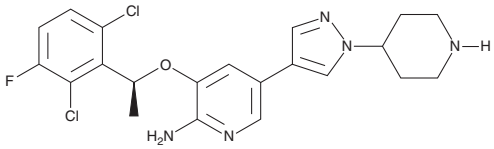
PRODUCT INFORMATION

(S)-Crizotinib

Item No. 31114

CAS Registry No.:	1374356-45-2
Formal Name:	3-[(1S)-1-(2,6-dichloro-3-fluorophenyl) ethoxy]-5-[1-(4-piperidinyl)-1H-pyrazol-4-yl]-2-pyridinamine
MF:	$C_{21}H_{22}Cl_2FN_5O$
FW:	450.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 270 nm
Supplied as:	A 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

(S)-Crizotinib is supplied as a solid. A stock solution may be made by dissolving the (S)-crizotinib in the solvent of choice, which should be purged with an inert gas. (S)-Crizotinib is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of (S)-crizotinib is approximately 0.5 mg/ml in ethanol and DMSO and approximately 5 mg/ml in DMF.

(S)-Crizotinib is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, (S)-crizotinib should first be dissolved in DMF and then diluted with the aqueous buffer of choice. (S)-Crizotinib 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

(S)-Crizotinib is an inhibitor of muT homolog 1 (MTH1; IC_{50} = 72 nM).[1] It is selective for MTH1 over a panel of 456 kinases at 1 µM. (S)-Crizotinib (30 µM) induces oxidative DNA damage and apoptosis in SGC-7901 and BGC-823 human gastric cancer cells.[2] It inhibits proliferation in a panel of 14 cancer cell lines expressing wild-type or various mutant forms of K-Ras and p53 (IC_{50}s = 0.52-7.4 µM).[1] (S)-Crizotinib (25 mg/kg, s.c.) reduces tumor growth in an SW480 mouse xenograft model.[1]

References

1. Huber, K.V.M., Salah, E., Radic, B., *et al.* Stereospecific targeting of MTH1 by (S)-crizotinib as an anticancer strategy. *Nature* **508(7495)**, 222-227 (2014).
2. Ji, J., Chen, W., Lian, W., *et al.* (S)-crizotinib reduces gastric cancer growth through oxidative DNA damage and triggers pro-survival akt signal. *Cell Death Dis.* **9(6)**, 660 (2018).

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