

PRODUCT DATA SHEET

N-Hexanoyl-biotin-D-erythro-dihydrospingosine

Catalog number: 2212

Synonyms: N-C6:0-Biotin-sphinganine;
N-C6:0-Biotin-dihydroceramide

Source: synthetic

Solubility: chloroform/methanol 4:1

CAS number: N/A

Molecular Formula: C₃₄H₆₄N₄O₅S

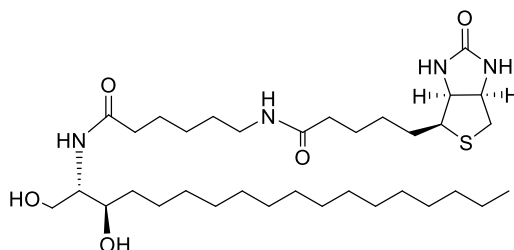
Molecular Weight: 641

Storage: -20°C

Purity: TLC >98%, identity confirmed by MS

TLC System: chloroform/methanol (80:20 by vol.)

Appearance: solid



Application Notes:

This dihydroceramide analogue contains a biotin unit attached to the amine of the dihydrospingosine moiety via a hexanoic acid linker and is ideal for use in sphingolipid studies. The biotin structure allows for attachment of the ceramide to streptavidin and avidin making it extremely useful for binding to substrates and for toxin detection¹.

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 and is subsequently converted to ceramide *via* the enzyme dihydroceramide desaturase or into phytosphingosine *via* the enzyme C4-hydroxylase.² Inhibition of ceramide synthase by some fungal toxins (such as fumonisin B1) causes an accumulation of dihydrospingosine and sphinganine-1-phosphate and a decrease in dihydroceramide and other dihydrospingolipids, leading to a number of diseases including oesophageal cancer.³ The dihydroceramide desaturase inhibitor N-(4-Hydroxyphenyl)-retinamide (4-HPR) has been tested as an anti-cancer agent; It inhibits the dihydroceramide desaturase enzyme in cells, resulting in a high concentration of dihydroceramide and dihydrospingolipids and this is thought to be the cause of its anti-cancer effects.⁴ Oxidative stress in cells causes an increase in the amount of dihydroceramide by potentially inhibiting the desaturase enzyme.⁵ Dihydroceramide inhibits the formation of channels by ceramides and may thus reduce ceramide induced apoptosis in cells.⁶ While ceramide is well known for promoting apoptosis, dihydroceramide has long been considered to be inactive. However, there has recently been evidence that an accumulation of dihydroceramide can induce cell cycle arrest.⁷

Selected References:

1. A. Mukhopadhyay et al. "Direct interaction between the inhibitor 2 and ceramide *via* sphingolipid-protein binding is involved in the regulation of protein phosphatase 2A activity and signaling" *FASEB*, Vol. 23(3) pp. 751-763, 2009
2. Y. Mizutani, A. Kihara, and Y. Igarashi "Identification of the human sphingolipid C4-hydroxylase, hDES2, and its up-regulation during keratinocyte differentiation" *FEBS Letters*, vol. 563 pp. 93-97, 2004
3. 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
4. W. Zheng "Fenretinide increases dihydroceramide and dihydrospingolipids due to inhibition of dihydroceramide desaturase" Georgia Institute of Technology, 2006
5. J. Idkowiak-Baldys et al. "Dihydroceramide Desaturase Activity is Modulated by Oxidative Stress" *Biochem. J.*, Vol. 427(2) pp. 265-274, 2010
6. J. Stiban et al. "Dihydroceramide hinders ceramide channel formation: Implications on apoptosis" *Apoptosis*, Vol. 11(5) pp. 773-780, 2006
7. J. Kraveka et al. "Involvement Of The Dihydroceramide Desaturase In Cell Cycle Progression In Human Neuroblastoma Cells" *Journal of Biological Chemistry*, Vol. 282(23) pp. 16718-16728, 2007

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