The change of functional roles of sulfatide in early and chronic inflammatory pain.

Motoki Morita^{1,2}, Shun Watanabe^{1,2}, Natsumi Nomura^{1,2}, Misa Oyama^{1,2}, Takashi Iwai^{1,2}, Mitsuo Tanabe^{1,2}

¹Lab. Pharm., Sch. Pharm., kitasato Univ., ²Medical Reserch Labolatory, Sch. Pharm., Kitasato Univ.

Glycosphingolipids (GSLs) are abundant in the nervous system and play important roles in cellular interaction and intracellular signal transduction. We found that gene expressions of several glycosyltransferases in GSLs biosynthesis pathway were increased in the spinal cord one day after inflammation caused by complete Freund's adjuvant (CFA). In naïve mice, intrathecal injection of GSLs, sulfatide and b-series gangliosides, synthesized from enzymes encoded by upregulated genes caused allodynia. Inhibitors of glial activation blocked or TNF α blocked sulfatide-induced allodynia. On the other hand, gene expressions of glycosyltransferases including sulfatide synthase were decreased 15 days (chronic inflammation) after CFA injection. Thus, we examined the effects of intrathecal sulfatide injection on chronic inflammatory pain. 21 days after CFA treatment, mice that were received daily injection of sulfatide showed reduced inflammatory pain. It appears that sulfatides play the different roles in early or chronic inflammatory pain. However, the molecular mechanisms underlying the change of function of sulfatide remain unclear. Future studies are needed to reveal how sulfatide induced both algesic effects in early inflammatory pain and analgesic effects in chronic inflammatory pain.