

**“Omega-3 Fatty Acids and Psychiatry: Theory and Practice,” Lake J.,
Psychiatric Times, Oct, 2002.**

Certain essential fatty acids (EFA) are now broadly used in Europe and North America to treat many medical and psychiatric disorders. This article reviews the proposed mechanism of action, efficacy and safety issues pertaining to uses of Omega-3 essential fatty acids in the treatment of psychiatric disorders.

Since the 1930s it has been established that certain essential fatty acids are required for normal human fetal and neonatal development (Uauy et al), and that an inadequate supply of these essential fatty acids during critical developmental periods results in pathological changes in immune function, degenerative changes in the lungs, liver and kidneys, and abnormalities in CNS maturation. It has been hypothesized that chronic deficiencies in dietary essential fatty acids (EFAs) may result in an increased incidence of multiple sclerosis, arthritis, enteritis, immune system dysfunction, heart disease, cancer, schizophrenia, bipolar disorder, diabetes and many other diseases (Rudin).

Essential fatty acids are highly unsaturated (containing many carbon-carbon double bonds) molecules that play important roles in normal cellular metabolism, growth, muscle physiology and CNS maturation and functioning. Fatty acids are named according to the position of double bonds. For example, in Omega-3 EFAs the first double bond is three carbons from the terminal (omega) position. Omega-3s and Omega-6s are the two classes of essential fatty acids of greatest relevance to the understanding of normal and pathological processes in the human body. Omega-3 essential fatty acids are concentrated in two principle forms: deep-dwelling cold water fishes like salmon and halibut, and products of cold-adapted plants including flax seed oil. The Omega-6 EFAs are concentrated in heat-adapted plants, including olive, peanut, safflower and sunflower oils.

To clearly understand the role of EFAs in brain structure and function, it is important to briefly review phospholipid metabolism. Phospholipids are comprised of a 3-carbon glycerol backbone with a terminal phosphorus group. The final step in the synthesis of phospholipids in nerve cell membranes is the attachment of highly unsaturated fatty acids by phospholipases to the glycerol molecule. The four essential fatty acids that are the normal structural elements of brain phospholipids are DGLA, and arachidonic acid (the omega-6 EFAs), and EPA and DHA (the omega-3 EFAs).

(Insert illustration here: fig. 1.1, in Horrobin, D., “The phospholipid concept of psychiatric disorders and its relationship to the neurodevelopmental concept of schizophrenia,” ch. 1, in *Phospholipid Spectrum Disorder in Psychiatry*, ed. Peet, Glen and Horrobin; Marius Press; ? date. I will fax separately)

DHA is an omega-3 fatty acid that is essential to normal fetal and neonatal maturation of the brain where it is synthesized into phospholipids that are fundamental components of nerve cell membranes. Infants who are not provided adequate amounts of necessary EFAs in utero or during the neonatal period are at risk for numerous medical complications including peripheral neuropathy, abnormal visual development, and

reduced slow-wave sleep if the deficiency is not corrected early. If the required EFAs are not present in adequate amounts, other fatty acids will be incorporated into nerve cell membranes, possibly predisposing the infant to a range of medical and psychiatric disorders. Pre-term infants are especially at risk, as they do not receive intrauterine DHA during the third trimester, and if they are not breast-fed or provided formula containing DHA, they will be susceptible to many disorders. EPA is the other Omega-3 essential for brain functioning. This fatty acid plays a central role in maintenance of nerve cell membranes from early development through adulthood.

It is important to note that essential fatty acids cannot be manufactured *de novo* by the human body, and must be acquired through diet in the form of their parent essential fatty acids: Linoleic acid (LA) and alpha-linolenic acid (ALA), which are metabolically transformed into longer chain EFAs (the Omega-3s and Omega-6s) in the liver with the aid of many co-factors, including insulin, zinc and several vitamins. EFAs enter the blood following conversion from LA and ALA in the liver, and eventually diffuse across the blood-brain barrier where they are incorporated into nerve cell membranes. At several points in their synthesis or supply to the brain, EFAs are susceptible to oxidative damage, metabolic stress mediated by cortisol, hormonal changes during puberty or menopause, viral infection, or other factors that can potentially interfere with the availability of EFAs to the body and brain, manifesting in a range of medical or psychiatric symptoms.

The Phospholipid Deficiency Hypothesis

It has been suggested (Rudin) that in industrialized countries widespread deficiencies of Omega-3 EFAs in the average diet resulting from trends in food selection and processing starting in the early 1900s, has led to increases in the prevalence of many medical and psychiatric disorders in these countries compared to less developed countries or more traditional cultures where dietary preferences or industrialized food processing have not occurred. This observation is strengthened by many studies that show significantly reduced risks of cardiac arrest, cardiac arrhythmias, sudden cardiac death and other diseases (Severus et al) when the diet is supplemented with certain Omega-3 EFAs. The phospholipid deficiency hypothesis states that increases in relative amounts of omega-6 EFAs in the average industrialized diet have resulted from increased consumption of plant-derived products high in these Omega-6 EFAs concurrent with dietary deficiencies in sources of the Omega-3 EFAs. The resulting increased ratio of omega-6 to omega-3 EFAs available for metabolism has important consequences for the balance of opposing processes in normal human physiology, as fatty acids are precursors of prostaglandins, the body's principle regulatory molecules. The Omega-6 EFAs are crucial to synthesis of many cytokines that mediate inflammation, including several interleukins, tumor necrosis factor alpha (TNF-alpha), interferon-gamma (Maes, 1997). In contrast, diets high in the Omega-3 EFAs are correlated with reduced overall production of these inflammatory cytokines (Caughey et al, 1996).

According to the phospholipid deficiency hypothesis, the relative shift in consumption of Omega-6 and Omega-3 EFAs has led to wide spread increases in the prevalence of many

endemic medical and psychiatric disorders mediated by chronic inflammatory changes. This hypothesis asserts that many disorders result from pathological effects of chronic EFA deficiencies on phospholipid metabolism that affect cell membrane function in all major organs, including the brain (Severus; Rudin; Horrobin 1996). An important point of the hypothesis is that disorders resulting from deficiencies of dietary essential fatty acids mimic a panhypovitaminosis B syndrome in that these vitamins (especially B-6, pyridoxine) are enzyme cofactors required for the conversion of essential fatty acids into prostaglandins. Thus, the same kinds of medical or psychiatric symptoms that result from chronic vitamin B deficiencies are also believed to manifest from chronic deficiencies of essential fatty acids. Disorders associated with B vitamin deficiencies include neuropathies, dermatoses, enteritis, diabetes, immune system dysfunction, and a range of psychiatric symptoms including psychosis and depression.

EFAs are essential to normal brain structure and function

The synthesis and breakdown of phospholipids in the brain is integral to normal growth of axons and dendrites, as well as the formation of new synapses and the pruning of old ones. Phospholipids comprise approximately 25% of the dry weight of the human brain, and together, arachidonic acid (AA) and docosahexanoic acid (DHA) account for roughly half of total brain phospholipids. Brain phospholipids are essential for fluidity of nerve cell membranes and provide the physicochemical environment in which cell-membrane associated proteins are embedded, influencing their tertiary structure and therefore their capacity to function as neurotransmitter receptors. The fatty acid components of neuron membrane phospholipids are also central to the integrity of cell signaling systems involving protein kinases and other second messenger systems believed to play an important role in the pathogenesis of many psychiatric disorders.

EFAs in the pathogenesis of Schizophrenia and other psychiatric disorders

Horrobin (Horrobin, 1996; 1998) has proposed a “membrane phospholipid” model of schizophrenia, which argues that abnormal metabolism of phospholipids resulting from genetic and environmental factors manifests as a range of symptoms that are classified as schizophrenia. Several case reports (Puri et al, 2000) and double-blind studies (Laugharne et al, 1996; Mellor et al, 1995; Peet et al, 1997) have consistently demonstrated sustained improvement of both positive and negative symptoms in chronic schizophrenic patients consuming certain EFAs who were not being treated concurrently with conventional antipsychotic medications. However, a recent double-blind placebo-controlled study revealed no differences in response between EPA (3gm/day) and placebo in a group of 87 schizophrenics concurrently taking antipsychotic medications (Fenton et al, 2001). In contrast to earlier studies, those patients were treated for residual psychotic symptoms, and were not treated while in the early acute phase of illness.

Horrobin has argued that the membrane phospholipid hypothesis may provide a unifying conceptual framework for understanding not only schizophrenia, but also bipolar disorder, and possibly dyslexia, schizotypal personality disorder, other schizophrenia-like syndromes, and possibly other psychiatric disorders. The membrane

phospholipid hypothesis is compatible with the dominant paradigm in biological psychiatry, which ascribes the etiology of psychiatric disorders to dysfunction at the level of neurotransmitters and their receptors. These seemingly disparate views may in fact complement one another. For example, abnormal phospholipid metabolism in nerve cell membranes indirectly affects the functional integrity of neurotransmitter receptors and intra-neuronal signaling systems that are believed to be centrally involved in the pathogenesis of schizophrenia, bipolar disorder and other psychiatric disorders. According to Horrobin's theory, an inadequate dietary supply of EFAs, or metabolic factors interfering with the normal conversion of parent EFAs (LA or ALA) into DHA or EPA, would ultimately restrict the supply of Omega-3 EFAs to the brain for incorporation into nerve cell membranes resulting in abnormal phospholipid composition and sub-optimal functioning of a range of membrane-based neurotransmitter systems.

The membrane phospholipid hypothesis suggests that a spectrum of psychiatric disorders is associated with abnormalities at the level of the neuronal membranes, and that nature and severity of symptoms are related to the magnitude and type of metabolic errors leading to abnormal phospholipid metabolism. Severe psychiatric syndromes like schizophrenia develop when genetic errors of metabolism resulting in chronic brain deficiencies of dietary EFAs are combined with other metabolic abnormalities that result in errors of EFA incorporation in phospholipid membranes or abnormally high rates of removal of EFAs from nerve cell membranes by phospholipases. Certain less severe psychiatric disorders might result from chronic dietary EFA deficiencies or metabolic errors interfering with the normal biosynthetic pathways that incorporate EFAs into nerve cell membranes. These include dyslexia (Richardson et al), which is often associated with schizotypal personality (a mild schizophrenia-like syndrome), and possibly attention-deficit disorder (Stevens et al, 1995). In contrast to disorders resulting from abnormalities of phospholipid synthesis, bipolar disorder may result from an abnormally high rate of remodeling of nerve cell membranes affecting the functional integrity of certain neurotransmitter receptors or cell signaling systems. According to Horrobin, remodeling errors might be mediated by excess activity of phospholipase A2, the enzyme that removes EFAs from nerve cell membranes. Horrobin cites the following in support of the membrane phospholipid hypothesis:

- Increased blood levels of an enzyme (phospholipase A2) that is known to remove EFAs from phospholipids in nerve cell membranes in schizophrenic patients
- Reduced levels of arachidonic acid and DHA in red cell membranes of schizophrenics
- MRI data showing relatively increased rates of phospholipid breakdown in the brains of never-medicated schizophrenics
- Reduced electro-retinogram (ERG) response in schizophrenics, an indicator of reduced retinal DHA
- Clozapine has been shown to increase red blood cell phospholipid AA and DHA levels, suggesting that this may be a mechanism of action for clozapine, or other atypical antipsychotic drugs, in addition to its dopamine blocking effects.

- Clozapine is known to act like a prostaglandin E analogue, which may relate to its antipsychotic mechanism of action in regulating neuron membrane lipid metabolism
- The gene for lipoprotein lipase, the enzyme that regulates supply of EFAs to the brain, is on chromosome 8, where there is evidence for a gene predisposing to schizophrenia. The activity of this enzyme is typically diminished during puberty, which is often associated with the full expression of schizophrenia
- Children diagnosed with ADHD have been found to have reduced blood concentrations of EFAs required for normal brain development

The emerging role of Omega-3s in depressed mood

Food preferences influencing EFA consumption may be directly related to observed differences in the rate of depression when industrialized countries are compared to more traditional cultures. Epidemiological surveys have demonstrated a correlation between major depression and fish oil consumption. Countries where fish is a mainstay of the average diet are characterized by significantly lower rates of major depression and post-partum depression (Hibbeln, 1998).

For example, in Japan, where fish consumption is very high, only 0.12% of the population experienced depressed mood in a given year. In contrast, New Zealanders, who consume relatively little fish, reported a 6% annual rate of depression. It is important to note that whereas epidemiological studies show correlations, definitive causal proof of an association between essential fatty acids found in fish oil or other foods and depression can only be provided by well controlled studies.

To date, only one small double-blind study on EFAs and depression has been completed (Nemets et al, 2002). This four week study compared two groups of adults with uncomplicated depression (ie, no comorbid medical or psychiatric disorders) who received a placebo or purified EPA (2gm/d) while continuing their anti-depressant medications. None of the patients included in the study met criteria for refractory depression, and all but one had been successfully treated with conventional antidepressants previously. Hamilton depression ratings were done at the start of the study and weekly thereafter. Average baseline scores were 18 or higher. By week 2, Hamilton scores were significantly different between the two groups, and by the end of the study EPA-treated patients showed a mean reduction of 12.4 points, compared to a mean reduction of 1.6 points in patients receiving a placebo. Overall, six of the ten patients assigned to the EPA group showed a 50% reduction in Hamilton depression score compared to only one of ten patients in the placebo group. EPA had a significant effect on several core depressive symptoms, including guilt feelings, worthlessness and insomnia. There were no reports of significant side effects in either group, and only one patient dropped out (placebo group) because of worsening depression. The authors noted that the results do not clarify whether EPA has an independent antidepressant effect or augments antidepressants via second messenger systems in a manner that is similar to the postulated mechanism for lithium augmentation. Confirmation of the significance of an

antidepressant effect, and clarification of the mechanism of action of EPA will require a long-term prospective design that includes study arms assigned to receive antidepressants alone, Omega-3s alone, combined antidepressants and Omega-3s, and a broader range of depression subtypes.

A recent case report claimed rapid dramatic improvement in a severely depressed, suicidal patient who had been refractory to multiple antidepressant trials, including lithium augmentation (Puri, 2002). That patient showed sustained improvement in mood over a 9-month period while maintained on EPA (4gm/d) alone. No adverse effects were reported. A multi-center NIMH-sponsored study on the effects of Omega-3 fatty acids in the treatment of major depression and Bipolar Disorder (clinicaltrials.gov—cite website document) is now under way. The 240 patients recruited for this study will be randomly assigned to receive Omega-3 fatty acids (6gm/d) or placebo during a 16-week period, followed by an optional 8-month open trial on Omega-3 fatty acids. All patients will continue on their current anti-depressant or mood stabilizing medications, and will be assessed for mood changes every two weeks during the study period.

Accumulating laboratory evidence suggests a plausible link between the dietary ratio of Omega-6 to Omega-3 EFAs and the incidence of depression. A recent study (Adams et al, 1996) demonstrated a positive correlation between the severity of depression and the ratio of arachidonic acid (an Omega-6) to EPA (an Omega-3) in erythrocytes. Maes, et al (Maes, 1996) have demonstrated that in the initial period or “acute phase” of major depression, an increased production of pro-inflammatory cytokines takes place. In support of this theory, direct administration of the same cytokines into the brain causes dysregulation in serotonin metabolism that is consistent with changes observed in depressed individuals. Further, tri-cyclic antidepressants and the SSRIs are known to suppress release of many pro-inflammatory cytokines by immune cells in the blood, consistent with the view that these drugs may perform a similar therapeutic role in the brain, resulting in improved mood. (Maes et al, 1998). Recent findings that Omega-3 EFAs may reduce the incidence of coronary artery disease (Hibbeln, 1995) by influencing the production of pro-inflammatory cytokines in the heart may explain the observed correlation between heart disease and major depressive disorder.

EFAs in Bipolar Disorder

Considerable indirect evidence and one small double-blind trial (Stoll, et al 1999) suggest that Omega-3 EFAs improve both depressive and manic symptoms in bipolar patients by inhibiting the activity of CNS phospholipases, thereby reducing the release of unsaturated EFAs from nerve cell phospholipids, and limiting the production of specific prostaglandins (eg, PGE1) that are known to be associated with mania or depression. It has been postulated that lithium, dopamine antagonists and serotonin blocking agents are effective in the treatment of mania through a similar mechanism of correcting such “over-activity” in cell membrane signal transduction processes.

Until now there have been no large studies on the efficacy of EFAs in the treatment of bipolar disorder, and all studies done to date have been designed to assess the efficacy of

Omega-3 EFAs in combination with mood stabilizing medications but not Omega-3s alone. However, evidence from one small double-blind study (Stoll et al, 1999) suggests that Bipolar patients taking Omega-3 fatty acids alone remain in remission significantly longer than matched patients receiving a placebo. In this 4-month placebo-controlled double blind study, 30 bipolar patients were treated with Omega-3 EFAs (9.6gm/d) or placebo (olive oil) in combination with their usual mood stabilizing medications (including lithium, valproic acid, carbamazepine, and others). Significantly, a post-hoc analysis determined that four out of eight patients who took *only* Omega-3 fatty acids remained in remission significantly longer than three patients who received *only* placebo. Further, among the remaining 22 patients taking mood stabilizing drugs, those treated with Omega-3s performed significantly better on all outcome measures than patients in the placebo arm.

A double-blind study still in the recruitment phase at the time of writing, is being sponsored by the National Center for Complementary and Alternative Medicine, National Institutes of Health (cite clinicaltrials.gov webdoc). The study will examine the efficacy of Omega-3 fatty acids as a maintenance therapy in 120 Bipolar I patients over a 12 month period. Patients will be randomized to receive Omega-3s or placebo in combination with their on-going mood stabilizing medication. The study design will include one group of stable Bipolar patients who will remain off mood stabilizers while taking Omega-3s. Results of this study will help to clarify the role of Omega-3 fatty acids in maintenance treatment of Bipolar I Disorder, and may also provide useful insights about safe and appropriate ways to combine mood stabilizing agents with Omega-3s for different Bipolar patient populations.

Other possible indications for Omega-3 fatty acids in psychiatry

a. Reduced cognitive decline in the elderly

Emerging evidence suggests that regular intake of Omega-3 fatty acids is inversely related to cognitive impairment or rate of cognitive decline in non-demented elderly males. An epidemiological study (Kalmijn et al, 1997) compared cognitive impairment scores over a three-year period in two groups of elderly men (69 to 89 years of age) with different dietary preferences. High intake of foods rich in linoleic acid, an Omega-6 fatty acid, was associated with higher rates of cognitive decline. In contrast, high fish consumption (containing large amounts of Omega-3 fatty acids) was inversely correlated with cognitive impairment. The authors inferred that high dietary intake of Omega-6 polyunsaturated fatty acids, including linoleic acid, likely contributes to increased oxidative stress in the brain indirectly promoting atherosclerosis and thrombosis which eventually manifest as declines in cognitive functioning. Conversely, high intake of Omega-3 rich foods, especially fish, may reduce oxidative stress and associated atherosclerotic changes in the brain, mitigating factors known to be associated with cognitive decline.

b. violent and impulsive behavior

Recent observational studies (Hibbeln et al, 1998a; Hibbeln et al, 1998b) suggest that low plasma DHA (a Omega-3 essential fatty acid) levels, and therefore presumably low CNS levels, may increase the predisposition of some individuals to violent or impulsive behavior. This effect appears to be larger in certain groups, especially males who develop alcohol dependence before age 20. One proposed explanation is that genetic abnormalities in essential fatty acid metabolism result in a higher turnover rate of serotonin in the central nervous system, associated with higher CSF 5-HIAA levels. A placebo-controlled double-blind study (Hamazaki, T., et al) compared DHA (1.8gm/d) with placebo (soybean oil) in matched cohorts of Japanese students. A significant rate of “aggression against others” was reported in the placebo group at times of peak academic stress. In contrast, students taking DHA were not observed to exhibit increased aggressive behavior. The researchers concluded that DHA supplementation prevents aggressive behavior “at times of mental stress.” To date, no confirmatory studies have been done to determine whether supplementation with Omega-3 fatty acids differentially reduces violent or impulsive behavior in high-risk males compared with normal populations.

c. dyslexia

Two randomized double-blind studies on the effects of Omega-3 fatty acids in dyslexia are presently under way. At the time of writing, data analysis had not been completed. However, anecdotal reports of improvements in dyslexia with DHA supplementation have long suggested that abnormal phospholipid metabolism is associated with this disorder. This view is consistent with Horrobin’s theory (Horrobin, D., et al, 1995) relating abnormalities in phospholipid metabolism to neurodevelopmental disorders in general, including schizophrenia. Findings of several case studies along with a detailed discussion of the two on-going studies (above) are reviewed in Richardson et al, in “Phospholipid Spectrum Disorder in Psychiatry,” by Peet, Glen and Horrobin.

d. attention-deficit hyperactivity disorder and learning disabilities

Certain behavioral and learning problems in children may be associated with low plasma levels of Omega-3 fatty acids. In a descriptive study, parents and teachers assessed behavior of one hundred 6 to 12 year old boys using standardized scales measuring conduct, impulsivity-hyperactivity, anxiety, psychosomatic complaints, and learning problems (Stevens, et al., 1996). Boys found to have lower plasma Omega-3 levels also reported higher frequencies of symptoms associated with fatty acid deficiencies, including increased thirst and dry skin. Boys with lower total omega-3 fatty acid levels consistently scored higher on behavior scales measuring anxiety, hyperactivity and impulsivity. Lower serum omega-3 levels also correlated with more frequent temper tantrums, and problems going to sleep and getting up in the morning. Teacher ratings of overall academic ability were significantly lower for boys with lower omega-3 serum levels, but there were no significant differences in reading or writing ability between boys with normal and low omega-3 levels. These results need to be confirmed by well controlled prospective studies—including age-matched girls—before conclusions can be

reached regarding the possible efficacy or clinical role of omega-3 fatty acid supplementation as treatments of behavioral or learning problems in children.

Safety considerations

A recent review article suggests that no significant safety issues are associated with consumption of saturated fatty acids, including essential fatty acids, as long as these substances do not account for more than 10% of total caloric intake. Above this level, there is evidence linking high intake of certain essential fatty acids with reduced glycemic control in type 2 diabetics, and also with a tendency for increased bleeding. There is also evidence of a slight overall increase in liver enzyme activity with large amounts of omega-3 EFAs, which could affect metabolic clearance of certain medications. Anecdotal reports of a possible correlation between high doses of Omega-3s and hypomania have been reported (Kuan-Pin Su, 2000; Stoll, 2000), including ten cases in a series of more than 300 patients (Stoll, 2000) treated with various open-label preparations of flaxseed oil or fish oil. Stoll observed that almost all cases of apparent hypomania induction were associated with flaxseed oil, not fish oil. This effect was first noticed more than 20 years ago (Rudin, D., 1981), and was also associated with high doses of flaxseed oil, but not fish oil products. This effect is still under investigation, but may be related to the relatively higher content of alpha-linolenic acid (ALA), a short-chain Omega-3 fatty acid, in flaxseed oil compared to fish oil. According to Stoll (Stoll, 2000), higher ALA might be expected to result in significant increases in EPA but not DHA levels based on described metabolic pathways that transform essential fatty acids in the human body. Stoll speculates (Stoll, 2000) that high amounts of ALA in flaxseed oil might predispose susceptible depressed or Bipolar patients to hypomania by causing a reduced ratio of DHA to EPA in the serum, and presumably also in the central nervous system. The resulting imbalance could trigger hypomania in genetically susceptible individuals.

There have been case reports that certain fish oil supplements can result in hypervitaminosis A (Grubb), and other reports have suggested an increased incidence of hypertension and stroke (Kenny, 1990) in individuals who consume large amounts of omega-3-containing supplements. It is important to comment that controlled studies have not confirmed the presence or magnitude of these risks. Some patients complain of gastrointestinal distress when taking flaxseed oil or fish oil products. Eight of 13 (62%) patients taking Omega-3s in the Stoll study (Stoll et al, 1999) reported mild gastrointestinal side effects, compared to 8 of 15 (53%) placebo-treated subjects. The difference between the groups was not statistically significant, and no other side effects were reported frequently.

The emerging role of Serum and RBC lipid analysis

Lipid analysis will have an increasingly important role in differential diagnosis and treatment decisions regarding essential fatty acid supplementation for a range of medical

and psychiatric disorders. Plasma fatty acid analysis reflects dietary intake, in contrast to analysis of red blood cell membranes, which is an indicator of fatty acid metabolism. This difference may prove to be of central importance in determining the most appropriate treatment strategy for a given psychiatric disorder, as on-going research suggests that certain neuro-developmental disorders, including schizophrenia, dyslexia, and autism, are probably related to errors of fatty acid metabolism, whereas other disorders, including unipolar depression, likely result from dietary imbalances of specific Omega-6 and Omega-3 essential fatty acids. In the future, errors of metabolism will be approached through genetic engineering and corrective metabolic nutritional “therapies.” Conversely, disorders resulting from dietary fatty acid imbalances will be treated with targeted ratios of specific fatty acids. Accurate laboratory determination of the specific biochemical type and magnitude of dysfunction in the metabolic or dietary pathways of essential fatty acids will guide future clinicians in formulating treatments that address the etiological basis of a given psychiatric symptom or syndrome. Basic research in this area has been on-going jointly for many years between Dr. Patricia Kane PhD, of BodyBio, and the Kennedy Krieger Institute (location?). That work has resulted in protocols currently being used at BodyBio to perform both plasma and RBC membrane lipid analysis. (I am sure there are several others involved in this work, don’t have names, locations at this time...include contact information?). Lipid analysis panels are also available through Great Smokies Diagnostic Laboratories (GSDL), and.... (I believe these are the two principle ones).

Recommended reading

An excellent comprehensive resource for psychiatrists that summarizes important research and theoretical work, is “Phospholipid Spectrum Disorder in Psychiatry,” Marius Press, (date?), edited by Peet, Glen and Horrobin. A less technical overview of the role fatty acids in health in general, is “The Omega-3 Connection,” by Andrew Stoll. This book will be useful for patients who are considering taking Omega-3 fatty acids, and are seeking an overview of current theory, research, and treatment applications of essential fatty acids for medical or psychiatric disorders.

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