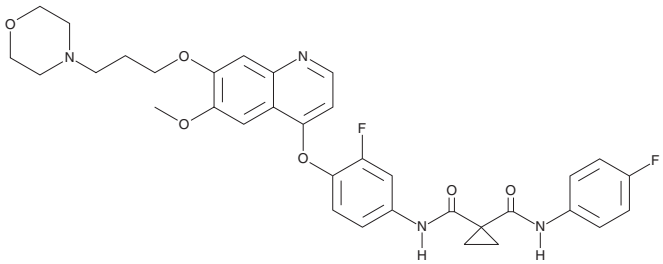
PRODUCT INFORMATION

Foretinib

Item No. 16249

CAS Registry No.:	849217-64-7
Formal Name:	N-[3-fluoro-4-[[6-methoxy-7-[3-(4-morpholinyl) propoxy]-4-quinolinyl]oxy]phenyl]-N'-(4-fluorophenyl)-1,1-cyclopropanedicarboxamide
Synonyms:	GSK1363089, XL880
MF:	$C_{34}H_{34}F_2N_4O_6$
FW:	632.7
Purity:	≥98%
UV/Vis.:	λ_{max}: 242 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

Foretinib is supplied as a crystalline solid. A stock solution may be made by dissolving the foretinib in the solvent of choice. Foretinib is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of foretinib in ethanol and DMF is approximately 25 mg/ml and approximately 16 mg/ml in DMSO.

Foretinib is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, foretinib should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Foretinib has a solubility of approximately 0.25 mg/ml in a 1:3 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Foretinib is a broad-spectrum tyrosine kinase (TK) inhibitor that targets members of the HGF and VEGF receptor TK families, as well as KIT, Flt-3, PDGFRβ, and Tie-2, at nanomolar concentrations.[1,2] It blocks HGF-induced Met phosphorylation and VEGF-induced ERK phosphorylation, preventing HGF-induced responses in tumor cells and HGV/VEGF-induced responses in endothelial cells.[1,3] Foretinib is functional *in vivo*, reducing tumor burden in an experimental model of lung metastasis.[1]

References

1. Qian, F., Engst, S., Yamaguchi, K., *et al.* Inhibition of tumor cell growth, invasion, and metastasis by EXEL-2880 (XL880, GSK1363089), a novel inhibitor of HGF and VEGF receptor tyrosine kinases. *Cancer Res.* **69(20)**, 8009-8016 (2009).
2. Davis, M.I., Hunt, J.P., Herrgard, S., *et al.* Comprehensive analysis of kinase inhibitor selectivity. *Nat. Biotechnol.* **29(11)**, 1046-1051 (2011).
3. Mughal, A., Aslam, H.M., Sheikh, A., *et al.* c-Met inhibitors. *Infect. Agent. Cancer* **8**, 13 (2013).

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