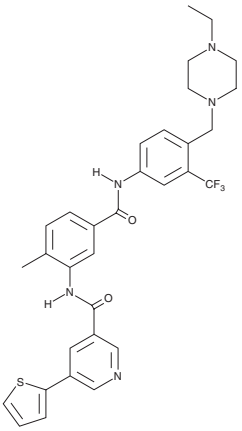
PRODUCT INFORMATION

ALW-II-41-27

Item No. 25275



CAS Registry No.:	1186206-79-0
Formal Name:	N-[4-[(4-ethyl-1-piperazinyl)methyl]-3-(trifluoromethyl)phenyl]-4-methyl-3-[[[5-(2-thienyl)-3-pyridinyl]carbonyl]amino]-benzamide
MF:	$C_{32}H_{32}F_3N_5O_2S$
FW:	607.7
Purity:	≥95%
UV/Vis.:	λ_{max}: 277 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

ALW-II-41-27 is supplied as a crystalline solid. A stock solution may be made by dissolving the ALW-II-41-27 in the solvent of choice, which should be purged with an inert gas. ALW-II-41-27 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of ALW-II-41-27 in ethanol is approximately 0.5 mg/ml and approximately 10 mg/ml in DMSO and DMF.

ALW-II-41-27 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, ALW-II-41-27 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. ALW-II-41-27 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

ALW-II-41-27 is a multi-kinase inhibitor that inhibits EphB2, EphA3, Kit, FMS, VEGFR2/KDR, FLT1, FGR, Src, Lyn, BMX, and Bcr-Abl in tyrosine kinase-transformed Ba/F3 cells (EC_{50}s = <500 nM).[1] ALW-II-41-27 also inhibits DDR2 and Src (IC_{50}s = 51 and 14 nM, respectively) as well as wild-type and mutant RET kinases (IC_{50}s = 24.7, 94.2, and 15.8 nM for wild-type, RET^{V804L}, and RET^{V804M}, respectively).[2,3] It reduces growth of NCI-H2286 and HCC-366 cancer cells (GI_{50}s = 0.51 and 0.65 µM, respectively) and RAT1 cells transformed by RET^{C634R} or RET^{M918T} (IC_{50}s = 44 and 56 nM, respectively). ALW-II-41-27 also inhibits growth of MDA-MB-231 breast cancer cells in a concentration-dependent manner and inhibits tumor growth *in vivo* in a mouse patient-derived xenograft (PDX) model of EphA2-overexpressing triple-negative breast cancer (TNBC).[4]

References

1. Choi, Y., Syeda, F., Walker, J.R., *et al.* Discovery and structural analysis of Eph receptor tyrosine kinase inhibitors. *Bioorg. Med. Chem. Lett.* **19(15)**, 4467-4470 (2009).
2. Terai, H., Tan, L., Beauchamp, E.M., *et al.* Characterization of DDR2 inhibitors for the treatment of DDR2 mutated nonsmall cell lung cancer. *ACS Chem. Biol.* **10(12)**, 2687-2696 (2015).
3. Moccia, M., Liu, Q., Guida, T., *et al.* Identification of novel small molecule inhibitors of oncogenic RET kinase. *PLoS One* **10(6)**, e0128364 (2015).
4. Song, W., Hwang, Y., Youngblood, V.M., *et al.* Targeting EphA2 impairs cell cycle progression and growth of basal-like/triple-negative breast cancers. *Oncogene* **36(40)**, 5620-5630 (2017).

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