AdipoRon

Cat. No.: HY-15848

CAS No.: 924416-43-3

Molecular Formula: $C_{27}H_{28}N_2O_3$

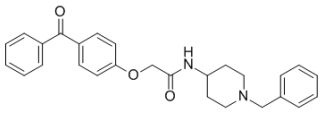
Molecular Weight: 428.52

Target: Adiponectin Receptor

Pathway: GPCR/G Protein

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 : ≥ 44 mg/mL (102.68 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3336 mL	11.6681 mL	23.3361 mL
5 mM	0.4667 mL	2.3336 mL	4.6672 mL
10 mM	0.2334 mL	1.1668 mL	2.3336 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution
5. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AdipoRon is an orally active adiponectin receptor (AdipoR) agonist, binding to AdipoR1 and AdipoR2 with K_ds of 1.8 and 3.1

	μM, respectively.
IC_{50} & Target	Kd: 1.8 μM (AdipoR1), 3.1 μM (AdipoR2)[1]
In Vitro	AdipoRon is an orally active and specific AdipoR agonist, binds to AdipoR1 and AdipoR2, with K_ds of 1.8 and 3.1 μM. AdipoRon (50 nM-50 μM) increases AMPK phosphorylation via AdipoR1[1]. AdipoRon (50 μM) dose-dependently attenuates the expression of TNF-α and TGF-β1 in the L02 cells. AdipoRon exhibits significant and dosage-dependent growth suppression on macrophages[2]. AdipoRon treatment significantly improves cardiac functional recovery after reperfusion, and inhibits post-MI apoptosis[3]. AdipoRon exerts vasodilation by mechanisms distinct to adiponectin and induces vasorelaxation without a marked decrease in VSMC $[Ca^{2+}]_i$[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	AdipoRon (50 mg/kg, i.v.) cuases significant phosphorylation of AMPK in skeletal muscle and liver of wild-type mice but not Adipor1$^{-/-}$ Adipor2$^{-/-}$ double-knockout mice[1]. AdipoRon (0.02, 0.1, and 0.5 mg/kg, i.g.) alleviates D-GalN induced hepatotoxicity in mice, and prevents hepatic architecture distortion against D-GalN challenge. The hepatoprotective potential of AdipoRon is particularly evident in higher dosages (0.1 and 0.5 mg/kg)[2]. Enhanced cardiomyocyte apoptosis in APN-deficient mice is rescued by AdipoRon (50 mg/kg, p.o.) administration. Antiapoptotic effect of AdipoRon is attenuated but not lost in AMPK-DN mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	The effects of AdipoRon on the proliferation of parenchymal and non-parenchymal hepatocytes are evaluated in vitro via L02 and RAW264.7, by MTT assay as described with slight modification: 100 μL cells suspension (6×10^4/mL) are seeded in a 96-well plate and incubated for 18 h. Fresh media with AdipoRon are added at specified concentrations, and the incubations continue for a further 24 h. Then cells are incubated for 4 h with 0.5 mg/mL of MTT, and analyzed in a microplate reader at 490 nm. Each group is performed in six replications. The mean absorbance values corrected for a blank (medium only) are calculated as percentages of survival[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] After 3 days of acclimation, mice are randomLy divided into six groups (9 mice in each): control, model, bicyclol (20 mg/kg), AdipoRon (0.02 mg/kg, 0.1 mg/kg, 0.5 mg/kg). The synthetic AdipoRon and bicyclol are dissolved in DMSO and diluted by saline containing 0.5% sodium carboxymethyl cellulose (CMC-Na) [final vehicle: 5% DMSO (v/v) saline solution]. All test groups are administered with vehicle (control and model groups) or therapeutic agents (bicyclol or AdipoRon groups) at a dosing volume of 10 mL/kg, by intragastric (i.g.) gavage twice per day for three consecutive days prior to D-GalN administration. 2 h after last treatment, mice are challenged with a single intraperitoneal (i.p.) administration of D-GalN saline solution at a dose of 600 mg/kg to induce acute liver injury, while the control group mice receive saline instead. Then mice are fasted for 20 h before orbital blood collection. Finally, all animals are sacrificed by cervical dislocation, and livers are harvested for biochemical or histopathology analysis[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Mol Psychiatry. 2020 Mar 4.
- J Biol Chem. 2018 Apr 20;293(16):6064-6074.
- J Cell Biochem. 2020 Jun;121(5-6):3333-3344.
- J Neurotrauma. 2019 Mar 19;36(6):903-918.

- Neurochem Res. 2019 May;44(5):1214-1227.

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REFERENCES

[1]. Okada-Iwabu M, et al. A small-molecule AdipoR agonist for type 2 diabetes and short life in obesity. Nature. 2013 Nov 28;503(7477):493-9.

[2]. Wang Y, et al. Hepatoprotective effects of AdipoRon against d-galactosamine-induced liver injury in mice. Eur J Pharm Sci. 2016 Aug 9;93:123-131.

[3]. Zhang Y, et al. AdipoRon, the first orally active adiponectin receptor activator, attenuates postischemic myocardial apoptosis through both AMPK-mediated and AMPK-independent signalings. Am J Physiol Endocrinol Metab. 2015 Aug 1;309(3):E275-82.

[4]. Hong K, et al. Adiponectin Receptor Agonist, AdipoRon, Causes Vasorelaxation Predominantly Via a Direct Smooth Muscle Action. Microcirculation. 2016 Apr;23(3):207-20.

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