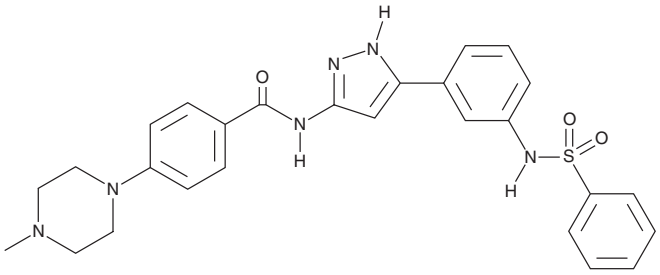
PRODUCT INFORMATION

BPR1J-097

Item No. 29805

CAS Registry No.:	1327167-19-0
Formal Name:	4-(4-methyl-1-piperazinyl)-N-[5-[3-[(phenylsulfonyl)amino]phenyl]-1H-pyrazol-3-yl]-benzamide
MF:	$C_{27}H_{28}N_6O_3S$
FW:	516.6
Purity:	≥95%
UV/Vis.:	λ_{max}: 298 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

BPR1J-097 is supplied as a crystalline solid. A stock solution may be made by dissolving the BPR1J-097 in the solvent of choice, which should be purged with an inert gas. BPR1J-097 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of BPR1J-097 in these solvents is approximately 10 and 30 mg/ml, respectively.

BPR1J-097 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, BPR1J-097 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. BPR1J-097 has a solubility of approximately 0.14 mg/ml in a 1:6 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

BPR1J-097 is an inhibitor of FMS-related tyrosine kinase 3 (FLT3; IC_{50} = 11 nM).[1] It is selective for FLT3 over FLT1, KDR, Aurora A, and Aurora B kinases (IC_{50}s = 211, 129, 340, and 876 nM, respectively). BPR1J-097 inhibits FLT3 activity and phosphorylation of STAT5 in MV4-11 acute myeloid leukemia (AML) cells containing the FLT3 activating mutant FLT3-ITD (IC_{50}s = ~10 and 1 nM, respectively). It inhibits the growth of FLT3-dependent MOLM-13 and MV4-11 AML cells (GI_{50}s = 21 and 46 nM, respectively) but not *FLT3*$^{-/-}$ U937 and K562 cells (GI_{50}s = >20,000 nM for both). BPR1J-097 (25 mg/kg) inhibits tumor growth in a MOLM-13 mouse xenograft model.

Reference

1. Lin, W.-H., Jiaang, W.-T., Chen, C.-W., *et al.* BPR1J-097, a novel FLT3 kinase inhibitor, exerts potent inhibitory activity against AML. *Br. J. Cancer* **106(3)**, 475-481 (2012).

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