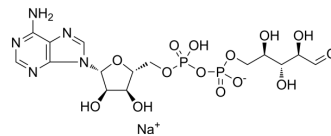


Adenosine 5'-diphosphoribose sodium

Cat. No.:	HY-100973A
CAS No.:	68414-18-6
Molecular Formula:	C ₁₅ H ₂₂ N ₅ NaO ₁₄ P ₂
Molecular Weight:	581.3
Target:	TRP Channel; Autophagy
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Autophagy
Storage:	-20°C, sealed storage, away from moisture * In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	H ₂ O : 125 mg/mL (215.04 mM; Need ultrasonic)					
	DMSO : 25 mg/mL (43.01 mM; ultrasonic and warming and heat to 80°C)					
	Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
		Concentration				
		1 mM		1.7203 mL	8.6014 mL	17.2028 mL
5 mM			0.3441 mL	1.7203 mL	3.4406 mL	
	10 mM		0.1720 mL	0.8601 mL	1.7203 mL	
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (172.03 mM); Clear solution; Need ultrasonic					

BIOLOGICAL ACTIVITY

Description	Adenosine 5'-diphosphoribose sodium (ADP ribose sodium) is a nicotinamide adenine nucleotide (NAD ⁺) metabolite. Adenosine 5'-diphosphoribose sodium is the most potent and primary intracellular Ca ²⁺ -permeable cation TRPM2 channel activator. Adenosine 5'-diphosphoribose sodium also can enhance autophagy ^{[1][2]} .
IC₅₀ & Target	TRPM2 channel ^{[1][2]} Autophagy ^[1]
In Vitro	In mouse embryonic fibroblasts (MEFs), H ₂ O ₂ treatment demonstrates that the activation of poly(ADP-ribose) (PAR) polymerase-1 (PARP-1) produced Adenosine 5'-diphosphoribose (ADP ribose), which is an activating signal for TRPM2 channels, thereby promoting Ca ²⁺ elevation through extracellular Ca ²⁺ influx and (or) lysosomal Ca ²⁺ release. This process eventually activates early or late autophagy in response to different degrees of oxidative stress ^{[1][1]} . ?TRPM2 channels are activated by binding of Adenosine 5'-diphosphoribose (ADP ribose) to the intracellular NUDT9-homology (NUDT9-H) domain unique to TRPM2 and located at its C terminus. In addition to ADPR, intracellular Ca ²⁺ is an

essential coactivator: TRPM2 channels open only in the combined presence of both ligands^[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

- [1]. Zhang DX, et al. The potential regulatory roles of NAD(+) and its metabolism in autophagy. *Metabolism*. 2016 Apr;65(4):454-62.
- [2]. Tóth B, et al. Pore collapse underlies irreversible inactivation of TRPM2 cation channel currents. *Proc Natl Acad Sci U S A*. 2012 Aug 14;109(33):13440-5.
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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