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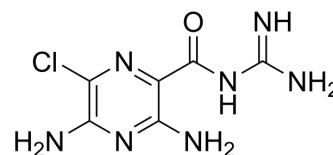
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Amiloride

Cat. No.:	HY-B0285		
CAS No.:	2609-46-3		
Molecular Formula:	C ₆ H ₈ ClN ₇ O		
Molecular Weight:	229.63		
Target:	Sodium Channel; TRP Channel; Apoptosis		
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (435.48 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	4.3548 mL	21.7742 mL	43.5483 mL
		5 mM	0.8710 mL	4.3548 mL	8.7097 mL
10 mM		0.4355 mL	2.1774 mL	4.3548 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 (10.89 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (10.89 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (10.89 mM); Clear solution 				

BIOLOGICAL ACTIVITY

Description	Amiloride (MK-870) is an inhibitor of both epithelial sodium channel (ENaC ^[1]) and urokinase-type plasminogen activator receptor (uTPA ^[2]). Amiloride is a blocker of polycystin-2 (PC2; TRPP2 ^[3]) channel.
IC₅₀ & Target	ENaC ^[1] ; uTPA ^[2] ; polycystin-2(TRPP2) ^[3]
In Vitro	Amiloride blocks δβγ channels with an IC ₅₀ of 2.6 μM (58, 71, 75, 134, 148). The K _i of amiloride for δβγ ENaC is 26-fold that of αβγ channels (0.1 μM for αβγ ENaC). Amiloride blockade of δβγ ENaC is much more voltage dependent compared with the

$\alpha\beta\gamma$ channel. The K_i of amiloride for $\delta\alpha\beta\gamma$ channels is 920 and 13.7 μM at -120 and +80 mV, respectively, which significantly differs from that of both $\alpha\beta\gamma$ and $\delta\beta\gamma$ channels^[1]. Amiloride is a relatively selective inhibitor of the epithelial sodium channel (ENaC) with an IC_{50} (the concentration required to reach 50% inhibition of an ion channel) in the concentration range of 0.1 to 0.5 μM . Amiloride is a relatively poor inhibitor of the the Na^+/H^+ exchanger (NHE) with an IC_{50} as low as 3 μM in the presence of a low external $[\text{Na}^+]$ but as high as 1 mM in the presence of a high $[\text{Na}^+]$. Amiloride is an even weaker inhibitor of the $\text{Na}^+/\text{Ca}^{2+}$ exchanger (NCX), with an IC_{50} of 1 mM. Amiloride (1 μM) and submicromolar doses of Benzamil (30 nM), doses known to inhibit the ENaC, inhibit the myogenic vasoconstriction response to increasing perfusion pressure by blocking the activity of ENaC proteins. Amiloride completely inhibits Na^+ influx in doses known to be relatively specific for ENaC (1.5 μM) in vascular smooth muscle cells (VSMC)^[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Amiloride (1 mg/kg/day) subcutaneously is found to reverse the initial increases in collagen deposition and prevent any further increases in the DOCA-salt hypertensive rat. Amiloride delays the onset of proteinuria and improved brain and kidney histologic scores in the saline-drinking, stroke-prone spontaneously hypertensive rats (SHRSP) compared with controls. Amiloride antagonizes or prevents actions of aldosterone in these cells and in cardiovascular and renal tissues in animals with salt-dependent forms of hypertension^[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Metab. 2022 Dec 6;34(12):2018-2035.e8.
- ACS Nano. 2023 Apr 14.
- Adv Sci (Weinh). 2023 Dec 14:e2307880.
- J Am Chem Soc. 2018 Dec 12;140(49):17234-17240.
- Biomaterials. 2022 May;284:121529.

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REFERENCES

- [1]. Ji, H.L., et al. delta ENaC: a novel divergent amiloride-inhibitable sodium channel. Am J Physiol Lung Cell Mol Physiol, 2012. 303(12): p. L1013-26.
- [2]. Teiwes J, et al. Epithelial sodium channel inhibition in cardiovascular disease. A potential role for amiloride. Am J Hypertens. 2007 Jan;20(1):109-17.
- [3]. Giamarchi A, et al. A polycystin-2 (TRPP2) dimerization domain essential for the function of heteromeric polycystin complexes. EMBO J. 2010 Apr 7;29(7):1176-91.

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

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