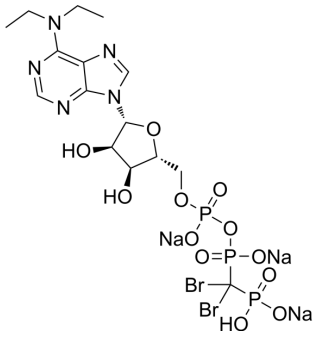
ARL67156 trisodium

Cat. No.:	HY-103265
CAS No.:	1021868-83-6
Molecular Formula:	$C_{15}H_{21}Br_2N_5Na_3O_{12}P_3$
Molecular Weight:	785.05
Target:	Phosphatase
Pathway:	Metabolic Enzyme/Protease
Storage:	-20°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 25 mg/mL (31.85 mM; Need ultrasonic)
DMSO : < 1 mg/mL (ultrasonic) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2738 mL	6.3690 mL	12.7380 mL
5 mM	0.2548 mL	1.2738 mL	2.5476 mL
10 mM	0.1274 mL	0.6369 mL	1.2738 mL

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

BIOLOGICAL ACTIVITY

Description

ARL67156 (FPL 67156) trisodium is a selective small molecular inhibitor, targeting to ecto-ATPase, CD39, and CD73. ARL67156 trisodium is also a competitive inhibitor of NTPDase1 (CD39), NTPDase3 and NPP1, with K_is of 11, 18 and 12?μM, respectively. ARL67156 trisodium can be used in the research of 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 trisodium (1-100 μM) potentiates neurogenic contractions in a concentration-dependent manner[4].
?ARL67156 trisodium (10 μg/mL, 24 h) increases the surface expression of CXCR3 on ATP-treated HMC-1 cells[5].
?ARL67156 trisodium (30 μM, 5s) potentiates the norepinephrine release promoted by ATP in guinea pig heart synaptosomes [6].
?ARL67156 trisodium (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 trisodium (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 trisodium (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.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. 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.

[2]. 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.

[3]. 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.

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

[5]. 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.

[6]. Lévesque 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.

[7]. 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.

[8]. 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.

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

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