PRODUCT DATA SHEET

N-Hexanoyl-biotin-disialoganglioside GD₃

**Catalog number:** 2055  
**Synonyms:** Biotin-C₆:0-GD₃  
**Source:** semi-synthetic, bovine buttermilk  
**Solubility:** chloroform/methanol/DI water, 2:1:0.1  
**CAS number:** N/A  
**Molecular Formula:** C₆₈H₁₁₆N₆O₃₁S  
**Molecular Weight:** 1546  
**Storage:** -20°C  
**Purity:** TLC: >98%; identity confirmed by MS  
**TLC System:** chloroform/methanol/2.5N ammonium hydroxide (60:40:9)  
**Appearance:** solid

**Application Notes:**
This ganglioside GD₃ analogue contains a biotin unit attached to the amine of the sphingosine moiety via a hexanoic acid linker and is ideal for use in ganglioside studies. The biotin structure allows for attachment of the ganglioside to streptavidin and avidin substrates making it extremely useful for binding to substrates and for toxin detection.¹

Gangliosides are acidic glycosphingolipids containing one or more sialic acids that generally form lipid rafts in the outer leaflet of the cell plasma membrane, especially in neuronal cells in the central nervous system.²,³ They participate in many cellular activities including proliferation, differentiation, adhesion, signal transduction, cell-to-cell interactions, tumorigenesis, and metastasis.⁴ The accumulation of gangliosides has been linked to several diseases including Tay-Sachs and Sandhoff disease while an autoimmune response against gangliosides can lead to Guillain-Barre syndrome. Gangliosides act as receptors for various toxins and bacteria, accumulate in various tumors, and aid in many neuronal functions.

Disialoganglioside GD₃ is predominantly expressed during neuronal development and its expression becomes very limited in adult tissues. GD₃ expression is unusually high in basal cell carcinomas and malignant melanomas and is thought to be a human melanoma-specific antigen. Although GD₃ is not immunogenic it has been investigated as a tool for immunotargeting human melanoma cells.⁵ Over expression of GD₃ has led to apoptosis by recruiting mitochondria to apoptotic pathways and suppressing NF-κB activation and subsequent κB-dependent gene induction.⁶ Increased levels of GD₃ have also been found to be associated with proliferative diseases, such as atherosclerosis.

**Selected References:**

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