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Oral presentation

Evidence from in vivo 31-phosphorus magnetic resonance spectroscopy phosphodiesters that exhaled ethane is a biomarker of cerebral n-3 polyunsaturated fatty acid peroxidation in humans

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This study tested the hypothesis that exhaled ethane is a biomarker of cerebral n-3 polyunsaturated fatty acid (PUFA) peroxidation in humans. Ethane is released specifically following peroxidation of n-3 PUFAs, probably via: abstraction of a hydrogen of the unsaturated carbon closest to the methyl end; isomerization to a diene radical; addition of oxygen to form a hydroperoxide; and β-scission to a hydroxyl and an alkoxy radical, the latter forming ethane by hydrogen addition. We reasoned that the cerebral source of ethane would be the docosahexaenoic acid component of membrane phospholipids. Breakdown of the latter also releases phosphorylated polar head groups, giving rise to glycerophosphorylcholine and glycerophosphorylethanolamine which can be measured from the 31-phosphorus neurospectroscopy phosphodiester peak. Schizophrenia patients were chosen because of evidence of increased free radical-mediated damage and cerebral lipid peroxidation in this disorder. Breath samples from eight patients were analyzed using mass spectrometry. Cerebral 31-phosphorus spectra were obtained from the same patients from 70 70 70 mm3 voxels using an image-selected in vivo spectroscopy pulse sequence. Ethane and percentage phosphodiester levels were positively correlated ($rs = 0.714$, $p < 0.05$), thus supporting the hypothesis that the measurement of exhaled ethane levels indexes cerebral n-3 lipid peroxidation.