

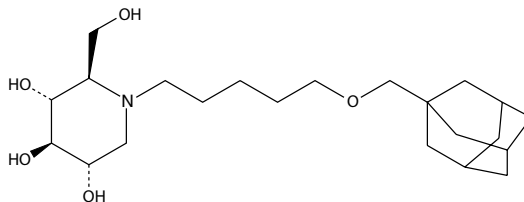
# Product Information



## AMP-Deoxynojirimycin

Item No. 10010332

**CAS Registry No.:** 216758-20-2  
**Formal Name:** 2R-(hydroxymethyl)-1-[5-(tricyclo[3.3.2.1.3,7]dec-1-ylmethoxy)pentyl]-3R,4R,5S-piperidinetriol  
**Synonyms:** Adamantane-pentyl-dNM, AMP-dNM, N-(5-adamantane-1-yl-methoxy-pentyl)-Deoxynojirimycin  
**MF:** C<sub>22</sub>H<sub>39</sub>NO<sub>5</sub>  
**FW:** 397.6  
**Purity:** ≥95%  
**Stability:** ≥1 year at -20°C  
**Supplied as:** A solution in ethanol



### Laboratory Procedures

For long term storage, we suggest that AMP-deoxynojirimycin (AMP-dNM) be stored as supplied at -20°C. It should be stable for at least one year.

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.

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),<sup>1</sup> cleaves the glucosyl moiety from glucosylceramide, liberating ceramide, which can be converted into sphingomyelin.<sup>2</sup> AMP-dNM is a hydrophobic derivative of dNM. It potently inhibits BGD (IC<sub>50</sub> = 0.3 nM),<sup>3</sup> less potently antagonizes GCS (IC<sub>50</sub> = 25 nM),<sup>2</sup> but only poorly inhibits other GCase isoforms. AMP-dNM has been shown to strongly suppress inflammation in a murine model of hapten-induced colitis,<sup>4</sup> enhance insulin sensitivity in murine and rat models of insulin resistance,<sup>5</sup> and induce sterol regulatory element-binding protein-regulated gene expression and cholesterol synthesis in HepG2 cells.<sup>1</sup>

### References

1. Bijl, N., Scheij, S., Houten, S., *et al.* The glucosylceramide synthase inhibitor N-(5-adamantane-1-yl-methoxy-pentyl)-deoxynojirimycin induces sterol regulatory element-binding protein-regulated gene expression and cholesterol synthesis in HepG2 cells. *J. Pharmacol. Exp. Ther.* **326**(3), 849-855 (2008).
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3. Overkleeft, H.S., Renkema, G.H., Neele, J., *et al.* Generation of specific deoxynojirimycin-type inhibitors of the non-lysosomal glucosylceramidase. *J. Biol. Chem.* **273**(41), 26522-26527 (1998).
4. Shen, C., Bullens, D., Kasran, A., *et al.* Inhibition of glycolipid biosynthesis by N-(5-adamantane-1-yl-methoxy-pentyl)-deoxynojirimycin protects against the inflammatory response in hapten-induced colitis. *International Immunopharmacology* **4**, 939-951 (2004).
5. Aerts, J.M., Ottenhoff, R., Powlson, A.S., *et al.* Pharmacological inhibition of glucosylceramide synthase enhances insulin sensitivity. *Diabetes* **56**, 1341-1349 (2007).

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