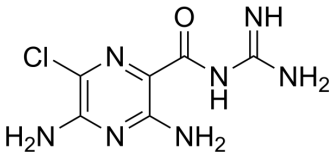
Amiloride

Cat. No.:	HY-B0285
CAS No.:	2609-46-3
Molecular Formula:	$C_6H_8ClN_7O$
Molecular Weight:	229.63
Target:	Sodium Channel; TRP Channel; Apoptosis
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



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.
IC_{50} & 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 αβγ channel. The K_i of amiloride for δαβγ channels is 920 and 13.7 μM at -120 and +80 mV, respectively, which significantly differs from that of both αβγ and δβγ 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 [Na^+] but as high as 1 mM in the presence of a high [Na^+]. Amiloride is an even weaker inhibitor of the Na^+/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.
- J Am Chem Soc. 2018 Dec 12;140(49):17234-17240.

- Small Methods. 2020 Dec 18.
- 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.

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