

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-piperidiny]-ethanesulfonamide

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

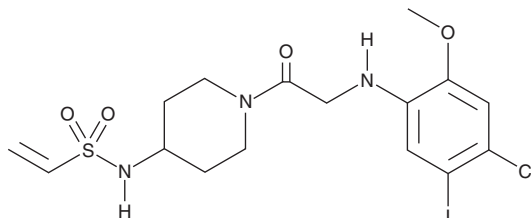
Purity: $\geq 98\%$

UV/Vis.: λ_{max} : 222, 260, 308 nm

Supplied as: A crystalline solid

Storage: $-20^{\circ}C$

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).

WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the [complete](#) Safety Data Sheet, which has been sent via email to your institution.

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