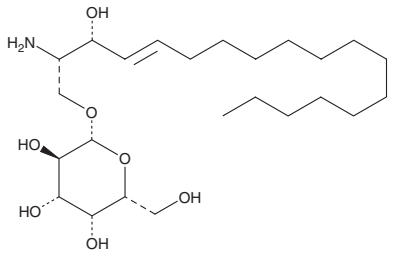
PRODUCT INFORMATION

Galactosylsphingosine (d18:1)

Item No. 20338

CAS Registry No.:	2238-90-6
Formal Name:	2S-amino-3R-hydroxy-4E-octadecen-1-yl-β-D-galactopyranoside
Synonyms:	Galactosylsphingosine, Psychosine
MF:	$C_{24}H_{47}NO_7$
FW:	461.6
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

Galactosylsphingosine (d18:1) is supplied as a crystalline solid. A stock solution may be made by dissolving the galactosylsphingosine (d18:1) in the solvent of choice, which should be purged with an inert gas. Galactosylsphingosine (d18:1) is soluble in the organic solvent ethanol at a concentration of approximately 1 mg/ml.

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 galactosylsphingosine (d18:1) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of galactosylsphingosine (d18:1) in PBS, pH 7.2, is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Galactosylsphingosine is a bioactive sphingolipid.[1-3] It potentiates LPS-induced production of inflammatory cytokines, decreases the mitochondrial membrane potential, and induces cell death in mouse astrocytes, effects that can be reversed by the sphingosine-1-phosphate receptor agonist FTY720 phosphate (Item No. 10008639).[2] Galactosylsphingosine (0.1 and 1 µM) induces demyelination of mouse cerebellar slices. It inhibits PDGF-induced translocation of PKC to the cell surface and induces apoptosis in MO3.13 oligodendrocytes.[5] Galactosylsphingosine (10 and 15 µM) inhibits gene transcription mediated by peroxisome proliferator-activated receptor α (PPARα) in reporter assays using C6 glial cells.[4] Levels of galactosylsphingosine are increased in postmortem brain from patients with Krabbe disease, a lysosomal storage disorder characterized by a β-galactosylceramidase deficiency.[1-4]

References

1. Hawkins-Salsbury, J., Parameswar, A.R., Jiang, X., *et al. J. Lipid. Res.* **54(12)**, 3303-3311 (2013).
2. O'Sullivan, C. and Dev, K.K. *J. Cell. Sci.* **128(21)**, 3878-3887 (2015).
3. Hannun, Y. A., Bell, R. M. *Science* **235(4789)**, 670-674 (1987).
4. Haq, E., Contreras, M.A., Giri, S., *et al. Biochem. Biophys. Res. Commun.* **343(1)**, 229-238 (2006).
5. Wang, L., Yang, Z., Lu, F., *et al. Molecules* **19(8)**, 12676-12689 (2014).

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