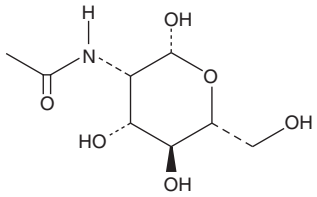
PRODUCT INFORMATION

N-acetyl-D-Mannosamine

Item No. 10011060

CAS Registry No.: 7772-94-3
Formal Name: 2-(acetylamino)-2-deoxy-b-D-mannopyranose
Synonym: ManNAc
MF: $C_8H_{15}NO_6$
FW: 221.2
Purity: ≥95%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

ManNAc is supplied as a crystalline solid. A stock solution may be made by dissolving the ManNAc in the solvent of choice, which should be purged with an inert gas. ManNAc is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of ManNAc in these solvents is approximately 0.2, 5, and 2 mg/ml, respectively.

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 ManNAc can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of ManNAc in PBS (pH 7.2) is approximately 5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Sialic acids, commonly present as terminal carbohydrates on glycoconjugates, are essential for a variety of cellular functions including cell adhesion and signal recognition as well as the formation and progression of tumors.[1] Disruption of sialic acid biosynthesis can result in severe glomerular proteinuria or neuromuscular disorders such as hereditary inclusion body myopathy (HIBM).[2] ManNAc is the precursor of all physiological sialic acids. Intraperitoneal injection of ManNAc twice daily at 1,000 mg/kg in C57BL/6 mice for 13 days leads to increased sialylation in kidney, liver, blood cells, brain, spinal cord, muscle, heart, lung, and spleen.[3] ManNAc reverses hyposialylation and improves glomerular integrity in *Gne*$^{M712T/M712T}$ mice whose key enzyme for sialic acid production has been deleted and may prove therapeutic in the treatment of HIBM.[2]

References

1. Schwarzkopf, M., Knobeloch, K.-P., Rohde, E., *et al.* Sialylation is essential for early development in mice. *Proc. Natl. Acad. Sci. USA* **99(8)**, 5267-5270 (2002).
2. Galeano, B., Klootwijk, R., Manoli, I., *et al.* Mutation in the key enzyme of sialic acid biosynthesis causes severe glomerular proteinuria and is rescued by N-acetylmannosamine. *J. Clin. Invest.* **117(6)**, 1585-1594 (2007).
3. Gagiannis, D., Gossrau, R., Reutter, W., *et al.* Engineering the sialic acid in organs of mice using N-propanoylmannosamine. *Biochem. Biophys. Acta* **1770**, 297-306 (2007).

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