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Corrigendum

Omega-3 Fatty Acid Ethyl-Eicosapentaenoate Attenuates IL-1β-Induced Changes in Dopamine and Metabolites in the Shell of the Nucleus Accumbens: Involved with PLA2 Activity and Corticosterone Secretion

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Following the online publication of this article, the authors noted errors in the abstract. The correct abstract is below.

Previously, we have reported that interleukin-1 beta (IL-1) induces changes in dopaminergic (DA) and serotonergic systems in the core of the nucleus accumbens (NAc). We have also demonstrated that n-3 fatty acid ethyl-eicosapentaenoate (EPA) can significantly reduce stress and anxiety-like behaviors, corticosterone concentrations, and peripheral inflammatory response induced by IL-1 administration. Compared to the core, the shell is involved more in emotion, stress, and psychiatric diseases. However, the relationship between inflammation and the functions of DA system in the shell has not been studied. Since phospholipase (PL) A2 is a key enzyme in the arachidonic acid–eicosanoids–prostaglandin (PG)E2 pathway, and the change in NAc DA system has been associated with glucocorticoid stimulation; therefore, the hypotheses of this study are (1) that IL-1 induced changes in DA neurotransmission in the shell may be through PLA2–PGE2–corticosterone pathway; (2) EPA may attenuate IL-1 effects via inhibiting PLA2 activities, which blocks PGE2 stimulation of corticosterone. Using an *in vivo* microdialysis method, the present study showed that IL-1 administration significantly increased extracellular levels of DA, and its metabolites 3,4-dihydroxyphenylacetic acid, and homovanillic acid in the shell of the NAc. IL-1 also increased blood concentration of corticosterone and PGE2, and increased the activities of cytosolic and secretory PLA2. IL-1-induced changes were significantly attenuated by EPA treatment. Furthermore, glucocorticoid receptor antagonist mifepristone (RU486) pretreatment significantly blocked IL-1-induced changes in DA and metabolites. Quinacrine, a PLA2 antagonist significantly blocked IL-1-induced increase in PGE2 and corticosterone concentrations. These results demonstrated the hypotheses that IL-1 effects may be via PLA2–PGE2–corticosterone pathway and EPA attenuated IL-1 effects may be through the suppression of PLA2 expression, which then reduced PGE2 synthesis and corticosterone secretion.