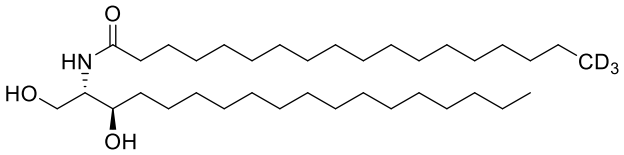
PRODUCT DATA SHEET

N-*omega*-CD_3-Octadecanoyl-D-*erythro*-dihydrosphingosine

Catalog number: 2202
Synonyms: N-C18:0-CD_3-D-*erythro*-Dihydroceramide; N-Stearoyl-CD_3-D-*erythro*-sphinganine
Source: synthetic
Solubility: hot ethanol, DMF, DMSO, chloroform/methanol 2:1

CAS number: N/A
Molecular Formula: $C_{36}H_{70}D_3NO_3$
Molecular Weight: 571
Storage: -20°C
Purity: TLC: >98%, GC: >98%, HPLC >98%
Identity confirmed by MS
TLC System: chloroform/methanol (95:5 by vol.)
Appearance: solid



Application Notes:

This product is a well-defined dihydroceramide containing a deuterated stearic acid acylated to a sphinganine base making it an ideal stable isotope-labeled standard for lipidomic studies using mass spectrometry. Stable isotope-labeled tracers are ideal for studies involving the metabolism and various metabolites of a lipid and can be used for the quantitative evaluation of major lipid pathways.[1] Lipidomics has shown great success in the use of deuterium labeled compounds in identifying and quantifying individual molecular species by the use of tandem mass spectrometry.[2]

Dihydroceramide is a critical intermediate in the *de novo* synthesis of ceramide, leading to many complex sphingolipids. It is synthesized by the acylation of sphinganine (dihydrosphingosine) and is subsequently converted to ceramide *via* the enzyme dihydroceramide desaturase or into phytosphingosine *via* the enzyme C4-hydroxylase.[3] Inhibition of ceramide synthase by some fungal toxins (such as fumonisin B1) causes an accumulation of sphinganine and sphinganine-1-phosphate and a decrease in dihydroceramide and other dihydrosphingolipids, leading to a number of diseases including oesophageal cancer.[4] The dihydroceramide desaturase inhibitor N-(4-Hydroxyphenyl) retinamide (4-HPR) has been tested as an anti-cancer agent by inhibiting the dihydroceramide desaturase enzyme in cells resulting in a high concentration of dihydroceramide and dihydro-sphingolipids and this is thought to be the cause of its anti-cancer effects.[5] Oxidative stress in cells causes an increase in the amount of dihydroceramide by potently inhibiting the desaturase enzyme.[6] Dihydroceramide inhibits the formation of channels by ceramides and may thus reduce ceramide induced apoptosis in cells.[7]

Selected References:

1. Magkos, F. and Mittendorfer, B., "Stable isotope-labeled tracers for the investigation of fatty acid and triglyceride metabolism in humans in vivo" *Clin Lipidol*. Vol. 4 pp. 215–230, *2009*
2. Byun, H. and Bittman, R. Selective deuterium labeling of the sphingoid backbone: facile syntheses of 3,4, 5-trideuterio-d-*erythro*-sphingosine and 3-deuterio-d-*erythro*-sphingomyelin" *Chem Phys Lipids*, Vol. 163(8) pp. 809-813, *2010*
3. Y. Mizutani, A. Kihara, and Y. Igarashi "Identifcation of the human sphingolipid C4-hydroxylase, hDES2, and its up-regulation during keratinocyte differentiation" *FEBS Letters*, vol. 563 pp. 93-97, *2004*
4. J. Soriano et al. "Mechanism of action of sphingolipids and their metabolites in the toxicity of fumonisin B1" *Progress in Lipid Research*, Vol. 44 pp. 345-356, *2005*
5. W. Zheng "Fenretinide increases dihydroceramide and dihydrosphingolipids due to inhibition of dihydroceramide desaturase" Georgia Institute of Technology, *2006*
6. J. Idkowiak-Baldys et al. "Dihydroceramide Desaturase Activity is Modulated by Oxidative Stress" Biochem. J., Vol. 427(2) pp. 265-274, 2010
7. J. Stiban et al. "Dihydroceramide hinders ceramide channel formation: Implications on apoptosis" *Apoptosis*, Vol. 11(5) pp. 773-780, *2006*

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