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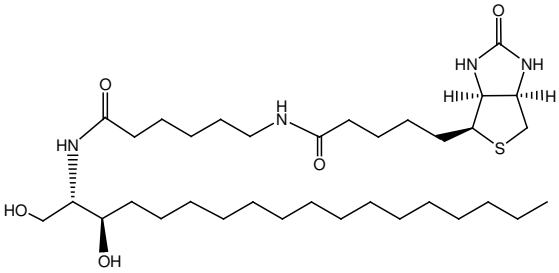
# PRODUCT DATA SHEET

## N-Hexanoyl-biotin-D-*erythro*-dihydrosphingosine

**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:** $C_{34}H_{64}N_4O_5S$
**Molecular Weight:** 641
**Storage:** -20°C
**Purity:** TLC >98%, identity confirmed by MS
**TLC System:** chloroform/methanol 80:20
**Appearance:** solid




## Application Notes:

This dihydroceramide analogue contains a biotin unit attached to the amine of the dihydrosphingosine 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 detection[1].

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 dihydrosphingosine and sphinganine-1-phosphate and a decrease in dihydroceramide and other dihydrosphingolipids, 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 dihydro-sphingolipids 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 potently 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:

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2. 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*
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 dihydrosphingolipids 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*