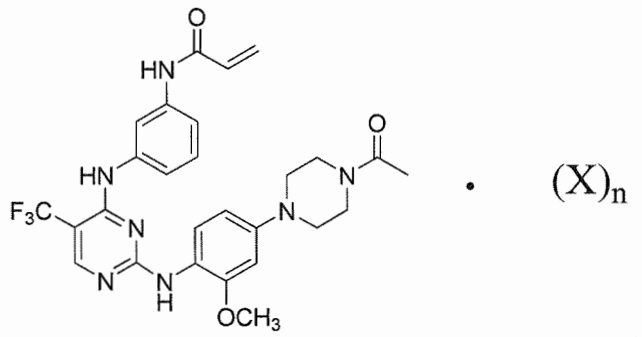
mutation who never received gefitinib treatment. In addition, germline EGFR T790M mutations are linked with certain familial lung cancers.

[0007] Current drugs in development, including second-generation covalent inhibitors, such as BIBW2992, HKI-272 and PF-0299804, are effective against the T790M resistance mutation but exhibit dose-limiting toxicities due to concurrent inhibition of WT EGFR. Accordingly, there remains a need to find mutant-selective EGFR kinase inhibitors useful as therapeutic agents.

SUMMARY ~~OF THE INVENTION~~

[0007] It has now been found that the novel benzenesulfonic acid, camphor sulfonic acid, 1,2-ethane disulfonic acid, hydrobromic acid, hydrochloric acid, maleic acid, methanesulfonic acid, naphthalene-2-sulfonic acid, 1,5-naphthalene disulfonic acid, oxalic acid, 4-toluenesulfonic acid or 2,4,6-trihydroxybenzoic acid salts of the present ~~invention~~disclosure, and compositions thereof, are useful as mutant-selective inhibitors of one or more EGFR kinases and exhibits desirable characteristics for the same. In general, these salts, and pharmaceutically acceptable compositions thereof, are useful for treating or lessening the severity of a variety of diseases or disorders as described in detail herein.

[0007a] In a first aspect of the present disclosure, there is provided a compound **2**:



wherein:

n is 1 or 2; and

X is hydrobromic acid, benzenesulfonic acid, camphor sulfonic acid, 1,2-ethane disulfonic acid, hydrochloric acid, maleic acid, methanesulfonic acid, naphthalene-2-sulfonic acid, 1,5-naphthalene disulfonic acid, oxalic acid, 4-toluenesulfonic acid or 2,4,6-trihydroxybenzoic acid.

[0007b] In a second aspect of the present disclosure, there is provided a composition comprising the compound as described herein and a pharmaceutically acceptable carrier or excipient.

[0007c] In a third aspect of the present disclosure, there is provided a method for inhibiting at least one mutant of EGFR selectively as compared to wild type (WT) EGFR in a biological sample, comprising contacting the biological sample with a compound as described herein, or a composition thereof.

[0007d] In a fourth aspect of the present disclosure, there is provided a use of a compound as described herein, or a composition thereof, in the manufacture of a medicament for inhibiting at least one mutant EGFR selectively as compared to wild type EGFR in a biological sample or in a patient.

[0007e] In a fifth aspect of the present disclosure, there is provided a use of a compound as described herein, or a composition thereof, in the manufacture of a medicament for treating a mutant EGFR-mediated disorder or condition in a patient.

BRIEF DESCRIPTION OF THE DRAWINGS

[0008] **Figure 1** depicts the x-ray powder diffraction (XRPD) pattern for a bis-besylate salt of Compound **1**.

[0009] **Figure 2** depicts the thermogravimetric analysis (TGA) pattern for a bis-besylate salt of Compound **1**.

[0010] **Figure 3** depicts the thermogravimetric analysis (TGA) pattern for a further dried sample of a bis-besylate salt of Compound **1**.

[0011] **Figure 4** depicts the differential scanning calorimetry (DSC) pattern for a bis-besylate salt of Compound **1**.

[0012] **Figure 5** depicts the infrared (IR) spectrum of a bis-besylate salt of Compound **1**.

[0013] **Figure 6** depicts the ^{1}H-NMR spectrum of a bis-besylate salt of Compound **1.**

[0014] **Figure 7** depicts the dynamic vapor sorption (DVS) pattern of a bis-besylate salt of Compound **1**.

19. The compound of claim 1, wherein X is hydrochloric acid.

20. The compound of claim 1, wherein X is maleic acid.

21. The compound of claim 1, wherein X is methanesulfonic acid.

22. The compound of claim 1, wherein X is naphthalene-2-sulfonic acid.

23. The compound of claim 1, wherein X is 1,5-naphthalene disulfonic acid.

24. The compound of claim 1, wherein X is oxalic acid.

25. The compound of claim 1, wherein X is p-toluenesulfonic acid.

26. The compound of claim 1, wherein X is 2,4,6-trihydroxybenzoic acid.

27. A composition comprising the compound of claim 1 and a pharmaceutically acceptable carrier or excipient.

28. A method for inhibiting at least one mutant of EGFR selectively as compared to wild type (WT) EGFR, in a biological sample ~~or in a patient~~, comprising contacting the biological sample with~~, or administering to the patient,~~ a compound according to claim 1, or a composition thereof.

29. The method according to claim 28, wherein said method is sparing for WT EGFR.

30. The method according to claim 28, wherein the at least one mutant is an activating mutant, a deletion mutant, a point mutation, or a mutant selected from T790M, delE746-A750, L858R, G719S.

~~31. A method for treating a mutant EGFR-mediated disorder or condition in a patient, comprising administering to the patient a composition according to claim 27.~~

~~32. The method of claim 31, wherein the disorder or condition is a cancer.~~

31. A composition comprising the compound of any one of claims 2 to 13 and a pharmaceutically acceptable carrier or excipient.

32. A composition comprising the compound of claim 2 and a pharmaceutically acceptable carrier or excipient.

33. A composition comprising the compound of claim 3 and a pharmaceutically acceptable carrier or excipient.

34. A composition comprising the compound of claim 4 and a pharmaceutically acceptable carrier or excipient.

35. A composition comprising the compound of claim 5 and a pharmaceutically acceptable carrier or excipient.

36. A composition comprising the compound of claim 6 and a pharmaceutically acceptable carrier or excipient.

37. A composition comprising the compound of claim 7 and a pharmaceutically acceptable carrier or excipient.

38. A composition comprising the compound of claim 8 and a pharmaceutically acceptable carrier or excipient.

39. A method for inhibiting at least one mutant of EGFR selectively as compared to wild type EGFR (WT EGFR) in a biological sample comprising contacting the biological sample with a compound according to any one of claims 2 to 13, or a composition thereof.

40. The method according to claim 39, wherein said method is sparing for WT EGFR.

41. The method according to claim 39, wherein the at least one mutant is an activating mutant, a deletion mutant, a point mutation, or a mutant selected from T790M, delE746-A750, L858R or G719S.

42. Use of a compound according to claim 1, or a composition thereof, in the manufacture of a medicament for inhibiting at least one mutant EGFR selectively as compared to wild type EGFR in a biological sample or in a patient.

43. Use of a compound according to claim 1, or a composition thereof, in the manufacture of a medicament for treating a mutant EGFR-mediated disorder or condition in a patient.

44. The use according to claim 43, wherein the disorder or condition is a cancer.

45. The use according to claim 44, wherein the cancer is a non-small cell lung cancer.

46. Use of a compound according to any one of claims 2 to 13, or a composition thereof, in the manufacture of a medicament for inhibiting at least one mutant EGFR selectively as compared to wild type EGFR (WT EGFR), in a biological sample or in a patient.

47. Use of a compound according to any one of claims 2 to 13, or a composition thereof, in the manufacture of a medicament for treating a mutant EGFR-mediated disorder or condition in a patient.

48. The use according to claim 47, wherein the disorder or condition is a cancer.

49. The use according to claim 48, wherein the cancer is a non-small cell lung cancer.