WHAT IS CLAIMED IS:

1. A method for altering at least one of: (i) formation; (ii) proliferation; (iii) maintenance; (iv) EMT; or (v) MET of a PKC-θ-overexpressing cell, the method comprising, consisting or consisting essentially of contacting the PKC-θ-overexpressing cell with a formation-, proliferation-, maintenance-; EMT- or MET-modulating amount of a PKC-θ inhibitor, wherein the PKC-θ-overexpressing cell is a cancer stem cell (CSC).

2. A method according to claim 1, wherein the PKC-θ-overexpressing cell is contacted with a PKC-θ-overexpressing cell formation-inhibiting amount of the PKC-θ inhibitor.

3. A method according to claim 1, wherein the PKC-θ-overexpressing cell is contacted with a PKC-θ-overexpressing cell proliferation-inhibiting amount of the PKC-θ inhibitor.

4. A method according to claim 1, wherein the PKC-θ-overexpressing cell is contacted with a PKC-θ-overexpressing cell EMT-inhibiting amount of the PKC-θ inhibitor.

5. A method according claim 1, wherein the PKC-θ-overexpressing cell is contacted with a PKC-θ-overexpressing cell MET-stimulating or -inducing amount of the PKC-θ inhibitor.

6. A method according to any one of claims 1 to 5, wherein the CSC is a breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma CSC.

7. A method according to claim 6, wherein the CSC is a breast CSC.

8. A method according to claim 7, wherein the breast CSC is a breast epithelial CSC.

9. A method according to claim 8, wherein the breast epithelial CSC is a breast ductal epithelial CSC.

10. A method according to any one of claims 1 to 9, wherein the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

11. A method according to any one of claims 1 to 10, wherein the CSC expresses one or more CSC markers selected from ABCB5, ALDH1, ABCG2, α6 integrin, α2 β1 integrin, β-catenin activity, CD15, CD13, CD20, CD24, CD26, CD29, CD44, CD90, CD133, CD166, CD271, c-Met, Hedgehog-Gli, Nestin, CXCR4, LGR5, Trop2 and Nodal-Activin.

12. A method according to any one of claims 1 to 10, wherein the CSC expresses one or more CSC markers selected from ALDH1, CD24, CD44, CD90, CD133, Hedgehog-Gli, α_6 integrin.

13. A method according to any one of claims 1 to 12, wherein the CSC expresses CD24 and CD44 (*e.g.*, $CD44^{high}$, $CD24^{low}$).

14. A method according to any one of claims 1 to 13, wherein the PKC-θ inhibitor is a selective PKC-θ inhibitor.

15. A method according to any one of claims 1 to 13, wherein the PKC-θ inhibitor is a non-selective PKC-θ inhibitor .

16. A method according to any one of claims 1 to 15, wherein the PKC-θ inhibitor reduces the expression of a *PKC-θ* gene or the level or functional activity of an expression product of that gene.

17. A method according to any one of claims 1 to 15, wherein the PKC-θ inhibitor inhibits the function of the PKC-θ, including reducing or abrogating the activity of at least one of its ligand-binding sites.

18. A method according to any one of claims 1 to 17, further comprising detecting overexpression of a *PKC-θ* gene in the PKC-θ-overexpressing cell prior to contacting the PKC-θ-overexpressing cell with the PKC-θ inhibitor.

19. A method according to any one of claims 1 to 18, further comprising detecting presence or an increased amount of PKC-θ in the nucleus of the PKC-θ-overexpressing cell prior to contacting the PKC-θ-overexpressing cell with the PKC-θ inhibitor.

20. A method according to any one of claims 1 to 19, further comprising detecting binding of PKC-θ to the promoter of *CD44* or *uPAR* in the PKC-θ-overexpressing cell prior to contacting the PKC-θ-overexpressing cell with the PKC-θ inhibitor.

21. A method according to any one of claims 1 to 20, further comprising detecting binding of PKC-θ to chromatin in the PKC-θ-overexpressing cell prior to contacting the PKC-θ-overexpressing cell with the PKC-θ inhibitor.

22. A method according to any one of claims 1 to 21, further comprising detecting that the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

23. A method according to any one of claims 1 to 22, further comprising detecting that the PKC-θ-overexpressing cell expresses one or more CSC markers (as defined for example in any one of claims 11 to 13).

24. A method for treating or preventing a cancer by inhibiting formation, proliferation, maintenance of a CSC, and/or by inhibiting EMT of a CSC, and/or by stimulating or inducing MET of a CSC in a subject, the method comprising administering to the subject a PKC-θ inhibitor in an effective amount to treat or prevent the cancer, wherein the cancer comprises the CSC.

25. A method according to claim 24, wherein the cancer is a metastatic cancer.

26. A method according to claim 25, further comprising identifying that the subject has or is at risk of development of the metastatic cancer, suitably prior to administration of the PKC-θ inhibitor .

27. A method according to any one of claims 24 to 26, further comprising detecting that the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

28. A method according to any one of claims 24 to 27, further comprising detecting that the PKC-θ-overexpressing cell expresses one or more CSC markers (as defined in any one of claims 11 to 13).

29. A method according to any one of claims 24 to 28, further comprising co-administering to the subject at least one cancer therapy that inhibits the proliferation of a non-CSC tumor cell or treats or ameliorates the symptoms or reverses or inhibits the development or progression of the cancer in the subject.

30. A method according to claim 29, wherein the cancer therapy is selected from radiation therapy, chemotherapy, stem cell transplant; and antibody therapy.

31. A method according to any one of claims 24 to 30, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.

32. A method according to any one of claims 24 to 29, wherein the cancer is breast cancer.

33. A method for treating or preventing a cancer in a subject, wherein the cancer comprises a CSC and a non-CSC tumor cell, the method comprising administering to the subject a PKC-θ inhibitor in an effective amount to inhibit formation, proliferation, maintenance or EMT of the CSC or to stimulate or induce MET of the CSC.

34. A method according to claim 33, wherein the cancer is a metastatic cancer.

35. A method according to claim 33 or claim 34, further comprising identifying that the subject has or is at risk of developing a cancer comprising a CSC and a non-CSC tumor cell prior to the administration of the PKC-θ inhibitor.

36. A method according to any one of claims 33 to 35, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.

37. A method according to any one of claims 33 to 35, wherein the cancer is breast cancer.

38. A method according to any one of claims 33 to 37, further comprising detecting overexpression of a *PKC-θ* gene in a tumor sample obtained from the subject, wherein the tumor sample comprises the CSC and/or the non-CSC tumor cell, prior to administering the PKC-θ inhibitor to the subject.

39. A method according to any one of claims 33 to 38, further comprising detecting that the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

40. A method according to any one of claims 32 to 39, further comprising detecting expression of one or more CSC markers (as defined for example in any one of claims 11 to 13) in a tumor sample obtained from the subject, wherein the tumor sample comprises the CSC and/or the non-CSC tumor cell, prior to administering the PKC-θ inhibitor to the subject.

41. A method for treating or preventing a cancer in a subject, wherein the cancer comprises a CSC and a non-CSC tumor cell, the method comprising concurrently administering to the subject a PKC-θ inhibitor in an effective amount to inhibit formation, proliferation, maintenance or EMT of the CSC or to stimulate or induce MET of the CSC, and a cancer therapy or agent that inhibits the proliferation, survival or viability of the non-CSC tumor cell, to thereby treat or prevent the cancer.

42. A method according to claim 41, wherein the cancer therapy or agent is selected from radiotherapy, surgery, chemotherapy, hormone ablation therapy, pro-apoptosis therapy and immunotherapy.

43. A method according to claim 41 or claim 42, wherein the cancer therapy or agent targets rapidly dividing cells or disrupts the cell cycle or cell division.

44. A method according to any one of claims 41 to 43, wherein the cancer is a metastatic cancer.

45. A method according to any one of claims 41 to 44, further comprising identifying that the subject has or is at risk of developing a cancer comprising the CSC and the non-CSC tumor cell prior to the co-administration.

46. A method according to any one of claims 41 to 45, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.

47. A method according to any one of claims 41 to 45, wherein the cancer is breast cancer.

48. A method according to any one of claims 41 to 47, further comprising detecting overexpression of a *PKC-θ* gene in a tumor sample obtained from the subject, wherein the tumor sample comprises the CSC and/or the non-CSC tumor cell, prior to administering the PKC-θ inhibitor to the subject.

49. A method according to any one of claims 41 to 48, wherein the CSC is a breast CSC.

50. A method according to any one of claims 41 to 49, further comprising detecting that the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

51. A method according to any one of claims 41 to 50, further comprising detecting that the CSC expresses one or more CSC markers (as defined for example in any one of claims 11 to 13) prior to administering the PKC-θ inhibitor to the subject.

52. A method according to any one of claims 41 to 51, wherein the PKC-θ inhibitor and the cancer therapy agent are administered in synergistically effective amounts.

53. A method for identifying agents that are useful for inhibiting formation, proliferation, maintenance or EMT of a CSC or for stimulating or inducing MET of a CSC, or for treating or preventing a metastatic cancer in a subject, wherein the metastatic cancer comprises a CSC, the method comprising contacting a preparation with a test agent, wherein the preparation comprises (i) a polypeptide comprising an amino acid sequence corresponding to at least a biologically active fragment of a PKC-θ, or to a variant or derivative thereof; or (ii) a polynucleotide comprising a nucleotide sequence from which a transcript of a *PKC-θ* gene or portion thereof is producible, or (iii) a polynucleotide comprising at least a portion of a genetic sequence (*e.g.*, a transcriptional element) that regulates the expression of a *PKC-θ* gene, which is operably linked to a reporter gene; and detecting a reduction in the level and/or functional activity of the polypeptide, transcript or transcript portion or an expression product of the reporter gene, relative to a normal or reference level and/or functional activity in the absence of the test agent, indicates that the agent is useful for inhibiting formation, proliferation, maintenance or EMT of the CSC or for stimulating or inducing MET of the CSC, or for treating or preventing the metastatic cancer.

54. A method according to claim 53, wherein the agent inhibits the binding between a PKC-θ and a PKC-θ ligand, as determined by: contacting the PKC-θ and the ligand with the agent and measuring the binding of the PKC-θ with the ligand.

55. A method of producing an agent for inhibiting formation, proliferation, maintenance or EMT of a CSC or for stimulating or inducing MET of a CSC, or for treating or preventing a cancer (*e.g.*, a metastatic cancer) that comprises a CSC, the method comprising: testing an agent suspected of inhibiting the function of PKC-θ as defined in claim 53 of claim 54; and synthesizing the agent on the basis that it tests positive for the inhibition, stimulation/induction or treatment/prevention.

56. A method according to claim 55, further comprising derivatizing the agent, and optionally formulating the derivatized agent with a pharmaceutically acceptable carrier and/or diluent, to improve the efficacy of the agent for inhibiting formation, proliferation,

maintenance or EMT of a CSC or for stimulating or inducing MET of a CSC, or for treating or preventing a cancer (*e.g.*, a metastatic cancer) that comprises a CSC.

57. Use of a PKC-θ inhibitor for inhibiting formation, proliferation, maintenance or EMT of a CSC or for stimulating or inducing MET of a CSC, or for treating or preventing a cancer (*e.g.*, a metastatic cancer) that comprises a CSC.

58. Use of a PKC-θ inhibitor in the manufacture of a medicament for inhibiting formation, proliferation, maintenance or EMT of a CSC or for stimulating or inducing MET of a CSC, or for treating or preventing a cancer (*e.g.*, a metastatic cancer) that comprises a CSC.

59. A use according to claim 57 or claim 58, wherein the PKC-θ inhibitor is contained or otherwise associated with a vehicle selected from the group consisting of liposomes, micelles, dendrimers, biodegradable particles, artificial DNA nanostructure, lipid-based nanoparticles, and carbon or gold nanoparticles.

60. A use according to claim 59, wherein the PKC-θ inhibitor is administered to the subject by a vehicle selected from the group consisting of Poly(lactic acid) (PLA), Poly(glycolic acid) (PGA); polymer poly(lactic-co-glycolic acid) (PLGA); poly(ethylene glycol) (PEG), and PLA-PEG copolymers, or any combinations thereof.

61. A method for enhancing the biological effects of a cancer therapy or agent on a cancer comprising a CSC and a non-CSC tumor cell, comprising administering to a subject in need thereof along with the cancer therapy or agent an effective amount of a PKC-θ inhibitor.

62. A method according to claim 61, wherein the cancer therapy or agent chemotherapeutic drug or irradiation.

63. Use of a PKC-θ inhibitor and a cancer therapy or agent that inhibits the proliferation, survival or viability of a non-CSC tumor cell for treating or preventing a cancer that comprises a CSC and a non-CSC tumor cells.

64. A use according to claim 63, wherein the PKC-θ inhibitor and optionally the cancer therapy or agent are prepared or manufactured as medicaments for this purpose.

WHAT IS CLAIMED IS:

1. Use of a protein kinase C theta (PKC-θ) inhibitor in the manufacture of a medicament for altering at least one of: (i) formation; (ii) proliferation; (iii) maintenance; (iv) epithelial to mesenchymal transition (EMT); or (v) mesenchymal to epithelial transition (MET) of a PKC-θ-overexpressing cell, wherein the PKC-θ-overexpressing cell is a cancer stem cell (CSC).

2. The use according to claim 1, wherein the CSC is a breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma CSC.

3. The use according to claim 2, wherein the CSC is a breast CSC.

4. The use according to claim 3, wherein the breast CSC is a breast epithelial CSC.

5. The use according to claim 4, wherein the breast epithelial CSC is a breast ductal epithelial CSC.

6. The use according to any one of claims 1 to 5, wherein the CSC has impaired or abrogated expression of the pluripotent stem cell markers Oct4 or Sox2.

7. The use according to any one of claims 1 to 6, wherein the CSC expresses one or more CSC markers selected from ABCB5, ALDH1, ABCG2, α6 integrin, α2 β1 integrin, β-catenin activity, CD15, CD13, CD20, CD24, CD26, CD29, CD44, CD90, CD133, CD166, CD271, c-Met, Hedgehog-Gli, Nestin, CXCR4, LGR5, Trop2 and Nodal-Activin.

8. The use according to any one of claims 1 to 7, wherein the CSC expresses CD24 and CD44 (*e.g.*, $CD44^{high}$, $CD24^{low}$).

9. The use according to any one of claims 1 to 8, wherein the PKC-θ inhibitor is a selective PKC-θ inhibitor.

10. The use according to any one of claims 1 to 8, wherein the PKC-θ inhibitor is a non-selective PKC-θ inhibitor .

11. Use of a PKC-θ inhibitor in the manufacture of a medicament for treating or preventing a cancer by inhibiting formation, proliferation, or maintenance of a CSC, and/or by inhibiting EMT of a CSC, and/or by stimulating or inducing MET of a CSC wherein the cancer comprises the CSC.

12. The use according to claim 11, wherein the cancer is a metastatic cancer.

13. The use according to claim 11 or claim 12, wherein the PKC-θ inhibitor is co-administered with at least one cancer therapy that inhibits the proliferation of a non-CSC tumor cell or treats or ameliorates the symptoms or reverses or inhibits the development or progression of the cancer.

14. The use according to claim 13, wherein the cancer therapy is selected from radiation therapy, chemotherapy, stem cell transplant; and antibody therapy.

15. The use according to any one of claims 11 to 14, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.

16. The use according to any one of claims 11 to 15, wherein the cancer is breast cancer.

17. Use of a PKC-θ inhibitor in the manufacture of a medicament for treating or preventing a cancer, wherein the cancer comprises a CSC and a non-CSC tumor cell.

18. The use according to claim 17, wherein the cancer is a metastatic cancer.

19. The use according to claims 17 or claim 18, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.

20. The use according to any one of claims 17 to 19, wherein the cancer is breast cancer.

21. Use of a PKC-θ inhibitor and a cancer therapy or agent that inhibits the proliferation, survival or viability of the non-CSC tumor cell in the manufacture of a medicament for treating or preventing a cancer.

22. The use according to claim 21, wherein the cancer therapy or agent is selected from radiotherapy, surgery, chemotherapy, hormone ablation therapy, pro-apoptosis therapy and immunotherapy.

23. The use according to claim 21 or claim 22, wherein the cancer therapy or agent targets rapidly dividing cells or disrupts the cell cycle or cell division.

24. The use according to any one of claims 21 to 23, wherein the cancer is a metastatic cancer.

25. The use according to any one of claims 21 to 24, wherein the cancer is selected from breast, prostate, lung, bladder, pancreatic, colon, melanoma, liver or glioma cancer.