Product Data Sheet

Product Name: ARL 67156 trisodium salt
Cat. No.: GC17225

Chemical Properties

Cas. No.	1021868-83-6		
SMILES	CCN(C1=C(N=CN2[C@@]3([H])[C@@](O)([H])[C@@](O)([H])[C@@](O3)([H])COP(O)(OP(C(Br)(P([O-])([O-])=O)Br)([O-])=O)=O)C2=NC=N1)CC.[Na+].[Na+].[Na+]		
Formula	$C_{15}H_{21}Br_2N_5O_{12}P_3.3Na$	M.Wt	785.06
Solubility	<15.78mg/ml in Water	Storage	Desiccate at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
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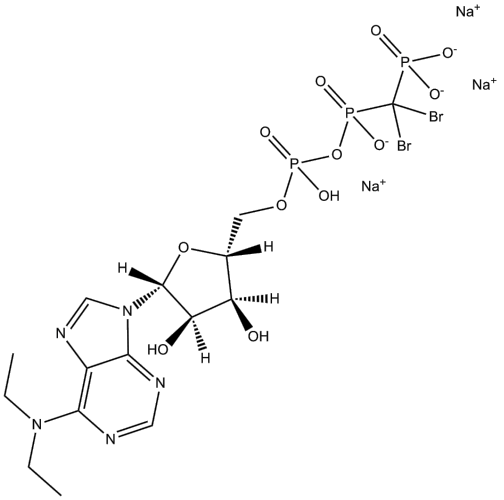
Structure



Protocol

Cell experiment [1]:

Cell lines	HMC-1 cell
Preparation Method	HMC-1 cells were placed into 6-well plates. After reaching 70% confluence, the cells were stimulated with ATP(10mM) in the absence or presence of exogenous CD39 or ARL 67156 trisodium salt (10μg/ml).

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Reaction Conditions	10μg/ml; 24h
Applications	CD39 decreased the surface expression of CXCR3 on both control and ATP-treated HMC-1 cells. ARL 67156 trisodium salt increased the surface expression of CXCR3 on ATP-treated HMC-1 cells.

Animal experiment [2]:

Animal models	Male C57BL/6 mice
Preparation Method	Mice were pre-treated with ARL 67156 trisodium salt(2mg/kg) intraperitoneally 1h before treatment with FBP (Fructose 1,6-bisphosphate; 100mg/kg) and blood was withdrawn from by cardiac punction 24h later. Then, serum adenosine quantification was performed.
Dosage form	2mg/kg; i.p.
Applications	ARL 67156 trisodium salt completely prevented the increase of serum adenosine concentration induced by FBP

References:
[1] Gao YD, Cao J, Li P, Huang G, Yang J. Th2 cytokine-primed airway smooth muscle cells induce mast cell chemotaxis via secretion of ATP. *J Asthma*. 2014;51(10):997-1003.
[2] Veras FP, Peres RS, Saraiva AL, et al. Fructose 1,6-bisphosphate, a high-energy intermediate of glycolysis, attenuates experimental arthritis by activating anti-inflammatory adenosinergic pathway. *Sci Rep*. 2015;5:15171.

Background

ARL 67156 trisodium salt is a selective inhibitor of ecto-ATPases [1]. The Ki values of ARL 67156 trisodium salt against human NTPDase1(CD39), NTPDase3, and NPP1 were 11±3, 18±4, and

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12±3μM, respectively[1].

In vitro, treatment with 10μg/ml ARL 67156 trisodium salt for 24 hours increased the surface expression of CXCR3 on ATP-treated HMC-1 cells[2]. Treatment with 100μM ARL 67156 trisodium salt for 4 hours reduced HIV-1 replication in macrophages[3]. Treatment with 30μM ARL 67156 trisodium salt for 5 seconds enhanced ATP-promoted norepinephrine release in the synaptosome of guinea pig hearts[4].

In vivo, administration of ARL 67156 trisodium salt(2mg/kg; i.p.) for 24 hours completely prevented the FBP-induced increase in serum adenosine concentration in mice[5].

References:

[1] Lévesque SA, Lavoie EG, Lecka J, Bigonnesse F, Sévigny J. Specificity of the ecto-ATPase inhibitor ARL 67156 on human and mouse ectonucleotidases. *Br J Pharmacol*. 2007;152(1):141-150.
[2] Gao YD, Cao J, Li P, Huang G, Yang J. Th2 cytokine-primed airway smooth muscle cells induce mast cell chemotaxis via secretion of ATP. *J Asthma*. 2014;51(10):997-1003.
[3] Schachter J, Delgado KV, Barreto-de-Souza V, Bou-Habib DC, Persechini PM, Meyer-Fernandes JR. Inhibition of ecto-ATPase activities impairs HIV-1 infection of macrophages. *Immunobiology*. 2015;220(5):589-596.
[4] Sesti C, Broekman MJ, Drosopoulos JH, Islam N, Marcus AJ, Levi R. EctoNucleotidase in cardiac sympathetic nerve endings modulates ATP-mediated feedback of norepinephrine release. *J Pharmacol Exp Ther*. 2002;300(2):605-611.
[5]Veras FP, Peres RS, Saraiva AL, et al. Fructose 1,6-bisphosphate, a high-energy intermediate of glycolysis, attenuates experimental arthritis by activating anti-inflammatory adenosinergic pathway. *Sci Rep*. 2015;5:15171.

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