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- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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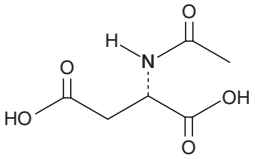
PRODUCT INFORMATION

N-Acetyl-L-aspartic Acid

Item No. 34635

CAS Registry No.:	997-55-7
MF:	$C_6H_9NO_5$
FW:	175.1
Purity:	≥98%
Supplied as:	A solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

N-Acetyl-L-aspartic acid is supplied as a solid. A stock solution may be made by dissolving the N-acetyl-L-aspartic acid in the solvent of choice, which should be purged with an inert gas. N-Acetyl-L-aspartic acid is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of N-acetyl-L-aspartic acid in ethanol is approximately 20 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-acetyl-L-aspartic acid can be prepared by directly dissolving the solid in aqueous buffers. The solubility of N-acetyl-L-aspartic acid in PBS (pH 7.2) is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

N-Acetyl-L-aspartic acid is an acetylated form of L-aspartic acid.[1] It is formed from L-aspartate and acetyl-CoA (Item No. 16160) by L-aspartate N-acetyltransferase (Asp-NAT) in neuronal mitochondria.[2] N-Acetyl-L-aspartic acid is hydrolyzed to L-aspartate and acetate by aspartoacylase (ASPA), which is expressed by oligodendrocytes. N-Acetyl-L-aspartic acid levels are increased in the brain of patients with Canavan disease, an autosomal recessive disorder caused by loss-of-function *ASPA* mutations and characterized by cognitive and motor impairments.[1-3]

References

1. Baslow, M.H. A review of phylogenetic and metabolic relationships between the acylamino acids, N-acetyl-L-aspartic acid and N-acetyl-L-histidine, in the vertebrate nervous system. *J. Neurochem.* **68(4)**, 1335-1344 (1997).
2. Madhavarao, C.N., Arun, P., Moffett, J.R., *et al.* Defective N-acetylaspartate catabolism reduces brain acetate levels and myelin lipid synthesis in Canavan's disease. *Proc. Natl. Acad. Sci. USA* **102(14)**, 5221-5226 (2004).
3. Bannerman, P., Guo, F., Chechneva, O., *et al.* Brain Nat8l knockdown suppresses spongiform leukodystrophy in an aspartoacylase-deficient Canavan disease mouse model. *Mol. Ther.* **26(3)**, 793-800 (2018).

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