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Omega-3 fatty acid deficiencies in aggression and violence.

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Omega-3 highly unsaturated fatty acids (n-3 HUFAs), are selectively concentrated in neuronal membranes and are required for optimal neuronal function, promoting neurite outgrowth and preventing apoptosis. Seafood is the predominant source of these essential fatty acids which cannot be made de novo. Deficient dietary intakes increase risk of affective disorders including major depression, bipolar disorder and postpartum depression according to epidemiological studies. Excessive intakes of omega-6 fatty acids, e.g. linoleic acid from seed oils, can reduce tissue levels of n-3 HUFAs 10-fold. Greater linoleic acid intakes ranging from 1% energy to 9 % of energy correlate with 100-fold greater risk of homicide mortality across 5 countries (1960-1999). Conversely, greater seafood consumption across 36 countries correlates with 30-fold lower rates of homicide mortality. Chronic alcohol consumption depletes n-3 HUFAs from frontal cortex in primate models, and n-3 deficient diets impair frontal cortex serotonergic release in rodent models. Thus, aggression among alcoholics may, in part, be attributable to neuronal depletions in n-3 HUFAs. The causal inference of these correlational observations- that greater n-3 consumption might reduce aggression- is currently being evaluated in a randomized placebo-controlled trial of aggressive alcoholics. Here we examine if supplementation with 1.88 g/d n-3 HUFAs or placebo ameliorates psychometric measures of aggression, impulsivity, and depression among aggressive alcoholics with low fish consumption in early recovery. Psychological measures include aggression, anger/hostility, stress, impulsivity, depression, personality, risk taking behavior and emotional learning. Cerebrospinal fluid is collected at baseline and after 12 weeks of intervention to assess changes in dopamine (CSF HVA) and serotonin (CSF 5-HIAAA) metabolites. Heart rate variability is measured both at rest and during the Point Subtraction Aggression Paradigm as a marker of autonomic reactivity. Preliminary results of this study will be presented.