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Oleic Acid Enhances Hippocampal Synaptic Efficacy

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Abstract : Oleic acid is a cis unsaturated fatty acid and is known to be a partially essential fatty acid due to its limited endogenous synthesis during pregnancy and lactation. Previous studies have demonstrated the role of oleic acid in neuronal differentiation and brain phospholipid synthesis. These evidences indicate a major role for oleic acid in learning and memory. Interestingly, oleic acid has been shown to enhance hippocampal long term potentiation (LTP), the physiological correlate of long term synaptic plasticity. However the effect of oleic acid on short term synaptic plasticity has not been investigated. Short term potentiation (STP) is the physiological correlate of short term synaptic plasticity which is the key underlying molecular mechanism of short term memory and neuronal information processing. STP in the hippocampal CA1 region has been known to require the activation of N-methyl-D-aspartate receptors (NMDARs). The NMDAR dependent hippocampal STP as a potential mechanism for short term memory has been a subject of intense interest for the past few years. Therefore in the present study the effect of oleic acid on NMDAR dependent hippocampal STP was determined in mouse hippocampal slices (in vitro) using Multi-electrode array system. STP was induced by weak tetanic Stimulation (one train of 100 Hz stimulations for 0.1s) of the Schaffer collaterals of CA1 region of the hippocampus in slices treated with different concentrations of oleic acid in presence or absence of NMDAR antagonist D-AP5 (30 µM) . Oleic acid at 20 (mean increase in fEPSP amplitude = ~135 % Vs. Control = 100%; P<0.001) and 30 µM (mean increase in fEPSP amplitude = ~ 280% Vs. Control = 100%); P<0.001) significantly enhanced the STP following weak tetanic stimulation. Lower oleic acid concentrations at 10 µM did not modify the hippocampal STP induced by weak tetanic stimulation. The hippocampal STP induced by weak tetanic stimulation was completely blocked by the NMDA receptor antagonist D-AP5 (30µM) in both oleic acid and control treated hippocampal slices. This lead to the conclusion that the hippocampal STP elicited by weak tetanic stimulation and enhanced by oleic acid was NMDAR dependent. Together these findings suggest that oleic acid may enhance the short term memory and neuronal information processing through the modulation of NMDAR dependent hippocampal short-term synaptic plasticity. In conclusion this study suggests the possible role of oleic acid to prevent the short term memory loss and impaired neuronal function throughout development.

Keywords : oleic acid, short-term potentiation, memory, field excitatory post synaptic potentials, NMDA receptor
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