Phospholipid disruption in the brain links mouse models of repetitive mild traumatic brain injury and alzheimer's disease

Purpose

To examine longitudinal changes in brain phospholipid profiles in mouse models of TBI and AD (PSAPP mice), to determine their role.

Participants

Researchers collected samples from mouse models of TBI and AD.

How was the study conducted?

Samples from the cortex/hippocampus were collected at 'pre', 'peri' and 'post' onset of amyloid pathology (i.e. 3, 9, 15 months of age) and at timepoints post-injury (i.e. 24hrs, 3, 6, 9 and 12 months).

Findings

Total levels of phosphatidylcholine (PC), phosphatidylethanolamine (PE), LysoPE, and phosphatidylinositol (PI), including their mono/poly-unsaturated and saturated fatty acid containing species were significantly increased at acute and/or chronic time points post-injury. These lipid species in AD mice were unchanged in the hippocampus, while in the cortex the levels were significantly decreased at time points post onset of amyloidopathy. LysoPC and sphingomyelin levels showed coincidental trends in both TBI and AD models in the hippocampus, an increase at early and/or later time points examined. Arachidonic acid to decosahexaenoic acid ratio for PE containing species was increased at acute time points in the hippocampus post-TBI, and in PSAPP mice there was an increase at all time points examined.

Military Impact

Exploration of the secondary mechanisms triggered by the irregular changes in phospholipids and their regulation at the appropriate time-windows of opportunity could help to identify new and potent therapeutic strategies to effectively improve the consequences of TBI/AD pathogenesis.

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