

Abstract

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Reductions of acetylcholine release and nerve growth factor expression are correlated with memory impairment induced by interleukin-1beta administrations: effects of omega-3 fatty acid EPA treatment.

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BACKGROUND: Interleukin (IL)-1beta may play an important role in Alzheimer's disease. However, the relationships between glucocorticoids and acetylcholine (ACh), and between neurotrophins and ACh in IL-1-induced memory deficits are unknown.

OBJECTIVE AND METHODS: While ethyl-eicosapentaenoate (E-EPA) has recently been reported to reduce inflammation and improve memory, cholinergic and neurotrophic mechanisms by which E-EPA improves memory is unclear. This study evaluated: (i) the correlation between ACh release and memory impairment; (ii) the effect of glucocorticoids on ACh release; (iii) the relationship between nerve growth factor (NGF) and inflammation; and (iv) the effects of E-EPA treatment on IL-1beta-induced changes.

RESULTS: Intracerebroventricular IL-1beta administrations produced a significant reduction in hippocampal ACh release in rats fed control diet, which was partially attenuated by mifepristone (RU 486) and completely blocked by IL-1 receptor antagonist. In eight-arm radial maze, significantly less ACh release was correlated with the memory deficits after IL-1beta administrations. mRNA expression of hippocampal NGF was lower, whereas IL-1beta was higher when compared with controls. E-EPA treatment significantly improved the memory, which was correlated with normalizing ACh release, and expressions of NGF and IL-1beta.

CONCLUSIONS: This study revealed important mechanisms by which IL-1beta impairs, while E-EPA improves memory through IL-1-glucocorticoid-ACh release and IL-1-NGF-ACh release pathways.

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