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Produktinformation

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- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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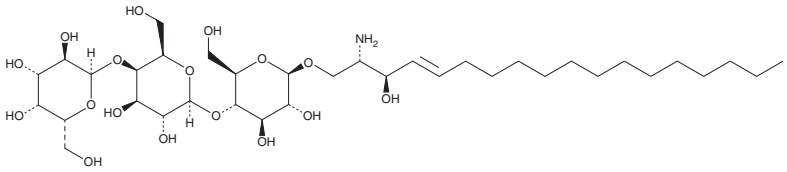
PRODUCT INFORMATION

Lyso-Globotriaosylceramide (d18:1)

Item No. 24873

CAS Registry No.:	126550-86-5
Formal Name:	(2S,3R,4E)-2-amino-3-hydroxy-4-octadecen-1-yl O-α-D-galactopyranosyl-(1→4)-O-β-D-galactopyranosyl-(1→4)-β-D-glucopyranoside
Synonyms:	Globotriaosylsphingosine (d18:1), Lyso-Ceramide Trihexoside, Lyso-Gb_3
MF:	$C_{36}H_{67}NO_{17}$
FW:	785.9
Purity:	≥98%
Supplied as:	A 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

Lyso-globotriaosylceramide (d18:1) is supplied as a solid. A stock solution may be made by dissolving the lyso-globotriaosylceramide (d18:1) in the solvent of choice. Lyso-globotriaosylceramide (d18:1) is soluble in a 4:3:1 solution of chloroform:methanol:water.

Description

Lyso-globotriaosylceramide is a form of globotriaosylceramide that is lacking the fatty acyl group. It binds to Shiga toxin 1 (Stx1) in the presence of cholesterol and phosphatidylcholine but does not bind Stx2.[1] It also reduces viability and aggregation of human neutrophils induced by phorbol 12-myristate 13-acetate (PMA; Item No. 10008014) when used at concentrations of 50 and 1 µM, respectively.[2] Lyso-globotriaosylceramide accumulates in the brain, heart, kidney, liver, lung, and spleen in a mouse model of Fabry disease, a lysosomal storage disorder characterized by a deficiency in the enzyme α-galactosidase A.[3] It also accumulates in the urine, kidney, and plasma of patients with Fabry disease.[4] Lyso-globotriaosylceramide levels decrease in response to administration of the α-galactosidase inhibitor 1-deoxygalactonojirimycin (migalastat; Item No. 17179) in a transgenic mouse model of Fabry disease. Decreases in plasma and urine concentrations of lyso-globotriaosylceramide have been used as a biomarker for efficacy of enzyme replacement therapy (ERT) and other therapies in the treatment of Fabry disease.

References

1. Gallegos, K.M., Conrady, D.G., Karve, S.S., *et al.* Shiga toxin binding to glycolipids and glycans. *PLoS One* **7(2)**, e30368 (2012).
2. Fiore, S., Nicolaou, K.C., Caulfield, T., *et al.* Evaluation of synthetic sphingosine, lysosphingolipids and glycosphingolipids as inhibitors of functional responses of human neutrophils. *Biochem. J.* **266(1)**, 25-31 (1990).
3. Kamani, M.A., Provençal, P., Boutin, M., *et al.* Glycosphingolipid storage in Fabry mice extends beyond globotriaosylceramide and is affected by ABCB1 depletion. *Future Sci. OA.* **2(4)**, FS0147, (2016).
4. Young-Gqamana, B., Brignol, N., Chang-H.H., *et al.* Migalastat HCl reduces globotriaosylsphingosine (lyso-Gb3) in Fabry transgenic mice and in the plasma of Fabry patients. *PLoS One* **8(3)**, e57631 (2013).

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