Platelet-activating factor (PAF) is increased in neuropathic pain mice: different regulation of PAF levels between the spinal cord and the dorsal root ganglia

Shota Yamamoto[1], Tomomi Hashidate-Yoshida[1], Hideo Shindou[1,2,3,4], Takao Shimizu[1]

[1]Department of Lipid Signaling, National Center for Global Health and Medicine, [2]Department of Lipid Science, Graduate School of Medicine, The University of Tokyo, [3]AMED-CREST, [4]AMED-P-CREATE

Platelet-activating factor (PAF) is a potent phospholipid mediator, which is involved in the pathology of neuropathic pain after peripheral nerve injury (PNI). In PAF receptor- and its biosynthetic enzyme lysophosphatidylcholine acyltransferase 2 (LPCAT2)- deficient mice, neuropathic hypersensitivity was significantly attenuated. However, regulation of PAF level after PNI remains to be elucidated. Here, we show that PNI increases PAF levels in the spinal cord and in the dorsal root ganglia (DRG). While PAF biosynthetic activity and mRNA expression of LPCAT2 were increased in both tissues, enzymatic activity of PAF degradation and mRNA expression of plasma type of PAF-acetylhydrolase (PAF-AH), which is one of PAF degradation enzymes, were decreased only in the DRG. These results suggest that distinct mechanisms exist between the spinal cord and the DRG to regulate PAF levels after PNI, and then increased PAF levels may contribute to neuropathic pain.