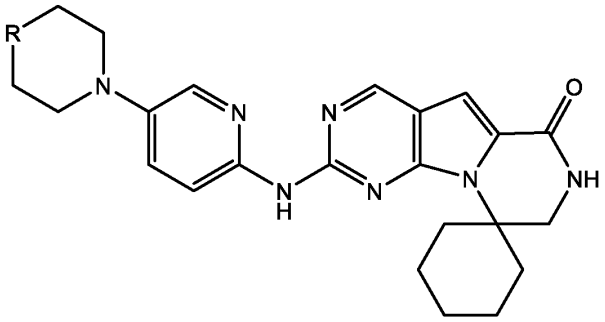
**Claims**

What is claimed is:

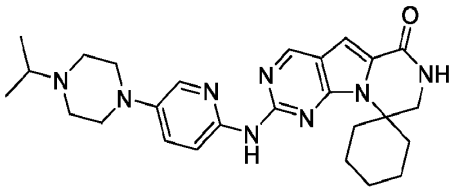
1. A method of treating a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI), wherein the CDK4/6 inhibitor is




wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

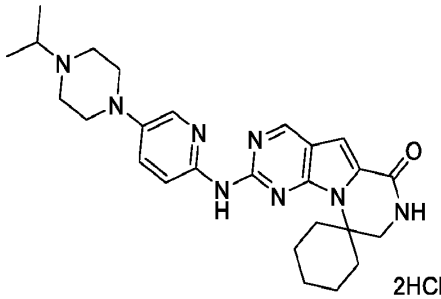
or a pharmaceutically acceptable salt thereof.

2. The method of claim 1, wherein the host is a human.
3. The method of claim 2, wherein the non-small cell lung cancer has an FGFR1 amplification or FGFR1 is overexpressed.
4. The method of claim 2, wherein the non-small cell lung cancer has an FGFR2 amplification or FGFR2 is overexpressed.
5. The method of any of claims 1 to 4, wherein the non-small cell lung cancer is large cell lung carcinoma.
6. The method of any of claims 1 to 4, wherein the non-small cell lung cancer is squamous cell carcinoma.
7. The method of any of claim 1 to 6, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, GSK3052230, and FIIN-2.
8. The method of any of claims 1 to 7, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

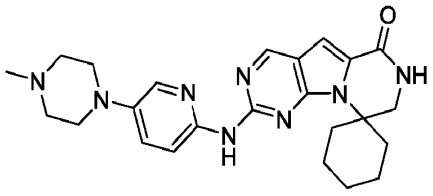
9. The method of any of claims 1 to 7, wherein the CDK4/6 inhibitor is



2HCl (Compound IA).

10. The method of claim 9, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

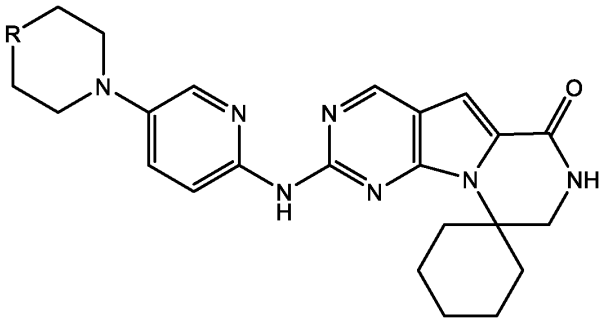
11. The method of any of claims 1 to 7, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

12. The method of any of claims 1 to 11, wherein the CDK4/6 inhibitor and the FGFR inhibitor are administered to the host at least once a day for at least 28 consecutive days.

13. The method of any of claims 1 to 11, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.
14. The method of any of claims 1 to 11, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.
15. The method of any of claims 1 to 14, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.
16. The method of any of claims 1 to 15, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.
17. The method of any of claims 1 to 16, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKI.
18. A method of reducing the development of acquired resistance to the inhibitory effects of a selective FGFR-TKI in a host with a non-small cell lung cancer having an FGFR1 or FGFR2 aberration, wherein the FGFR1 or FGFR2 aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is:




wherein R is NX, and wherein X is hydrogen, methyl or isopropyl,
or a pharmaceutically acceptable salt thereof.

19. The method of claim 18, wherein the host is a human.

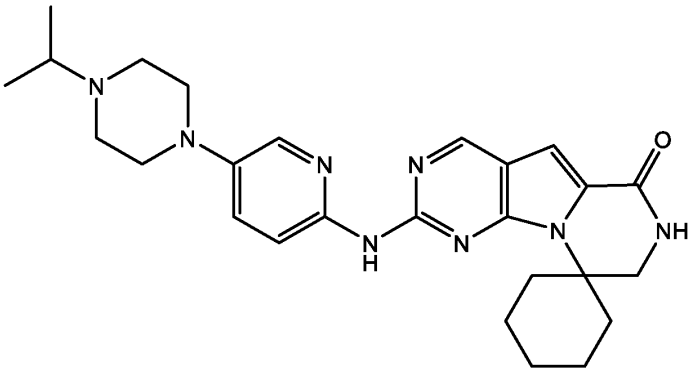
20. The method of any of claims 18 to 19, wherein the FGFR1 aberration or FGFR 2 aberration is an overexpression or amplification.

21. The method of any of claims 18 to 20, wherein the non-small cell lung cancer is large cell lung carcinoma.

22. The method of any of claims 18 to 20, wherein the non-small cell lung cancer is squamous cell carcinoma.

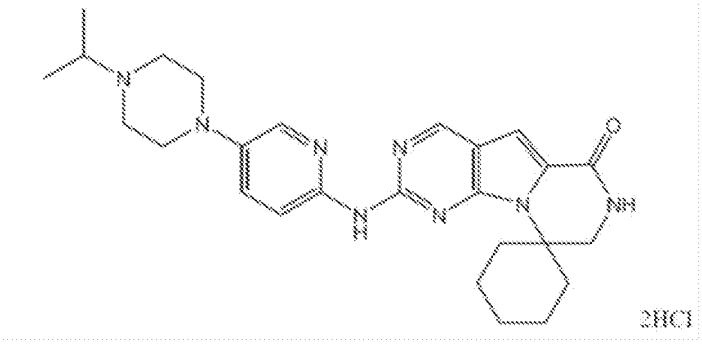
23. The method of any of claim 18 to 22, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, GSK3052230, and FIIN-2.

24. The method of any of claims 18 to 23, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

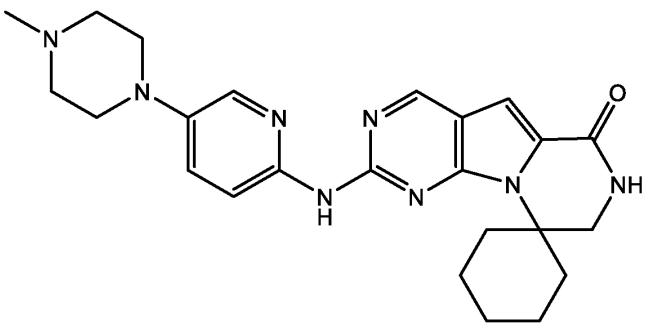
25. The method of any of claims 18 to 23, wherein the CDK4/6 inhibitor is



(Compound IA).

26. The method of claim 25, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

27. The method of any of claims 18 to 23, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

28. The method of any of claims 18 to 27, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

29. The method of any of claims 18 to 27, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

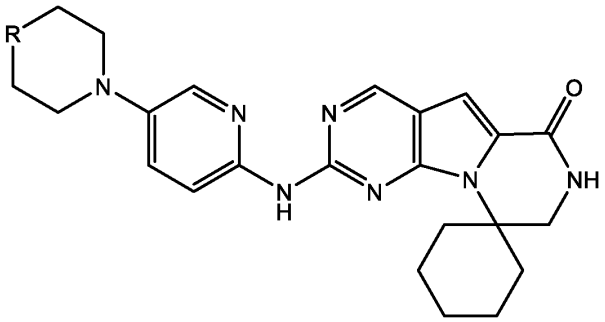
30. The method of any of claims 18 to 27, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

31. The method of any of claims 18 to 30, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

32. The method of any of claims 18 to 31, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

33. The method of any of claims 18 to 32, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

34. A method of treating a host with a gastric adenocarcinoma having a dysregulated FGFR signaling pathway caused by an FGFR2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

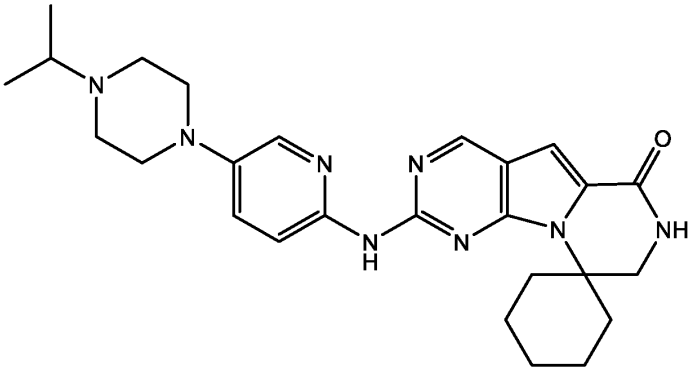
or a pharmaceutically acceptable salt thereof.

35. The method of claim 34, wherein the host is a human.

36. The method of claim 34 or 35, wherein the FGFR2 aberration is a result of an FGFR2 overexpression or amplification.

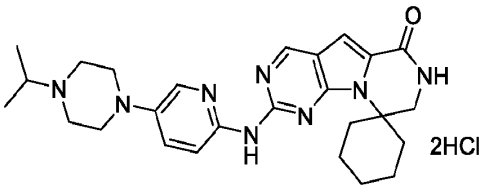
37. The method of any of claims 34 to 36, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, and FIIN-2.

38. The method of any of claims 34 to 37, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

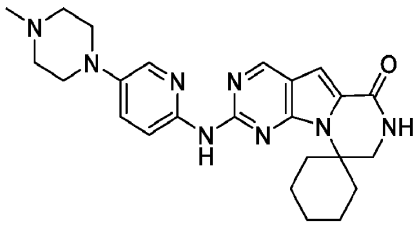
39. The method of any of claims 34 to 37, wherein the CDK4/6 inhibitor is




(Compound IA).

40. The method of claim 34 to 37, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

41. The method of any of claims 34 to 37, wherein the CDK4/6 inhibitor has the structure:




, or a pharmaceutically acceptable salt thereof.

42. The method of any of claims 34 to 41, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

43. The method of any of claims 34 to 41, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

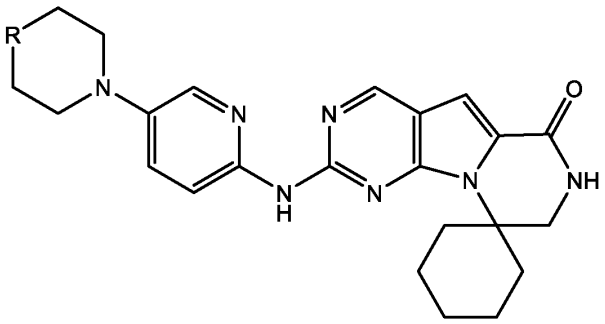
44. The method of any of claims 34 to 41, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

45. The method of any of claims 34 to 44, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

46. The method of any of claims 34 to 45, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

47. The method of any of claims 34 to 46, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

48. A method of reducing the development of acquired resistance to the inhibitory effects of a selective FGFR-TKI in a host with gastric adenocarcinoma having an FGFR 2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is:



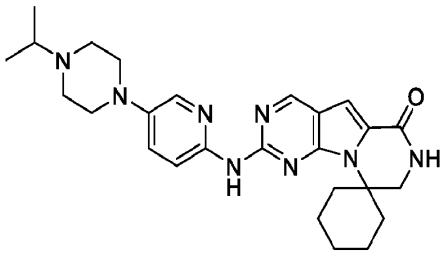

wherein R is NX, and wherein X is methyl or isopropyl,
or a pharmaceutically acceptable salt thereof.

49. The method of claim 48, wherein the host is human.

50. The method of any of claims 48 to 49, wherein the FGFR aberration is an FGFR overexpression or amplification.

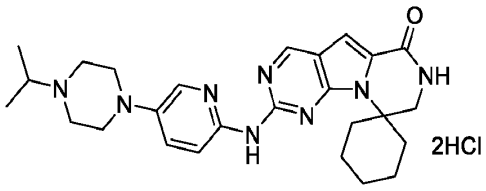
51. The method of any of claim 48-50, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, and FIIN-2.

52. The method of any of claims 48 to 51, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

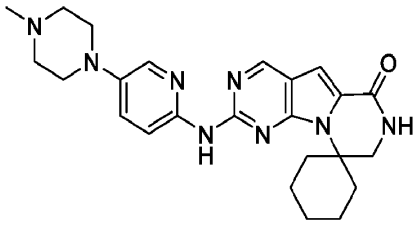
53. The method of any of claims 48 to 51, wherein the CDK4/6 inhibitor is



(Compound IA).

54. The method of claim 53, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

55. The method of any of claims 48 to 51, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

56. The method of any of claims 48 to 55, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

57. The method of any of claims 48 to 55, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

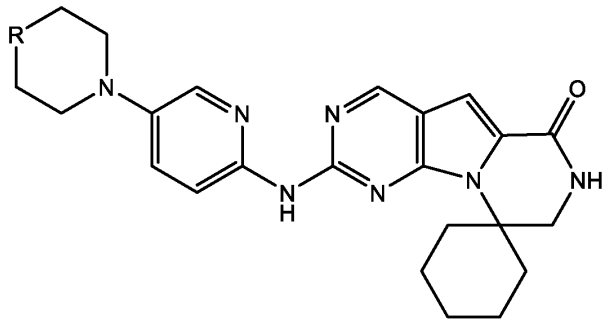
58. The method of any of claims 48 to 55, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

59. The method of any of claims 48 to 58, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

60. The method of any of claims 48 to 59, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

61. The method of any of claims 48 to 60, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

62. A method of treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 amplification or overexpression comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is



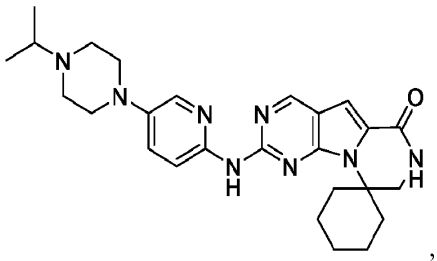

wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof,

and wherein the cancer is selected from the group consisting of non-small cell lung cancer, small cell lung cancer, triple negative breast cancer, osteosarcoma, pilocytic astrocytoma, and glioblastoma.

63. The method of claim 62, wherein the host is a human.

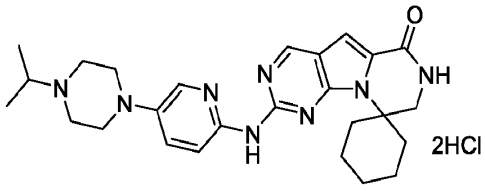
64. The method of any of claim 62 to 63, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, Debio1347, PRN1371, FIIN, 2, GSK3052230, and PD173074.

65. The method of any of claims 62 to 64, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

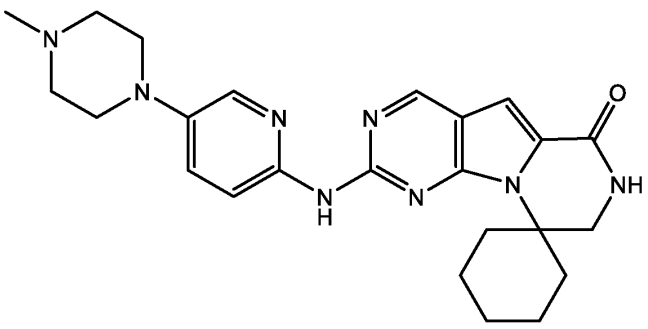
66. The method of any of claims 62 to 64, wherein the CDK4/6 inhibitor is



(Compound IA).

67. The method of claim 66, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

68. The method of any of claims 62 to 64, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

69. The method of any of claims 62 to 68, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

70. The method of any of claims 62 to 68, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

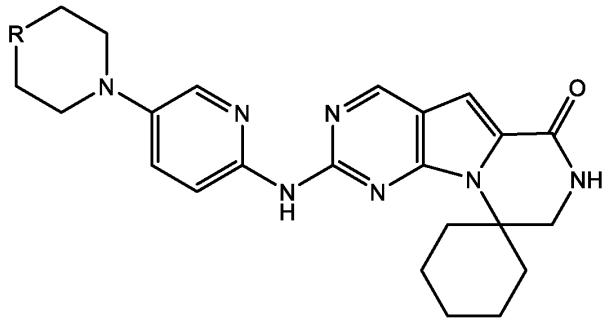
71. The method of any of claims 62 to 68, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

72. The method of any of claims 62 to 71, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

73. The method of any of claims 62 to 72, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

74. The method of any of claims 62 to 73, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

75. A method of treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR2 aberration, wherein the FGFR2 aberration is selected from the group consisting of an FGFR2 amplification or overexpression, an FGFR2 mutation, and an FGFR2 translocation or fusion, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is



wherein R is NX, and wherein X is methyl or isopropyl, or a pharmaceutically acceptable salt thereof,

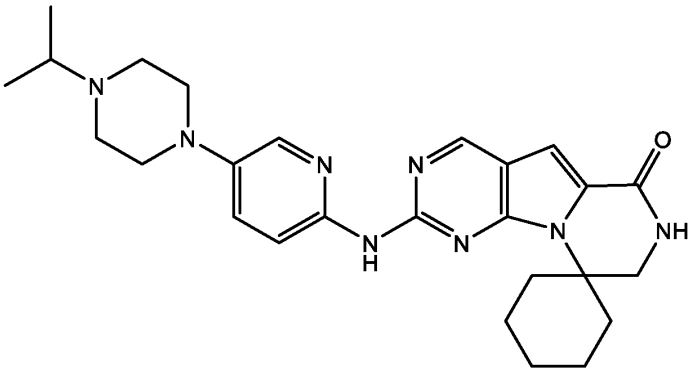
and wherein the cancer is selected from the group consisting of endometrial cancer, non-small cell lung cancer, gastric cancer, intrahepatic cholangiocarcinoma, and thyroid cancer.

76. The method of claim 75, wherein the host is a human.

77. The method of claim 77, wherein the FGFR2 aberration is an FGFR2 amplification or overexpression.

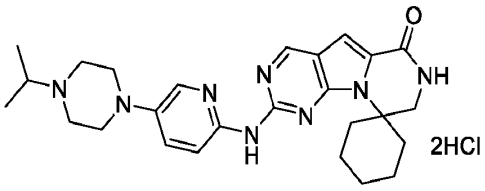
78. The method of any of claim 75 to 77, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, and FIIN-2.

79. The method of any of claims 75 to 78, wherein the CDK4/6 inhibitor is:



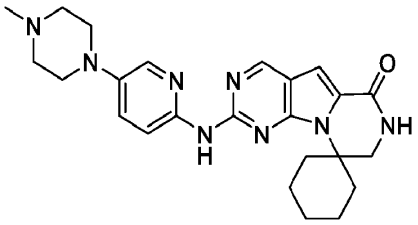
, or a pharmaceutically acceptable salt thereof.

80. The method of any of claims 75 to 78, wherein the CDK4/6 inhibitor is




(Compound IA).

81. The method of claim 80, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.
82. The method of any of claims 75 to 78, wherein the CDK4/6 inhibitor has the structure:

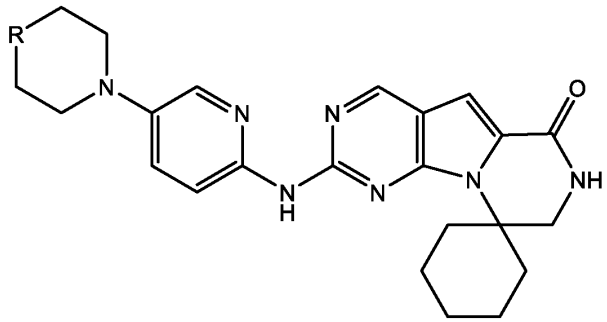



, or a pharmaceutically acceptable salt thereof.

83. The method of any of claims 75 to 82, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.
84. The method of any of claims 75 to 82, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.
85. The method of any of claims 75 to 82, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.
86. The method of any of claims 75 to 85, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.
87. The method of any of claims 75 to 86, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

88. The method of any of claims 75 to 87, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

89. A method of treating a host with a cancer with a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR3 aberration, wherein the FGFR3 aberration is selected from the group consisting of an FGFR3 amplification or overexpression, an FGFR3 mutation, and an FGFR3 translocation or fusion, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective FGFR-TKI, wherein the CDK4/6 inhibitor is



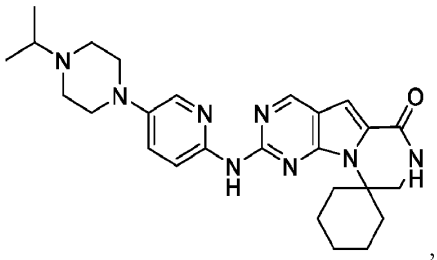

wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof,

and wherein the cancer is selected from the group consisting of glioblastoma, non-small cell lung cancer, cervical cancer, and multiple myeloma.

90. The method of claim 89, wherein the FGFR3 aberration is an FGFR3 translocation or fusion.

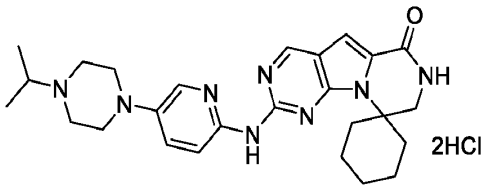
91. The method of any of claim 89 to 90, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, MGFR1877S, vofatamab, and FIIN-2.

92. The method of any of claims 89 to 91, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

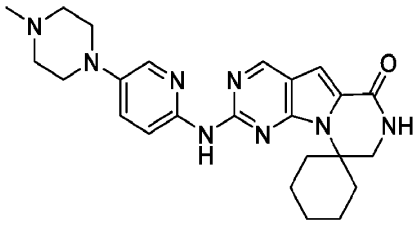
93. The method of any of claims 89 to 91, wherein the CDK4/6 inhibitor is



(Compound IA).

94. The method of claim 93, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

95. The method of any of claims 89 to 91, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

96. The method of any of claims 89 to 95, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

97. The method of any of claims 89 to 95, wherein the CDK4/6 inhibitor and the EGFR-TKI is administered to the host at least once a day for at least 35 consecutive days.

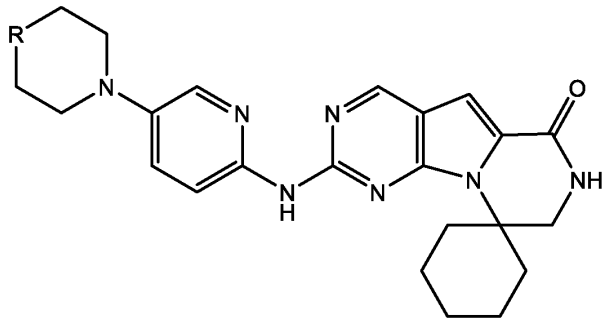
98. The method of any of claims 89 to 95, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

99. The method of any of claims 89 to 99, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

100. The method of any of claims 89 to 99, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

101. The method of any of claims 89 to 100, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

102. A method of treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR4 aberration or FGF aberration wherein the FGFR4 aberration is selected from the group consisting of an FGFR4 amplification, an FGFR4 mutation, and an FGFR4 translocation and the FGF aberration is an overexpression or amplification, comprising administering to the host an effective amount of a short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor (FGFR) inhibitor, wherein the CDK4/6 inhibitor is



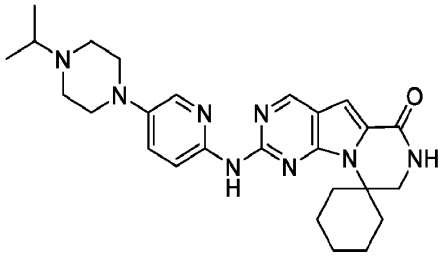

wherein R is NX, and wherein X is methyl or isopropyl, or a pharmaceutically acceptable salt thereof,

and wherein the cancer is selected from the group consisting of hepatocellular carcinoma, rhabdomyosarcoma, endometrial cancer, and ovarian cancer.

103. The method of claim 102, wherein the host is human.

104. The method of any of claim 102 to 103, wherein the selective FGFR inhibitor is selected from the group consisting of infigratinib, futibatinib, derazantinib, LY287445, INCB062079, BLU9931, H3-6527, fisogatinib, roblitinib, Debio1347, PRN1371, and FIIN-2.

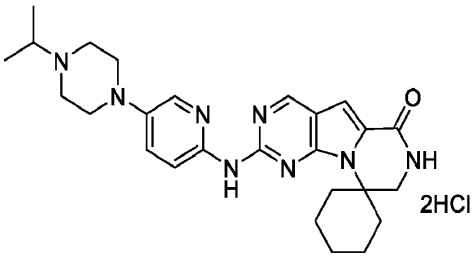
105. The method of any of claims 102 to 104, wherein the CDK4/6 inhibitor is:



,

or a pharmaceutically acceptable salt thereof.

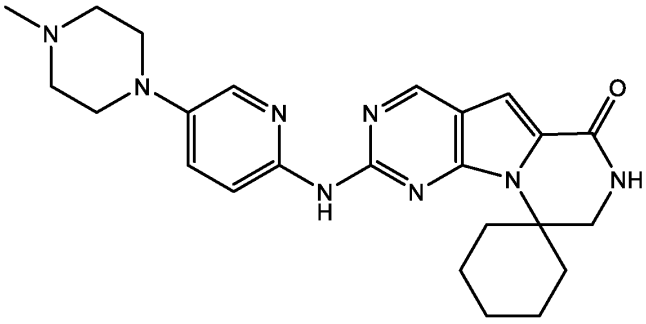
106. The method of any of claims 102 to 104, wherein the CDK4/6 inhibitor is



(Compound IA).

107. The method of claim 106, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

108. The method of any of claims 102 to 104, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

109. The method of any of claims 102 to 108, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 28 consecutive days.

110. The method of any of claims 102 to 108, wherein the CDK4/6 inhibitor and the FGFR inhibitor are administered to the host at least once a day for at least 35 consecutive days.

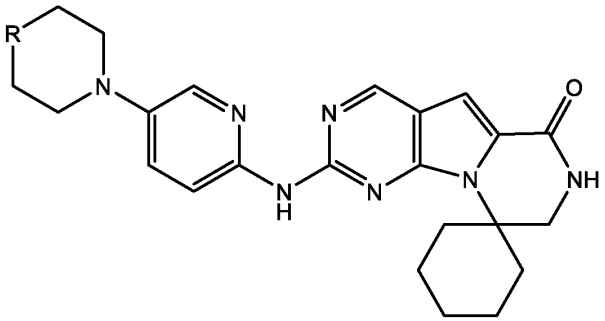
111. The method of any of claims 102 to 108, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

112. The method of any of claims 102 to 111, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

113. The method of any of claims 102 to 112, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

114. The method of any of claims 102 to 113, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKIs.

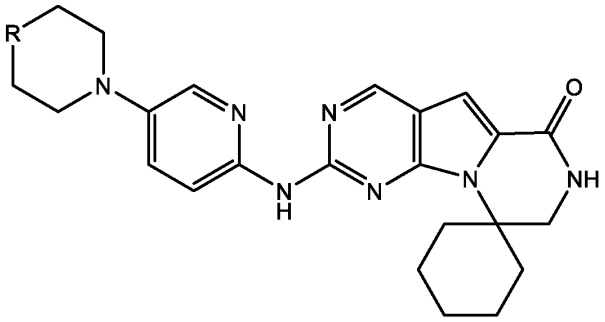
115. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in the treatment of a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

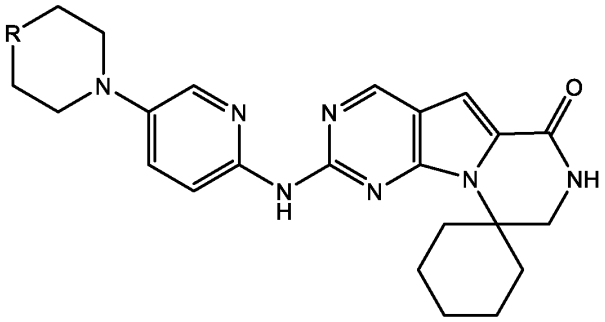
116. A short acting CDK4/6 inhibitor compound of the formula:




wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in reducing the development of acquired resistance to the inhibitory effects of a selective FGFR-TKI in a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

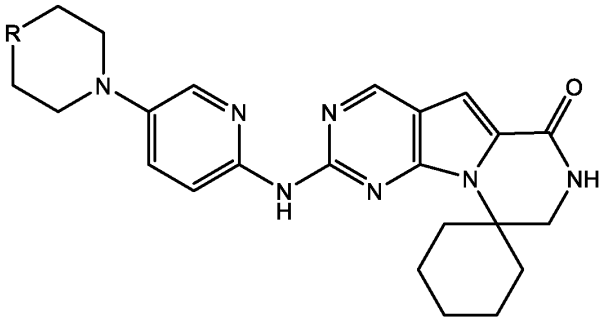
117. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in treating a host with a gastric adenocarcinoma having a dysregulated FGFR signaling pathway caused by an FGFR2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation,, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

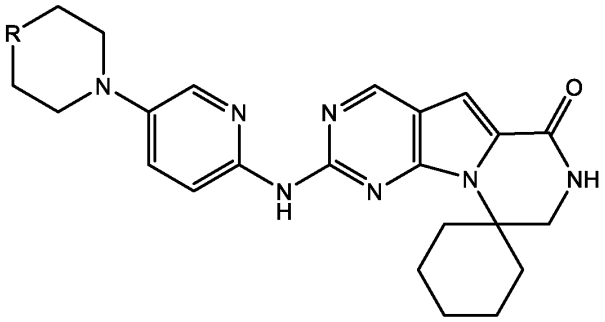
118. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in treating a host with gastric adenocarcinoma having an FGFR 2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

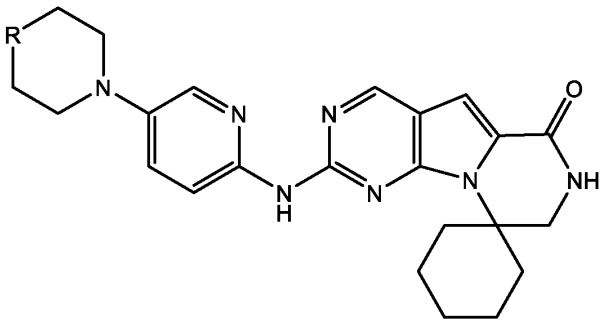
119. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 amplification or overexpression comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

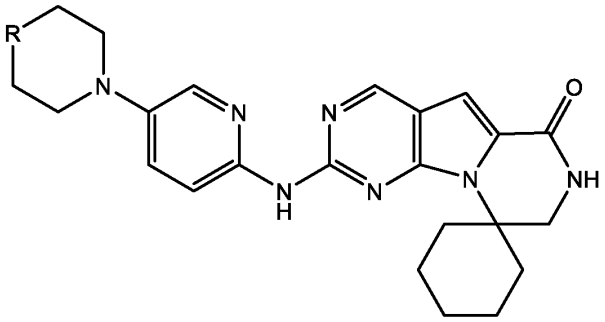
120. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR2 aberration, wherein the FGFR2 aberration is selected from the group consisting of an FGFR2 amplification or overexpression, an FGFR2 mutation, and an FGFR2 translocation or fusion, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

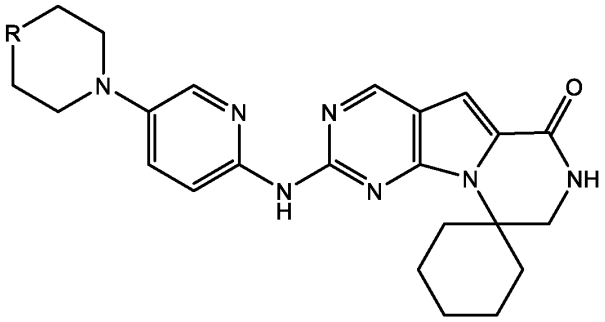
121. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

or a pharmaceutically acceptable salt thereof, for use in treating a host with a cancer with a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR3 aberration, wherein the FGFR3 aberration is selected from the group consisting of an FGFR3 amplification or overexpression, an FGFR3 mutation, and an FGFR3 translocation or fusion, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

122. A short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl,

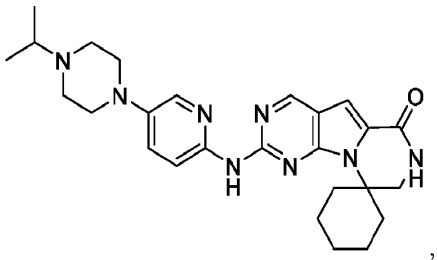
or a pharmaceutically acceptable salt thereof, for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR4 aberration or FGF aberration wherein the FGFR4 aberration is selected from the group consisting of an FGFR4 amplification, an FGFR4 mutation, and an FGFR4 translocation and the FGF aberration is an overexpression or amplification, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an

effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

123. The composition of claim 115 to 122, wherein the host is a human.

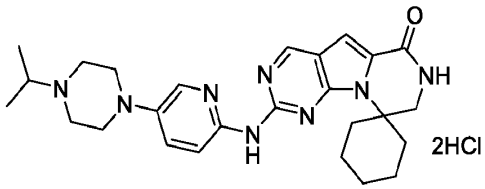
124. The composition of any of claim 115 to 123, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, GSK3052230, FIIN-2, INCB062079, BLU9931, H3-6527, fisogatinib, and roblitinib.

125. The composition of any of claims 115 to 124, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

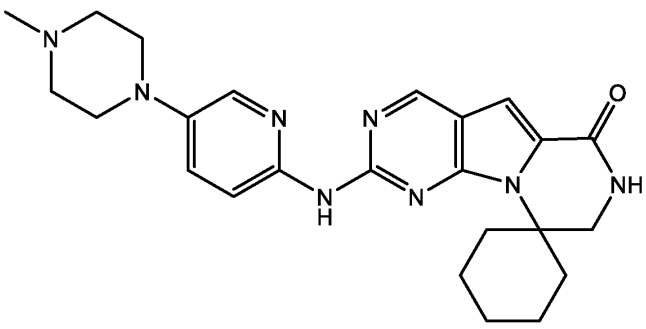
126. The composition of any of claims 115 to 124, wherein the CDK4/6 inhibitor is




(Compound IA).

127. The composition of claim 126, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

128. The composition of any of claims 115 to 124, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

129. The composition of any of claims 115 to 128, wherein the CDK4/6 inhibitor and the FGFR inhibitor are administered to the host at least once a day for at least 28 consecutive days.

130. The composition of any of claims 115 to 128, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

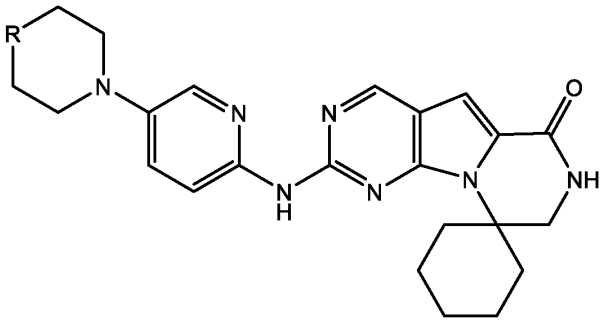
131. The composition of any of claims 115 to 128, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

132. The composition of any of claims 115 to 131, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

133. The composition of any of claims 115 to 132, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

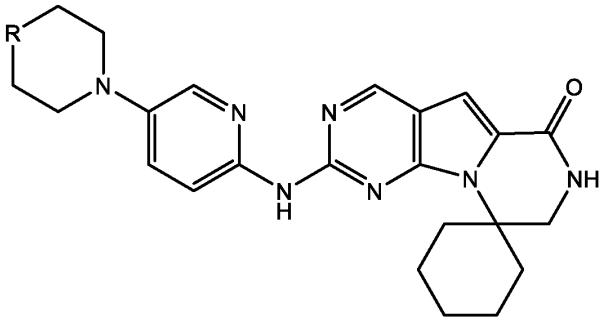
134. The composition of any of claims 115 to 133, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKI.

135. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in the treatment of a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

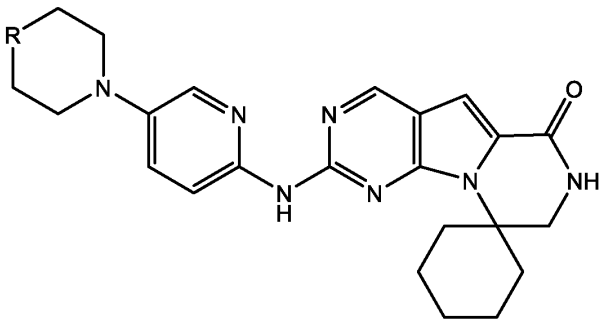
136. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in reducing the development of acquired resistance to the inhibitory effects of a selective FGFR-TKI in a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an

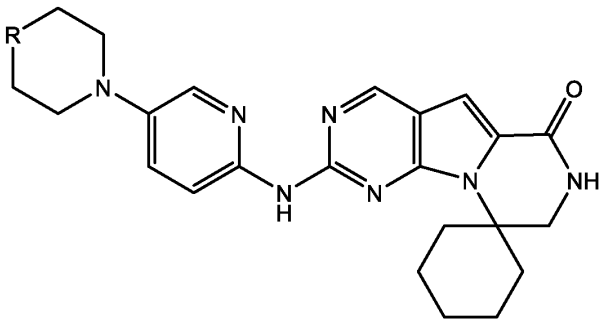
effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

137. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with a gastric adenocarcinoma having a dysregulated FGFR signaling pathway caused by an FGFR2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation,, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

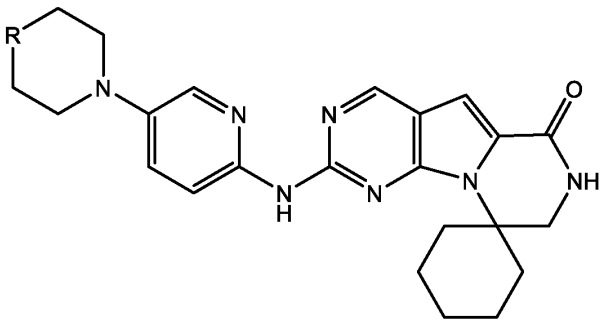
138. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with gastric adenocarcinoma having an FGFR 2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or

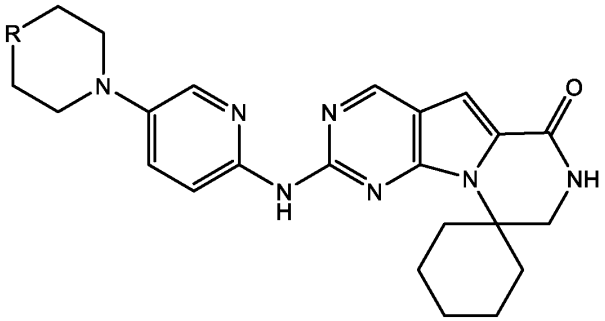
mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

139. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 amplification or overexpression comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

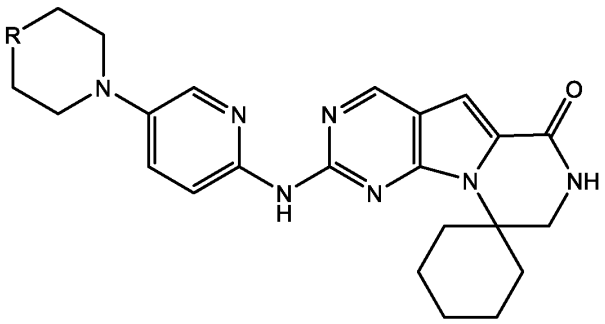
140. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR2 aberration, wherein the FGFR2 aberration is selected from the group consisting of an FGFR2 amplification or overexpression, an FGFR2 mutation, and an

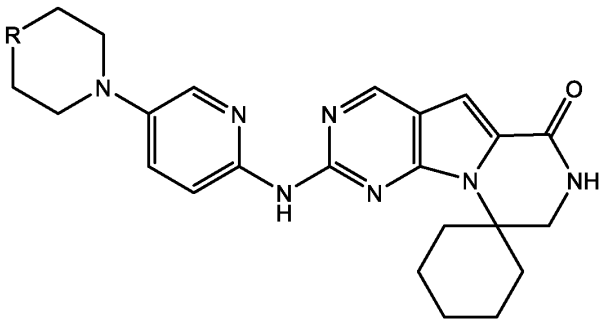
FGFR2 translocation or fusion, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

141. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with a cancer with a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR3 aberration, wherein the FGFR3 aberration is selected from the group consisting of an FGFR3 amplification or overexpression, an FGFR3 mutation, and an FGFR3 translocation or fusion, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

142. Use of a short acting CDK4/6 inhibitor compound of the formula:



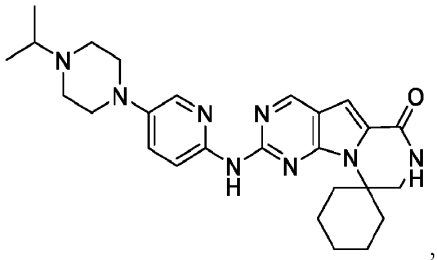
wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, in the manufacture of a medicament for use in treating a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR4 aberration or FGF aberration wherein the FGFR4 aberration is

selected from the group consisting of an FGFR4 amplification, an FGFR4 mutation, and an FGFR4 translocation and the FGF aberration is an overexpression or amplification, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

143. The composition of claim 135 to 142, wherein the host is a human.

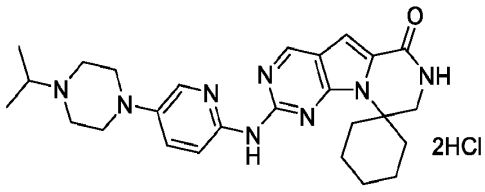
144. The use of any of claim 135 to 143, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, GSK3052230, FIIN-2, INCB062079, BLU9931, H3-6527, fisogatinib, and roblitinib.

145. The composition of any of claims 135 to 144, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

146. The use of any of claims 135 to 144, wherein the CDK4/6 inhibitor is

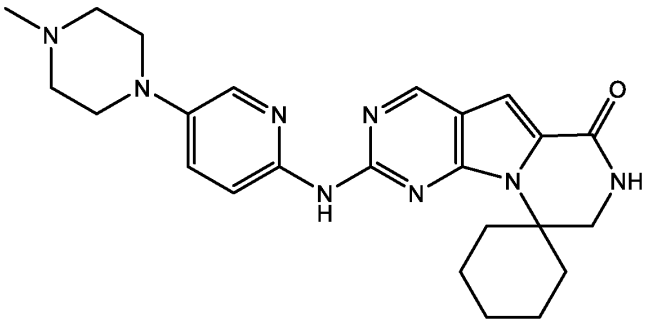


(Compound IA).

147. The use of claim 146, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values

selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

148. The use of any of claims 135 to 143, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

149. The use of any of claims 135 to 148, wherein the CDK4/6 inhibitor and the FGFR inhibitor are administered to the host at least once a day for at least 28 consecutive days.

150. The use of any of claims 135 to 148, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

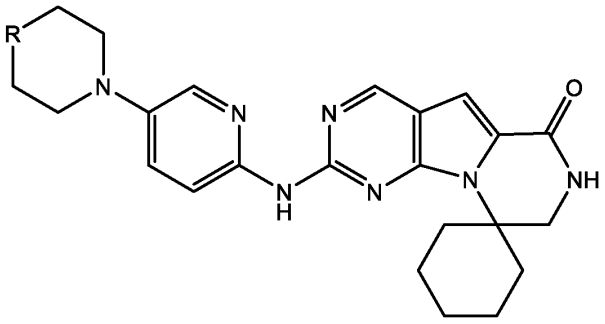
151. The use of any of claims 135 to 148, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

152. The use of any of claims 135 to 151, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

153. The use of any of claims 135 to 152, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

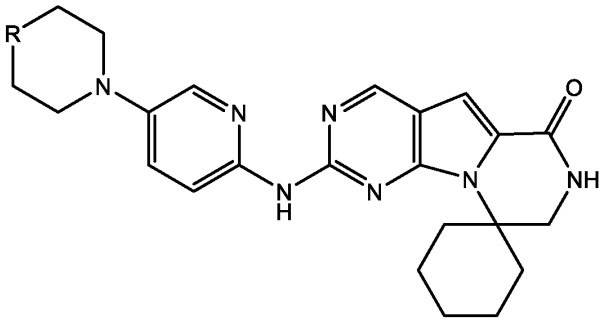
154. The use of any of claims 135 to 153, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKI.

155. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, the treatment comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

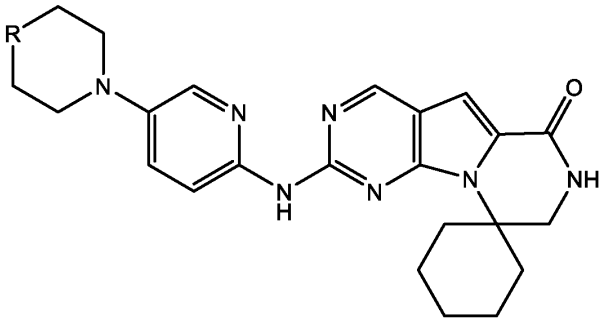
156. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for reducing the development of acquired resistance to the inhibitory effects of a selective FGFR-TKI in a host with a non-small cell lung cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 or FGFR2 aberration mutation, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6

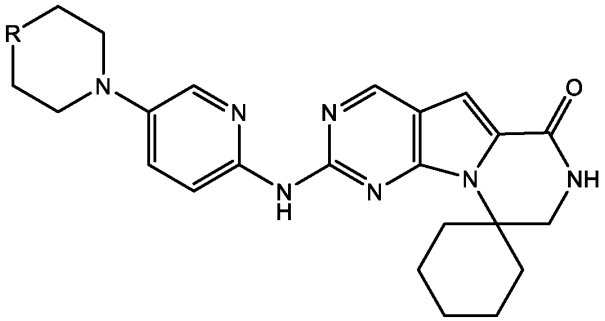
inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

157. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a gastric adenocarcinoma having a dysregulated FGFR signaling pathway caused by an FGFR2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation,, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

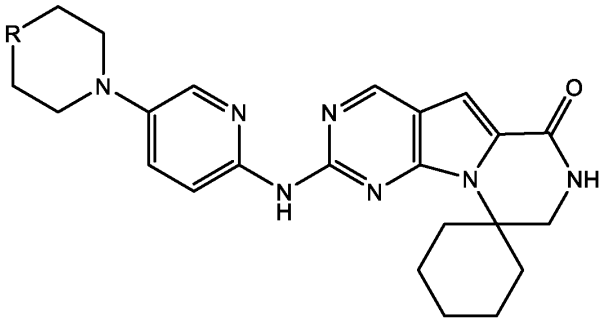
158. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with gastric adenocarcinoma having an FGFR 2 aberration, wherein the aberration is selected from the group consisting of an overexpression, amplification, translocation, fusion, or mutation, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to

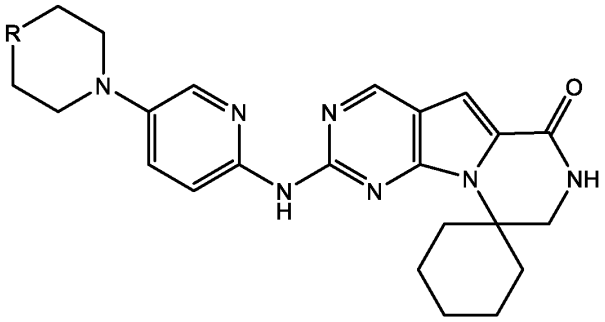
the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

159. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR1 amplification or overexpression comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

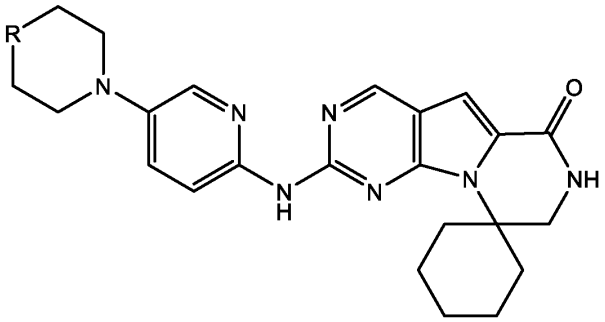
160. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR2 aberration, wherein the FGFR2 aberration is selected from the group consisting of an FGFR2 amplification or overexpression, an FGFR2 mutation, and an FGFR2 translocation or fusion, comprising administering to the host an effective amount of the short acting

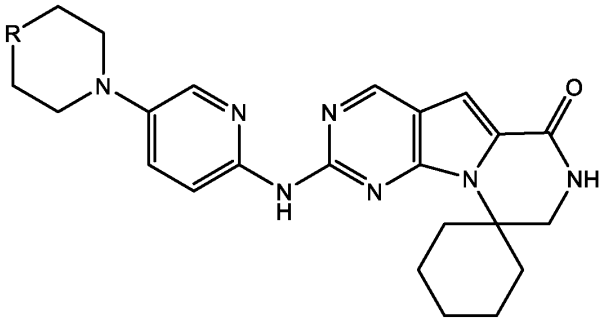
CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

161. Use of a short acting CDK4/6 inhibitor compound of the formula:



wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a cancer with a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR3 aberration, wherein the FGFR3 aberration is selected from the group consisting of an FGFR3 amplification or overexpression, an FGFR3 mutation, and an FGFR3 translocation or fusion, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

162. Use of a short acting CDK4/6 inhibitor compound of the formula:



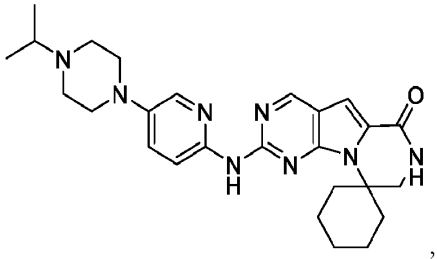
wherein R is NX, and wherein X is hydrogen, methyl, or isopropyl, or a pharmaceutically acceptable salt thereof, for the treatment of a host with a cancer having a dysregulated fibroblast growth factor receptor (FGFR) signaling pathway caused by an FGFR4 aberration or FGF aberration wherein the FGFR4 aberration is selected from the group consisting of an FGFR4 amplification, an FGFR4 mutation, and an FGFR4 translocation

and the FGF aberration is an overexpression or amplification, comprising administering to the host an effective amount of the short acting CDK4/6 inhibitor, and administering to the host an effective amount of a selective fibroblast growth factor receptor-tyrosine kinase inhibitor (FGFR-TKI).

163. The composition of claim 155 to 162, wherein the host is a human.

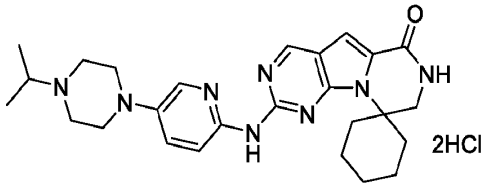
164. The use of any of claim 155 to 163, wherein the selective FGFR-TKI is selected from the group consisting of erdafitinib, pemigatinib, infigratinib, futibatinib, derazantinib, LY287445, Debio1347, PRN1371, alofanib, bemarituzumab, GSK3052230, FIIN-2, INCB062079, BLU9931, H3-6527, fisogatinib, and roblitinib.

165. The composition of any of claims 155 to 164, wherein the CDK4/6 inhibitor is:



, or a pharmaceutically acceptable salt thereof.

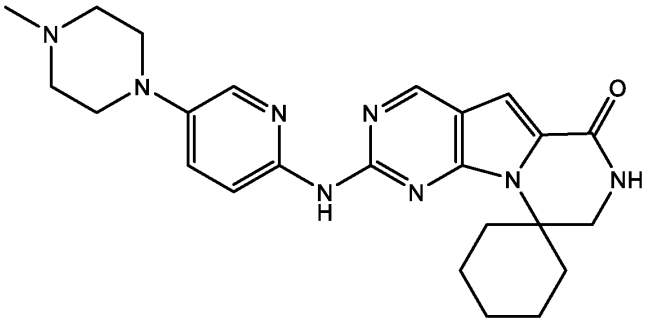
166. The use of any of claims 155 to 164, wherein the CDK4/6 inhibitor is



(Compound IA).

167. The use of claim 166, wherein the CDK4/6 inhibitor is Compound IA, Form B, wherein Form B is characterized by an XRPD pattern comprising at least two 2theta values selected from 6.5±0.2°, 9.5±0.2°, 14.0±0.2°, 14.4±0.2°, 18.1±0.2°, 19.9±0.2°, and 22.4±0.2°.

168. The use of any of claims 155 to 164, wherein the CDK4/6 inhibitor has the structure:



, or a pharmaceutically acceptable salt thereof.

169. The use of any of claims 155 to 168, wherein the CDK4/6 inhibitor and the FGFR inhibitor are administered to the host at least once a day for at least 28 consecutive days.

170. The use of any of claims 155 to 168, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 35 consecutive days.

171. The use of any of claims 155 to 168, wherein the CDK4/6 inhibitor and the FGFR-TKI are administered to the host at least once a day for at least 56 consecutive days.

172. The use of any of claims 155 to 171, wherein the host, at the time of the first administration of the CDK4/6 inhibitor, is CDK4/6 inhibitor treatment naïve.

173. The use of any of claims 155 to 172, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired resistance to one or more previously administered FGFR inhibitors.

174. The use of any of claims 155 to 173, wherein the cancer, at the time of the first administration of the CDK4/6 inhibitor, has acquired a mutation rendering the cancer susceptible to developing resistance to one or more FGFR-TKI.