneration (wet AMD), comprising administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof and (b) an VEGF antagonist, wherein (a) and (b) are administered in an amount that is effective for treating or preventing wet AMD, and wherein the administering occurs once every month, ±about seven days, for a first administration period of at least 3 consecutive months, followed by administering (a) and (b) for a second administration period at a frequency of at least every other month±about seven days beginning at two months±about seven days after the day of the last month of the first administration period on which (a) and (b) are administered.

2. The method of claim **1**, wherein (a) and (b) are administered within about 60 minutes of each other.

3. The method of claim **1**, wherein the VEGF antagonist is ranibizumab, bevacizumab, pegaptanib sodium, ESBA1008 or aflibercept.

4. The method of claim **1**, wherein the VEGF antagonist is ranibizumab or bevacizumab, wherein (a) and (b) are administered at a frequency of once every month±about seven days during the second administration period and wherein the second administration period is at least about nine months.

5. The method of claim **4**, further comprising measuring the subject's visual acuity.

6. The method of claim **5**, further comprising administering to the subject (a) and (b) in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity on the last two of any three consecutive months is ≤a five-ETDRS-letter difference from the subject's visual acuity on the first of the three consecutive months.

7. The method of claim **5**, further comprising administering to the subject (a) and (b) every other month in an amount that is effective for treating or preventing wet AMD, wherein the subject's visual acuity on the last two of any three consecutive months is 5 a five-ETDRS-letter difference from the subject's visual acuity on the first of the three consecutive months.

8. The method of claim **7**, further comprising administering to the subject (a) and (b) every month in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity on the last two of any three consecutive months is ≤a five-ETDRS-letter difference from the subject's visual acuity on the first of the three consecutive months.

9. The method of claim **1**, wherein the VEGF antagonist is aflibercept.

10. The method of claim **1**, wherein the total number of months does not exceed 24.

11. The method of claim **4**, wherein the subject has intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid at one month, ±about seven days, immediately following the second administration period.

12. The method of claim **11**, further comprising: administering to the subject on each month±about seven days, beginning on the month that immediately follows the second administration period (a) and (b) in an amount that is effective for treating or preventing wet AMD, until the subject's visual acuity on the last two of any three consecutive months that follow the 12 consecutive months is ≤a five-ETDRS-letter difference from the subject's visual acuity on the first of the three consecutive months.

13. The method of claim **12**, wherein the total number of months does not exceed 24.

14. The method of claim **1**, wherein Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally in an amount of about 1.5 mg/eye.

15. The method of claim **4**, wherein the VEGF antagonist is bevacizumab and is administered intravitreally in an amount of about 1.25 mg/eye.

16. The method of claim **9**, wherein the VEGF antagonist is administered intravitreally in an amount of about 2 mg/eye.

17. The method of claim **4**, wherein the VEGF antagonist is ranibizumab and is administered intravitreally in an amount of about 0.5 mg/eye.

18. The method of claim **1**, further comprising administering an anti-C5 agent.

19. The method of claim **1**, further comprising administering (a) and (b) on a month in which the subject has intraretinal or sub-retinal hemorrhage or a ≥50 μm increase in foveal intraretinal fluid.

20. A method for treating or preventing sub-retinal fibrosis, comprising administering to a subject in need thereof (a) Antagonist A or another pharmaceutically acceptable salt thereof in an amount that is effective for treating or preventing sub-retinal fibrosis.

21. The method of claim **20**, further comprising administering to the subject (b) a VEGF antagonist, wherein (a) and (b) are administered in an amount that is effective for treating or preventing sub-retinal fibrosis.

22. The method of claim **20**, wherein the subject has wet age-related macular degeneration (wet AMD).

23. The method of claim **22**, wherein the sub-retinal fibrosis is associated with the wet AMD.

24. The method of claim **20**, wherein administering Antagonist A or another pharmaceutically acceptable salt thereof results in a decrease in the size of sub-retinal hyperreflective material (SHRM) as evidenced by spectral domain optical coherence tomography (SD-OCT) or results in stabilization of the subject's vision.

25. The method of claim **20**, wherein Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally in an amount of about 1.5 mg/eye.

26. The method of claim **21**, wherein the VEGF antagonist is bevacizumab, ranibizumab, aflibercept, pegaptanib sodium or ESBA1008.

27. The method of claim **26**, wherein the VEGF antagonist is bevacizumab and is administered intravitreally in an amount of about 1.25 mg/eye.

28. The method of claim **26**, wherein the VEGF antagonist is aflibercept and is administered intravitreally in an amount of about 2 mg/eye.

29. The method of claim **26**, wherein the VEGF antagonist is ranibizumab and is administered intravitreally in an amount of about 0.5 mg/eye.

30. The method of claim **21**, further comprising administering an anti-C5 agent.

31. A method for treating or preventing von Hippel-Lindau (VHL) disease, comprising administering to a subject in need thereof. Antagonist A or another pharmaceutically acceptable salt thereof in an amount that is effective for treating or preventing VHL disease.

32. The method of claim **31**, further comprising administering a VEGF antagonist.

33. The method of claim **31**, wherein Antagonist A or another pharmaceutically acceptable salt thereof is administered intravitreally in an amount of about 1.5 mg/eye.

34. The method of claim **32**, wherein the VEGF antagonist is bevacizumab and is administered intravitreally in an amount of about 1.25 mg/eye.

35. The method of claim **32**, wherein the VEGF antagonist is aflibercept and is administered intravitreally in an amount of about 2 mg/eye.

36. The method of claim **32**, wherein the VEGF antagonist is ranibizumab and is administered intravitreally in an amount of about 0.5 mg/eye.

37. The method of claim **32**, further comprising administering an anti-C5 agent.

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