テクノケミカル
〒113-0021 東京都文京区本駒込 1 丁目 27-9
TEL: 03-3947-7310 FAX: 03-3947-7306
E-mail: info@technochemical.com
HP:www.technochemical.com

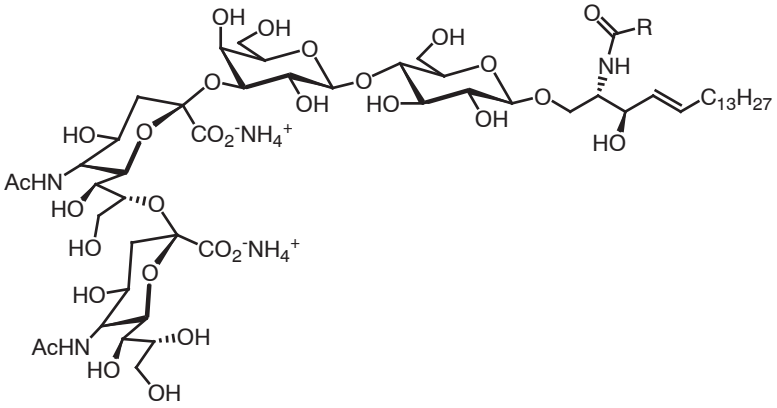
MATREYA LLC
168 Tressler Street
Pleasant Gap, PA 16823
Tel: 800-342-3595 / 814-359-5060
Fax: 814-359-5062
Email: customerservice@matreya.com
Web: www.matreya.com

PRODUCT DATA SHEET

製品名: Disialoganglioside GD3 (NH_4^+ salt)

カタログ番号: 1504
別名:
由来: Natural, bovine buttermilk
溶解度: Chloroform/Methanol (2: 1)
Forms micellar solution in water
CAS 番号: 62010-37-1

分子式: $C_{75}H_{125}N_3O_{29}\cdot 2NH_3$
分子量: 1543+2NH_3
(tricosanoyl, NH_4^+ salt)
保存温度: -20 ºC
純度: TLC >98%
TLC 溶媒: Chloroform/ Methanol/ 2.5 N
Ammonium hydroxide (60: 40: 9 by Vol.)
形状: Solid



Application notes:

Gangliosides[1] are acidic glycosphingolipids that form lipid rafts in the outer leaflet of the cell plasma membrane, especially in neuronal cells in the central nervous system.[2] They participate in cellular proliferation, differentiation, adhesion, signal transduction, cell-to-cell interactions, tumorigenesis, and metastasis. The accumulation of gangliosides has been linked to several diseases including Tay-Sachs and Sandhoff disease. 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.[3] 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.[4] Increased levels of GD3 have also been found to be associated with proliferative diseases, such as atherosclerosis.

アプリケーションノート
ガングリオシドは酸性のスフィンゴ糖脂質であり、特に中枢神経系神経細胞の外側の単細胞質に脂質ラフトを形成します。細胞増殖、細胞分化、接着、シグナル変換、細胞間相互作用、腫瘍形成や癌細胞の転移に関与します。ガングリオシドの蓄積は、テイサックス（Tay-Sachs）病やサンドホフ（Sandhoff）病を含む様々な疾病に関連があります。GD3は神経細胞発達の間、高率に発現しますが、成体組織ではその発現は非常に限られています。GD3の発現は基底細胞癌と悪性黒色種では異常に高く、ヒト黒色腫特有抗原と考えられています。GD3は免疫発生性ではないものの、ヒト黒色腫細胞の免疫標的のツールとして研究されてきました。GD3の過剰発現によって、ミトコンドリアがアポトーシス代謝にシフトしたり、NF-κB 活性が抑制されて κB 制御下遺伝子の発現誘導が起きたりします。GD3量の増加は、アテローム性動脈硬化症のような増殖性疾患とも関連性があることも解っています。

Selected References:

1. L. Svennerholm et al. (eds.), "Structure and Function of Gangliosides" *New York, Plenum, 1980*
2. T. Kolter, R. Proia, K. Sandhoff, "Combinatorial Ganglioside Biosynthesis" *J. Biol. Chem*., Vol. 277:29, pp. 25859-25862, *2002*
3. H. Jennings et al. "Bioengineering of Surface GD3 Ganglioside for Immunotargeting Human Melanoma Cells" *Journal of Biological Chemistry*, Vol.279:24 pp. 25390, *2004*
4. J. Fernandez-Checa et al. "Ganglioside GD3 Sensitizes Human Hepatoma Cells to Cancer Therapy" *Journal of Biological Chemistry*, Vol. 277:51 pp. 49870, *2002*