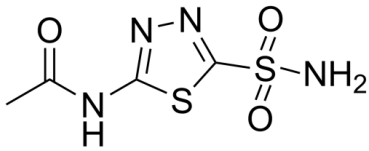
Data Sheet

Product Name:	Acetazolamide
Cat. No.:	CS-3568
CAS No.:	59-66-5
Molecular Formula:	$C_4H_6N_4O_3S_2$
Molecular Weight:	222.25
Target:	Autophagy; Bacterial; Carbonic Anhydrase
Pathway:	Anti-infection; Autophagy; Metabolic Enzyme/Protease
Solubility:	DMSO : 50 mg/mL (ultrasonic);H_2O : < 0.1 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Acetazolamide is a **carbonic anhydrase** (**CA**) **IX** inhibitor with an **IC_{50}** of 30 nM for **hCA IX**. Acetazolamide has diuretic, antihypertensive and anti-gonococcal activities[1][4][5][6]. IC50 & Target: IC50: 30 nM (hCA IX), 130 nM (hCA II)[1] ***In Vitro:*** Acetazolamide also inhibits hCA II with an IC_{50} of 130 nM[1].

Acetazolamide (Ace) is a small heteroaromatic sulfonamide that binds to various carbonic anhydrases with high affinity, acting as a carbonic anhydrase (CA) inhibitor[2].

Compared with the control group, the high Acetazolamide concentration (AceH, 50 nM), Cisplatin (Cis; 1 μg/mL) and Cis combined with the low Acetazolamide concentration (AceL, 10 nM) treatments significantly reduces viability of Hep-2 cells[2].

Treatment with the Acetazolamide/Cis combination significantly increases the expression levels of P53, as both AceL+Cis and AceH+Cis treatments result in significantly increased P53 protein expression levels compared with the control group. The Ace/Cis combination treatment significantly reduces the bcl-2/bax expression ratio, and increases the expression of caspase-3 protein, compared with the control group. AceL, AceH, Cis and AceL+Cis treatments significantly reduce the bcl-2/bax ratio compared with the control group[2].

Combined Ace and Cis treatment effectively promotes apoptosis in Hep-2 cells[2].

Combined treatment with Ace/Cis markedly decreases the expression of AQP1 mRNA in Hep-2 cells. Both AceH and AceL+Cis treatments decrease the expression of aquaporin-1 (AQP1) mRNA in Hep-2 cells compared with the control group[2].

In Vivo: Acetazolamide (40 mg/kg) significantly potentiates the inhibitory effect of MS-275 on tumorigenesis in neuroblastoma (NB) SH-SY5Y xenografts[3].

Acetazolamide (40 mg/kg) and/or MS-275 treatment reduce expression of HIF1-α and CAIX in NB SH-SY5Y xenograft[3].

Acetazolamide (40 mg/kg), MS-275 and Acetazolamide+MS-275 reduce expression of mitotic and proliferative markers in NB SH-SY5Y xenografts[3].

Acetazolamide (50 mg/kg; PO, for 3 days) significantly reduces the gonococcal load in the vagina of infected mice by 90%[6].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: Cell Viability Assay[2]

Cell line: Hep-2 cells and HUVECs

Concentration: 10 nM and 50 nM

Incubation time: 48 h

Assay: The cell viability of Hep-2 cells and HUVECs is measured by MTT assay. Hep-2 cells and HUVECs in logarithmic growth phase are plated in 96-well plates. Following 48 h of drug treatment as indicated, 200 μL MTT (5 mg/mL) is added to each well. Cells

are incubated with the MTT solution at 37°C for 4 h. Then, 150 μL DMSO is added for 5 min. The optical density (OD) values are measured at 490 nm with a Versamax Microplate reader.
Note: Combined treatment effectively reduced viability in Hep-2 cells.
Animal Administration: In vivo studies[3]
Animal model: 4-6 weeks-old female NOD/SCID mice
Dosage: 40 mg/kg, intraperitoneal injection, every day for 2 weeks
Administration: Mice are randomized into four groups (5 mice per group). The control and treatment groups receive intraperitoneal injections of vehicle (PBS) or Acetazolamide (40 mg/kg), MS-275 (20 mg/kg) or the combination, respectively, every day for 2 weeks. Experiments are terminated when tumor sizes exceed 2 cm3 in volume or animals show signs of morbidity. Tumor diameters are measured on a daily basis until termination.
Note: Inhibited tumor growth of NB xenografts with significant anti-tumor growth potentiation effect.

References:

[1]. Hou Z, et al. Dual-tail approach to discovery of novel carbonic anhydrase IX inhibitors by simultaneously matching the hydrophobic and hydrophilic halves of the active site. Eur J Med Chem. 2017 May 26;132:1-10.

[2]. Gao H, et al. Combined treatment with acetazolamide and cisplatin enhances chemosensitivity in laryngeal carcinoma Hep-2 cells. Oncol Lett. 2018 Jun;15(6):9299-9306.

[3]. Bayat Mokhtari R, et al. Acetazolamide potentiates the anti-tumor potential of HDACi, MS-275, in neuroblastoma. BMC Cancer. 2017 Feb 24;17(1):156.

[4]. Kassamali R, et al. Acetazolamide: a forgotten diuretic agent. Cardiol Rev. 2011 Nov-Dec;19(6):276-8.

[5]. Jabeen E, et al. Interaction of antihypertensive acetazolamide with nonsteroidal anti-inflammatory drugs. J Photochem Photobiol B. 2013 Aug 5;125:155-63.

[6]. Abutaleb NS, et al. In vivo efficacy of acetazolamide in a mouse model of Neisseria gonorrhoeae infection. Microb Pathog. 2022 Mar;164:105454.

CAIndexNames:

Acetamide, N-[5-(aminosulfonyl)-1,3,4-thiadiazol-2-yl]-

SMILES:

CC(NC1=NN=C(S(=O)(N)=O)S1)=O

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