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Abstract

Gene knockout mice of glycosyltransferases have clearly showed roles of their products in the bodies, while there are examples where phenotype of knockout were much less severe than expected probably due to functional redundancy. The most striking novel finding obtained from ganglioside-deficient mice was that progressive inflammatory reaction took place, leading to neurodegeneration. In particular, dysfunction of complement-regulatory proteins due to deteriorated architecture of lipid rafts seemed to be essential mechanisms for the inflammation. Furthermore, roles of gangliosides in neurons were demonstrated by neuron-specific transgenic of B4galnt1 with genetic background of B4galnt1 deficiency. From study of gene knockout mice of St8sia1, new roles of b-series gangliosides in leptin secretion from adipocytes, and roles of a-series gangliosides in leptin receptor, ObR in hypothalamus were demonstrated, leading to apparent intact balance of energy. Essential roles of b-series gangliosides in malignant properties of gliomas were also shown, suggesting their roles in the regulation of inflammation and proliferation in nervous tissues. How to apply these findings for the control of newly discovered patients with ganglioside deficiency remains to be investingated.

Keywords: ganglioside; knockout; neurodegeneration; inflammation; complement; astrocyte; glioma