研究業績 英文表記

和文	
表題	エイコサペンタエン酸強化ホスファチジルコリンはオートファジー・インフラマソーム経路 を介して A β 1・42 誘発の神経毒性を軽減させた
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英文	
Title	Eicosapentaenoic Acid-Enriched Phosphatidylcholine Mitigated Aβ1-42-Induced Neurotoxicity via Autophagy-Inflammasome Pathway
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Abstract	Recent studies indicated that neuroinflammation contributes to the exacerbation of Alzheimer's disease (AD) and plays an important role in AD. The NOD-like receptor protein 3 (NLRP3) inflammasome, which is an important component of innate immune system, is associated with a wide range of human central nervous system disorders, including AD. Most of the studies focus on the protective effects of docosahexaenoic acid (DHA) in AD, but eicosapentaenoic acid (EPA) has rarely been involved. Here, we investigate the effects of EPA in the forms of phosphatidylcholine (EPA-PC) and ethyl esters (EPA-EE) in improving $A\beta1-42$ -induced neurotoxicity. The spatial memory ability and the biochemical changes in the hippocampus were measured, including glial cell activation, tumor necrosis factor α production, NLRP3 inflammasome activation, and autophagic flux. The present results showed that the AD rats were significantly protected from spatial memory loss by the supplementation (EPA + DHA = 60 mg/kg, i.g., 20 days) of EPA-PC, while EPA-EE showed no significant benefit. Further mechanism studies suggested that EPA-PC could inhibit A β -induced neurotoxicity by alleviating NLRP3 inflammasome activation and enhancing autophagy. These findings indicate that EPA could improve cognitive deficiency in A $\beta1$ -42-induced AD rats via autophagic inflammasomal pathway and the bioactivity differs in its molecular form.
keyword	β -amyloid1-42, EPA enriched phosphatidylcholine, NOD-like receptor protein 3 inflammasome, autophagy