Oxamic acid sodium

Cat. No.: HY-W013032A

CAS No.: 565-73-1

Molecular Formula: $C_2H_2NNaO_3$

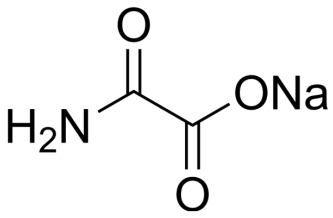
Molecular Weight: 111.03

Target: Lactate Dehydrogenase; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

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 : 12.5 mg/mL (112.58 mM; Need ultrasonic)

DMSO : 3.23 mg/mL (29.09 mM; ultrasonic and warming and adjust pH to 5 with HCl and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	9.0066 mL	45.0329 mL	90.0658 mL
5 mM	1.8013 mL	9.0066 mL	18.0132 mL
10 mM	0.9007 mL	4.5033 mL	9.0066 mL

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

In Vivo

1. Add each solvent one by one: Saline
   Solubility: 100 mg/mL (900.66 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Oxamic acid (oxamate) sodium salt is a lactate dehydrogenase-A (LDH-A) inhibitor. Oxamic acid sodium salt shows anti-tumor activity, and anti-proliferative activity against cancer cells, and can induce apoptosis[1][2][3].

In Vitro

Oxamic acid suppresses the proliferation, migration and invasion of both A2780 and SKOV3 cells[1].
Oxamic acid (10 μM; 24-72 h) inhibits cell proliferation in a dose- and time-dependent manner in both NPC cancer cells[2].
Oxamic acid (0-100 mM; 24 h) induces cell cycle arrest in the G2/M phase in CNE-1 and CNE-2 cells[2].
Oxamic acid (0-100 mM; 48 h) induces apoptosis via caspase-3 activation and the mitochondrial pathway in NPC cells[2].
Oxamic acid (0-100 mM; 24 h) increases ROS levels in NPC cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	CNE-1 and CNE-2 cells

Concentration:	10 μM
Incubation Time:	24-72 hours
Result:	Showed IC_{50}s of 74.6, 32.4 and 17.8 mM and 62.3, 44.5, 31.6 mM at 24, 48 and 72 h in the CNE-1 and CNE-2 cancer cells, respectively.

Apoptosis Analysis[2]

Cell Line:	NPC cells
Concentration:	0, 20, 50 and 100 mM
Incubation Time:	48 hours
Result:	Showed the increasement of early and late apoptotic cells in a dose-dependent manner. Increased the expression of pro-apoptotic Bax and cleaved-caspase-3, while reduced the anti-apoptotic signals of Bcl-2 and pro-caspase-3.

Cell Cycle Analysis[2]

Cell Line:	CNE-1 and CNE-2 cells
Concentration:	0, 20, 50 and 100 mM
Incubation Time:	24 hours
Result:	Showed a dose-dependent increase in the numbers of CNE-1 and CNE-2 cells in the G2/M phase.

In Vivo

Oxamic acid (intraperitoneal injection; 750 mg/kg; once daily; 3 w) treatment improves the efficacy of tumor inhibition in vivo when combined with irradiation treatment[2].

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

Animal Model:	Female Balb/c nude mice injected with CNE-1 cells[2]
Dosage:	750 mg/kg
Administration:	Intraperitoneal injection; 750 mg/kg; once daily; 3 weeks
Result:	Inhibited the tumor growth when compared to either oxamate alone or irradiation alone.

CUSTOMER VALIDATION

- Theranostics. 2023 Jul 3;13(11):3856-3871.
- J Ginseng Res. 2023 Dec 27.
- Research Square Preprint. 2023 Sep 15.

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REFERENCES

[1]. Xiang J, et al. LDH-A inhibitors as remedies to enhance the anticancer effects of PARP inhibitors in ovarian cancer cells. Aging (Albany NY). 2021 Dec 16;13(24):25920-25930.

[2]. Zhai X, et al. Inhibition of LDH-A by oxamate induces G2/M arrest, apoptosis and increases radiosensitivity in nasopharyngeal carcinoma cells. Oncol Rep. 2013 Dec;30(6):2983-91.

[3]. Muramatsu H, et al. Targeting lactate dehydrogenase□A promotes docetaxel□induced cytotoxicity predominantly in castration□resistant prostate cancer cells. Oncol Rep. 2019 Jul;42(1):224-230.

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