Aliskiren hemifumarate

Cat. No.: HY-12177

CAS No.: 173334-58-2

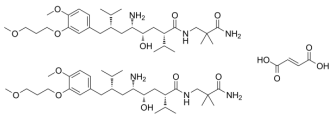
Molecular Formula: $C_{64}H_{110}N_6O_{16}$

Molecular Weight: 1219.59

Target: Renin; Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

Storage: 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

H_2O : ≥ 50 mg/mL (41.00 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.8199 mL	4.0997 mL	8.1995 mL
5 mM	0.1640 mL	0.8199 mL	1.6399 mL
10 mM	0.0820 mL	0.4100 mL	0.8199 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 50 mg/mL (41.00 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Aliskiren (CGP 60536; CGP60536B; SPP 100) hemifumarate is an orally active and selective renin inhibitor, with IC_{50} of 1.5 nM. Aliskiren hemifumarate can be used for the research of hypertension, cardiovascular diseases and cancer cachexia[1][2][3].

IC_{50} & Target

IC50: 1.5 nM (renin)[1]; 0.6 nM (human renin), 2 nM (marmoset renin), 80 nM (rat renin), 7 nM (dog renin), 11 nM (rabbit renin), 63 nM (guinea pig renin), 150 nM (pig renin)[2]

In Vitro

Aliskiren hemifumarate inhibits plasma renin activity (PRA) in vitro with IC_{50}s of 2.9 nM (human PRA), 8.0 nM (monkey PRA), respectively[1].
Aliskiren hemifumarate (5 μM; 24 h) inhibits prorenin-induced human aortic smooth muscle cell migration[2].
Aliskiren hemifumarate (1-10 μM; 24 h) inhibits both the lamellipodia formation and morphological changes induced by prorenin with no significant effect on PDGF-BB activity[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	Smooth muscle cell (SMC)
Concentration:	1-10 μM
Incubation Time:	24 hours
Result:	Inhibited human aortic smooth muscle cell migration induced by prorenin (10 nM) at 10 μM.

In Vivo

Aliskiren hemifumarate (3 mg/kg, 10 mg/kg; p.o.; daily; 0-12 d) inhibit renin and lower blood pressure without affecting heart rate in sodium-depleted marmosets[3].
Aliskiren hemifumarate (10 mg/kg; p.o.; single dose) delays cachexia development, reduces tumor, and prolongs mouse survival. And also improves whole□body strength, mobility and coordination, enhances locomotor activity, and inhibits muscle wasting[4].
Aliskiren hemifumarate (10 mg/kg; p.o.; single dose; 20 d after C26 injection) reduces oxidative stress associated with cancer cachexia[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Sodium-depleted marmosets[3]
Dosage:	3 mg/kg, 10 mg/kg
Administration:	Oral gavage; once daily; 12 days
Result:	Increased plasma immunoreactive renin levels, and lowered blood pressure without affecting heart rate. Showed no rebound increase in BP following the end of treatment with either dose of aliskiren. Inhibited the RAS and controls the upregulation of pro□inflammatory cytokines.

Animal Model:	Cancer cachexia model in BALB/c mice injected with C26 mouse colon carcinoma cells[4]
Dosage:	10 mg/kg
Administration:	Oral gavage; on day 5 (as a preventive strategy, AP group) or on day 12 (as a therapeutic strategy, AT group) after C26 injection; for 20 days after C26 injection
Result:	Enhanced grip strength, coordination, and locomotor activity. Inhibited serum Ang I and □ levels and both serum and muscular tumor necrosis factor□α (TNF□α) and inter□ leukin□6 (IL□6) levels.

CUSTOMER VALIDATION

- Neurobiol Dis. 2014 Nov;71:292-304.
- Lipids Health Dis. 2018 Jul 31;17(1):183.
- Front Biosci-Landmrk. 2023 Oct 17, 28(10), 238.
- Toxicology Research and Application. 2018, 2:239784731880115.
- Toxicology Research and Application. September 25, 2018.

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REFERENCES

[1]. Wang C, et al. Aliskiren targets multiple systems to alleviate cancer cachexia. Oncol Rep. 2016 Nov;36(5):3014-3022.

[2]. Ferri N, et al. Aliskiren inhibits prorenin-induced human aortic smooth muscle cell migration. J Renin Angiotensin Aldosterone Syst. 2015 Jun;16(2):284-91.

[3]. Yuji Nakamura, et al. Discovery of DS-8108b, a Novel Orally Bioavailable Renin Inhibitor. ACS Med. Chem. Lett., 2012, 3 (9), pp 754-758

[4]. Buczko W, et al. Pharmacokinetics and pharmacodynamics of aliskiren, an oral direct renin inhibitor. Pharmacol Rep. 2008 Sep-Oct;60(5):623-31.

[5]. Wood JM, et al. Structure-based design of aliskiren, a novel orally effective renin inhibitor.Biochem Biophys Res Commun, 2003, 308(4), 698-705.

[6]. Gradman AH, et al.Aliskiren, a novel orally effective renin inhibitor, provides dose-dependent antihypertensive efficacy and placebo-like tolerability in hypertensive patients. Circulation, 2005, 111(8), 1012-1018.

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