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Commentary

Role of Phospholipid Metabolites in Neuronal Signaling and Memory Acquisition

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DESCRIPTION

Polyunsaturated Free Fatty Acids (FFAs), such as arachidonic acid, are known modulators of neurotransmission and synaptic plasticity and are released by phospholipase activity on membrane phospholipids. The precise nature of other FFA and phospholipid alterations in specific parts of the brain during learning, on the other hand, is uncertain. They observed that the largest concentrations of these analytes were identified in parts of the brain traditionally engaged in fear learning and memory, such as the amygdala, utilizing a targeted lipidomics technique to characterize FFAs and phospholipids across the rat brain. In the amygdala and prefrontal cortex, auditory fear conditioning increased saturated (especially myristic and palmitic acids) and, to a lesser extent, unsaturated FFAs.

Fear training and FFA alterations both needed N-methyl-D-aspartate receptor activation. These findings point to saturated FFAs playing a function in memory acquisition. One of the major issues in neurobiology is elucidating the molecular mechanisms that underlie learning and memory formation. Several decades of research have revealed that a variety of protein and gene responses contribute to learning and memory acquisition. Despite the obvious abundance of phospholipids and their metabolites in the brain, as well as their proven involvement in neuronal signaling, little is known about their contribution to these critical activities. In the brain, lipids perform a variety of structural, signaling, developmental, and metabolic roles.

Phospholipids, in particular, are critical components of neuronal plasma and synaptic vesicle membranes, thought to be necessary for neurotransmission, synaptic plasticity, and memory formation. The vesicular trafficking that underpins these processes is characterized by tightly regulated dynamic modulation of phospholipid membrane fluidity, curvature, and surface chemistry, in conjunction with protein/protein and protein/lipid interactions at the pre and post-synapse. These activities are aided in part by the action of particular phospholipases, which can alter the phospholipid landscape locally.

Enzymatic phospholipid remodeling produces phospholipid metabolites such as diacylglycerols, inositol triphosphate, lysophospholipids, and free fatty acids, which can influence membrane dynamics and act as lipid signaling molecules. Protein lipidation, in particular myristoylation and palmitoylation, which involve the transfer of saturated myristic and palmitic acids to synaptic proteins, is prevalent in the nervous system and essential for synaptic plasticity. The study of the involvement of phospholipid metabolites in regulating fundamental processes in memory formation is thus an important complement to protein, gene, and structural investigations in this field.

Ester-linked glycerophospholipids make up the vast bulk of phospholipids in neuronal membranes. Fatty acids are the two hydrophobic tails on the phospholipid glycerol backbone, with a hydrophilic sn-3 head group identifying the phospholipid class. Canonical phospholipids are thought to be rich in saturated fatty acids like palmitic acid (C16:0) and stearic acid (C18:0) at the sn-1 position, and unsaturated fatty acids like arachidonic acid (C20:4) and docosahexaenoic acid (DHA, C22:6) at the sn-2 position.

Unsaturated fatty acids have long been thought to be beneficial to health, and early studies linked a decrease in brain polyunsaturated fatty acids to aging and memory impairment. Phospholipase A2 (PLA2) mediates multiple neural processes that result in the release of unsaturated FFAs (especially arachidonic acid) from the sn-2 position of certain phospholipids. Arachidonic acid has been shown to modulate neurotransmitter release modulate membrane fluidity, influence LTP *via* its ability to diffuse through the synaptic cleft regulate synaptic transmission alone or in combination with other FFAs and lysophospholipids and initiate inflammation.

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