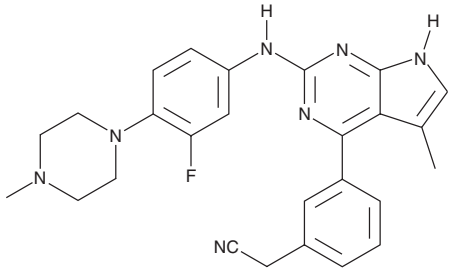
PRODUCT INFORMATION

SGI-7079

Item No. 20111

CAS Registry No.: 1239875-86-5
Formal Name: 3-[2-[[3-fluoro-4-(4-methyl-1-piperazinyl) phenyl]amino]-5-methyl-7H-pyrrolo[2,3-d] pyrimidin-4-yl]-benzeneacetonitrile
MF: $C_{26}H_{26}FN_7$
FW: 455.5
Purity: ≥98%
UV/Vis.: λ_{max}: 285 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

SGI-7079 is supplied as a crystalline solid. A stock solution may be made by dissolving the SGI-7079 in the solvent of choice, which should be purged with an inert gas. SGI-7079 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of SGI-7079 in these solvents is approximately 30 mg/ml.

SGI-7079 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SGI-7079 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. SGI-7079 has a solubility of approximately 0.25 mg/ml in a 1:3 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

SGI-7079 is an inhibitor of the receptor tyrosine kinase Axl.[1] It inhibits proliferation of inflammatory breast cancer cells (IC_{50}s = 0.43 and 0.15 µM for SUM149 and KPL-4 cells, respectively), decreases invasion, and halts the cell cycle in the G_1 phase. SGI-7079 synergistically increases the potency of erlotinib (Item No. 10483) on EGFR inhibition.[2] In a mouse xenograft model of non-small cell lung cancer, SGI-7079 dose-dependently inhibits tumor growth, an effect that is greater when used in combination with erlotinib.

References

1. Wang, X., Saso, H., Iwamoto, T., *et al.* TIG1 promotes the development and progression of inflammatory breast cancer through activation of Axl kinase. *Cancer Res.* **73(21)**, 6516-6525 (2013).
2. Byers, L.A., Diao, L., Wang, J., *et al.* An epithelial-mesenchymal transition gene signature predicts resistance to EGFR and PI3K inhibitors and identifies Axl as a therapeutic target for overcoming EGFR inhibitor resistance. *Clin. Cancer Res.* **19(1)**, 279-290 (2013).

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