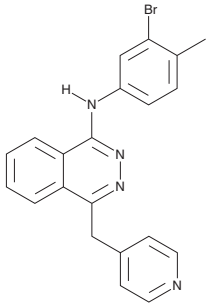
PRODUCT INFORMATION

NVP-ACC789

Item No. 23458

CAS Registry No.:	300842-64-2
Formal Name:	N-(3-bromo-4-methylphenyl)-4-(4-pyridinylmethyl)-1-phthalazinamine
MF:	$C_{21}H_{17}BrN_4$
FW:	405.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 215, 337 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

NVP-ACC789 is supplied as a crystalline solid. A stock solution may be made by dissolving the NVP-ACC789 in the solvent of choice, which should be purged with an inert gas. NVP-ACC789 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of NVP-ACC789 in these solvents is approximately 0.2, 1, and 0.5 mg/ml, respectively.

NVP-ACC789 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, NVP-ACC789 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. NVP-ACC789 has a solubility of approximately 0.09 mg/ml in a 1:10 solution of DMF:PBS (pH 7) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

NVP-ACC789 is an inhibitor of VEGF receptor tyrosine kinases (VEGFRs; IC_{50}s = 0.38, 0.02, 0.18, and 0.23 µM for human VEGFR1, 2, 3, and mouse Vegfr2, respectively).[1] It is selective for VEGFRs over FGFRs and PDGFRα (IC_{50}s = >10 µM) but has activity at PDGFRβ (IC_{50} = 1.4 µM) in an enzyme assay. NVP-ACC789 inhibits VEGF-induced VEGFR2 autophosphorylation (IC_{50} = 11.5 nM in CHO cells transfected with the human receptor). It also inhibits VEGF- and bFGF-induced angiogenesis and cell migration of BAE and BME cells. *In vivo*, NVP-ACC789 blocks bFGF- and VEGF-induced angiogenesis (ED_{50}s = 9 and 26 mg/kg, respectively) in a mouse model of growth factor-induced angiogenesis.

Reference

1. Tille, J.C., Wood, J., Mandriota, S.J., *et al.* Vascular endothelial growth factor (VEGF) receptor-2 antagonists inhibit VEGF- and basic fibroblast growth factor-induced angiogenesis in vivo and in vitro. *J. Pharmacol. Exp. Ther.* **299(3)**, 1073-1085 (2001).

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