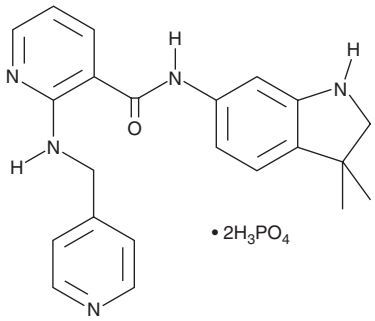
PRODUCT INFORMATION

AMG 706

Item No. 17671

CAS Registry No.:	857876-30-3
Formal Name:	N-(2,3-dihydro-3,3-dimethyl-1H-indol-6-yl)-2-[(4-pyridinylmethyl)amino]-3-pyridinecarboxamide, diphosphate
Synonym:	Motesanib
MF:	$C_{22}H_{23}N_5O \bullet 2H_3PO_4$
FW:	569.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 253, 333 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

AMG 706 is supplied as a crystalline solid. A stock solution may be made by dissolving the AMG 706 in the solvent of choice, which should be purged with an inert gas. AMG 706 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of AMG 706 in these solvents is approximately 3 and 1 mg/ml, respectively.

AMG 706 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, AMG 706 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. AMG 706 has a solubility of approximately 0.1 mg/ml in a 1:9 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

AMG 706 is a multikinase inhibitor that predominantly targets receptor tyrosine kinases, including VEGFR1, VEGFR2, VEGFR3, c-Kit, PDGFR, and RET (IC_{50}s = 2, 3, 6, 8, 84, and 59 nM, respectively).[1] It also potently inhibits CSF1R and ZAK (IC_{50}s = 5.6 and 8 nM, respectively), as well as several mutants of c-Kit.[2] AMG 706 inhibits the proliferation of human endothelial cells induced by VEGF but not FGF.[1] Oral administration of AMG 706 blocks VEGF-induced angiogenesis in the rat corneal model and induces regression of established A431 xenografts in mice.[1]

References

1. Polverino, A., Coxon, A., Starnes, C., *et al.* AMG 706, an oral, multikinase inhibitor that selectively targets vascular endothelial growth factor, platelet-derived growth factor, and kit receptors, potently inhibits angiogenesis and induces regression in tumor xenografts. *Cancer Res.* **66(17)**, 8715-8721 (2006).
2. Davis, M.I., Hunt, J.P., Herrgard, S., *et al.* Comprehensive analysis of kinase inhibitor selectivity. *Nat. Biotechnol.* **29(11)**, 1046-1051 (2011).

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