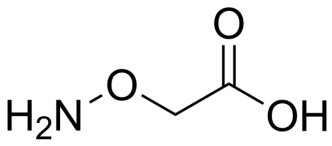
Aminoxyacetic acid

Cat. No.:	HY-107994A
CAS No.:	645-88-5
Molecular Formula:	$C_2H_5NO_3$
Molecular Weight:	91.07
Target:	GABA Receptor
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Aminooxyacetic acid (Carboxymethoxylamine) is a malate-aspartate shuttle (MAS) inhibitor. Aminooxyacetic acid also inhibits the GABA degradating enzyme GABA-T.
IC_{50} & Target	MAS[1], GABA-T[2]
In Vitro	Aminooxyacetic acid hemihydrochloride (AOAA) dose-dependently decreases the survival of C6 glioma cells. Aminooxyacetic acid hemihydrochloride treatment produces a significant increase in the percentage of the cells arrested in the stage of G0/G1, as well as a significant decrease in the percentage of the cells at S phase and G2/M phase. Aminooxyacetic acid hemihydrochloride treatment leads to an obvious decrease in the number of the cells in the phase of cell division. Aminooxyacetic acid hemihydrochloride significantly increases the percentage of the cells in both early-stage apoptosis and necrosis. Treatment of the cells with 1 mM or 5 mM Aminooxyacetic acid hemihydrochloride leads to decreased levels of aging of the cells[1]. Glutamine-dependent cell lines show greater inhibition of cell growth by Aminooxyacetic acid hemihydrochloride (AOA) compare with cells that are less glutamine dependent[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The accumulation of GABA in cerebellum and whole brain is initially very rapid, being significantly increased already 5 min after the injection of Aminooxyacetic acid (AOAA). The rapid initial accumulation becomes gradually slower and maximal levels (400 to 600 % of the control levels) reached 2 to 6 h after Aminooxyacetic acid treatment. Still 24 h after Aminooxyacetic acid treatment, the GABA levels are elevated by about 250%. From 2 to 6 h after Aminooxyacetic acid treatment, the convulsions are completely blocked. 24 h after Aminooxyacetic acid treatment, the convulsions are almost identical to the controls[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2023 Feb 15;8(1):65.
- Nat Metab. 2022 Feb;4(2):239-253.
- Theranostics. 2021 Mar 24;11(12):5650-5674.
- Cancer Res. 2022 Jul 27;CAN-22-0042.

- CNS Neurosci Ther. 2022 Dec 27.

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REFERENCES

[1]. Wang C, et al. Malate-aspartate shuttle inhibitor aminooxyacetic acid leads to decreased intracellular ATP levels and altered cell cycle of C6 glioma cells by inhibiting glycolysis. Cancer Lett. 2016 Aug 1;378(1):1-7.

[2]. Pagliusi SR, et al. Aminooxyacetic acid induced accumulation of GABA in the rat brain. Interaction with GABA receptors and distribution in compartments. Naunyn Schmiedebergs Arch Pharmacol. 1983 Apr;322(3):210-5.

[3]. Korangath P, et al. Targeting Glutamine Metabolism in Breast Cancer with Aminooxyacetate. Clin Cancer Res. 2015 Jul 15;21(14):3263-73.

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