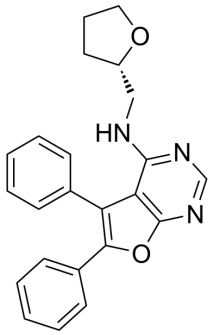
AIM-100

Cat. No.:	HY-15290
CAS No.:	873305-35-2
Molecular Formula:	$C_{23}H_{21}N_3O_2$
Molecular Weight:	371.43
Target:	Ack1
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (134.61 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6923 mL	13.4615 mL	26.9230 mL
	5 mM	0.5385 mL	2.6923 mL	5.3846 mL
	10 mM	0.2692 mL	1.3461 mL	2.6923 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: ≥ 2.5 mg/mL (6.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.73 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AIM-100 is a potent and selective Ack1 inhibitor with an IC_{50} of 21.58 nM. AIM-100 also inhibits Tyr^{267} phosphorylation. AIM-100 does not inhibits other kinases including PI3-kinase and AKT subfamily members. AIM-100 has an anticancer effect[1][2].

IC_{50} & Target

IC50: 21.58 nM (Ack1)[2]

In Vitro

AIM-100 (2-10 μM; 48 hours) treatment not only inhibits Ack1 activation but also suppresses AKT tyrosine phosphorylation, leading to cell cycle arrest in the G1 phase. AIM-100 not only inhibits Ack1/AKT Tyr-phosphorylation but also suppressed

	growth of cell lines derived from pancreatic, breast, and lung tumors[1]. The Ack1 inhibitor AIM-100 not only inhibited Ack1 activity but also was able to suppress AR Tyr^{267} phosphorylation and its recruitment to the ataxia-telangiectasia mutated kinase (ATM) enhancer[2]. AIM-100 is able to suppress pTyr^{267}-AR phosphorylation, binding of androgen receptor (AR) to PSA, NKX3.1, and TMPRSS2 promoters, and inhibit AR transcription activity[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In male nude castrated mice, AIM-100 (4 mg/kg) suppresses growth of radioresistant castration-resistant prostate cancer (CRPC) xenograft tumors by decreasing ataxia-telangiectasia mutated kinase (ATM) expression[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Mol Med. 2023 Jan 16;29(1):6.
- Cell Biochem Funct. 2020 Jul;38(5):642-650.
- Oncotarget. 2015 Dec 1;6(38):40622-41.

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REFERENCES

[1]. Mahajan K, et al. Ack1 tyrosine kinase activation correlates with pancreatic cancer progression. Am J Pathol. 2012 Apr;180(4):1386-93.

[2]. Mahajan K, et al. Ack1-mediated androgen receptor phosphorylation modulates radiation resistance in castration-resistant prostate cancer. J Biol Chem. 2012 Jun 22;287(26):22112-22.

[3]. Mahajan K, et al. Effect of Ack1 tyrosine kinase inhibitor on ligand-independent androgen receptor activity. Prostate. 2010 Sep 1;70(12):1274-85.

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