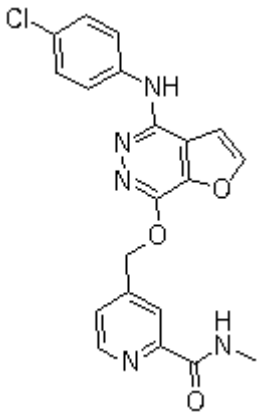
Product Introduction

Telatinib

Telatinib is a potent inhibitor of VEGFR2/3**,** c-Kit **and PDGFRβ with** IC50 **of 6 nM/4 nM, 1 nM and 15 nM, respectively. Phase 2.**

Technical Data:

Molecular Weight (MW):	409.83	
Formula:	$C_{20}H_{16}ClN_5O_3$	
Solubility (25 ℃) * <1 mg/ml means slightly soluble or insoluble:	DMSO 82 mg/mL	
	Water <1 mg/mL	
	Ethanol 1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	332012-40-5	

Biological Activity

Telatinib has 0.66, 0.17, and 2.5 times higher IC50 values for VEGFR3, c-Kit, and PDGFRβ than VEGFR2, respectively, while Vatalanib exhibits 18, 20, and 16 times higher IC50 values, respectively, indicating that Telatinib has potential benefit over Vatalanib. Telatinib inhibits VEGFR2 autophosphorylation in a whole-cell assay with an IC50 of 19 nM, suppresses VEGF-dependent proliferation of human umbilical vein endothelial cells with an IC50 of 26 nM, and blocks PDGF-stimulated growth of human aortic smooth muscle cells with an IC50 of 249 nM. [3] Telatinib displays little inhibitory activity against the Raf kinase pathway, epidermal growth factor receptor family, the fibroblast growth factor receptor (FGFR) family, and

the Tie-2 receptor. [4]

Given that tumor development and metastasis are ascribed to deregulated VEGFR signal pathway, Telatinib treatment significantly inhibits tumor growth and metastasis by blocking the VEGFR signaling and subsequently tumor angiogenesis. In addition to the significant inhibition of tumor angiogenesis, Telatinib treatment induces a significant decrease in endothelium-dependent and endothelium-independent vasodilation, as well as reduction in capillary density, leading to an increase in systolic and diastolic blood pressure. [1] Administration of Telatinib as a single agent exhibits a potent anti-tumor activity in multiple human tumor xenograft models including MDA-MB-231 breast cancer, Colo-205 colon cancer, DLD-1 colon cancer, and H460 non-small cell lung cancer, as well as pancreatic and prostate carcinoma in a dose-dependent manner. [2]

References

[1] Steeghs N, et al. Clin Cancer Res, 2008, 14(11), 3470-3476.
[2] Strumberg D, et al. Br J Cancer, 2008, 99(10), 1579-1585.
[3] Eskens FA, et al. J Clin Oncol, 2009, 27(25), 4169-4176.
[4] Langenberg MH, et al. Clin Cancer Res, 2010, 16(7), 2187-2197.

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