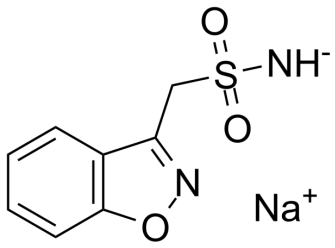
Zonisamide sodium

Cat. No.:	HY-B0124A
CAS No.:	68291-98-5
Molecular Formula:	$C_8H_7N_2NaO_3S$
Molecular Weight:	234.21
Target:	Carbonic Anhydrase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Zonisamide (AD 810) sodium is an orally active carbonic anhydrase inhibitor, with K_is of 35.2 and 20.6 nM for hCA II and hCA V , respectively. Zonisamide sodium exerts neuroprotective effects through anti-apoptosis and upregulating MnSOD levels. Zonisamide sodium also increases the expression of Hrd1, thereby improving cardiac function in AAC rats. Zonisamide sodium can be used in studies of seizure, parkinson's disease and cardiac hypertrophy[1][2][3][4].

IC_{50} & Target

Ki: 35.2μM (hCA II) , 20.6 nM (hCA V)[2]

In Vitro

Zonisamide sodium (10, 50, 100, 200 μM; 24 h) increases viability of SH-SY5Y cells via an anti-apoptotic effect[1].
Zonisamide sodium (100 μM; 24 h) shows neuroprotective effects in PD-cellular models. (PD: parkinson's disease)[1].
Zonisamide sodium (100 μM; 24 h) reduces levels of proapoptotic molecules, and upregulates levels of MnSOD (MnSOD over-expression attenuates MPTP toxicity and protects cells from apoptosis)[1].
Zonisamide sodium (0.1, 0.3, 1 μM; 24 h) inhibits cardiac hypertrophy and fibrosis in vitro[3].
Zonisamide sodium markedly increases the expression of Hrd1 in Ang II-treated NRCMs[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	SH-SY5Y cells
Concentration:	10, 50, 100, 200 μM
Incubation Time:	24 h
Result:	Induced an increase of cell viability, and with the greatest effect being at 100 μM. Exhibited neuroprotective effect on SH-SY5Y cells (PD-cellular models) when at 100 μM.

Apoptosis Analysis[1]

Cell Line:	SH-SY5Y cells
Concentration:	100 μM
Incubation Time:	24 h
Result:	Showed an effect of anti-apoptotic.

RT-PCR[3]

Cell Line:	NRCMs and cardiac fibroblasts (expose to Ang II for cardiomyocyte hypertrophy and fibrosis model)
Concentration:	0.1, 0.3, 1 μM
Incubation Time:	24 h
Result:	Decreased the expression of atrial natriuretic factor (ANF) and cardiomyosin heavy chain β (β-MHC) but increased the expression of cardiac myosin heavy chain α (α-MHC) in NRCMs. Decreased cardiac expression of the fibrosis-related gene Collagen 1A1 (Col1A1) in cardiac fibroblasts.

Western Blot Analysis[1]

Cell Line:	SH-SY5Y cells
Concentration:	100 μM
Incubation Time:	24 h
Result:	Reduced the proapoptotic molecules levels of cleaved caspase-9, -3, and p-JNK, and blocked the activation of proapoptotic molecules in SH-SY5Y cells. Induced an increase in MnSOD levels.(MnSOD over-expression attenuates MPTP toxicity and protects cells from apoptosis).

In Vivo

Zonisamide sodium (40 mg/kg; i.p.; single daily for 14 days) prevents seizures in $FeCl_3$-induced chronic amygdalar seizures model[2].

Zonisamide sodium (14, 28, 56 mg/kg; i.p.; single daily for 6 weeks) alleviates cardiac hypertrophy and improved cardiac function in rats subjected to AAC (abdominal aortic constriction)[3].

Zonisamide sodium (14, 28, 56 mg/kg; i.p.; single daily for 6 weeks) upregulates Hrd1 expression and accelerates ERAD in the hearts of AAC rats[3].

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

Animal Model:	Male Wistar rats (200-250 g; $FeCl_3$-induced chronic amygdalar seizures)[2].
Dosage:	40 mg/kg
Administration:	Intraperitoneal injection; single daily for 14 days.
Result:	Showed activity of anti-seizures. Significantly down-regulated GABA transporters GAT-1 in the hippocampus.

Animal Model:	Adult male Sprague-Dawley rats (100-120 g; cardiac hypertrophy model)[3].
Dosage:	14, 28, 56 mg/kg (dissolved in 1% DMSO)
Administration:	Intraperitoneal injection; single daily for 6 weeks.
Result:	Significantly attenuated cardiac hypertrophy and fibrosis. Increased LV ejection fraction (EF), fractional shortening (FS) and E/A ratio. Markedly increased the expression of Hrd1 in the hearts of AAC rats.

REFERENCES

[1]. Kawajiri S, et al. Zonisamide reduces cell death in SH-SY5Y cells via an anti-apoptotic effect and by upregulating MnSOD. Neurosci Lett. 2010 Sep 6;481(2):88-91.

[2]. Ueda Y, et al. Effect of zonisamide on molecular regulation of glutamate and GABA transporter proteins during epileptogenesis in rats with hippocampal seizures. Brain Res Mol Brain Res. 2003 Aug 19;116(1-2):1-6.

[3]. Wu Q, et al. Zonisamide alleviates cardiac hypertrophy in rats by increasing Hrd1 expression and inhibiting endoplasmic reticulum stress. Acta Pharmacol Sin. 2021 Oct;42(10):1587-1597.

[4]. De Simone G, et al. Carbonic anhydrase inhibitors. Zonisamide is an effective inhibitor of the cytosolic isozyme II and mitochondrial isozyme V: solution and X-ray crystallographic studies. Bioorg Med Chem Lett. 2005 May 2;15(9):2315-20.

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