Ansornitinib

Cat. No.: HY-147243

CAS No.: 1448874-96-1

Molecular Formula: $C_{30}H_{32}N_6O_4$

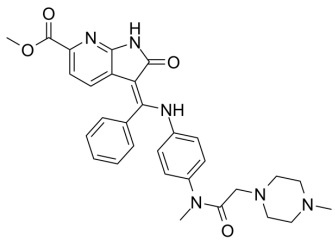
Molecular Weight: 540.61

Target: VEGFR; PDGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 40 mg/mL (73.99 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8498 mL	9.2488 mL	18.4976 mL
	5 mM	0.3700 mL	1.8498 mL	3.6995 mL
	10 mM	0.1850 mL	0.9249 mL	1.8498 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 5 mg/mL (9.25 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 5 mg/mL (9.25 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Ansornitinib is an orally active dual kinase inhibitor that inhibits platelet-derived growth factor receptor (PDGFR) and vascular endothelial growth factor receptor (VEGFR2). Ansornitinib can be used as an antifibrotic agent in lung, liver, kidney, and gastrointestinal fibrotic diseases[1].

In Vitro

Ansornitinib (compound I) (0.1 nM-10 μM, 2 h) can significantly inhibit the phosphorylation of PDGFRβ in human hepatic stellate cells and KDR phosphorylation in human umbilical vein endothelial cells (HUVEC)[1].
Ansornitinib (compound I) (0.1-13 μM, 48 h) can induce a decrease in the expression of many different inflammation-related markers, such as macrophage colony-stimulating factor (M-CSF), soluble interleukin 8, and fibrosis-related markers, such as N-calmodulin, α-SMA, etc., in a dose-dependent manner[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Ansornitinib (compound I) (25 mg/kg, p.o., twice a day, 4 weeks) can reduce fibrosis in TGFβ positive female mice[1].
Ansornitinib (compound I) (5-45 mg/kg, p.o., twice a day, 4 days) can reduce inflammatory bowel disease (IBD) in TNBS-induced IBD/acute colitis male CD-1 mice[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	8-10 weeks TGFβ positive female mice[1]
Dosage:	25 mg/kg
Administration:	Oral administration; twice a day; 4 weeks
Result:	Reduced lung fibrosis score, lung hydroxyproline levels and αSMA, a marker of early pulmonary fibrosis.

Animal Model:	TNBS-induced IBD/acute colitis male CD-1 mice[1]
Dosage:	5, 15, 45 mg/kg
Administration:	Oral administration; twice a day; 4 days
Result:	Improved colonic histology and reduced the TNBS-induced loss of cupped cells at 15 mg/kg and 45 mg/kg significantly. Reduced the expression level of myeloperoxidase (MPO) at 5 mg/kg and 15 mg/kg significantly while there was no statistical difference at 45 mg/kg.

Animal Model:	Male Sprague Dawley rats[1]
Dosage:	30 mg/kg
Administration:	Intravenous injection; once
Result:	The pharmacokinetic parameters of Ansornitinib (compound I)

Parameter	Ansornitinib (compound I)
$t_{1/2}$	4.1 h
T_{max}	0.518 h
C_{max}	7860 ng/mL
Clearance	173 mL/kg/min
steady-state volume	18.2 L/kg
$AUC_{0\text{-}last}$	3180 ng/mL*h
$AUC_{0\text{-}inf\_obs}$	3200 ng/mL*h

REFERENCES

[1]. Shakil ASLAM, et al. Reducing fibrosis and treating related diseases, disorders, and conditions. WO2022006278

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