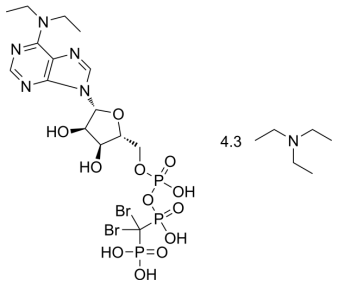
ARL67156 triethylamine

Cat. No.:	HY-103265D
Molecular Formula:	$C_{15}H_{24}Br_2N_5O_{12}P_3\cdot(_{4\cdot3}C_6H_{15}N)$
Molecular Weight:	1154.23
Target:	Phosphatase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

Methanol : 125 mg/mL (108.30 mM; Need ultrasonic)
DMSO : 100 mg/mL (86.64 mM; Need ultrasonic)
H_2O : 100 mg/mL (86.64 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.8664 mL	4.3319 mL	8.6638 mL
	5 mM	0.1733 mL	0.8664 mL	1.7328 mL
	10 mM	0.0866 mL	0.4332 mL	0.8664 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 (2.17 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.17 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ARL67156 (FPL 67156) triethylamine is a selective small is a selective samll molecular inhibitor, targeting to ecto-ATPase, CD39, and CD73. ARL67156 triethylamine is also a competitive inhibitor of NTPDase1 (CD39), NTPDase3 and NPP1, with K_is of 11, 18 and 12?μM, respectively. ARL67156 triethylamine can be used in the research of disease like calcific aortic valve disease, asthma[1][2].

IC_{50} & Target

Ki: 11 μM (NTPDase1), 18 μM (NTPDase3), 12 μM (NPP1)[1]; 0.97 μM (CD39), 0.45 μM (CD73)[8]

In Vitro

ARL67156 triethylamine (1-100 μM) potentiates neurogenic contractions in a concentration-dependent manner[4].
?ARL67156 triethylamine (10 μg/mL, 24 h) increases the surface expression of CXCR3 on ATP-treated HMC-1 cells[5].

?ARL67156 triethylamine (30 μM, 5s) potentiates the norepinephrine release promoted by ATP in guinea pig heart synaptosomes[6].
?ARL67156 triethylamine (100 μM, 4h) significantly decreases HIV-1replication in macrophages[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

ARL67156 triethylamine (1.1 μg/kg/day, administered with osmotic pumps implanted subcutaneously, for 28 days) prevents the development of calcific aortic valve disease in Warfarin (HY-B0687)-treated rats[2].
?ARL67156 triethylamine (intraperitoneal injection, 2?mg/kg) prevents the increase of serum adenosine concentration induced by Fructose 1,6-bisphosphate (FBP)[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Warfarin-induced mineralization rat model[2]
Dosage:	1.1 μg/kg/day
Administration:	Administered with osmotic pumps implanted subcutaneously, for 28 days
Result:	Prevented the development of aortic stenosis by lowering the level of apoptosis and mineralization of the aortic valve/aorta. Normalized the level of pAkt (an important kinase involved in the survival pathway).

Animal Model:	C57BL/6 mice[3]
Dosage:	2 mg/kg
Administration:	Intraperitoneal injection, 1 h before administration of FBP (100 mg/kg)
Result:	Completely abolished the anti-inflammatory effects of FBP (observed by the neutrophil infiltration, hyperalgesia and oedema of the joint).

CUSTOMER VALIDATION

- Neuron. 2021 Dec 17;S0896-6273(21)00988-0.
- Front Immunol. 04 October 2022.

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REFERENCES

[1]. évesque SA, et al. Specificity of the ecto-ATPase inhibitor ARL 67156 on human and mouse ectonucleotidases. Br J Pharmacol. 2007 Sep;152(1):141-50.

[2]. Nancy Côté, et al. Inhibition of Ectonucleotidase With ARL67156 Prevents the Development of Calcific Aortic Valve Disease in Warfarin-Treated Rats. Eur J Pharmacol. 2012 Aug 15;689(1-3):139-46.

[3]. Flávio P Veras, et al. Fructose 1,6-bisphosphate, a high-energy intermediate of glycolysis, attenuates experimental arthritis by activating anti-inflammatory adenosinergic pathway. Sci Rep. 2015 Oct 19;5:15171.

[4]. C Kennedy, et al. ATP as a co-transmitter with noradrenaline in sympathetic nerves--function and fate. Ciba Found Symp. 1996;198:223-35; discussion 235-8.

[5]. Ya-Dong Gao, et al. Th2 cytokine-primed airway smooth muscle cells induce mast cell chemotaxis via secretion of ATP. J Asthma. 2014 Dec;51(10):997-1003.

[6]. Casilde Sesti, et al. EctoNucleotidase in cardiac sympathetic nerve endings modulates ATP-mediated feedback of norepinephrine release. J Pharmacol Exp Ther. 2002 Feb;300(2):605-11.

[7]. Julieta Schachter, et al. Inhibition of ecto-ATPase activities impairs HIV-1 infection of macrophages. Immunobiology. 2015 May;220(5):589-96.

[8]. Schäkel L, et al. Nucleotide Analog ARL67156 as a Lead Structure for the Development of CD39 and Dual CD39/CD73 Ectonucleotidase Inhibitors. Front Pharmacol. 2020 Sep 8;11:1294.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA