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Commentary

Importance of Gangliosides and its Function in Central Nervous System

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DESCRIPTION

Gangliosides are important components of biological membranes, consisting of amphipathic molecules with a negatively charged silica acid containing hydrophilic glycan head group linked to a hydrophobic ceramide tail. There are hundreds of gangliosides identified and described in all vertebrate cells, with a high concentration in the nervous system, due to the numerous variations in the length, saturation, and hydroxylation of the lipid moiety and the composition and structure of the sugars in the oligosaccharide chain.

A series of particular glycosyltransferases gradually add monosaccharaides to the ceramide that has been gradually formed in the endoplasmic reticulum before being transported to the Golgi apparatus to begin the biosynthesis of gangliosides. Except for GM4, which is created from galactosylceramide, most gangliosides are derived from lactosylceramide. The simple gangliosides GM3, GD3, and GT3 are produced by the sequential addition of 1, 2, or 3 silica-acid molecules to the galactose residue of lactosylceramide by sialyltransferases. These simple gangliosides act as precursors for the synthesis of more complex gangliosides with their glycan facing the extracellular space, gangliosides are mostly inserted in the no cytosolic monolayer and guided to the plasma membrane. Lipid rafts are specialized membrane micro domains that are stabilized by Tran's association with other sphingolipids, cholesterol, and membrane proteins. This interaction is made possible by the molecular structure and biophysical characteristics of lipid rafts.

Roles of gangliosides in nervous system ganglioside GM1 in the adult central nervous system (CNS), GM1, an a series complex ganglioside produced from the simple ganglioside GM3, is the most prevalent ganglioside. The plasma membrane of glial cells and neurons include GM1, which makes up 10–20% of the total ganglioside content in the human CNS. In recent years, GM1 has received the most research attention. GM1 production in humans starts off at low levels at birth, rises gradually through adulthood, and then gradually declines as people age, which has been related to cognitive loss and neurodegenerative disorders. The pattern of GM1 expression suggests that it may be significant in late-phase physiological and/or developmental processes.

The regeneration of the adult peripheral nervous system (PNS) appears to be assisted by GM1. Atomized neurons of the dorsal root ganglia showed increased production of Neu3 sialitase, the enzyme that transforms GD1a, GT1b, and GD1b to GM1, and inhibition of this enzyme decreased axonal regrowth. Neu3 sialidase in central neurons is not altered after injury, but when this enzyme is added exogenously to retinal explants, axonal development is stimulated, indicating a critical role for GM1 in regeneration.

The CNS may suffer damage from both GM1 overexpression and deficiency. The crucial link between GM1 levels and CNS development and function is further supported by the fact that GM1 is metabolized by galactosidase, and mutations in the galactosidase encoding gene (GLB1) induce GM1 accumulation in lysosomes, resulting in GM1 gangliosidosis, and unique and severely degenerative condition. Through its interaction with tropomyosin receptor kinase receptors in lipid rafts, GM1 has been shown in numerous studies to have a significant neurotrophic effect on various cell types. Exogenous GM1 injection can influence the survival of CNS dopaminergic, glutamatergic, and cholinergic neurons, suggesting a potential function for this molecule in the treatment of PD, AD, and other neurodegenerative diseases. Numerous preclinical models of brain injury and neurodegeneration have been used to thoroughly study the neuroprotective properties of GM1. General anesthetics with a higher risk of cognitive impairment in young children, such as chloroform and remifentanil, were introduced to NSCs in laboratory and found to cause cell death.

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