Reference:

Selective Deficits in the Omega-3 Fatty Acid Docosahexaenoic Acid in the Postmortem Orbitofrontal Cortex of Patients with Major Depressive Disorder


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Summary:

The pioneering work by Dr. Joseph Hibbeln and others have reported an inverse relationship between the per capita consumption of fish/seafood containing DHA/EPA omega-3 fatty acids and the prevalence of major depressive disorder (MDD) based on cross-national and cross-sectional epidemiological surveys. Since the region of the brain known as the orbito-frontal cortex (OFC) plays a key role in the emotional and other processes involved in the pathophysiology of MDD, the present study determined the concentrations of the principle fatty acids in the postmortem OFC in 15 patients with MDD and compared these to 27 normal control subjects. The average age of the MDD and normal groups was 46 years at death; the MDD patients first experienced disease onset at a mean age of 34 years with a subsequent average duration of disease lasting 13 years.

After correcting for multiple comparisons, the authors observed DHA omega-3 as the only fatty acid that was significantly different in the OFC of the MDD patients relative to the normal controls. The DHA concentration was 22% lower in the MDD patients with the deficits in DHA concentration being greater in female patients (32% below controls) than in male patients (16% below controls). The authors conclude that their results demonstrate a selective deficit in DHA omega-3 fatty acid in the OFC of patients with MDD and suggest that their findings add to the body of evidence implicating DHA omega-3 deprivation in the pathophysiology and overall pathogenesis of MDD.
Dr. Holub's Comments:

Based on their interesting findings, the authors suggest that the OFC deficits in DHA which they observed in the MDD patients may be due to deficient intakes of dietary DHA. However, estimates of dietary DHA intakes were not included as part of this current investigation. It is possible that impairments in DHA uptake and transport from the circulation into the brain/OFC may be involved. Furthermore, an accelerated catabolism (breakdown) of DHA in the brain of MDD patients as compared to healthy controls may also contribute to the observed differences in DHA concentrations. Future randomized-controlled clinical trials using supplemental sources of DHA and/or DHA/EPA at or before the onset of MDD will be of considerable interest to determine if the rate of progression of this disorder can be attenuated via targeted omega-3 supplementation.