D-Glutamine

Cat. No.: HY-100587

CAS No.: 5959-95-5

Molecular Formula: $C_5H_{10}N_2O_3$

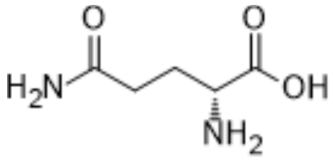
Molecular Weight: 146.14

Target: Autophagy; Mitophagy; Endogenous Metabolite; Ferroptosis

Pathway: Autophagy; Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

H_2O : 20 mg/mL (136.86 mM; ultrasonic and adjust pH to 2 with HCl)

DMSO : 4 mg/mL (27.37 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	6.8428 mL	34.2138 mL	68.4275 mL
5 mM	1.3686 mL	6.8428 mL	13.6855 mL
10 mM	0.6843 mL	3.4214 mL	6.8428 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 12.5 mg/mL (85.53 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

D-Glutamine is a cell-permeable D type stereoisomer of Glutamine.

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Glutamine is a key amino acid in the central nervous system (CNS), playing an important role in the glutamate/GABA-Glutamine cycle (GGC). In the GGC,Glutamine is transferred from astrocytes to neurons, where it will replenish the inhibitory and excitatory neurotransmitter pools[1].

D-Glutamine has been used to study its role in conferring protection against acetaldehyde-induced disruption of barrier function in Caco-2 cell monolayer. Role of L-Glutamine in the protection of intestinal epithelium from acetaldehyde-induced disruption of barrier function is evaluated in Caco-2 cell monolayer. L-Glutamine reduced the acetaldehyde-induced decrease in transepithelilal electrical resistance and increase in permeability to inulin and lipopolysaccharide in a time- and

dose-dependent manner; D-Glutamine, L-aspargine, L-arginine, L-lysine, or L-alanine produced no significant protection. D-Glutamine also fails to influence the acetaldehyde-induced decrease in TER and increase in inulin flux. D-Glutamine or glutaminase inhibitor by themselves did not influence TER or inulin flux in control or acetaldehyde-treated cell monolayers. Lack of effect of D-Glutamine in protection from acetaldehyde indicates that the L-Glutamine-mediated protection is stereospecific[2].

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

PROTOCOL

Cell Assay [2]

Effect of D-Glutamine and glutaminase inhibitor on acetaldehyde-induced permeability. Caco-2 cell monolayers are incubated for 4 h without or with acetaldehyde (600 μM) and L-Glutamine or D-Glutamine (2 mM) in the absence or presence of 6-diazo-5-oxo-L-norleucine (DON). Transepithelial electrical resistance (TER) and FITC-inulin flux are measured. Values are means±SE (n=6)[2].

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

CUSTOMER VALIDATION

- Microbiome. 2019 Mar 20;7(1):43.
- Laurea Magistrale in Biomedical Engineering, Politecnico di Milano. 2019 Jun.

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REFERENCES

[1]. Leke R, et al. The Glutamine Transporters and Their Role in the Glutamate/GABA-Glutamine Cycle. Adv Neurobiol. 2016;13:223-257.

[2]. Seth A, et al. L-Glutamine ameliorates acetaldehyde-induced increase in paracellular permeability in Caco-2 cellmonolayer. Am J Physiol Gastrointest Liver Physiol. 2004 Sep;287(3):G510-7.

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