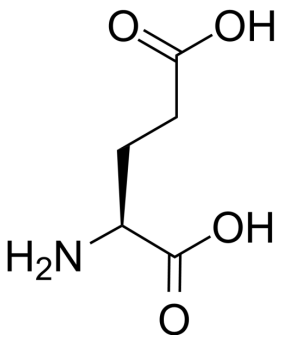
L-Glutamic acid



Cat. No.: HY-14608

CAS No.: 56-86-0

Molecular Formula: $C_5H_9NO_4$

Molecular Weight: 147.13

Target: Endogenous Metabolite; iGluR; Ferroptosis; Apoptosis

Pathway: Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

H_2O : 6.25 mg/mL (42.48 mM; Need ultrasonic)

DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.7967 mL	33.9836 mL	67.9671 mL
5 mM	1.3593 mL	6.7967 mL	13.5934 mL
10 mM	0.6797 mL	3.3984 mL	6.7967 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 9.09 mg/mL (61.78 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

L-Glutamic acid is an excitatory amino acid neurotransmitter that acts as an agonist for all subtypes of glutamate receptors (metabolic rhodophylline, NMDA, and AMPA). L-Glutamic acid has an agonist effect on the release of DA from dopaminergic nerve endings. L-Glutamic acid can be used in the study of neurological diseases[1][2][3][4][5].

IC_{50} & Target

DA	Human Endogenous Metabolite	Microbial Metabolite

In Vitro

L-Glutamic acid (120, 500, 750, 1000 mg/dL) can reduce the harmful effect of lithium on the embryonic development of Xenopus Xenopus[3].

L-Glutamic acid (2, 5, 10, 20 mM, 24-48 h) can induce neuroexcitotoxicity in neuroblastoma[4].

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

Cell Viability Assay[4]

Cell Line:	SH-SY5Y, IMR-32, SK-N-BE(2)
Concentration:	2, 5, 10, 20 mM
Incubation Time:	24 and 48 h
Result:	Reduced cell viability in a dose-dependent manner.

In Vivo

L-Glutamic acid (3 g/kg, subcutaneous injection) can promote excitotoxic degeneration of retinal ganglion cells in mice[1].
L-Glutamic acid (750 mg/kg, intraperitoneal injection) can reduce and inhibit oxidative stress induced by chlorpyrifos (CPF) in rats[5].

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

Animal Model:	Crv4 mice model[1]
Dosage:	3 g/kg
Administration:	s.c., single dose
Result:	Reduced the number of Brn-3a$^+$ RGCs by >70%. In the absence of mGlu1 receptor, MSG-induced retinal damage is diminished.

Animal Model:	CPF-induced rat model[5]
Dosage:	750 mg/kg
Administration:	i.p.
Result:	Reduced CPF-induced oxidative stress by increasing the level of GSH and activity of GSH-related enzymes.

CUSTOMER VALIDATION

- Redox Biol. 2024 Mar 4:71:103112.
- J Transl Med. 2024 Feb 18;22(1):178.
- Neurochem Int. 2023 Jul 24;105587.

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REFERENCES

[1]. Liberatore F, et al. Permissive role for mGlu1 metabotropic glutamate receptors in excitotoxic retinal degeneration. Neuroscience. 2017 Nov 5;363:142-149.

[2]. Giorguieff MF, et al. Presynaptic effect of L-glutamic acid on the release of dopamine in rat striatal slices. Neurosci Lett. 1977 Oct;6(1):73-7.

[3]. Boga Pekmezekmek A, et al. L-Glutamic acid monosodium salt reduces the harmful effect of lithium on the development of Xenopus laevis embryos. Environ Sci Pollut Res Int. 2020 Nov;27(33):42124-42132.

[4]. Croce N, et al. Hydrochloric acid alters the effect of L-glutamic acid on cell viability in human neuroblastoma cell cultures. J Neurosci Methods. 2013 Jul 15;217(1-2):26-

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[5]. Salyha N, et al. Protective role of l-glutamic acid and l-cysteine in mitigation the chlorpyrifos-induced oxidative stress in rats. Environ Toxicol Pharmacol. 2018 Dec;64:155-163.

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