The Importance of Magnesium to Human Nutrition

by Michael B. Schachter M.D., F.A.C.A.M.

Magnesium is an extremely important and valuable mineral, whose value for good health is just being recognized by conventional physicians.

Virtually, all chemical reactions in the body require an enzyme system to help the biochemical reaction take place. An enzyme system generally consists of three parts. They are a specific protein molecule, another smaller organic compound, which is often a vitamin, such as pyridoxine or vitamin B6, and finally a charged mineral, such as zinc, copper, manganese or magnesium. Magnesium is a critical co-factor in more than 300 enzymatic reactions in the human body. Each mineral when dissolved in fluids has a characteristic electrical charge, called its valance. Minerals with a charge of plus 1, or univalent cations, include sodium and potassium. Minerals with a charge of plus 2, or divalent cations, include copper, zinc, manganese and magnesium. Potassium and magnesium are the most abundant cations found within the cells of the body with magnesium being the most abundant divalent cation.

In the USA, magnesium supplementation is dramatically under utilized by conventional physicians and is more important in patient therapy than most physicians realize. There are over 200 published clinical studies documenting the need for magnesium. In fact, at the 1992 American College of Cardiology annual meeting, a limited biography on magnesium was the most often requested item at the National Council on Magnesium and Cardiovascular booth.

Up until recently, conventional medicine's interest in magnesium has been only by obstetricians, who have used injectable magnesium sulfate extensively in the treatment of high blood pressure and pre-eclampsia and eclampsia of pregnancy. But, recently conventional physicians have become interested in treating patients with acute heart attacks, chronic cardiovascular disease, heart arrhythmias, diabetes, asthma, chronic fatigue syndrome and many other disorders.

Symptoms of Magnesium Deficiency?
What are some of the symptoms of magnesium deficiency? They are outlined beautifully in a recent article by Dr. Sidney Baker. Magnesium deficiency can affect virtually every organ system of the body. With regard to skeletal muscle, one may experience twitches, cramps, muscle tension, muscle soreness, including back aches, neck pain, tension headaches and jaw joint (or TMJ)

dysfunction. Also, one may experience chest tightness or a peculiar sensation that he can't take a deep breath. Sometimes a person may sigh a lot.

Symptoms involving impaired contraction of smooth muscles include constipation; urinary spasms; menstrual cramps; difficulty swallowing or a lump in the throat-especially provoked by eating sugar; photophobia, especially difficulty adjusting to oncoming bright headlights in the absence of eye disease; and loud noise sensitivity from stapedius muscle tension in the ear.

Other symptoms and signs of magnesium deficiency and discuss laboratory testing for this common condition. Continuing with the symptoms of magnesium deficiency, the central nervous system is markedly affected. Symptoms include insomnia, anxiety, hyperactivity and restlessness with constant movement, panic attacks, agoraphobia, and premenstrual irritability. Magnesium deficiency symptoms involving the peripheral nervous system include numbness, tingling, and other abnormal sensations, such as zips, zaps and vibratory sensations.

Symptoms or signs of the cardiovascular system include palpitations, heart arrhythmias, angina due to spasms of the coronary arteries, high blood pressure and mitral valve prolapse. Be aware that not all of the symptoms need to be present to presume magnesium deficiency; but, many of them often occur together. For example, people with mitral valve prolapse frequently have palpitations, anxiety, panic attacks and premenstrual symptoms. People with magnesium deficiency often seem to be "uptight." Other general symptoms include a salt craving, both carbohydrate craving and carbohydrate intolerance, especially of chocolate, and breast tenderness.

Diagnosing Magnesium Deficiency

Aside from the signs and symptoms of magnesium deficiency, how can a physician diagnose magnesium deficiency? Unfortunately, laboratory testing is of limited value. Since magnesium is found primarily in the cells, the serum magnesium may be normal in spite of a significant magnesium deficiency. The red blood cell magnesium is a little bit better. Probably the best test, although certainly not full proof, is the magnesium loading test. In this test, the patient collects a 24-hour urine sample and the total magnesium is measured. The patient is then given an injection of a specified amount of magnesium and another 24-hour urine specimen is collected. The magnesium is again measured. If the body retains more than a certain amount of magnesium, then it is concluded that the body is magnesium deficient and is holding on to the magnesium that has been injected. Perhaps the best method of diagnosing magnesium deficiency, however, is the combination of signs and symptoms of magnesium deficiency, which improve with a therapeutic trial of either oral or injected magnesium.

How can one get magnesium from foods? The best way of insuring enough magnesium is to eat a variety of whole foods, including whole grains, nuts, seeds and vegetables, preferably food grown on naturally composted soil. The green color of green vegetables is due to chlorophyll, which is a molecule that contains magnesium. Avoid refined processed foods, especially white sugar and white flour products, as most magnesium is removed from them.

Prevention and Treatment of Magnesium Deficiency Using Oral and Injectable Magnesium

For people who suffer from chronic magnesium deficiency and also to prevent the development of this condition, oral magnesium supplements can be quite useful. Magnesium is available in many forms. The cheapest is probably magnesium oxide, but this form is not absorbed as well as some other forms, which include chelated magnesium, magnesium glycinate and magnesium aspartate. Dr. Baker feels that the prescription form of magnesium chloride, known as Slow-mag, has been most useful for his patients. I have found that magnesium taurate, an unusual form of magnesium in which magnesium is chemically combined with the amino acid derivative taurine, is particularly well utilized and beneficial. This is because some of the same effects that one hopes to get from magnesium, such as the calming effect on the nervous system, and the strengthening effect on heart muscle, is also gotten with taurine. So, the two are synergistic together. I use it in all forms of cardiac and nervous system disorders.

What about dosage? The recommended daily allowance or RDA for magnesium is 350 milligrams of elemental magnesium. An important point here is that when reading the label of a supplement containing magnesium, it is important to distinguish between the number of milligrams per tablet or capsule of the entire magnesium complex versus the number of milligrams of elemental magnesium or pure magnesium. For example, one label of a chelated magnesium states that 4 tablets contain 4,000 mg of the chelated magnesium complex with 500 mg of elemental magnesium. The important number is the one that refers to the elemental magnesium. The other 3,500 mg in this case refers to the amino acid complex that is bound to the magnesium.

Keeping this definition of elemental magnesium in mind, many people do not even get the RDA of 350 mg of magnesium daily. A therapeutic dosage could easily run between 400 mg and 1000 mg daily of elemental magnesium in divided doses. In people with normal kidneys, it is difficult to reach toxic levels of magnesium. However, too much oral magnesium will result in diarrhea. Recall that milk of magnesia is a laxative containing a magnesium salt. Patients suffering from chronic kidney failure must be much more careful because their kidneys have difficulty eliminating magnesium and a toxic buildup may occur. Toxic levels of magnesium may lead to depression of the entire nervous system and even coma and death. But, this is extraordinarily rare and occurs only in

patients with severe kidney function impairment. In general, magnesium doses of 1000 mg per day or less are extremely safe.

Magnesium Supplementation for Various Medical Disorders
Oral magnesium supplementation may be helpful to a wide variety of medical disorders including: high blood pressure, asthma, angina pectoris, coronary artery disease, cardiac arrhythmias, chronic fatigue syndrome, all types of musculoskeletal disorders, epilepsy, mitral valve prolapse, anxiety, panic disorder and many other medical and psychiatric conditions.

For many conditions, such as acute heart attacks, magnesium given by either an intramuscular injection or as an intravenous drip, is the preferred method of treatment. Studies show it reduces the death rate and complications of acute heart attacks. In spite of its low cost or perhaps as a result of its low cost, it is not yet given routinely to heart attack victims. Other patients, such as those suffering from chronic fatigue syndrome also seem to do better with magnesium given by injection. This may be due to the superior absorption of injectable magnesium or because high concentrations in the body are necessary for maximal therapeutic effects. In our office, we use injectable magnesium extensively, as part of our EDTA chelation bottle, and for many of the conditions I've mentioned previously.

Increased use of oral and injectable magnesium, along with a diet rich in magnesium, should greatly improve therapeutic results for many patients.

Cod Liver Oil to Heal Autism (Vaccinations)

By Mary Megson, MD

..."When these children get the MMR vaccine, their vitamin A stores are depleted and they cannot compensate for blocked pathways. Lack of vitamin A, which has been called “the anti-infective agent,” leaves them immunosuppressed. They lack cell-mediated immunity. T cell activation, important for long term immune memory, requires 14-hydroxy retro-retinol. **On cod liver oil, the only natural source of this natural substance, the children get well.** The parasympathetic nervous system is blocked by the second G protein defect."...

http://www.newmediaexplorer.org/chris/2004/09/20/cod_liver_oil_to_heal_autism_vaccinations.htm

By Mary Megson, MD

I have practiced pediatrics for twenty-two years, the last fifteen years seeing only children with developmental disabilities, which include learning disabilities, attention deficit hyperactivity disorder, cerebral palsy, mental retardation and autism.

In 1978, I learned as a resident at Boston Floating Hospital that the incidence of autism was one in 10,000 children. Over the last ten years I have watched the incidence of autism skyrocket to 1/300-1/600 children.

Over the last nine months, I have treated over 1,200 children in my office. Ninety percent of these children are autistic and from the Richmond area alone. Yet the State Department of Education reports that there are only 1,522 autistic students in the entire state of Virginia.

MHMR (Mental Health Mental Retardation) agencies have created local infant intervention programs, and they have had a hard time keeping up with the numbers of delayed infants and toddlers. I have served as advisor to the City of Richmond and the surrounding counties as they have established entire programs for autistic children that fill multiple classes in several schools in each district.

The segment of children with “regressive autism,” the form where children develop normally for a period of time then lose skills and sink into autism, most commonly at 18-24 months of age, is increasing at a phenomenal rate. I am seeing several children in the same family affected, including in the last week four cases of “autistic regression” developing in four-year-old children after their MMR and DPT vaccination. In the past, this was unheard of.

In the vast majority of these cases, one parent reports night blindness or other rarer disorders which are caused by a genetic defect in a G protein, where they join cell membrane receptors, which are activated by retinoids, neurotransmitters, hormones, secretin and other protein messengers. G proteins are cellular proteins that upgrade or downgrade signals in sensory organs that regulate touch, taste, smell, hearing and vision. They are found all over the body, in high concentration in the gut and the brain. They turn on or off multiple metabolic pathways including those for glucose, lipid and protein metabolism as well as cell growth and survival.

Close to the age of “autistic regression,” we add pertussis toxin, which completely disrupts G alpha signals. The opposite G proteins are turned on without inhibition leading to the following:

1. Glycogen breakdown or gluconeogenesis. Many of these children have elevated blood sugars. There is a 68 percent incidence of diabetes in parents and grandparents of these children.

2. Lipid breakdown which increases blood fats that lead to hyperlipidemia. One-third of families has either a parent or grandparent who died from myocardial infarction at less than 55 years of age and was diagnosed with hyperlipidemia.
3. Cell growth differentiation and survival which leads to uncontrolled cell growth. There are 62 cases of malignancies associated with ras-oncogene [a cancer gene] in 60 families of these autistic children.

The measles antibody cross reacts with intermediate filaments which are the glue that hold cells together in the gut wall. The loss of cell-to-cell connection interrupts aproptosis or the ability of neighboring cells to kill off abnormal cells. The MMR vaccine at 15 months precedes the DPT at 18 months, which turns on uncontrolled cell growth differentiation and survival.

Most families report cancer in the parents or grandparents, the most common being colon cancer. The genetic defect, found in 30-50 percent of adult cancers, is a cancer gene (ras-oncogene). It is the same defect as that for congenital stationary night blindness.

G-protein defects cause severe loss of rod function in most autistic children. They lose night vision, and light-to-dark shading on objects in the daylight. They sink into a “magic eye puzzle,” seeing only color and shape in all of their visual field, except for a “box” in the middle, the only place where they get the impression of the three dimensional nature of objects.

Only when they look at television or a computer do they predictably hear the right language for what they see. They try to make sense of the world around them by lining up toys, sorting by color. They have to “see” objects by adding boxes together, thus “thinking in pictures.” Their avoidance of eye contact is an attempt to get light to land off center in the retina where they have some rod function.

Suddenly mother’s touch feels like sand-paper on their skin. Common sounds become like nails scraped on a blackboard. We think they cannot abstract, but we are sinking these children into an abstract painting at 18 months of age and they are left trying to figure out if the language they are hearing is connected to what they are looking at.

The defect for congenital stationary night blindness on the short arm of the X chromosome affects cell membrane calcium channels which, if not functioning, block NMDA/glutamate receptors in the hippocampus where pathways connect the left and right brain with the frontal lobe.

Margaret Bauman has described a lack of cell growth and differentiation in the hippocampus seen on autopsy in autistic children. The frontal lobe is the seat of attention, inhibition of impulse, social judgment and all executive function.

When stimulated, these NMDA receptors through G proteins stimulate nuclear vitamin A receptors discovered by Ron Evans and his colleagues in December, 1998. When blocked, in the animal model, mice are unable to learn and remember changes in their

environment. They act as if they have significant visual perceptual problems and have spatial learning deficits.

Of concern is the fact that the hepatitis B virus protein sequence was originally isolated in the gene for a similar retinoid receptor (RAR beta), which is the critical receptor important for brain plasticity and retinoid signaling in the hippocampus. After the mercury is removed, I understand we will restart hepatitis B vaccine at day one of life. Studies need to be done to determine if this plays an additive role in the marked increase in autism.

I am using natural lipid soluble concentrated cis form of vitamin A in cod liver oil to bypass blocked G protein pathways and turn on these central retinoid receptors. In a few days, most of these children regain eye contact and some say their “box” of clear vision grows. After two months on vitamin-A treatment some of these children, when given a single dose of bethanechol [a drug related to acetlycholene, a substance that transmits nerve impulses] to stimulate pathways in the parasympathetic system in the gut, focus, laugh, concentrate, show a sense of humor and talk after 30 minutes, as if reconnected.

This improves cognition, but they are still physically ill. When these children get the MMR vaccine, their vitamin A stores are depleted and they cannot compensate for blocked pathways. Lack of vitamin A, which has been called “the anti-infective agent,” leaves them immunosuppressed. They lack cell-mediated immunity. T cell activation, important for long term immune memory, requires 14-hydroxy retro-retinol. On cod liver oil, the only natural source of this natural substance, the children get well. The parasympathetic nervous system is blocked by the second G protein defect.

These children are unable to relax, focus and digest their food. Instead, they are in sympathetic overdrive with a constant outpouring of adrenaline and stress hormones. They are anxious, pace, have dilated pupils, high blood pressure and rapid heart rate. These and other symptoms of attention deficit hyperactivity disorder are part of this constant “fight or flight” response. These symptoms improve on bethanechol.

I live in a small middle class neighborhood with twenty-three houses. I recently counted thirty children who live in this community who are on medication for ADHD. One week ago my oldest son, who is gifted but dyslexic, had twelve neighborhood friends over for dinner. As I looked around the table, all of these children but one had dilated pupils. After two-and-one-half months of taking vitamin A and D in cod liver oil, my son announced, “I can read now! The letters don’t jump around on the page anymore!” He is able to focus and his handwriting has improved dramatically. In his high school for college-bound dyslexic students, 68 of 70 teenagers report seeing headlights with starbursts, a symptom of congenital stationary nightblindness.

I think we are staring a disaster in the face that has affected thousands of Americans. The children with autism or dyslexia/ADHD are lucky. There are many other children not identified, just disconnected.

We must direct all of our resources and efforts to establish multi-disciplinary centers to treat these children. Insurance companies should pay for evaluations, both medical and psychiatric, and treatment. These children are physically ill, immunosuppressed with a chronic autoimmune disorder affecting multiple organ systems. Funding to look at etiology of autism, to identify children at risk prior to “autistic regression,” and to prevent this disorder is imperative.

Implementing vaccine policies that are safe for all children should become our first priority.

Mothers from all over the country have brought pictures of their autistic children to Washington this weekend. Most of these children were born normal and were lost to “autistic regression.” Look into their eyes and you will hear their silence.

Editor’s note: In addition to cod liver oil, children with developmental disorders should be given a nutrient-dense diet that includes plenty of calcium and other minerals. Additional vitamin D may also be helpful. (See page 11.)

About the Author

Dr. Mary Megson is a board-certified pediatrician, trained in child development, a member of the American Academy of Pediatrics and assistant professor of pediatrics at the Medical College of Virginia. This testimony was given April 6, 2000 at Senate hearings on autisim and vaccinations. Parts of this article are technical.

The Vitamin D Connection

Six years ago, Professor John McGrath and his colleagues at the University of Queensland in Australia, pointed out that vitamin D, "the neglected *neurosteroid*," was crucial for proper brain development. In the same paper, they reported that activated vitamin D increases *nerve growth factor* in the brain and the *vitamin D receptor* appears in a wide variety of brain tissue quite early in the development of the baby. These two facts alone led them to conclude that vitamin D deficiency "should be examined in more detail as a candidate risk factor for neurodevelopmental...disorders." McGrath J, Feron F, Eyles D, Mackay-Sim A. **Vitamin D: the neglected neurosteroid?** Trends Neurosci. 2001 Oct;24(10):570–2.

In 2006, Dr. Alan Kalueff and his colleagues went further, suggesting vitamin D offers "neuroprotection, possible interplay with several brain neurotransmitter system and hormones, as well as regulation of behaviors." In 2007, Dr. Kalueff, now at the National Institutes of Mental Health, reviewed the nootropic (brain-enhancing) properties of vitamin D in even more detail and concluded that the scientific data stress the importance of the mother having enough vitamin D while she is pregnant and the child having

enough vitamin D after birth for "normal brain functioning." There is no doubt vitamin D affects the brain, and does so profoundly. Kalueff AV, *et al*. **The vitamin D neuroendocrine system as a target for novel neurotropic drugs.** CNS Neurol Disord Drug Targets. 2006 Jun;5(3):363–71. Kalueff AV, Tuohimaa P. **Neurosteroid hormone vitamin D and its utility in clinical nutrition.** Curr Opin Clin Nutr Metab Care. 2007 Jan;10(1):12–9.

Predisposition - What Gene Should We Be Looking For?

Given what we know about neurosteroids, in our search for the genetics of autism it is reasonable to search for a gene which:

- is environmentally responsive.
- codes for a systemic *steroid* that is also a potent neurosteroid.
- profoundly affects brain development
- has had its levels decrease over the same time that autism has increased.
- is affected differently by *estrogen* and *testosterone*.
- has levels that are much lower in blacks than in whites.
- explains all the bizarre *epidemiology* of autism.

A tall order indeed.

Two clues: rare genetic malformations of the vitamin D system

An inborn error of *metabolism* that causes a rare form of rickets, pseudo-vitamin D deficiency rickets, involves the defective manufacture of *activated vitamin D*. While no one has assessed afflicted children for signs of autism, these children clearly display autistic markers such as hypotonia (flabby muscles), decreased activity, developmental motor delay, listlessness, and failure to thrive.

Much more interesting is the fact that children with Williams Syndrome (rare congenital disorder due to a missing piece of chromosome seven) often have greatly elevated activated vitamin D levels for several months in early life. They usually present in later life with remarkable sociability, overfriendliness, empathy, and willingness to initiate social interaction—strikingly the opposite personality of autistic children. Knudtzon J, Aksnes L, Akslen LA, Aarskog D. **Elevated 1,25-dihydroxyvitamin D and normocalcaemia in presumed familial Williams syndrome.** Clin Genet. 1987 Dec;32(6):369–74. Mervis CB, Klein-Tasman BP. **Williams syndrome: cognition, personality, and adaptive behavior.** Ment Retard Dev Disabil Res Rev. 2000;6(2):148–58.

So, abnormally-low activated vitamin D levels produce infants with symptoms of autism while abnormally-high levels produce children with personalities the exact opposite of autism.

What is the role of the vitamin D receptor in autism?

Variations in the DNA sequence of vitamin D receptor are common and called vitamin D receptor (VDR) *polymorphisms* (many-shaped receptors). No one has studied them in autism, but a highly significant association exists between one VDR polymorphism and larger head size. Larger head sizes are common in autism, especially in childhood

Essential Fatty Acids

Researched and Composed by Venom

Abstract:

Essential fatty acids have been shown to have positive effects on insulin sensitivity, thermogenesis, anabolism, and much more. These processes entail the use of many complex mechanisms, however. Of prime importance is their function in hormone synthesis. By passing through the cyclooxygenase, or lipoxygenase enzymatic pathways, fats are able to form several hormones known as eicosanoids. These hormones elicit many anabolic attributes. Highlighted features in this study include: how to maximize hormones, mechanisms of fatty acids functions, and optimal EFA ratios for the athlete.

Recommend readings:

Metabolic Primer Part II

Endocrine Insanity Part I

Endocrine Insanity Part II

Endocrine Insanity Part III

Nutrient Density Explored

Essential Nutrients

Approximately 50 ingredients which are necessary for maintenance, growth, health, and ultimately survival have been discovered, but cannot be manufactured by the body. The majority of these are nutrients that must be

supplied through diet because the body cannot provide them. Included are: oxygen, water, light, a source of energy, 13 vitamins, 8 essential amino acids (10 for children), 20-21 minerals, and 2 ***essential fatty acids (EFAs).*** If one is lacking, your performance will suffer. Later on I discuss the importance of, 'the complete package,' and its role in the optimization of essential fatty acids [53].

Hormones

EFAs play a vital role in hormone synthesis. Before we proceed, it is imperative that you understand certain terms, as they will be applied throughout the article. Additionally, I recommend you study Joe King's 3 Endocrine articles for a comprehensive understanding of hormones [78,79,80]. We'll begin with eicosanoids.

Eicosanoids

Eicosanoids function in intercellular (between cells) communications. They are modified 20-carbon fatty acids with a 5 carbon ring in the center. The center ring forces the molecule to bend over itself, producing 2 extended parallel chains, facing away from the ring. Furthermore, because they are lipids, they easily cross the plasma membrane, and are insoluble in water. The 3 classes of eicosanoids hormones are: prostaglandins, leukotrienes, and thromboxanes.

To form eicosanoids, the molecule of fat must go through one of two enzymatic pathways. That is, the cyclooxygenase, or lipoxygenase pathway. The former leads to the production of prostaglandins and thromboxanes. The latter produces leukotrienes. This trio of eicosanoids will be discussed subsequently [53].

Prostaglandins- Unlike most hormones, which circulate in the blood and function as messengers effecting tissues from specific glands, prostaglandins exert local effects in their area of synthesis. They are biologically active lipid hormones found within the plasma membrane of almost every cell. As hormone-like chemicals, they monitor cellular actions. Discovery was made in 1930 from the prostate gland of a sheep; hence, the name prostaglandins. About 30 PGs have been discovered.

Prostaglandins and their receptors have several effects on numerous physiological processes; these can be minor or major. Examples are: controlling local hormone response, blood clotting, inflammation, pain, fever, pepsin and HCI secretion in the stomach, nerve functions, calcium metabolism, and much more. However, these functions have been narrowed down to 3 phases: series one, two, and three. Lastly, the conversion of arachidonic acid through the cyclooxygenase pathway forms PGH2, which is the parent compound of other prostaglandins and thromboxanes. Other series will be discussed further on [53].

Thromboxanes- these are very similar to prostaglandins, and go through the

same pathway for formation. They can help reduce blood loss from injuries, assist blood clotting, and many other roles which help maintain homeostasis [16].

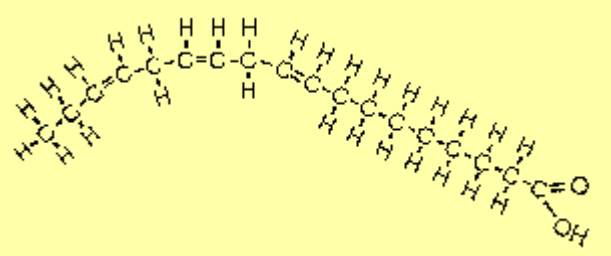
Leukotrienes- formed via the lipoxygenase pathway, leukotrienes possess many potent actions on essential organs and systems. These include: regulation of certain white blood cells, smooth muscle contraction, assistance of the immune system, cardiovascular system, and nervous system, among others.

Leukotrienes have been shown to be effective supplements for ailments such as: rheumatoid arthritis, psoriasis, and inflammatory bowel disease [58]. Additionally, studies display them to be effective anti-inflammatories, as well as assisting diseases such as asthma, rheumatoid arthritis, and inflammatory bowel disease [31].

And with that, we're ready to get down to the meat of the article: essential fatty acids!

Omega-3

Omegas-3s are polyunsaturated fats, but are commonly called super unsaturated to distinguish them from omega-6s. Its main component is Alpha-linolenic Acid (LNA). Scientifically, it is called cis-w3,6,9-octadectrienoic acid. Other synonyms are 18:3w3, or 18:3n3, w3, and n3 fatty acids. The number before the colon denotes the amount of carbon atoms, and the number after indicates total double bonds. The human body needs LNA for survival, but cannot manufacture it, which makes it an essential fatty acid.

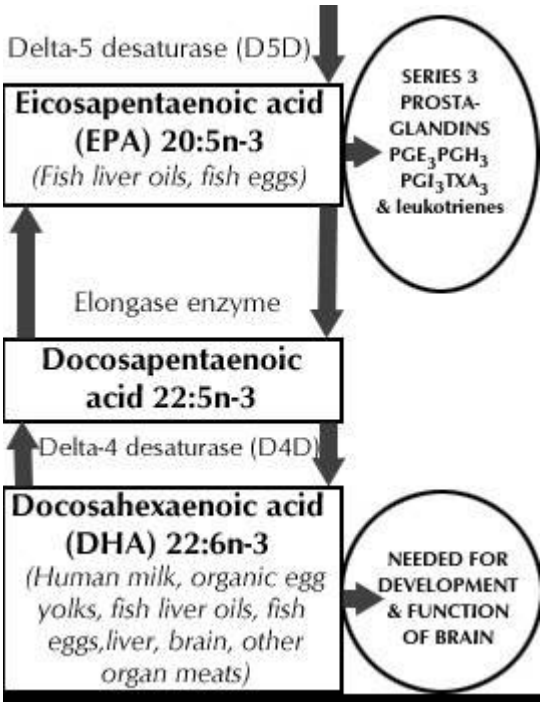


EFAs have several functions, such as cell membrane structure, energy via oxidation, and as mentioned previously, production of hormones, which brings us to our next topic: LNA derivatives.

Derivatives

LNA is the starting material for the biosynthesis of Docosahexaenoic Acid (DHA), and Eicosapentaenoic Acid (EPA). It converts to the derivative Stearidonic Acid (SDA), to eicosatetraenoic acid (ETA), and to Eicosapentaenoic Acid (EPA), respectively. From here, EPA forms DHA in a 4-step process of elongation,

elongation, desaturation, and chain shortening, in that order. Desaturation is the introduction of double bonds; the enzymes that do this are called desaturases. Elongation catalyzes the addition of 2-carbons to fatty acids [68]. From here, several eicosanoids are formed, producing an abundance of anabolic effects. EPA manufactures series 3 prostaglandins. We will primarily focus on the latter two derivates, EPA and DHA.



EPA and DHA

In an epidemiological (the study of diseases in populations and states) survey, fascinating observations were made on the Greenland Eskimos, using approximately 1800 people over a 25 year period. Results show that they have a lower risk and often a complete absence of diseases such as acute myocardial infarction, diabetes mellitus, thyrotoxicosis, bronchial asthma, multiple sclerosis, and psoriasis. Why would this be? The answer is clear: their diet, which is rich in EPA and DHA from fish [34].

EPA is termed 20:5n3, while DHA is 22:6n3. These oils play a vital role in our body. For example, EPA is the most potent factor in prevention of prostaglandin series 2, which can induce many side effects (discussed later). Many more beneficial attributes will be shown shortly. First, let's talk about conversions.

Most studies in humans have shown that high doses of LNA convert to EPA at a low rate, and conversion to DHA is severely restricted. Furthermore, a diet with a high omega-6-3 ratio can reduce this by 40 to 50%. Thus, it is suggested to

consume 2-3 portions of fatty fish per week, or 1.25 g EPA + DHA per day, while keeping your omega-6s under control [24]. The British Nutrition Foundation also recommends a daily intake of EPA and DHA in amounts corresponding to the intake of 3 to 4 g standardized fish oil or 2 to 3 portions of fatty fish weekly [25]. Many other journals testify to these reports as well. [46,47].

Good sources of EPA and DHA are fatty (at least 10%), fresh, cold-water fish such as sardines, trout, salmon, eel, and mackerel.

Anabolic effects

Lower PG2s

Series 2 prostaglandins can have serious adverse affects. We will get into more detail when we discuss omega-6 fatty acids, but suffice it to say, lowering its production would be of benefit to the athlete [77]. Consequently, omega-3s (particularly EPA, which as stated above, is the most potent omega-3 in series 2 prevention; EPA stops AA from being released so it can't form PG2S) have been shown to inhibit the production of prostaglandin series 2, which is produced by w6s [74]. The effect of consuming more w-3s and lowering w-6s is amazing. These include decreased water retention and inflammation, as well as cardiovascular health.

For example, they tested a diet rich in omega-3 oils--using fish oil--and a diet rich in omega-6 oils--using corn oil,--on female mice [54]. The results showed that dietary supplementation with w3s over w6s inhibited the production of pro-inflammatory cytokines and slowed progression of immune-complex-mediated kidney injury. This may be due to the enhanced ability of the cells to dispose of harmful reactive oxygen intermediates.

Another study compared fish oil to safflower oil over a 5 month period. The results showed splenic natural killer cells and lymphokine-activated killer cells were proportional to the concentration of n6s, but reduced by n3s [5].

Additionally, supplementation with n-3 has also been shown to enhance the immune system, largely due to decreased PG2s [27,56].

There are many more benefits to this method [67], and be sure that this information will be taken into account when ratios are discussed.

Anti-inflammatory

N3 fatty acids have been shown to suppress inflammation by decreasing the production of pro-inflammatory cytokines and series 2 prostaglandins, making them of use for several chronic inflammatory diseases [4]. It has been postulated that they may help relieve delayed onset muscular soreness (DOMS), but results

are not very convincing [44].

Lower triglyceride and cholesterol levels

Omega-3s--particularly EPA and DHA found in fish--have been shown to lower serum triglyceride, total cholesterol levels, and phospholipids [45]. From his study, Tato F. et al. states [69], 'We conclude that in FCH moderate doses of long-chain n-3 fatty acids are highly effective in lowering pathological VLDL triglycerides, VLDL cholesterol, and VLDL apo B.' In another study on the benefits of omega-3s, plasma triglycerides were reduced by 58% and plasma cholesterol concentration by 34% [70]! The results are incredible.

Cancer

Studies show that w3s can help prevent cancers, such as colon and breast cancer. In a study with fatty fish, they compared subjects using high (2 servings), and moderate (0.5 servings) amounts of fish weekly. The results showed supplementing with fish twice weekly was more effective in decreasing the risk of cancer [71]. These benefits may be related to reduced series 2 prostaglandins.

Additionally, in a series of case-control studies conducted in Italy and Switzerland between 1991 and 2001, the role of n-3 polyunsaturated fatty acid intake in the etiology of cancer of oral cavity and pharynx (736 cases, 1772 controls), esophagus (395 cases, 1066 controls), large bowel (1394 colon, 886 rectum, 4765 controls), breast (2900 cases, 3122 controls), and ovary (1031 cases, 2411 controls) cancers were tested. From this comprehensive experiment, it was concluded that, 'All the estimates were statistically significant, excluding that for rectal cancer, and consistent across strata of age and gender. These results suggest that n-3 PUFAs decrease the risk of several cancers [73].'

Kidneys

Using patients with IgA nephropathy--renal (kidney) diseases--it was tested how effective omega-3s were. They concluded that the n3s can slow the rate of renal function loss effectively [17]. Several other scientific authorities also recommend w3s for renal maintenance [18,26].

Insulin Sensitivity

If you have read any of our articles, you know just how valuable insulin sensitivity is. Simply put, increased sensitivity promotes a much greater anabolic response to insulin and increases your fat-burning ability immensely, while insulin resistance leads to elevated fat storage, reduced hypertrophy, and increased susceptibility to diseases such as diabetes. For more, study the following articles: Metabolic Primer Part I, and Endocrine Insanity Part III.

Here is the exciting part: studies show omega-3s can increase insulin sensitivity drastically, while its counterpart--omega-6s--in higher dosages may lead to insulin resistance.

For instance, a fascinating study was performed on rats using high-fat diets and various lipids to assess their effect on bodyweight regulation, adiposity, and metabolism. Results showed that rats who consumed high amounts of saturated or n-6 polyunsaturated fatty acids became obese, insulin resistant, and gained the most fat, while fish oils showed to be a superior fat in the experiment [57].

Another study stated that the negative effects of a high-sucrose diet, which induced insulin resistance and mild glucose intolerance, were counteracted by enhanced dietary intake of omega-3 polyunsaturated fatty acids [51].

Storlien LH et al. tested the effects of certain fats on rats. Subjects who had diets rich in polyunsaturated (omega-6) fatty acids developed severe insulin resistance. Afterward, they substituted 11% of the fatty acids in the polyunsaturated fat diet with long-chain omega-3 fatty acids from fish oils. The omega-3s were shown to effectively normalize insulin action [66].

Furthermore, Chicco A et al. composed a diet with 7% of the calories coming from cod liver oil--which is rich in omega-3 fatty acids--on male Wistar rats. The end results showed a significant reduction in plasma insulin levels throughout the day, due to enhanced insulin sensitivity [11].

Popp-Snijders C et al. performed an excellent study for the effects of Omega-3s on diabetics. Six non-insulin-dependent diabetics supplemented with just 3 g of the omega-3 fatty acids daily, over an 8 week time span. The subjects showed enhanced insulin sensitivity and lower plasma triglyceride levels [55].

Another experiment was performed on rats. First, they implemented a diet high in omega-6 and saturated fatty acid, which again lead to insulin resistance. Afterward, they replaced simply 6 percent of the linoleic omega-6 fatty acids from safflower oil with long-chain polyunsaturated omega-3 fatty acids from fish oil. This resulted in the prevention of insulin resistance [62].

It should be noted that in Western society diabetes has become a prevalent disease. This can be largely attributed to the lopsided ratio of omega-6:3 fatty acids. Diabetics will want to take close notice of these results, and adjust their diets accordingly [62].

So, as you see, a diet rich in Omega-6s can lead to insulin resistance, while a diet full of Omega-3s will inevitably increase insulin sensitivity [39,40,14].

Thermogenesis

The omega-3 has the distinctive ability of enhancing thermogenesis and lipid metabolism (increased usage of fat), thereby reducing body fat deposition. Clarke SD contributes to this, stating, '[Omega-3s exert their] effects on lipid metabolism and thermogenesis by up-regulating the transcription of the mitochondrial uncoupling protein-3, and inducing genes encoding proteins involved in fatty acid oxidation (e.g. carnitine palmitoyltransferase and acyl-CoA oxidase) while simultaneously down-regulating the transcription of genes encoding proteins involved in lipid synthesis (e.g. fatty acid synthase) [14].'

The effect of what they are saying is this: food, as well as our own body, contains what is known as 'potential chemical energy.' That is, energy held within the bonds of molecules. A good analogy is to think of a boulder placed on a 100 foot hill. By position, when the bolder is on top of the hill, it has the potential to turn into kinetic energy (the energy of movement) if someone were to tip it off the hill so that it began to roll. High-energy bonds within molecules are high-energy because when those bonds are broken, energy is released, which can be used to do work (force x distance=work).

They use the term Lipid Oxidation for a very specific purpose. The organelle in your body known as mitochondria is responsible for extracting energy from lipids (fats), and using it to synthesize or build our energy currency known as ATP (refer to Adam's tibialis article). Oxidation is referred to because oxygen is required for this process to occur (oxidation refers to an atom accepting electrons from another atom). The entire process is known as cellular respiration, because you need the respiratory system to consume the oxygen needed for the extraction of energy from these food groups, or stored energy deposits such as your own adipose tissue. Carnitine is an essential protein needed for fat breakdown, and thus, when the body encodes for more of it and other vital proteins required for this process, fat breakdown increases. Genes code for proteins. Likewise, by down-regulating certain genes which code for proteins that

enhance the formation of fatty molecules, you logically slow down the process. As a result, the above effects are additive to enhanced fat-burning.

Nervous system

N3s are high in biologic structures which require fast movement, like transport mechanisms in the brain and retina. Due to this, it appears that omega-3s may have functions with the nervous system benefiting vision and the brain, among others. [49]

Hypertension (high blood pressure)

Omegas-3s are potent supplements in the reduction of blood pressure. For example, a thorough study was composed using 31 placebo-controlled trials on 1356 subjects using fish oils. A significant drop in blood pressure was observed. They noted that its benefits are strongest for those with heart-related diseases [48].

Its great effectiveness may be due to alteration of prostaglandin metabolism, vascular endothelial function, increased vascular responses to pressure agents, and restriction of vascular smooth muscle production.

Joints

Bodybuilders apply tremendous pressure to their joints on a day-to-day basis. It is vital that we have a strong, smooth-running skeletal system to push ourselves to the max. In assistance of our goals, I present omega-3s.

W3s have shown tremendous results in soothing tender joints and stiffness. For example, w3s were tested on patients with arthritis. The results showed a significant reduction in disease activity, pain assessment, and number of weak joints. In addition, morning stiffness and several tender joints were relieved [35].

Another 24-week study was done on 17 patients with rheumatoid arthritis. Supplementation consisted of 54 mgs of EPA and 36 mgs of DHA. They reported a significant improvement in tender and swollen joints [36].

Several other scientific authorities attest to its benefits for joints as well [42,75,50]. These results are attributed to a reduction in prostaglandin series 2, its lubricating effects on our joints, construction of cell membranes, and w3's other anti-inflammatory influences displayed earlier.

Skin

Omega-3s have been shown to work wonders on skin inflammations, disease,

and overall skin perfection, such as softer, smoother, healthier skin. Dr. John A. Grossman, board certified aesthetic plastic surgeon, states [19], ‘For softer skin, get plenty of omega-3 fatty acids in your diet.’ Due to these findings, several companies have designed sprays, foods, and other supplements containing omega-3s for smoother skin [63,64,19].

Deficiency in omega-3s can produce several skin disorders such as dermatitis, skin atrophy, scaly dermatitis, edema, dry skin, and much more [16].

These benefits are attributed to enhanced skin blood flow, decreased PG2s, and its role in anti-inflammation [65].

Platelets

Platelets are disks circulating in our blood stream that aid in blood clotting. Sticky platelets form clots easier, while less-sticky platelets reduce clot formation. Sticky platelets promote heart attacks, along with other cardiovascular disorders. The former is induced by series 2 prostaglandins, while reduced stickiness is promoted by omega-3s.

Omega-3s and 6s were tested for their effects on platelet adhesion. The results showed w3s were inhibitory to platelet adhesion, while a diet rich in omega-6s stimulated it [2]. There are several other sources which testify to these effects as well [41,27].

Sources

Sources high in omega-3 fatty acids include flax (58%), chia (30%), hemp seed oil (20%), pumpkin (15%), fish (31%), canola oil (7%), and walnuts (5%). Other sources are flax meal, flax bread, omega-3 rich eggs, dark leafy greens such as spinach, and fish oil tablets, among others.

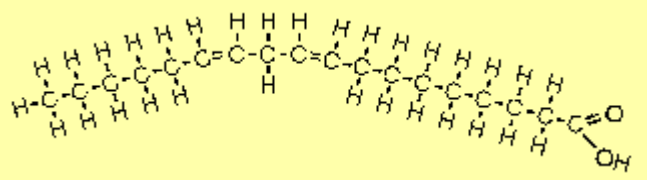
Deficiencies

Because EFAs are needed for the function of virtually all tissues, the list of side-effects for deficiencies is vast. This includes hemorrhagic dermatitis, weakness, impaired vision, tingling sensations, mood swings, edema, dry skin, sticky platelets, high blood pressure and triglycerides, hemorrhagic folliculitis, immune and mental deficiencies, skin atrophy, and scaly dermatitis, among others [3,33,30]. Some have suggested omega-3s for the reduction of symptoms in PMS, but most studies display poor results [15]. Lastly, EFAs in general are vital for growth and development [43].

Later on, I will discuss the recommended usage of Omega-3s in relation to their essential counterpart.

Omega-6

Omega-6 is a polyunsaturated fat. Its main component is Linoleic Acid (LA). Scientifically, it is called cis-w6,9-octadectrienoic acid. Other synonyms are 18:2w6, or 18:2n6, w6, and n6 fatty acids. Again, the number before the colon denotes the amount of carbon atoms, and the number after indicates total double bonds. It is the second essential fatty acid.

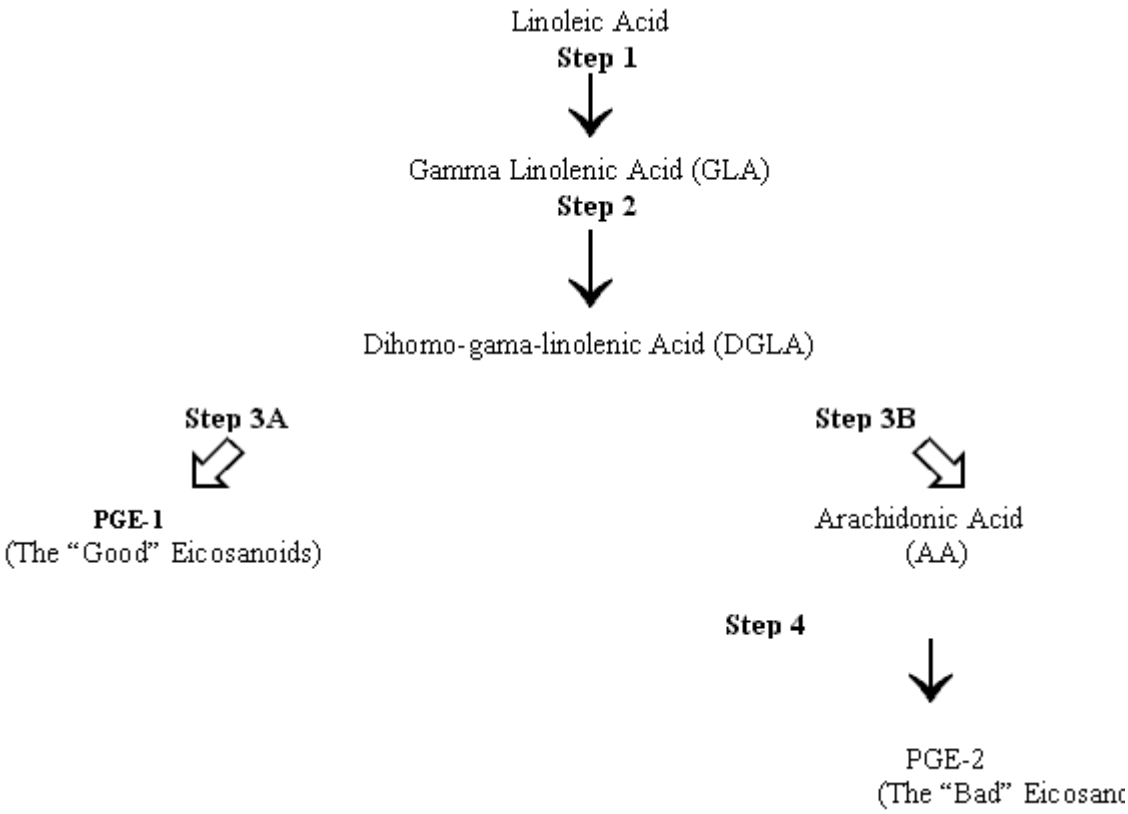


Derivatives

Linoleic Acid is the starting material for omega-6 derivatives. First, it is turned into gamma-linolenic acid (GLA, or 18:3w6), then to dihomo-gamma-linolenic acid (DGLA, 20:3W6), then to arachidonic acid (AA, or 20:4w6), and AA can be further converted to docosapentaenoic acid (DPA, or 22:5w6)). To turn AA into DPA, your body uses a 4-step process consisting of elongation, elongation, desaturation, and chain shortening, respectively. Again, desaturation is the introduction of double bonds. The enzymes that do this are called desaturases. Elongation catalyzes the addition of 2-carbons to fatty acids. This process occurs on the surface of the endoplasmic reticulum (excluding chain shortening), and leads to the formation of DPA [68].

DGLA forms series one prostaglandins, which are anabolic. AA forms series 2 prostaglandins, which serves some benefit, but for the most part generates unhealthy and catabolic results. This includes platelet aggregation, cardiovascular diseases, and inflammation, among other side-effects.

Fortunately, series one prostaglandins help to inhibit AA from being released to form PG2s, but the most potent prevention of PG2 is EPA, as discussed earlier.



In summary, omega-6s can convert to DGLA, and then to series one prostaglandins, which are good, but can additionally be converted to AA, and form series 2 prostaglandins, which are bad.

Anabolic effects

Cardiovascular health

N-6s have been shown to play an important role in cardiovascular maintenance. These effects are attributed to the metabolism of prostaglandin series one, which reduces platelet stickiness, relaxes blood vessels, enhances circulation, lowers blood pressure, and inhibits prostaglandin series 2 [54].

Anti-inflammation

Omega-6 has demonstrated anti-inflammatory effects. For example, patients with rheumatoid arthritis supplemented with GLA and, at the end of the experiment, there was a great reduction in joint inflammation [21]. Other studies testify to these results as well [22].

Lowers cholesterol

Horrobin DF et al. states [28], “For 30 years it has been known that linoleic acid can lower elevated cholesterol levels. However, the mechanisms, and exact derivative that accomplishes this, is still unclear.” But, from his research he showed that GLA--linoleic acid’s first derivative,--has cholesterol-lowering actions 170 times greater than the parent molecule, which means linoleic must be converted to GLA for these benefits to occur. The conversion is done by the enzyme delta-6-desaturase. This enzyme can be inhibited by aging, diabetes-mellitus, alcohol, catecholamines, trans-fatty acids, and saturated fats. Direct GLA consumption would therefore be beneficial for lower cholesterol.

Other journals testify to these benefits as well. For example, one showed omega-6s significantly lowered plasma total and low-density lipoprotein (LDL) cholesterol by 8 percent and 14 percent, respectively [59].

Other benefits

Omega-6s have even more benefits. Tests show they can increase thermogenesis [72], fat oxidation [61], play a role in sympathetic nervous system stimulation [52], and increase the usage of fatty acids for energy [13]. Mechanisms are very similar to the in-depth explanations I displayed under omega-3s.

Sources

Omega-6s are abundant in our society. Sources include chia (40%), evening primrose (81%), almonds (17%), flax (14%), natural peanut butter (30%), grape (71%), hemp (60%), pumpkin (50%), canola (30%), safflower oil (75%), sesame (45%), and walnuts (51%). 15% of turkey and chicken fat comes from omega-6. However, these are usually eaten without skin, which eliminates most of the fat. Good sources of GLA are evening primrose (9%), borage oil (20%), and black currant seed oil (18%).

Deficiencies

As with omega-3s, the list of side-effects for w6 deficiency is long. These include inflammatory skin, inherited skin condition, atopic dermatitis (eczema), growth retardation, poor wound healing, decreased immune system, and much more [20,29]. Note that w6s are vital for healthy skin, but not quite as potent compared to omega-3s. They also help maintain our cell membranes.

EFAS=Delicate!

Essential fatty acids are very sensitive. They must be handled with care or they are useless. Three important factors to monitor are heat, air, and light.

Heat- heating oils rich in EFAs at high temperatures will increase oxidation, and

can change the chemical structure, rendering its benefits useless. I recommend against high-temperature-frying and deep-frying foods rich in EFAs. Add the oils in after you are done cooking and then mix them within the dish. Boiling is much safer than frying, due to lower temperatures. Keeping the temperature around 100 C can help prevent oxidation and the other negative effects listed above. The best way to go is to get your EFAs raw, i.e. sushi.

Air- exposing these fats to air will promote rapid oxidation, leading to toxic, spoiled oils, which can harm your body. I recommend sealing them in tight containers; never leave them out in the air too long.

Light- when essential fatty acids are exposed to high amounts of light, they can be broken down into many toxic substances. Oxidation explodes, free radicals are produced, and the end result is rancid, toxic, worthless oils. To avoid these effects, EFAs need to be stored in dark, dense containers.

The complete package

In order for all the benefits of essential fatty acids to occur, you need to have a complete diet. Absence of any essential nutrient will inhibit your results. One valuable nutrient for this process is vitamin E.

Vitamin E

Vitamin E helps polyunsaturated fats (PUFUs) tremendously. It is effective at chain-breaking and, as an antioxidant, vitamin E is critical for preventing oxidation of PUFAs [60,76].

The commonly recommend ratio of vitamin E:PUFU is least 0.6 mg Vitamin E/g PUFA. Higher levels may be necessary for diets that are rich in fatty acids containing more than two double bonds [76].

However, vitamin E deficiency is unlikely, especially considering the fact that plants from which many EFAs are derived (I.e. safflower oil) contain high amounts of vitamin E.

Other important factors in the optimization of EFAs are vitamin B-complex, and ZMA. For this, I refer to the following articles: Supplement Review ZMA and How it Works, A comprehensive discussion on B-Complex & its relation to peak performance.

Essential Fatty Acid Ratios and Recommendations

There is no set number for total EFA consumption, or the ratio between omega-6s and omega-3s. This must be adapted according the person's requirements, goals, and results. Below I will present what must be considered.

There are several factors to be taken into account for ratios. First, on average, (this can be different, however) w6s are more frequent throughout our bodily tissues; overall it is near a 4:1 ratio in favor of omega-6. Also, the conversion of omega-3 is four times quicker than omega-6. Both EFAs compete for absorption, and can effect each other's metabolism [6,24]. N6 derivatives, however, are much more harmful to n3 production [38]. Also, EFAs have shown synergistic effects, suggesting they should both be used for optimal gain [7].

To avoid deficiency, results show that 1-4% of your calories need to come from n6s, and about 1% from n3s [8,32]. No toxicity level has been found for EFAs. Several studies have been performed on animals consuming large amounts, with no adverse effects. In addition, many cultures, such as the Eskimos, consumed great amounts of EFAs (primarily w3s), and were one of the healthiest societies ever; just make sure you consume your vitamins.

An interesting situation is in the United States (USA). An increase in vegetable oils, rich with w6s, has the USA's ratio of n6:n3 skyrocketed to about 10:1. It has been postulated that this is a major factor for the increased diseases (especially cardiovascular) within our society today [37].

With these statistics in mind, many authorities recommend a higher ratio of n6:n3, much lower than the United States average, however. Between 4:1 (In favor of n6) and 1:1 has been recommended [10], but this is based on just the minimal requirements and normal everyday function, not the athlete.

For optimal performance, around 10% of your total calories coming from essential fatty acids has been suggested [9].

Now, the real question is what is best for the athlete? It is the opinion of this author that the evidence clearly shows a higher ratio of Omega-3 to omega-6 is much more beneficial. The reasons I and several other athletes opt for this are:

1. Enhanced insulin sensitivity- as I displayed through several studies, n6 can promote insulin resistance, while n3 leads to increased insulin sensitivity. This in itself is more than enough reason to lower w6s and raise w3s.
2. Reduced PG2s- series two prostaglandins have been shown to promote several diseases, and are quite catabolic. The strongest agent against this is the omega-3, EPA. By increasing your omega-3s, you will decrease your risk for disease and avoid the negative effects of the aforementioned hormone. This will likewise decrease inflammation, improve your immune system, prevent cardiovascular diseases, and much more.
3. Increased Derivates- as stated earlier, w6s have a much stronger negative effect on w3 metabolism. By increasing this, you will help balance/optimize eicosanoid production.
4. Maximum results- overall, omega-3s have a stronger and wider list of benefits

than omega-6s. Your skin will improve drastically, nervous system will be enhanced, joints and inflammation will decrease at a higher rate, increased thermogenesis, and the prevention of several diseases, such as cardiac-related ones.

Now, let me reiterate, there are no set optimal ratios or daily recommended intakes for EFAs.

In several situations, people have opted for a higher ratio of n3:n6. For example, from a study on cancer, particularly breast cancer for women, it was concluded that, ‘These results are consistent with the hypothesis that a higher (n-3)/(n-6) PUFA ratio may reduce the risk of breast cancer, especially in premenopausal women [23].’ Diabetics also have adjusted their diets, due to omega-3s insulin-enhancing effects.

So going with a higher ratio of omega-3s is not new, and many have implemented this scheme into their diet plan.

I recommend around a 2:1 and 3:1 ratio of omega-3:omega-6. This can be adjusted, however, according to your results and goals. At least 30% of your daily fat should come from EFAs, and going higher is perfectly fine. I would monitor this by how your body responds.

If you are new to essential fatty acids, I suggest you start at a minimal dosage, and progressively increase them, in order to let your body adapt to it and avoid any gastrointestinal distress.

As far as what to consume, the main derivatives I would focus on are EPA and DHA. I recommend at least 3 grams total daily, along with your other omega-3s, such as flax. You can get this from fish or fish oil supplements.

A sample day for me may include: 3 Tbsp of flax, 2 Tbsp natty pb, 1 Tbsp safflower oil, plenty of dark leafy greens, and 10 oz of salmon.

Chart

Below I have included a vast essential fatty acid chart. I will give the percentage of fats, which come from omega 6 and omega 3 fatty acids for several foods. Enjoy!

Food	Omega 3	Omega 6
Cashew	0%	6%

Walnut	5%	51%
Safflower	0%	75%
Chia	30%	40%
flax	58%	14%
Pumpkin	15%	50%
Grape	0%	71%
Sunflower	0%	65%
Hemp	20%	60%
Wheat germ	5%	50%
Evening Promise	0%	81%
Corn	0%	59%
Sesame	0%	45%
Canola	7%	30%
Natural Peanut Butter	0%	30%
Almonds	0%	17%
Pistachio	0%	19%
Olive oil	0%	8%
Kukui	29%	40%
Soybean	7%	50%
Rice bran	1%	35%
Salmon	30%	0%
Borage oil	0%	34%

Conclusion

To conclude this article, I turn to the apostle Paul [1]:

Romans 8:31-39

31 What shall we then say to these things? If God be for us, who can be against us? **32** He that spared not his own Son, but delivered him up for us all, how shall he not with him also freely give us all things? **33** Who shall lay any thing to the charge of God's elect? It is God that justifieth. **34** Who is he that condemneth? It is Christ that died, yea rather, that is risen again, who is even at the right hand of God, who also maketh intercession for us. **35** Who shall separate us from the love of Christ? shall tribulation, or distress, or persecution, or famine, or nakedness, or peril, or sword? **36** As it is written, For thy sake we are killed all the day long; we are accounted as sheep for the slaughter. **37** Nay, in all these things we are more than conquerors through him that loved us. ***38 For I am persuaded, that neither death, nor life, nor angels, nor principalities, nor powers, nor things present, nor things to come, 39 Nor height, nor depth, nor any other creature, shall be able to separate us from the love of God, which is in Christ Jesus our Lord.***

Keep it Hardcore,

Venom
Executive of Bioenergetic Research
Venom@abcbodybuilding.com

References:

1. God.

2. Andrioli G, Carletto A, Guarini P, Galvani S, Biasi D, Bellavite P, Corrocher R.Differential effects of dietary supplementation with fish oil or soy lecithin on human platelet adhesion.

3. Bjerve KS, Fischer S, Wammer F, Egeland T. alpha-Linolenic acid and long-chain omega-3 fatty acid supplementation in three patients with omega-3 fatty acid deficiency: effect on lymphocyte function, plasma and red cell lipids, and prostanoid formation.

4. Blok WL, Katan MB, van der Meer JW Modulation of inflammation and cytokine production by dietary (n-3) fatty acids. J Nutr. 1996 Jun;126(6):1515-33. Review.

5. Berger A, German JB, Chiang BL, Ansari AA, Keen CL, Fletcher MP, Gershwin ME. Influence of feeding unsaturated fats on growth and immune status of mice.

6. Broughton KS, Whelan J, Hardardottir I, Kinsella JE. Effect of increasing the dietary (n-3) to (n-6) polyunsaturated fatty acid ratio on murine liver and peritoneal cell fatty acids and eicosanoid formation.

7. Bordoni A, Lopez-Jimenez JA, Spano C, Biagi P, Horrobin DF, Hrelia S. Metabolism of linoleic and alpha-linolenic acids in cultured cardiomyocytes: effect of different N-6 and N-3 fatty acid supplementation.

8. Bjerve KS. n-3 fatty acid deficiency in man.

9. Bezard J, Blond JP, Bernard A, Clouet P. The metabolism and availability of essential fatty acids in animal and human tissues.

10. Blank C, Neumann MA, Makrides M, Gibson RA. ptimizing DHA levels in piglets by lowering the linoleic acid to alpha-linolenic acid ratio.

11. Chicco A, D'Alessandro ME, Karabatas L, Gutman R, Lombardo YB. Effect of moderate levels of dietary fish oil on insulin secretion and sensitivity, and pancreas insulin content in normal rats.

12.. Chandrasekar B, Fernandes G.Decreased pro-inflammatory cytokines and increased antioxidant enzyme gene expression by omega-3 lipids in murine lupus nephritis.

13. Clandinin MT, Wang LC, Rajotte RV, French MA, Goh YK, Kielo ES. Increasing the dietary polyunsaturated fat content alters whole-body utilization of 16:0 and 10:0.

14. Clarke SD.Polyunsaturated fatty acid regulation of gene transcription: a mechanism to improve energy balance and insulin resistance.

15. Collins A, Cerin A, Coleman G, Landgren BM. Essential fatty acids in the treatment of premenstrual syndrome.

16. Calder PC.Long-chain n-3 fatty acids and inflammation: potential application in surgical and trauma patients.Braz J Med Biol Res. 2003 Apr;36(4):433-46. Epub 2003 Apr 08. Review.

17. Donadio JV Jr, Larson TS, Bergstralh EJ, Grande JP. A randomized trial of high-dose compared with low-dose omega-3 fatty acids in severe IgA nephropathy.

18. Donadio JV Jr, Grande JP, Bergstralh EJ, Dart RA, Larson TS, Spencer DC. The long-term outcome of patients with IgA nephropathy treated with fish oil in a controlled trial. Mayo Nephrology Collaborative Group.

19. Dry Skin Remedy, Get Plenty of Omega-3 Fatty Acids

20. Fleming CR, Smith LM, Hodges RE. Essential fatty acid deficiency in adults receiving total parenteral nutrition. Am J Clin Nutr. 1976 Sep;29(9):976-83.

21. Furse RK, Rossetti RG, Zurier RB. Gammalinolenic acid, an unsaturated fatty acid with anti-inflammatory properties, blocks amplification of IL-1 beta production by human monocytes. J Immunol. 2001 Jul 1;167(1):490-6.

22. Fan YY, Chapkin RS. Importance of dietary gamma-linolenic acid in human health and nutrition.

23. Goodstine SL, Zheng T, Holford TR, Ward BA, Carter D, Owens PH, Mayne ST. Dietary (n-3)/(n-6) fatty acid ratio: possible relationship to premenopausal but not postmenopausal breast cancer risk in U.S. women.

24. Gerster H. Can adults adequately convert alpha-linolenic acid (18:3n-3) to eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3)? Int J Vitam Nutr Res. 1998;68(3):159-73. Review.

25. Gerster H. The use of n-3 PUFAs (fish oil) in enteral nutrition.

26. Group.Donadio JV Jr, Bergstralh EJ, Offord KP, Spencer DC, Holley KE. controlled trial of fish oil in IgA nephropathy. Mayo Nephrology Collaborative

27. Gallai V, Sarchielli P, Trequattrini A, Franceschini M, Floridi A, Firenze C, Alberti A, Di Benedetto D, Stragliotto E. Cytokine secretion and eicosanoid production in the peripheral blood mononuclear cells of MS patients undergoing dietary supplementation with n-3 polyunsaturated fatty acids.

28. Horrobin DF, Manku MS. How do polyunsaturated fatty acids lower plasma cholesterol levels?

29. Horrobin DF. Essential fatty acid metabolism and its modification in atopic eczema.

30. Holman RT. How essential are essential fatty acids?

31. Henderson WR Jr.The role of leukotrienes in inflammation. Ann Intern Med. 1994 Nov 1;121(9):684-97. Review.

32. Innis SM. Essential fatty acids in growth and development.

33. Koletzko B, Cunnane S. Human alpha-linolenic acid deficiency.

34. Kromann N, Green A. Epidemiological studies in the Upernavik district, Greenland. Incidence of some chronic diseases 1950-1974.

35. Kremer JM, Bigauoette J, Michalek AV, Timchalk MA, Lininger L, Rynes RI, Huyck C, Zieminski J, Bartholomew LE.Effects of manipulation of dietary fatty acids on clinical manifestations of rheumatoid arthritis.

36. Kremer JM, Lawrence DA, Jubiz W, DiGiacomo R, Rynes R, Bartholomew LE, Sherman M. Dietary fish oil and olive oil supplementation in patients with rheumatoid arthritis. Clinical and immunologic effects.

37. Kinsella JE, Lokesh B, Stone RA. Dietary n-3 polyunsaturated fatty acids and amelioration of cardiovascular disease: possible mechanisms.

38. Lands WE. Biochemistry and physiology of n-3 fatty acids.

39. Liu S, Baracos VE, Quinney HA, Clandinin MT. Dietary omega-3 and polyunsaturated fatty acids modify fatty acyl composition and insulin binding in skeletal-muscle sarcolemma.

40. Luo J, Rizkalla SW, Boillot J, Alamowitch C, Chaib H, Bruzzo F, Desplanque N, Dalix AM, Durand G, Slama G. Dietary (n-3) polyunsaturated fatty acids improve adipocyte insulin action and glucose metabolism in insulin-resistant rats: relation to membrane fatty acids.

41. Li XL, Steiner M. Fish oil: a potent inhibitor of platelet adhesiveness.

42. Lau CS, Morley KD, Belch JJ. Effects of fish oil supplementation on non-steroidal anti-inflammatory drug requirement in patients with mild rheumatoid arthritis--a double-blind placebo controlled study.

43. Lands WE. Biochemistry and physiology of n-3 fatty acids.

44. Lenn J, Uhl T, Mattacola C, Boissonneault G, Yates J, Ibrahim W, Bruckner G.The effects of fish oil and isoflavones on delayed onset muscle soreness. ivision of Clinical Nutrition, College of Health Sciences, Department of Kinesiology & Health Promotion, University of Kentucky, Lexington, USA.

45. Leaf A, Weber PC. Cardiovascular effects of n-3 fatty acids.

46. Mantzioris E, James MJ, Gibson RA, Cleland LG. Dietary substitution with an alpha-linolenic acid-rich vegetable oil increases eicosapentaenoic acid concentrations in tissues.

47. Mantzioris E, James MJ, Gibson RA, Cleland LG. Differences exist in the relationships between dietary linoleic and alpha-linolenic acids and their respective long-chain metabolites.

48. Morris MC, Sacks F, Rosner B. Does fish oil lower blood pressure? A meta-analysis of controlled trials.

49. Moriguchi K, Yuri T, Yoshizawa K, Kiuchi K, Takada H, Inoue Y, Hada T, Matsumura M, Tsubura A. Dietary docosahexaenoic acid protects against N-methyl-N-nitrosourea-induced retinal degeneration in rats.

50. McCarthy GM, Kenny D. Dietary fish oil and rheumatic diseases.

51. Macho L, Fickova M, Sebokova E, Mitkova A, Klimes I. Effect of dietary fish oil on 2-deoxy-D-3H glucose uptake in isolated adipocytes of rats fed various diets.

52. Matsuo T, Shimomura Y, Saitoh S, Tokuyama K, Takeuchi H, Suzuki M. Sympathetic activity is lower in rats fed a beef tallow diet than in rats fed a safflower oil diet.

53. Principles of Human Physiology. William J German, Cindy L. Stanfield.

54. Pietinen P. Dietary fat and blood pressure. Ann Med. 1994 Dec;26(6):465-8. Review.

55. Popp-Snijders C, Schouten JA, Heine RJ, van der Meer J, van der Veen EA. Dietary supplementation of omega-3 polyunsaturated fatty acids improves insulin sensitivity in non-insulin-dependent diabetes.

56. Puertollano MA, de Pablo MA, Alvarez de Cienfuegos G. Modulatory effects of long-chain n-3 fatty acids on cell functions.

57. Pellizzon M, Buison A, Ordiz F Jr, Santa Ana L, Jen KL. Effects of dietary fatty acids and exercise on body-weight regulation and metabolism in rats. Department of Nutrition and Food Science, Wayne State University, Detroit, MI 48202, USA.

58. Proc Natl Acad Sci U S A. 1995 Jan 17;92(2):517-21. V Griffiths RJ, Pettipher ER, Koch K, Farrell CA, Breslow R, Conklyn MJ, Smith MA, Hackman BC, Wimberly DJ, Milici AJ, et al. Leukotriene B4 plays a critical role in the progression of collagen-induced arthritis.

59. Rassias G, Kestin M, Nestel PJ. Linoleic acid lowers LDL cholesterol without a proportionate displacement of saturated fatty acid.

60. Sijben JW, Schrama JW, Nieuwland MG, Hovenier R, Beynen AC, Verstegen MW, Parmentier HK. Interactions of dietary polyunsaturated fatty acids and vitamin E with regard to vitamin E status, fat composition and antibody responsiveness in layer hens.

61. Shimomura Y, Tamura T, Suzuki M. Less body fat accumulation in rats fed a safflower oil diet than in rats fed a beef tallow diet.

62. Storlien LH, Kraegen EW, Chisholm DJ, Ford GL, Bruce DG, Pascoe WS. Fish oil prevents insulin resistance induced by high-fat feeding in rats.

63. Sonya Dakar Omega - 3 Repair Complex, Scarred Skin by Joy Beauty

64. Sea Pet -- omega-3 fish oil, essential fatty acids for dogs, cats

65. Suzuki R, Shimizu T, Kudo T, Ohtsuka Y, Yamashiro Y, Oshida K.Effects of n-3 polyunsaturated fatty acids on dermatitis in NC/Nga mice.

66. Storlien LH, Jenkins AB, Chisholm DJ, Pascoe WS, Khouri S, Kraegen EW. Influence of dietary fat composition on development of insulin resistance in rats. Relationship to muscle triglyceride and omega-3 fatty acids in muscle phospholipid.

67. Sanderson P, Yaqoob P, Calder PC. Effects of dietary lipid manipulation upon graft vs host and host vs graft responses in the rat.

68. Sprecher H, Luthria DL, Mohammed BS, Baykousheva SP.J Reevaluation of the pathways for the biosynthesis of polyunsaturated fatty acids. Lipid Res. 1995 Dec;36(12):2471-7. Review.

69. Simons LA, Hickie JB, Balasubramaniam S. On the effects of dietary n-3 fatty acids (Maxepa) on plasma lipids and lipoproteins in patients with hyperlipidaemia.

70. Tato F, Keller C, Wolfram G. Medizinische Poliklinik, Universitat Munchen. Effects of fish oil concentrate on lipoproteins and apolipoproteins in familial combined hyperlipidemia.

71. Terry P, Wolk A, Vainio H, Weiderpass E. Fatty fish consumption lowers the risk of endometrial cancer: a nationwide case-control study in Sweden.

72. Takeuchi H, Matsuo T, Tokuyama K, Shimomura Y, Suzuki M. Diet-induced thermogenesis is lower in rats fed a lard diet than in those fed a high oleic acid safflower oil diet, a safflower oil diet or a linseed oil diet.

73. Tavani A, Pelucchi C, Parpinel M, Negri E, Franceschi S, Levi F, La Vecchia C. n-3 polyunsaturated fatty acid intake and cancer risk in Italy and Switzerland.

74. von Schacky C, Fischer S, Weber PC.Long-term effects of dietary marine omega-3 fatty acids upon plasma and cellular lipids, platelet function, and eicosanoid formation in humans.

75. van der Tempel H, Tulleken JE, Limburg PC, Muskiet FA, van Rijswijk MH. ffects of fish oil supplementation in rheumatoid arthritis.

76. Valk EE, Hornstra G. Relationship between vitamin E requirement and polyunsaturated fatty acid intake in man: a review.

77. Wu D, Meydani SN, Meydani M, Hayek MG, Huth P, Nicolosi RJ. Immunologic effects of marine- and plant-derived n-3 polyunsaturated fatty acids in nonhuman primates.

78. Yu Yevon, Endocrine Insanity Part I. Hyperplasia The Magazine.

79. Yu Yevon, Endocrine Insanity Part II . Hyperplasia The Magazine.

80. Yu Yevon, Endocrine Insanity Part III Hyperplasia The Magazine.