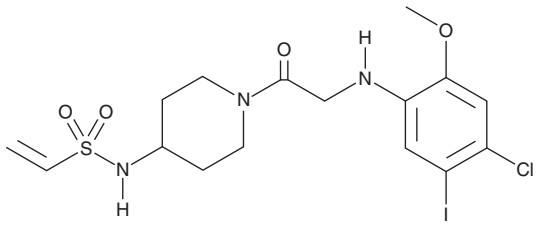
PRODUCT INFORMATION

K-Ras(G12C) Inhibitor 9

Item No. 18005

CAS Registry No.: 1469337-91-4
Formal Name: N-[1-[2-[(4-chloro-5-iodo-2-methoxyphenyl)amino]acetyl]-4-piperidinyl]-ethenesulfonamide
Synonyms: Ki-Ras(G12C) Inhibitor 9, c-Ki-ras(G12C) Inhibitor 9, K-Ras4A(G12C) Inhibitor 9, c-K-ras(G12C) Inhibitor 9, KRAS(G12C) Inhibitor 9, Kristen Rat Sarcoma Virus(G12C) Inhibitor 9
MF: $C_{16}H_{21}ClIN_3O_4S$
FW: 513.8
Purity: ≥98%
UV/Vis.: λ_{max}: 222, 260, 308 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

K-Ras(G12C) inhibitor 9 is supplied as a crystalline solid. A stock solution may be made by dissolving the K-Ras(G12C) inhibitor 9 in the solvent of choice, which should be purged with an inert gas. K-Ras(G12C) inhibitor 9 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of K-Ras(G12C) inhibitor 9 in these solvents is approximately 12 and 14 mg/ml, respectively.

K-Ras(G12C) inhibitor 9 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, K-Ras(G12C) inhibitor 9 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. K-Ras(G12C) inhibitor 9 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

K-Ras(G12C) inhibitor 9 is an irreversible, allosteric inhibitor of the K-Ras(G12C) mutant that causes 100% modification of the protein when used at 10 µM for 24 hours *in vitro*.[1] K-Ras is a small GTPase that cycles between a GTP-bound active state and a GDP-bound inactive state to turn on downstream Raf kinases to drive cell growth. A G12C mutation in K-Ras blocks GTP hydrolysis, activates K-Ras, and promotes carcinogenesis.[2,3] Similar K-Ras(G12C) inhibitors significantly reduce GTP affinity relative to GDP, decrease Raf binding, and lower cell viability while increasing apoptosis.[1]

References

1. Ostrem, J.M., Peters, U., Sos, M.L., *et al.* K-Ras(G12C) inhibitors allosterically control GTP affinity and effector interactions. *Nature* **503(7477)**, 548-551 (2013).
2. Lim, S.M., Westover, K.D., Ficarro, S.B., *et al.* Therapeutic targeting of oncogenic K-Ras by a covalent catalytic site inhibitor. *Angew. Chem. Int. Ed. Engl.* **53(1)**, 199-204 (2014).
3. Padavano, J., Henkhaus, R.S., Chen, J., *et al.* Mutant K-RAS promotes invasion and metastasis in pancreatic cancer through GTPase signaling pathways. *Cancer Growth Metastasis* **8 (suppl 1)**, 95-113 (2015).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM