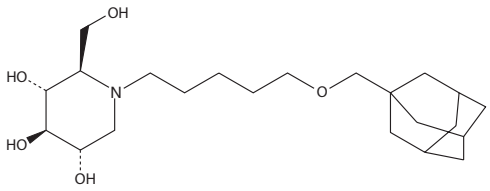
PRODUCT INFORMATION

AMP-Deoxynojirimycin

Item No. 10010332

CAS Registry No.: 216758-20-2
Formal Name: 2R-(hydroxymethyl)-1-[5-(tricyclo[3.3.2.13,7]dec-1-ylmethoxy) pentyl]-3R,4R,5S-piperidinetriol
Synonyms: Adamantane-pentyl-dNM, AMP-DNJ, AMP-dNM, N-(5-adamantane-1-yl-methoxy-pentyl)-Deoxynojirimycin
MF: $C_{22}H_{39}NO_5$
FW: 397.6
Purity: ≥95%
Supplied as: A solution in ethanol
Storage: -20°C
Stability: ≥2 years



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

Laboratory Procedures

AMP-dNM is supplied as a solution in ethanol. To change the solvent, simply evaporate the ethanol under a gentle stream of nitrogen and immediately add the solvent of choice. Solvents such as ethanol, DMSO, and dimethyl formamide (DMF) purged with an inert gas can be used. The solubility of AMP-dNM in ethanol is approximately 30 mg/ml and approximately 50 mg/ml in DMSO and DMF.

AMP-dNM is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, the ethanolic solution of AMP-dNM should be diluted with the aqueous buffer of choice. AMP-dNM has a solubility of approximately 0.5 mg/ml in a 1:1 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

The lipid messenger ceramide is converted to glucosylceramide by glucosylceramide synthase (GCS). In the reverse direction, non-lysosomal glucosylceramidase (GCase), also known as β-glucosidase 2 (BGD), cleaves the glucosyl moiety from glucosylceramide, liberating ceramide, which can be converted into sphingomyelin.[1,2] AMP-deoxynojirimycin (AMP-dNM) is a hydrophobic derivative of dNM. It potently inhibits BGD (IC_{50} = 0.3 nM), less potently antagonizes GCS (IC_{50} = 25 nM), but only poorly inhibits other GCase isoforms.[2,3] AMP-dNM has been shown to strongly suppress inflammation in a murine model of hapten-induced colitis, enhance insulin sensitivity in murine and rat models of insulin resistance, and induce sterol regulatory element-binding protein-regulated gene expression and cholesterol synthesis in HepG2 cells.[1,4,5]

References

1. Bijl, N., Scheij, S., Houten, S., *et al. J. Pharmacol. Exp. Ther.* **326(3)**, 849-855 (2008).
2. Aerts, J.M., Hollak, C., Boot, R., *et al. Philos. Trans. R. Soc. Lond. B Biol. Sci.* **358(1433)**, 905-914 (2003).
3. Overkleeft, H.S., Renkema, G.H., Neele, J., *et al. J. Biol. Chem.* **273(41)**, 26522-26527 (1998).
4. Shen, C., Bullens, D., Kasran, A., *et al. Int. Immunopharmacol.* **4(7)**, 939-951 (2004).
5. Aerts, J.M., Ottenhoff, R., Powlson, A.S., *et al. Diabetes* **56(5)**, 1341-1349 (2007).

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