Saralasin acetate hydrate

Cat. No.: HY-P0205A

CAS No.: 39698-78-7

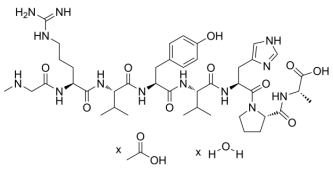
Molecular Formula: $C_{44}H_{71}N_{13}O_{13}$

Sequence Shortening: {Sar}-RVYVHPA

Target: Angiotensin Receptor

Pathway: GPCR/G Protein

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Saralasin ([Sar1,Ala8] Angiotensin II) acetate hydrate is an octapeptide analog of angiotensin II. Saralasin acetate hydrate is a competitive angiotensin II receptor antagonist with a K_i value of 0.32 nM for 74% of the binding sites, and has partial agonist activity as well. Saralasin acetate hydrate can be used for the research of renovascular hypertension, renin-dependent (angiotensinogenic) hypertension[1][3][6].

IC_{50} & Target

Ki: 0.32 nM (Angiotensin II receptor)[3]

In Vitro

Saralasin acetate hydrate (1 nM, 48 or 72 h) inhibits cell growth in 3T3 and SV3T3 cells[1].

Saralasin acetate hydrate (5 μM, 2h) restores $I_{to,\ fast}$ (Fast-Inactivating Transient Outward K+ Current in Mouse Ventricle) and I K, slow (Slow-Inactivating Transient Outward K+ Current in Mouse Ventricl) to control levels in myocytes[2].

Saralasin acetate hydrate (0.1-10 nM, 40 min) inhibits binding of FITC-Ang II to rat liver membrane preparation (used as the source of angiotensin receptors) with a K_i value of 0.32 nM for 74% of the binding sites and 2.7 nM for the remaining binding sites[3].

Saralasin acetate hydrate (1 μM, perfused rat ovary in vitro) inhibits the ovulation rate versus control and reduces prostaglandin E2 and 6-keto-prostaglandin $F_{1\alpha}$ levels[4].

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

Cell Proliferation Assay[1]

Cell Line:	3T3 and SV3T3 cells
Concentration:	1 nM
Incubation Time:	48 h, 72 h
Result:	Inhibited cell growth in 3T3 and SV3T3 cells and caused an increase of cellular renin concentration.

In Vivo

Saralasin acetate hydrate (intravenous injection, 5-50 μg/kg, a single dose) ameliorates the oxidative stress and tissue injury in cerulein-induced pancreatitis[5].

Saralasin acetate hydrate (subcutaneous injection, 10 and 30 mg/kg, a single dose) increases serum renin activity (SRA) in normal, conscious rats, without markedly altering blood pressure or heart rate[6].

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

Animal Model:	Cerulein-induced acute pancreatitis rats model[5]
Dosage:	5, 10, 20, and 50 μg/kg, a single dose.
Administration:	Intravenous injection
Result:	Restored the pancreatic morphological characteristics to the control level. Reduced pancreatic injury and suppressed the glutathione depletion induced by cerulean.

Animal Model:	Male Sprague-Dawley rats[6]
Dosage:	10 and 30 mg/kg, a single dose.
Administration:	Subcutaneous injection
Result:	Stimulated renin release without altering blood pressure or heart rate at the time of measuring serum renin levels 20 minutes after injection.

REFERENCES

[1]. P Schelling, et al. Effects of angiotensin II and angiotensin II antagonist saralasin on cell growth and renin in 3T3 and SV3T3 cells. J Cell Physiol. 1979 Mar;98(3):503-13.

[2]. Jeremy H Kim, et al. Pressure-overload-induced angiotensin-mediated early remodeling in mouse heart. PLoS One. 2017 May 2;12(5):e0176713.

[3]. Maziar Mohammad Akhavan, et al. A non-radioactive method for angiotensin II receptor binding studies using the rat liver. J Pharmacol Toxicol Methods. 2006 May-Jun;53(3):206-14.

[4]. M Mikuni, et al. Saralasin-induced inhibition of ovulation in the in vitro perfused rat ovary is not replicated by the angiotensin II type-2 receptor antagonist PD123319. Am J Obstet Gynecol. 1998 Jul;179(1):35-40.

[5]. Siu Po Ip, et al. Saralasin, a nonspecific angiotensin II receptor antagonist, attenuates oxidative stress and tissue injury in cerulein-induced acute pancreatitis. Pancreas. 2003 Apr;26(3):224-9. 6/

[6]. Campbell WB, et al. Saralasin-induced renin release: its blockade by prostaglandin synthesis inhibitors in the conscious rat. Hypertension. 1979;1(6):637-642.

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