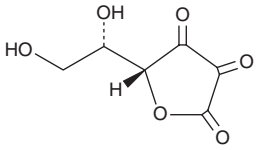
PRODUCT INFORMATION

Dehydroascorbic Acid

Item No. 29089

CAS Registry No.: 490-83-5
Formal Name: L-*threo*-2,3-hexodiulosonic acid, γ-lactone
Synonyms: L-Dehydroascorbic Acid, DHAA
MF: $C_6H_6O_6$
FW: 174.1
Purity: ≥95%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years
Special Conditions: A dimer in solid form but becomes a monomer in solution.



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

Laboratory Procedures

Dehydroascorbic acid is supplied as a crystalline solid. A stock solution may be made by dissolving the dehydroascorbic acid in the solvent of choice, which should be purged with an inert gas. Dehydroascorbic acid is soluble in the organic solvent DMSO at a concentration of approximately 10 mg/ml.

Dehydroascorbic acid is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, dehydroascorbic acid should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Dehydroascorbic acid has a solubility of approximately 0.16 mg/ml in a 1:5 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Dehydroascorbic acid is an oxidized form of ascorbic acid (Item No. 14656).[1] It is obtained through the diet or formed *via* oxidation of ascorbic acid in the gut and can also be reduced back to ascorbic acid in various cell types. Dehydroascorbic acid (0.5 and 1 mM) inhibits hydrogen peroxide-induced cell death in murine astrocytes by approximately 74 and 83%, respectively.[2] It also increases glutathione peroxidase (GPX) and glutathione reductase activity and inhibits production of reactive oxygen species (ROS) in hydrogen peroxide-treated astrocytes when used at a concentration of 1 mM. Dehydroascorbic acid (40 and 250 mg/kg) increases cerebral blood flow and reduces infarct volume and mortality in a mouse model of cerebral ischemia-reperfusion injury induced by transient middle cerebral artery occlusion (MCAO) when administered prior to ischemia.[3] It also reduces infarct volume in a permanent MCAO mouse model when administered pre- or post-ischemia at doses of 250 and 500 mg/kg, respectively.

References

1. Wilson, J.X. The physiological role of dehydroascorbic acid. *FEBS Lett.* **527(1-3)**, 5-9 (2002).
2. Kim, E.J., Park, Y.G., Baik, E.J., *et al.* Dehydroascorbic acid prevents oxidative cell death through a glutathione pathway in primary astrocytes. *J. Neurosci. Res.* **79(5)**, 670-679 (2005).
3. Huang, J., Agus, D.B., Winfree, C.J., *et al.* Dehydroascorbic acid, a blood-brain barrier transportable form of vitamin C, mediates potent cerebroprotection in experimental stroke. *Proc. Natl. Acad. Sci. USA* **98(20)**, 11720-11724 (2001).

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