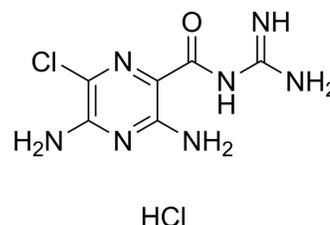


## Amiloride hydrochloride

<b>Cat. No.:</b>	HY-B0285A
<b>CAS No.:</b>	2016-88-8
<b>Molecular Formula:</b>	C <sub>6</sub> H <sub>9</sub> Cl <sub>2</sub> N <sub>7</sub> O
<b>Molecular Weight:</b>	266.09
<b>Target:</b>	Sodium Channel; Apoptosis; TRP Channel
<b>Pathway:</b>	Membrane Transporter/Ion Channel; Apoptosis; Neuronal Signaling
<b>Storage:</b>	4°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

DMSO : ≥ 100 mg/mL (375.81 mM)  
 H<sub>2</sub>O : 7.14 mg/mL (26.83 mM; Need ultrasonic)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	3.7581 mL	18.7906 mL	37.5813 mL
	5 mM	0.7516 mL	3.7581 mL	7.5163 mL
	10 mM	0.3758 mL	1.8791 mL	3.7581 mL

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

#### In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
Solubility: ≥ 2.5 mg/mL (9.40 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.5 mg/mL (9.40 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.5 mg/mL (9.40 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

Amiloride hydrochloride (MK-870 hydrochloride) is an inhibitor of both epithelial sodium channel (ENaC<sup>[1]</sup>) and urokinase-type plasminogen activator receptor (uTPA<sup>[2]</sup>). Amiloride hydrochloride is a blocker of polycystin-2 (PC2; TRPP2<sup>[3]</sup>) channel.

#### IC<sub>50</sub> & Target

ENaC<sup>[1]</sup>; uTPA<sup>[2]</sup>; polycystin-2(TRPP2)<sup>[3]</sup>

#### In Vitro

Amiloride blocks δβγ channels with an IC<sub>50</sub> of 2.6 μM. The K<sub>i</sub> 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 αβγ channel. The K<sub>i</sub> of

amiloride for  $\delta\alpha\beta\gamma$  channels is 920 and 13.7  $\mu\text{M}$  at  $-120$  and  $+80$  mV, respectively, which significantly differs from that of both  $\alpha\beta\gamma$  and  $\delta\beta\gamma$  channels<sup>[1]</sup>.

Amiloride is a relatively selective inhibitor of the epithelial sodium channel (ENaC) with an  $\text{IC}_{50}$  (the concentration required to reach 50% inhibition of an ion channel) in the concentration range of 0.1 to 0.5  $\mu\text{M}$ . Amiloride is a relatively poor inhibitor of the  $\text{Na}^+/\text{H}^+$  exchanger (NHE) with an  $\text{IC}_{50}$  as low as 3  $\mu\text{M}$  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  $\text{IC}_{50}$  of 1 mM. Amiloride (1  $\mu\text{M}$ ) 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  $\text{Na}^+$  influx in doses known to be relatively specific for ENaC (1.5  $\mu\text{M}$ ) in vascular smooth muscle cells (VSMC)<sup>[2]</sup>. 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<sup>[2]</sup>. 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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