of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of the at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy other than or in addition to a VEGF antagonist.

[0008a] In one aspect, there is provided a method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising: determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

[0009] Another embodiment of the invention provides methods of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF antagonist. The methods comprise: determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of the at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy other than or in addition to a VEGF antagonist.

[0009a] In one aspect, there is provided a method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising: determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

[0009b] In one aspect, there is provided a method of optimizing therapeutic efficacy for treatment of cancer, the method comprising determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

[0009c] In one aspect, there is provided a method of optimizing therapeutic efficacy for treatment of cancer, the method comprising determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

[0010] A further embodiment of the invention provides methods of predicting responsiveness of a patient suffering from cancer to treatment with an anti-cancer therapy other than or in addition to a VEGF antagonist. The methods comprise determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of the at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the anti-cancer therapy other than or in addition to a VEGF antagonist.

[0011] Yet another embodiment of the invention provides methods of predicting responsiveness of a patient suffering from cancer to treatment with an anti-cancer therapy other than or in addition to a VEGF antagonist. The methods comprise: determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of the at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the anti-cancer therapy other than or in addition to a VEGF antagonist.

[0012] Even another embodiment of the invention provides methods for determining the likelihood that a patient with cancer will exhibit benefit from anti-cancer therapy other than or in addition to a VEGF antagonist. The methods comprise: determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of the at least one gene in the sample as compared to a reference sample indicates that the patient

level of the at least one gene in a reference sample indicates that the patient may benefit from treatment with a VEGF-C antagonist. In some embodiments of the invention, the compounds are polynucleotides. In some embodiments of the invention, the compounds are proteins, such as, for example, antibodies.

[0103] One embodiment of the invention provides methods of identifying a patient suffering from cancer who may benefit from treatment with an EGF-like-domain, multiple 7 (EGFL7) antagonist. The methods comprise determining expression levels of at least one gene selected from: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

[0103a] In one aspect, there is provided a method of identifying a patient suffering from cancer who may benefit from treatment with an EGF-like-domain, multiple 7 (EGFL7) antagonist, the method comprising determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

[0104] Another embodiment of the invention provides methods of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

[0104a] In one aspect, there is provided a method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist

[0105] A further embodiment of the invention provides methods of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

[0106] Yet another embodiment of the invention provides methods of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of the at least one gene

in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

[0107] Even another embodiment of the invention provides methods of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

[0108] Yet a further embodiment of the invention provides methods of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

[0109] Even another embodiment of the invention provides methods of optimizing therapeutic efficacy of an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

[0109a] In one aspect, there is provided a method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from a patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

[0110] Yet a further embodiment of the invention provides methods of optimizing therapeutic efficacy of an EGFL7 antagonist. The methods comprise determining expression levels of at least one gene selected from: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of the at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

[0110a] In one aspect, there is provided a method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from a patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

[0111] Another embodiment of the invention provides methods for treating a cell proliferative disorder in a patient. The methods comprise determining

that a sample obtained from the patient has increased expression levels, as compared to a reference sample, of at least one gene selected from: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle, and administering to the patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

[0111a] In one aspect, there is provided use of an EGFL7 antagonist in the manufacture of a medicament for the treatment of a cell proliferative disorder in a patient, wherein the patient is one whose sample has increased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle.

[0112] A further embodiment of the invention provides methods for treating a cell proliferative disorder in a patient. The methods comprise determining that a sample obtained from the patient has decreased expression levels, as compared to a reference sample, of at least one gene selected from: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, and administering to the patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

[0112a] In one aspect, there is provided use of an EGFL7 antagonist in the manufacture of a medicament for the treatment of a cell proliferative disorder in a patient, wherein the patient is one whose sample has decreased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1.

[0113] In some embodiments of the invention, the sample obtained from the patient is selected from: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof. In some embodiments of the invention, the expression level is mRNA expression level. In some embodiments of the invention, the expression level is protein expression level. In some embodiments of the invention, the EGFL7 antagonist is an anti-EGFL7 antibody.

[0114] In some embodiments of the invention, the methods further comprises administering a VEGF-A antagonist to the patient. In some embodiments of the invention, the VEGF-A antagonist and the EGFL7 antagonist are administered concurrently. In some embodiments of the invention, the VEGF-A antagonist and the EGFL7 antagonist are administered sequentially. In some embodiments of the invention, the VEGF-A antagonist is an anti-VEGF-A antibody. In some embodiments of the invention, the anti-VEGF-A antibody is bevacizumab.

[0115] A further embodiment of the invention provides methods of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist. The methods comprise determining expression levels of VEGF-C in a sample obtained from the patient, wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

[0116] Another embodiment of the invention provides methods of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist. The methods comprise determining expression levels of VEGF-C in a sample obtained from the patient, wherein increased expression levels of VEGF-

CLAIMS

1. A method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

2. A method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

3. A method of optimizing therapeutic efficacy for treatment of cancer, the method comprising

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

4. A method of optimizing therapeutic efficacy for treatment of cancer, the method comprising

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

5. The method of any one of claims 1-4, wherein the sample obtained from the patient is a member selected from the group consisting of: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof.

6. The method of any one of claims1-4, wherein the expression level is selected from mRNA expression level, protein expression level, and combinations thereof.

7. The method of any one of claims 1-4, further comprising detecting the expression of at least a second, third, fourth, fifth, sixth, seventh, eighth, ninth, or tenth gene set forth in Table 1.

8. The method of any one of claims 1-4, wherein an effective amount of the anti-cancer therapy other than a VEGF-A antagonist is to be administered to said patient.

9. The method of claim 8, wherein the anti-cancer therapy is a member selected from the group consisting of: an antibody, a small molecule, and an siRNA.

10. The method of claim 8, wherein the anti-cancer therapy is a member selected from the group consisting of: an EGFL7 antagonist, a NRP1 antagonist, and a VEGF-C antagonist.

11. The method of claim 10, wherein the EGFL7 antagonist is an antibody.

12. The method of claim 10, wherein the NRP1 antagonist is an antibody.

13. The method of claim 10, wherein the VEGF-C antagonist is an antibody.

14. The method of claim 8 wherein the VEGF-A antagonist is to be administered to said patient.

15. The method of claim 14, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

16. The method of claim 15, wherein the anti-VEGF-A antibody is bevacizumab.

17. The method of claim 14, wherein the anti-cancer therapy and the VEGF-A antagonist are to be administered concurrently.

18. The method of claim 14, wherein the anti-cancer therapy and the VEGF-A antagonist are to be administered sequentially.

19. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGF-like-domain, multiple 7 (EGFL7) antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

20. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

21. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from a patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

22. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from a patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

23. The use of an EGFL7 antagonist in the manufacture of a medicament for the treatment of a cell proliferative disorder in a patient, wherein the patient is one whose sample has increased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle.

24. The use of an EGFL7 antagonist in the manufacture of a medicament for the treatment of a cell proliferative disorder in a patient, wherein the patient is one whose sample has decreased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1.

25. The method of any one of claims 19, 20, 21, or 22, wherein an EGFL7 antagonist is to be administered to the patient.

26. The use of claim 23 or 24, or the method of claim 25, wherein the EGFL7 antagonist is an anti-EGFL7 antibody.

27. The use of claim 23 or 24, or the method of claim 25, wherein a VEGF-A antagonist is to be administered to said patient.

~~WHAT IS CLAIMED IS:~~

1. A method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

2. A method of identifying a patient who may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy.

3. A method of predicting responsiveness of a patient suffering from cancer to treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the anti-cancer therapy.

4. A method of predicting responsiveness of a patient suffering from cancer to treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the anti-cancer therapy.

5. A method for determining the likelihood that a patient with cancer will exhibit benefit from anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from the anti-cancer therapy.

6. A method for determining the likelihood that a patient with cancer will exhibit benefit from anti-cancer therapy other than or in addition to a VEGF-A antagonist, the method comprising:

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from the anti-cancer therapy.

7. A method of optimizing therapeutic efficacy for treatment of cancer, the method comprising

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein an increased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

8. A method of optimizing therapeutic efficacy for treatment of cancer, the method comprising

determining expression levels of at least one gene set forth in Table 1 in a sample obtained from the patient, wherein a decreased expression level of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

9. A method for treating cancer in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels, as compared to a reference sample, of at least one gene set forth in Table 1, and

administering an effective amount of an anti-cancer therapy other than or in addition to a VEGF-A antagonist to said patient, whereby the cancer is treated.

10. A method for treating cancer in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels, as compared to a reference sample, of at least one gene set forth in Table 1, and

administering an effective amount of an anti-cancer therapy other than or in addition to a VEGF-A antagonist to said patient, whereby the cancer is treated.

11. The method of any one of claims 1 to 10, wherein the sample obtained from the patient is a member selected from the group consisting of: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof.

12. The method of any one of claims 1 to 10, wherein the expression level is mRNA expression level.

13. The method of any one of claims 1 to 10, wherein the expression level is protein expression level.

14. The method of any one of claims 1 to 10, further comprising detecting the expression of at least a second gene set forth in Table 1.

15. The method of claim 14, further comprising detecting the expression of at least a third gene set forth in Table 1.

16. The method of claim 15, further comprising detecting the expression of at least a fourth gene set forth in Table 1.

17. The method of claim 16, further comprising detecting the expression of at least a fifth gene set forth in Table 1.

18. The method of claim 17, further comprising detecting the expression of at least a sixth gene set forth in Table 1.

19. The method of claim 18, further comprising detecting the expression of at least a seventh gene set forth in Table 1.

20. The method of claim 19, further comprising detecting the expression of at least an eighth gene set forth in Table 1.

21. The method of claim 20, further comprising detecting the expression of at least a ninth gene set forth in Table 1.

22. The method of claim 21, further comprising detecting the expression of at least a tenth gene set forth in Table 1.

23. The method of any one of claims 1 to 8, further comprising administering an effective amount of the anti-cancer therapy other than a VEGF-A antagonist to said patient.

24. The method of claim 23, wherein the anti-cancer therapy is a member selected from the group consisting of: an antibody, a small molecule, and an siRNA.

25. The method of claim 23, wherein the anti-cancer therapy is a member selected from the group consisting of: an EGFL7 antagonist, a NRP1 antagonist, and a VEGF-C antagonist.

26. The method of claim 25, wherein the EGFL7 antagonist is an antibody.

27. The method of claim 25, wherein the NRP1 antagonist is an antibody.

28. The method of claim 25, wherein the VEGF-C antagonist is an antibody.

29. The method of claim 9, 10, or 23 further comprising administering the VEGF-A antagonist to said patient.

30. The method of claim 29, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

31. The method of claim 30, wherein the anti-VEGF-A antibody is bevacizumab.

32. The method of claim 29, wherein the anti-cancer therapy and the VEGF-A antagonist are administered concurrently.

33. The method of claim 29, wherein the anti-cancer therapy and the VEGF-A antagonist are administered sequentially.

34. A kit for determining whether a patient may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene set forth in Table 1 and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with an anti-cancer therapy in addition to a VEGF-A antagonist, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the anti-cancer therapy other than or in addition to a VEGF-A antagonist.

35. A kit for determining whether a patient may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene set forth in Table 1 and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with an anti-cancer therapy in addition to a VEGF-A antagonist, wherein a decrease in the expression level of at least one of said genes as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

36. A set of compounds for detecting expression levels of at least one gene set forth in Table 1, the set comprising

at least one compound capable of specifically hybridizing to at least one gene set forth in Table 1, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

37. A set of compounds for detecting expression levels of at least one gene set forth in Table 1, the set comprising

at least one compound that specifically hybridizes to at least one gene set forth in Table 1, wherein a decrease in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with an anti-cancer therapy other than or in addition to a VEGF-A antagonist.

38. The set of compounds of claim 36 or 37, wherein the compounds are polynucleotides.

39. The set of compounds of claim 38, wherein the polynucleotides comprise three sequences set forth in Table 2.

40. The set of compounds of claim 36 or 37, wherein the compounds are proteins.

41. The set of compounds of claim 40, wherein the proteins are antibodies.

42. A method of identifying a patient suffering from cancer who may benefit from treatment with a neuropilin-1 (NRP1) antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8 in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

43. A method of identifying a patient suffering from cancer who may benefit from treatment with a neuropilin-1 (NRP1) antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

44. A method of predicting responsiveness of a patient suffering from cancer to treatment with a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8 in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the NRP1 antagonist.

45. A method of predicting responsiveness of a patient suffering from cancer to treatment with a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the NRP1 antagonist.

46. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8 in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

47. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the NRP1 antagonist.

48. A method of optimizing therapeutic efficacy of a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8 in a sample obtained from a patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

49. A method of optimizing therapeutic efficacy of a NRP1 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1 in a sample obtained from a patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the NRP1 antagonist.

50. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8, and

administering to said patient an effective amount of a NRP1 antagonist, whereby the cell proliferative disorder is treated.

51. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1, and

administering to said patient an effective amount of a NRP1 antagonist, whereby the cell proliferative disorder is treated.

52. The method of any one of claims 42 to 51, wherein the sample obtained from the patient is a member selected from the group consisting of: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof.

53. The method of any one of claims 42 to 51, wherein the expression level is mRNA expression level.

54. The method of any one of claims 42 to 51, wherein the expression level is protein expression level.

55. The method of any one of claims 42 to 49, further comprising administering a NRP1 antagonist to the patient.

56. The method of any one of claims 42 to 51 or 55, wherein the NRP1 antagonist is an anti-NRP1 antibody.

57. The method of claim 50, 51, or 55 wherein the method further comprises administering a VEGF-A antagonist to said patient.

58. The method of claim 57, wherein the VEGF-A antagonist and the NRP1 antagonist are administered concurrently.

59. The method of claim 57, wherein the VEGF-A antagonist and the NRP1 antagonist are administered sequentially.

60. The method of claim 57, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

61. The method of claim 60, wherein the anti-VEGF-A antibody is bevacizumab.

62. A method of identifying a patient suffering from cancer who may benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient, wherein increased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

63. A method of predicting responsiveness of a patient suffering from cancer to treatment with a NRP1 antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient,

wherein increased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the NRP1 antagonist.

64. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient,

wherein increased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

65. A method of optimizing therapeutic efficacy of a NRP1 antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from a patient,

wherein increased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

66. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of PlGF as compared to a reference sample, and

administering to said patient an effective amount of a NRP1 antagonist, whereby the cell proliferative disorder is treated.

67. A method of identifying a patient suffering from cancer who may benefit from treatment with a neuropilin-1 (NRP1) antagonist, the method comprising

determining expression levels of Sema3A in a sample obtained from the patient, wherein increased expression levels of Sema3A in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

68. A method of predicting responsiveness of a patient suffering from cancer to treatment with a NRP1 antagonist, the method comprising

determining expression levels of Sema3A in a sample obtained from the patient, wherein increased expression levels of Sema3A in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the NRP1 antagonist.

69. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of Sema3A in a sample obtained from the patient, wherein increased expression levels of Sema3A in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

70. A method of optimizing therapeutic efficacy of a NRP1 antagonist, the method comprising

determining expression levels of Sema3A in a sample obtained from a patient, wherein increased expression levels of Sema3A in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

71. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of Sema3A as compared to a reference sample, and

administering to said patient an effective amount of a NRP1 antagonist, whereby the cell proliferative disorder is treated

72. A method of identifying a patient suffering from cancer who may benefit from treatment with a neuropilin-1 (NRP1) antagonist, the method comprising

determining expression levels of TGFβ1 in a sample obtained from the patient, wherein increased expression levels of TGFβ1 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

73. A method of predicting responsiveness of a patient suffering from cancer to treatment with a NRP1 antagonist, the method comprising

determining expression levels of TGFβ1 in a sample obtained from the patient, wherein increased expression levels of TGFβ1 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the NRP1 antagonist.

74. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a NRP1 antagonist, the method comprising

determining expression levels of TGFβ1 in a sample obtained from the patient, wherein increased expression levels of TGFβ1 in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

75. A method of optimizing therapeutic efficacy of a NRP1 antagonist, the method comprising

determining expression levels of TGFβ1 in a sample obtained from a patient, wherein increased expression levels of TGFβ1 in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the NRP1 antagonist.

76. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of TGFβ1 as compared to a reference sample, and

administering to said patient an effective amount of a NRP1 antagonist, whereby the cell proliferative disorder is treated

77. The method of any one of claims 62 to 65, 67 to 70, or 72 to 75, further comprising administering a NRP1 antagonist to the patient.

78. The method of any one of claims 62 to 77, wherein the NRP1 antagonist is an anti-NRP1 antibody.

79. The method of claim 66, 71, 76, or 77 wherein the method further comprises administering a VEGF-A antagonist to said patient.

80. The method of claim 79, wherein the VEGF-A antagonist and the NRP1 antagonist are administered concurrently.

81. The method of claim 79, wherein the VEGF-A antagonist and the NRP1 antagonist are administered sequentially.

82. The method of claim 79, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

83. The method of claim 82, wherein the anti-VEGF-A antibody is bevacizumab.

84. A kit for determining the expression levels of at least one gene selected from the group consisting of TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8, and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with a NRP1 antagonist, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

85. A kit for determining the expression levels of at least one gene selected from the group consisting of Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1 and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with a NRP1 antagonist, wherein a decrease in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the NRP1 antagonist.

86. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8, the set comprising

at least one compound capable of specifically hybridizing to at least one gene selected from the group consisting of: TGFβ1, Bv8, Sema3A, PlGF, LGALS1, ITGa5, CSF2, Vimentin, CXCL5, CCL2, CXCL2, Alk1, and FGF8, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with a NRP1 antagonist.

87. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1, the set comprising

at least one compound capable of specifically hybridizing to at least one gene selected from the group consisting of: Prox1, RGS5, HGF, Sema3B, Sema3F, LGALS7, FGRF4, PLC, IGFB4, and TSP1, wherein a decrease in the expression level of said at least gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with a NRP1 antagonist.

88. The set of compounds of claims 86 or 87, wherein the compounds are polynucleotides.

89. The set of compounds of claim 88, wherein the polynucleotides comprise three sequences set forth in Table 2.

90. The set of compounds of claims 86 or 87, wherein the compounds are proteins.

91. The set of compounds of claim 90, wherein the proteins are antibodies.

92. A method of identifying a patient suffering from cancer who may benefit from treatment with a Vascular Endothelial Growth Factor C (VEGF-C) antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and

CXCL2 in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

93. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

94. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2 in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

95. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

96. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2 in a sample obtained from the patient, wherein increased expression levels of

said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the VEGF-C antagonist.

97. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the VEGF-C antagonist.

98. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2 in a sample obtained from a patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the VEGF-C antagonist.

99. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1 in a sample obtained from a patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the VEGF-C antagonist.

100. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels, as compared to a reference sample, of at least one gene selected from the group

consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated.

101. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated.

102. The method of any one of claims 92 to 101, wherein the sample obtained from the patient is a member selected from the group consisting of: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof.

103. The method of any one of claims 92 to 101, wherein the expression level is mRNA expression level.

104. The method of any one of claims 92 to 101, wherein the expression level is protein expression level.

105. The method of any one of claims 92 to 99, further comprising administering a VEGF-C antagonist to the patient.

106. The method of any one of claims 92 to 10 or 105, wherein the VEGF-C antagonist is an anti-VEGF-C antibody.

107. The method of claim 100, 101, or 105, wherein the method further comprises administering a VEGF-A antagonist to said patient.

108. The method of claim 107, wherein the VEGF-A antagonist and the VEGF-C antagonist are administered concurrently.

109. The method of claim 107, wherein the VEGF-A antagonist and the VEGF-C antagonist are administered sequentially.

110. The method of claim 107, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

111. The method of claim 110, wherein the anti-VEGF-A antibody is bevacizumab.

112. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient, wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

113. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

114. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

115. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from a patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

116. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of VEGF-C as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated.

117. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-D in a sample obtained from the patient, wherein increased expression levels of VEGF-D in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

118. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-D in a sample obtained from the patient, wherein increased expression levels of VEGF-D in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

119. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-D in a sample obtained from the patient, wherein increased expression levels of VEGF-D in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

120. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-D in a sample obtained from a patient, wherein increased expression levels of VEGF-D in the sample as compared to a

reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

121. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of VEGF-D as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated

122. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGFR3 in a sample obtained from the patient, wherein increased expression levels of VEGFR3 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

123. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGFR3 in a sample obtained from the patient, wherein increased expression levels of VEGFR3 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

124. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGFR3 in a sample obtained from the patient, wherein increased expression levels of VEGFR3 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

125. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of VEGFR3 in a sample obtained from a patient, wherein increased expression levels of VEGFR3 in the sample as compared to

a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

126. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of VEGFR3 as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated

127. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein increased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

128. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein increased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

129. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein increased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

130. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from a patient, wherein increased expression levels of FGF2 in the sample as compared to a reference

sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

131. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of FGF2 as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated

132. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-A in a sample obtained from the patient, wherein decreased expression levels of VEGF-A in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

133. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-A in a sample obtained from the patient, wherein decreased expression levels of VEGF-A in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

134. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-A in a sample obtained from the patient, wherein decreased expression levels of VEGF-A in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

135. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of VEGF-A in a sample obtained from a patient, wherein decreased expression levels of VEGF-A in the sample as compared to a

reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

136. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of VEGF-A as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated.

137. A method of identifying a patient suffering from cancer who may benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient, wherein decreased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

138. A method of predicting responsiveness of a patient suffering from cancer to treatment with a VEGF-C antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient, wherein decreased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the VEGF-C antagonist.

139. A method of determining the likelihood that a patient will exhibit a benefit from treatment with a VEGF-C antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from the patient, wherein decreased expression levels of PlGF in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

140. A method of optimizing therapeutic efficacy of a VEGF-C antagonist, the method comprising

determining expression levels of PlGF in a sample obtained from a patient, wherein decreased expression levels of PlGF in the sample as compared to a reference

sample indicates that the patient has an increased likelihood of benefit from treatment with the VEGF-C antagonist.

141. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of PlGF as compared to a reference sample, and

administering to said patient an effective amount of a VEGF-C antagonist, whereby the cell proliferative disorder is treated.

142. The method of any one of claims 112 to 115, 117 to 120, 122 to 125, 127 to 130, 132 to 135, or 137 to 140, further comprising administering a VEGF-C antagonist to the patient.

143. The method of any one of claims 112 to 142, wherein the VEGF-C antagonist is an anti-VEGF-C antibody.

144. The method of claim 116, 121, 126, 131, 136, 141, or 142 wherein the method further comprises administering a VEGF-A antagonist to said patient.

145. The method of claim 144, wherein the VEGF-A antagonist and the VEGF-C antagonist are administered concurrently.

146. The method of claim 144, wherein the VEGF-A antagonist and the VEGF-C antagonist are administered sequentially.

147. The method of claim 144, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

148. The method of claim 147, wherein the anti-VEGF-A antibody is bevacizumab.

149. A kit for determining the expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2, and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with a VEGF-C antagonist, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

150. A kit for determining the expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1 and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with a VEGF-C antagonist, wherein a decrease in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the VEGF-C antagonist.

151. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2, the set comprising

at least one compound capable of specifically hybridizing to at least one gene selected from the group consisting of: VEGF-C, VEGF-D, VEGFR3, FGF2, RGS5/CDH5, IL-8, CXCL1, and CXCL2, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with a VEGF-C antagonist.

152. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1, the set comprising

at least one compound capable of specifically hybridizing to at least one gene selected from the group consisting of: VEGF-A, CSF2, Prox1, ICAM1, ESM1, PlGF, ITGa5, TGFβ, Hhex, Col4a1, Col4a2, and Alk1, wherein a decrease in the expression level of said at least gene as compared to the expression level of said at least one gene

in a reference sample indicates that the patient may benefit from treatment with a VEGF-C antagonist.

153. The set of compounds of claims 151 or 152, wherein the compounds are polynucleotides.

154. The set of compounds of claim 153, wherein the polynucleotides comprise three sequences set forth in Table 2.

155. The set of compounds of claims 151 or 152, wherein the compounds are proteins.

156. The set of compounds of claim 155, wherein the proteins are antibodies.

157. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGF-like-domain, multiple 7 (EGFL7) antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

158. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

159. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a

sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

160. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

161. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from the patient, wherein increased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

162. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from the patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

163. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle in a sample obtained from a patient, wherein increased expression levels of said at least

one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit from treatment with the EGFL7 antagonist.

164. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1 in a sample obtained from a patient, wherein decreased expression levels of said at least one gene in the sample as compared to a reference sample indicates that the patient has increased likelihood of benefit of treatment with the EGFL7 antagonist.

165. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

166. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels, as compared to a reference sample, of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

167. The method of any one of claims 157 to 166, wherein the sample obtained from the patient is a member selected from the group consisting of: tissue, whole blood, blood-derived cells, plasma, serum, and combinations thereof.

168. The method of any one of claims 157 to 166, wherein the expression level is mRNA expression level.

169. The method of any one of claims 157 to 166, wherein the expression level is protein expression level.

170. The method of any one of claims 157 to 164, further comprising administering an EGFL7 antagonist to the patient.

171. The method of any one of claims 157 to 166, or 170, wherein the EGFL7 antagonist is an anti-EGFL7 antibody.

172. The method of claim 165, 166, or 170 wherein the method further comprises administering a VEGF-A antagonist to said patient.

173. The method of claim 172, wherein the VEGF-A antagonist and the EGFL7 antagonist are administered concurrently.

174. The method of claim 172, wherein the VEGF-A antagonist and the EGFL7 antagonist are administered sequentially.

175. The method of claim 172, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

176. The method of claim 175, wherein the anti-VEGF-A antibody is bevacizumab.

177. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient, wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

178. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

179. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from the patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

180. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of VEGF-C in a sample obtained from a patient,

wherein increased expression levels of VEGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

181. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of VEGF-C as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

182. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of BV8 in a sample obtained from the patient, wherein increased expression levels of BV8 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

183. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of BV8 in a sample obtained from the patient, wherein increased expression levels of BV8 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

184. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of BV8 in a sample obtained from the patient, wherein increased expression levels of BV8 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

185. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of BV8 in a sample obtained from a patient, wherein increased expression levels of BV8 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

186. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of BV8 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated

187. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CSF2 in a sample obtained from the patient, wherein increased expression levels of CSF2 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

188. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CSF2 in a sample obtained from the patient, wherein increased expression levels of CSF2 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

189. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CSF2 in a sample obtained from the patient, wherein increased expression levels of CSF2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

190. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of CSF2 in a sample obtained from a patient, wherein increased expression levels of CSF2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

191. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of CSF2 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated

192. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of TNFα in a sample obtained from the patient, wherein increased expression levels of TNFα in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

193. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of TNFα in a sample obtained from the patient, wherein increased expression levels of TNFα in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

194. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of TNFα in a sample obtained from the patient, wherein increased expression levels of TNFα in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

195. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of TNFα in a sample obtained from a patient, wherein increased expression levels of TNFα in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

196. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has increased expression levels of TNFα as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated

197. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3B in a sample obtained from the patient, wherein decreased expression levels of Sema3B in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

198. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3B in a sample obtained from the patient, wherein decreased expression levels of Sema3B in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

199. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3B in a sample obtained from the patient, wherein decreased expression levels of Sema3B in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

200. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of Sema3B in a sample obtained from a patient, wherein decreased expression levels of Sema3B in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

201. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of Sema3B as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

202. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF9 in a sample obtained from the patient, wherein decreased expression levels of FGF9 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

203. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF9 in a sample obtained from the patient, wherein decreased expression levels of FGF9 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

204. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF9 in a sample obtained from the patient, wherein decreased expression levels of FGF9 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

205. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of FGF9 in a sample obtained from a patient, wherein decreased expression levels of FGF9 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

206. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of FGF9 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

207. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of HGF in a sample obtained from the patient, wherein decreased expression levels of HGF in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

208. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of HGF in a sample obtained from the patient, wherein decreased expression levels of HGF in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

209. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of HGF in a sample obtained from the patient, wherein decreased expression levels of HGF in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

210. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of HGF in a sample obtained from a patient, wherein decreased expression levels of HGF in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

211. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of HGF as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

212. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of RGS5 in a sample obtained from the patient, wherein decreased expression levels of RGS5 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

213. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of RGS5 in a sample obtained from the patient, wherein decreased expression levels of RGS5 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

214. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of RGS5 in a sample obtained from the patient, wherein decreased expression levels of RGS5 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

215. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of RGS5 in a sample obtained from a patient, wherein decreased expression levels of RGS5 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

216. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of RGS5 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

217. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

218. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

219. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

220. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from a patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

221. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of NRP1 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

222. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

223. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

224. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from the patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

225. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of NRP1 in a sample obtained from a patient, wherein decreased expression levels of NRP1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

226. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of NRP1 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

227. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein decreased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

228. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein decreased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

229. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from the patient, wherein decreased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

230. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of FGF2 in a sample obtained from a patient, wherein decreased expression levels of FGF2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

231. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of FGF2 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

232. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CXCR4 in a sample obtained from the patient, wherein decreased expression levels of CXCR4 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

233. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CXCR4 in a sample obtained from the patient, wherein decreased expression levels of CXCR4 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

234. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of CXCR4 in a sample obtained from the patient, wherein decreased expression levels of CXCR4 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

235. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of CXCR4 in a sample obtained from a patient, wherein decreased expression levels of CXCR4 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

236. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of CXCR4 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

237. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of cMet in a sample obtained from the patient, wherein decreased expression levels of cMet in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

238. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of cMet in a sample obtained from the patient, wherein decreased expression levels of cMet in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

239. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of cMet in a sample obtained from the patient, wherein decreased expression levels of cMet in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

240. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of cMet in a sample obtained from a patient, wherein decreased expression levels of cMet in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

241. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of cMet as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

242. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FN1 in a sample obtained from the patient, wherein decreased expression levels of FN1 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

243. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FN1 in a sample obtained from the patient, wherein decreased expression levels of FN1 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

244. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of FN1 in a sample obtained from the patient, wherein decreased expression levels of FN1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

245. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of FN1 in a sample obtained from a patient, wherein decreased expression levels of FN1 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

246. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of FN1 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

247. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin 2 in a sample obtained from the patient, wherein decreased expression levels of Fibulin 2 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

248. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin 2 in a sample obtained from the patient, wherein decreased expression levels of Fibulin 2 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

249. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin 2 in a sample obtained from the patient, wherein decreased expression levels of Fibulin 2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

250. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin 2 in a sample obtained from a patient, wherein decreased expression levels of Fibulin 2 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

251. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of Fibulin 2 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

252. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin4 in a sample obtained from the patient, wherein decreased expression levels of Fibulin4 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

253. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin4 in a sample obtained from the patient, wherein decreased expression levels of Fibulin4 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

254. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin4 in a sample obtained from the patient, wherein decreased expression levels of Fibulin4 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

255. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of Fibulin4 in a sample obtained from a patient, wherein decreased expression levels of Fibulin4 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

256. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of Fibulin4 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

257. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of MFAP5 in a sample obtained from the patient, wherein decreased expression levels of MFAP5 in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

258. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of MFAP5 in a sample obtained from the patient, wherein decreased expression levels of MFAP5 in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

259. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of MFAP5 in a sample obtained from the patient, wherein decreased expression levels of MFAP5 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

260. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of MFAP5 in a sample obtained from a patient, wherein decreased expression levels of MFAP5 in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

261. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of MFAP5 as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

262. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of PDGF-C in a sample obtained from the patient, wherein decreased expression levels of PDGF-C in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

263. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of PDGF-C in a sample obtained from the patient, wherein decreased expression levels of PDGF-C in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

264. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of PDGF-C in a sample obtained from the patient, wherein decreased expression levels of PDGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

265. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of PDGF-C in a sample obtained from a patient, wherein decreased expression levels of PDGF-C in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

266. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of PDGF-C as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

267. A method of identifying a patient suffering from cancer who may benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3F in a sample obtained from the patient, wherein decreased expression levels of Sema3F in the sample as compared to a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

268. A method of predicting responsiveness of a patient suffering from cancer to treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3F in a sample obtained from the patient, wherein decreased expression levels of Sema3F in the sample as compared to a reference sample indicates that the patient is more likely to be responsive to treatment with the EGFL7 antagonist.

269. A method of determining the likelihood that a patient will exhibit a benefit from treatment with an EGFL7 antagonist, the method comprising

determining expression levels of Sema3F in a sample obtained from the patient, wherein decreased expression levels of Sema3F in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

270. A method of optimizing therapeutic efficacy of an EGFL7 antagonist, the method comprising

determining expression levels of Sema3F in a sample obtained from a patient, wherein decreased expression levels of Sema3F in the sample as compared to a reference sample indicates that the patient has an increased likelihood of benefit from treatment with the EGFL7 antagonist.

271. A method for treating a cell proliferative disorder in a patient, the method comprising

determining that a sample obtained from the patient has decreased expression levels of Sema3F as compared to a reference sample, and

administering to said patient an effective amount of an EGFL7 antagonist, whereby the cell proliferative disorder is treated.

272. The method of any one of claims 177 to 180, 182 to 185, 187 to 190, 192 to 195, 197 to 200, 202 to 205, 207 to 210, 212 to 215, 217 to 220, 222 to 225, 227 to 230, 232 to 235, 237 to 240, 242 to 245, 247 to 250, 252 to 255, 257 to 260, 262 to 265, or 267 to 270 further comprising administering an EGFL7 antagonist to the patient.

273. The method of any one of claims 177 to 272, wherein the EGFL7 antagonist is an anti- EGFL7 antibody.

274. The method of any one of claims 181, 186, 191, 196, 201, 206, 211, 216, 221, 226, 231, 236, 241, 246, 251, 256, 261, 266, 271, or 272, wherein the method further comprises administering a VEGF-A antagonist to said patient.

275. The method of claim 274, wherein the VEGF-A antagonist and the EGFL7 antagonist are administered concurrently.

276. The method of claim 274, wherein the VEGF-A antagonist and the EGFL7 antagonist are administered sequentially.

277. The method of claim 274, wherein the VEGF-A antagonist is an anti-VEGF-A antibody.

278. The method of claim 277, wherein the anti-VEGF-A antibody is bevacizumab.

279. A kit for determining the expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle, and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with an EGFL7 antagonist, wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

280. A kit for determining the expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, the kit comprising

an array comprising polynucleotides capable of specifically hybridizing to at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, and

instructions for using said array to determine the expression levels of said at least one gene to predict responsiveness of a patient to treatment with an EGFL7 antagonist, wherein a decrease in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with the EGFL7 antagonist.

281. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle, the set comprising

at least one compound capable of specifically hybridizing to at least one gene selected from the group consisting of VEGF-C, BV8, CSF2, TNFα, CXCL2, PDGF-C, and Mincle: wherein an increase in the expression level of said at least one gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with an EGFL7 antagonist.

282. A set of compounds capable of detecting expression levels of at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, the set comprising

at least one compound that specifically hybridizes to at least one gene selected from the group consisting of: Sema3B, FGF9, HGF, RGS5, NRP1, FGF2, CXCR4, cMet, FN1, Fibulin 2, Fibulin4/EFEMP2, MFAP5, PDGF-C, Sema3F, and FN1, wherein a decrease in the expression level of said at least gene as compared to the expression level of said at least one gene in a reference sample indicates that the patient may benefit from treatment with an EGFL7 antagonist.

283. The set of compounds of claims 281 or 282, wherein the compounds are polynucleotides.

284. The set of compounds of claim 283, wherein the polynucleotides comprise three sequences from Table 2.

285. The set of compounds of claims 281 or 282, wherein the compounds are proteins.

286. The set of compounds of claim 285, wherein the proteins are antibodies.