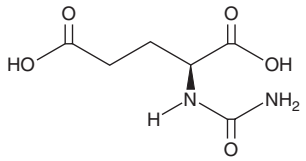
PRODUCT INFORMATION

N-Carbamyl-L-Glutamic Acid

Item No. 23512

CAS Registry No.: 1188-38-1
Formal Name: N-(aminocarbonyl)-L-glutamic acid
Synonym: Carglumic Acid
MF: $C_6H_{10}N_2O_5$
FW: 190.2
Purity: ≥98%
Supplied as: A crystalline solid
Storage: Room temperature
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

N-Carbamyl-L-glutamic acid is supplied as a crystalline solid. A stock solution may be made by dissolving the N-carbamyl-L-glutamic acid in the solvent of choice, which should be purged with an inert gas. N-Carbamyl-L-glutamic acid is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of N-carbamyl-L-glutamic acid in ethanol is approximately 10 mg/ml and approximately 30 mg/ml in DMSO and DMF.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of N-carbamyl-L-glutamic acid can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of N-carbamyl-L-glutamic acid in PBS, pH 7.2, is approximately 2 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

N-Carbamyl-L-glutamic acid is an activator of carbamoyl phosphate synthetase 1 (CPS1), the first enzyme in the urea cycle.[1] It is a structural analog of N-acetyl-glutamate, an endogenous CPS1 activator. N-Carbamyl-L-glutamic acid (500 mg/kg) reduces 2-ketoisocaproate-induced hyperammonemia in a mouse model of 3-hydroxy-3-methylglutaryl-CoA lyase deficiency.[2] It inhibits cell proliferation of a variety of cancer cell lines with IC_{50} values ranging from 5 to 7.5 nM.[3] It also inhibits tumor growth by 80 and 82% in orthotopic mouse models of pancreatic and triple-negative breast cancer, respectively, when administered at a dose of 120 mg/kg per day for 10 days. Formulations containing N-carbamyl-L-glutamic acid have been used in the treatment of primary N-acetyl-glutamate synthase deficiency.

References

1. Häberle, J. Carglumic acid for the treatment of N-acetylglutamate synthase deficiency and acute hyperammonemia. *Exp. Rev. Endocrinol. Metab.* **7(3)**, 263-271 (2012).
2. Gauthier, N., Wu, J.W., Wang, S.P., *et al.* A liver-specific defect of Acyl-CoA degradation produces hyperammonemia, hypoglycemia and a distinct hepatic Acyl-CoA pattern. *PLoS One* **8(7)**, e60581 (2013).
3. Chen, C.-T., Chen, Y.-C., Yamaguchi, H., *et al.* Carglumic acid promotes apoptosis and suppresses cancer cell proliferation *in vitro* and *in vivo*. *Am. J. Cancer Res.* **5(12)**, 3560-3569 (2015).

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