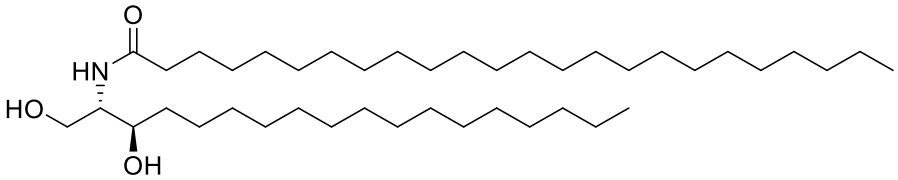
PRODUCT DATA SHEET

N-Tetracosanoyl-D-*erythro*-dihydrosphingosine

Catalog number: 2093
Synonyms: N-C24:0-D-*erythro*-dihydroceramide; N-Tetracosanoyl-D-*erythro*-sphinganine; N-Lignoceryl-D-*erythro*-dihydrosphingosine
Source: synthetic
Solubility: chloroform/methanol 2:1

CAS number: N/A
Molecular Formula: $C_{42}H_{85}NO_3$
Molecular Weight: 652
Storage: -20°C
Purity: TLC: >98%; HPLC; identity confirmed by MS
TLC System: chloroform/methanol (95:5 by vol.)
Appearance: solid



Application Notes:

Dihydroceramide is a critical intermediate in the *de novo* synthesis of ceramide, leading to many complex sphingolipids. It is synthesized by the acylation of dihydrosphingosine and is subsequently converted to ceramide *via* the enzyme dihydroceramide desaturase or into phytosphingosine *via* the enzyme C4-hydroxylase.[1] 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.[2] 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.[3] Oxidative stress in cells causes an increase in the amount of dihydroceramide by potently inhibiting the desaturase enzyme.[4] Dihydroceramide inhibits the formation of channels by ceramides and may thus reduce ceramide induced apoptosis in cells.[5] Skin cells contain significant amounts of long chain ceramides, such as dihydroceramides, that are vital for maintaining skin barrier functions.[6]

Selected References:

1. 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*
2. 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*
3. W. Zheng "Fenretinide increases dihydroceramide and dihydrosphingolipids due to inhibition of dihydroceramide desaturase" Georgia Institute of Technology, *2006*
4. J. Idkowiak-Baldys et al. "Dihydroceramide Desaturase Activity is Modulated by Oxidative Stress" Biochem. J., Vol. 427(2) pp. 265-274, 2010
5. J. Stiban et al. "Dihydroceramide hinders ceramide channel formation: Implications on apoptosis" *Apoptosis*, Vol. 11(5) pp. 773-780, *2006*
6. S. Grond et al., "PNPLA1 Deficiency in Mice and Humans Leads to a Defect in the Synthesis of Omega-O-Acylceramides" *J Invest Dermatol*. Vol. 137(2) pp. 394-402, *2017*