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Produktinformation

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- Diagnostik & molekulare Diagnostik
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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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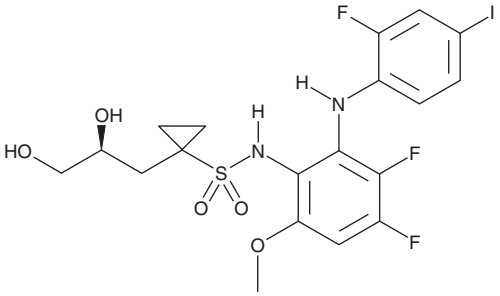
PRODUCT INFORMATION

Refametinib

Item No. 16997

CAS Registry No.: 923032-37-5
Formal Name: N-[3,4-difluoro-2-[(2-fluoro-4-iodophenyl)amino]-6-methoxyphenyl]-1-[(2S)-2,3-dihydroxypropyl]-cyclopropanesulfonamide
Synonyms: BAY 86-9766, RDEA119
MF: $C_{19}H_{20}F_3IN_2O_5S$
FW: 572.3
Purity: ≥95%
UV/Vis.: λ_{max}: 275, 299 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: As supplied, 2 years from the QC date provided on the Certificate of Analysis, when stored properly



Laboratory Procedures

Refametinib is supplied as a crystalline solid. A stock solution may be made by dissolving the refametinib in the solvent of choice. Refametinib is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of refametinib in these solvents is approximately 20, 1, 15 mg/ml, respectively.

Refametinib is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, refametinib should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Refametinib has a solubility of approximately 0.1 mg/ml in a 1:5 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Refametinib is an allosteric, selective inhibitor of MEK1 and MEK2 (IC_{50}s = 19 and 47 nM, respectively).[1] It blocks phosphorylation of ERK1/2 across several human cancer cell lines differing in tissue origin and BRAF mutational status (EC_{50}s = 2.5-16 nM), inhibiting cell cycling in cancer cells but not in primary cells.[1] Refametinib is orally available and active in human tumor xenograft models.[1] It has potential utility, particularly in combination therapy, in certain forms of cancer.[2-4]

References

1. Iverson, C., Larson, G., Lai, C., *et al.* RDEA119/BAY 869766: A potent, selective, allosteric inhibitor of MEK1/2 for the treatment of cancer. *Cancer Res.* **69(17)**, 6839-6847 (2009).
2. Diep, C.H., Munoz, R.M., Choudhary, A., *et al.* Synergistic effect between erlotinib and MEK inhibitors in KRAS wild-type human pancreatic cancer cells. *Clin. Cancer Res.* **17(9)**, 2744-2756 (2011).
3. Schmieder, R., Puehler, F., Neuhaus, R., **et al.** Allosteric MEK1/2 inhibitor refametinib (BAY 86-9766) in combination with sorafenib exhibits antitumor activity in preclinical murine and rat models of hepatocellular carcinoma. *Neoplasia* **15(10)**, 1161-1171 (2013).
4. Akinleye, A., Furgan, M., Mukhi, N., *et al.* MEK and the inhibitors: From bench to bedside. *J. Hematol. Oncol.* **6**, 1-11 (2013).

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