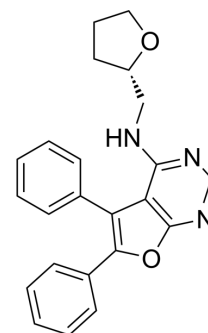


AIM-100

Cat. No.:	HY-15290		
CAS No.:	873305-35-2		
Molecular Formula:	C ₂₃ H ₂₁ N ₃ O 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)					
		Solvent Concentration	Mass	1 mg	5 mg	10 mg
	Preparing Stock Solutions	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	<ol style="list-style-type: none"> 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 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 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 inhibit other kinases including PI3-kinase and AKT subfamily members. AIM-100 has an anticancer effect ^{[1][2]} .
IC₅₀ & Target	IC ₅₀ : 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 Tyr²⁶⁷ phosphorylation and its recruitment to the ataxia-telangiectasia mutated kinase (ATM) enhancer^[2].

AIM-100 is able to suppress pTyr²⁶⁷-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.

Caution: Product has not been fully validated for medical applications. For research use only.

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