

PRODUCT DATA SHEET

N-Hexanoyl-biotin-sulfatide

Catalog number: 2207

Synonyms: N-C6:0-Biotin-sulfatide; N-Hexanoyl-biotin-sphingosyl-*beta*-D-galactoside-3-sulfate

Source: semisynthetic, bovine

Solubility: chloroform/methanol/water 2:1:0.1; methanol/water 9:1; DMF

CAS number: N/A

Molecular Formula: C₄₀H₇₂N₄O₁₃S₂

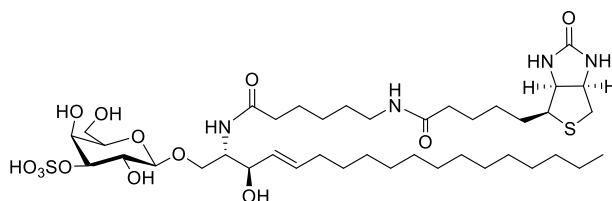
Molecular Weight: 881

Storage: -20°C

Purity: TLC: >98%, identity confirmed by MS

TLC System: chloroform/methanol/DI water (60:30:5 by vol.)

Appearance: solid



Application Notes:

N-Hexanoyl-biotin-sulfatide contains a biotin unit attached to the amine of the sphingosine via a hexanoic acid linker and is ideal for use in sphingolipid studies. The biotin structure allows for the attachment of the sulfatide to streptavidin, avidin, or any other biotin binding protein making it extremely useful for substrate and toxin detection¹. The avidin-biotin complex is the strongest known non-covalent interaction between a protein and ligand. The formation of the bond is very rapid and once formed is stable with regards to pH, temperature, organic solvents, and denaturing agents. The biotin label is attached via a 6-carbon linker reducing the interaction of the biotin with the sulfo-galactose.

Sulfatide is a type of sulfolipid that is found primarily in the central nervous system and is a myelin-specific sphingolipid. A deficiency of sulfatide in white and gray matter has been associated with Alzheimer's disease and other types of dementia. Apolipoprotein E plays an important regulating role in the metabolism of sulfatides.² The production of anti-sulfatide antibodies in the cerebrospinal fluid, leading to a deficiency in sulfatides, may be a cause of degeneration of the myelin sheath, leading to multiple sclerosis and other demyelinating diseases.³ Metachromatic leukodystrophy is an inherited disorder characterized by a deficiency of the lysosomal enzyme arylsulfatase A and the subsequent accumulation of sulfatide in neural and visceral tissues.⁴ Sulfatide also regulates the differentiation of oligodendroblasts. Central nervous system (CNS) myelin is strongly inhibitory to growing axons and sulfatides present in the myelin of the CNS have been identified as major myelin-associated axon growth inhibitors.⁵ Sulfatides in the myelin, especially *cis*-tetracosenoyl-sulfatides, stimulate a distinct population of CD1d-restricted natural killer T cells giving these sulfatides important implications for the design of therapeutics that target T cells reactive for myelin glycolipids in autoimmune diseases of the central nervous system.⁶

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. H. Cheng, Y. Zhou, D. M. Holtzman, X. Han "Apolipoprotein E mediates sulfatide depletion in animal models of Alzheimer's disease." *Neurobiology of Aging* August 2008
3. Ramesh C. Halder, A. Jahng, I. Maricic and Vipin Kumar "Mini Review: Immune Response to Myelin-Derived Sulfatide and CNS-Demyelination" *Neurochemical Research*, February, Vol. 32(2): 257, 2007
4. Phillip D. Whitfield, Peter C. Sharp, David W. Johnson, Paul Nelson and Peter J. Meikle "Characterization of Urinary Sulfatides in Metachromatic Leukodystrophy Using Electrospray Ionization-Tandem Mass Spectrometry" *Molecular Genetics and Metabolism*, May Vol. 73(1): 30, 2001
5. A. Winzeler et al. "The Lipid Sulfatide Is a Novel Myelin-Associated Inhibitor of CNS Axon Outgrowth" *The Journal of Neuroscience*, vol. 31 pp. 6481-6492, 2011
6. D. Zajonc et al. "Structural basis for CD1d presentation of a sulfatide derived from myelin and its implications for autoimmunity" *The Journal of Experimental Medicine*, vol. 202 pp. 1517-1526, 2005

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