

Omega 3 and Omega 6: The Ideal Ratio:

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There is a lot of confusion in the public's mind about the correct balance between Omega 6 and Omega 3 fatty acids, and how to design a diet to incorporate the "ideal" ratio for optimum benefit.

It has been estimated that the ratio of Omega-6 to Omega-3 fatty acids in the diet of early humans was 1:1. The ratio in the United States today has risen to 10:1 because of the combination of reduced Omega-3 fatty acid intake and the widespread use of vegetable oils rich in Linoleic Acid. Because of the well-known competition between Linoleic Acid (LA) and Alpha Linolenic Acid (ALA) for metabolic conversion to GLA/DGLA/AA & EPA/DHA: reducing the former while increasing the latter (or simply increasing the latter) is a strategy for increasing tissue levels of Omega-3 fatty acids.

Another obvious strategy is to simply consume more GLA and DHA, an approach that minimizes the significance of the ratio.

It is hypothesized that many of the health issues facing modern society such as cancer, heart disease, diabetes, obesity, Crohn's disease, etc. is because of the high ratio of Omega 6 to Omega 3 EFA's in the modern diet and that all we have to do is to increase dietary consumption of oils which contain higher amounts of Omega 3 such as flax oil or hemp oil and reduce the consumption of predominately Omega 6 oils such as soy oil, olive oil, corn oil and canola oil to reduce the incidence of these "lifestyle diseases" By using fear mongering as a marketing tactic to gain a larger share of the huge consumer market for vegetable oils, the growers and producers of flax seed and flax oil infer that excess Omega 6 vegetable oils are dangerous to health, and that flax seed oils high in Omega 3 should replace oils high in Omega 6.

The problem in this "logic" is that there is no distinction made about which Omega 6 oil and which Omega 3 oil they are referring to. Linoleic Acid (LA), the essential Omega 6 fatty acid obtained from vegetable oils, is not used by the body in that form but must first be transformed by a process called *desaturation* by the Delta 6 desaturase enzyme to form Gamma Linolenic Acid (GLA). GLA in turn is *elongated* by enzyme activity to form Dihomo- γ -linolenic acid (DGLA), a 20-carbon ω -6 fatty acid. DGLA is the direct precursor to the important Prostaglandin series 1. DGLA is further desaturated to form Arachidonic Acid (AA) which is the precursor to Prostaglandin series 2. The amount of AA produced by the body is limited by many factors and in many disease states very little GLA is produced, limiting the amount of DGLA and AA available for metabolic purposes. AA is sometimes referred to as the "bad" Omega 6 because excess AA has pro-inflammatory properties which can exacerbate many health issues. The truth is that AA is vital to human health but that an excess can produce inflammatory activity. The ideal balance of AA and all other EFA's is the dietary goal to correct deficiencies and to promote optimal health.

The current hypothesis that excess Omega 6 is the culprit for many of our health issues is not based on actual fact, but on marketing statements from the flax industry and from researchers who are often sponsored by the flax and Omega 3 fish oil industry.

The suggestion that excess Omega 6 can be harmful has to be qualified: Excess AA, an Omega 6 fatty acid, has been clinically shown to be a risk factor in some disease states, but the only dietary source of AA is from meat, eggs and dairy. AA does not occur in plant food. Excess Omega 6 in the form of AA can only be reduced by reducing the amount of meat, eggs and dairy products from the diet.

Therefore, increasing consumption of higher Omega 3 foods such as flax and reducing the higher Omega 6 foods such as soy, corn or wheat will not limit the damage from excess AA from meat, eggs and dairy.

Unfortunately, the public and many health professionals have been duped and they accept at face value the “urban myth” that excessive Omega 6 vegetable oils are the problem and they completely ignore the main source of the problem which is excess consumption of AA from meat, eggs and dairy, which has been increasing at an alarming rate in the past 50 years. Furthermore, meat, dairy and eggs are a major dietary source of saturated fat which is a known blocking factor of the enzyme Delta 6 desaturase’s ability to form GLA with predicable health consequences.

Vegetarian Diet

It has been overwhelmingly demonstrated in many scientific studies including the largest study, *The China Study*, that a vegetarian diet is the healthier diet for humans and that vegetarians have a longer life expectancy and lower rates of cancer, heart disease, diabetes, obesity and other “modern” diseases.

Following a vegetarian diet could mean you live more than nine years longer than you might by consuming meat based diets, according to new research findings. The study data, released by researchers at the *Loma Linda University, USA*, finds that people following a vegetarian diet have a number of health benefits compared to those who consume meat – and on top of those benefits is a longer lifespan, with vegetarian men living an average of 9.5 and women an average of 6.1 years longer than meat eaters

A study published on August 2, 2016 by the *JAMA Internal Medicine* shows that high animal protein intake was positively associated with mortality and high plant protein intake was inversely associated with mortality, especially among individuals with at least 1 lifestyle risk factor. Substitution of plant protein for animal protein especially that from processed red meat, was associated with lower mortality, suggesting the importance of protein source.

Despite having significantly lower dietary intakes of EPA/DHA (from fish consumption) blood levels of EPA/DHA were approximately the same as regular fish eaters, according to findings published in the *American Journal of Clinical Nutrition*. Vegetarians do not obtain AA or EPA/DHA from the diet, as a healthy body will make its own from dietary LA and ALA to satisfy its own metabolic requirements, as has been the case for millions of years.

Prostaglandins

Prostaglandins are lipid autacoids derived from arachidonic acid. They both sustain homeostatic functions and mediate pathogenic mechanisms, including the inflammatory response. They are generated from arachidonate by the action of cyclooxygenase (COX) isoenzymes and their biosynthesis is blocked by nonsteroidal anti-inflammatory drugs (NSAIDs), including those selective for inhibition of COX-2. Despite the clinical efficacy of NSAIDs, prostaglandins may function in both the promotion and resolution of inflammation.

Inflammation is the immune system’s response to infection and injury and has been implicated in the pathogenesis of arthritis, cancer and stroke, as well as in neurodegenerative and cardiovascular disease. Inflammation is an intrinsically beneficial event that leads to removal of offending factors and restoration of tissue structure and physiological function.

The acute phase of inflammation is characterized by the rapid influx of blood granulocytes, typically neutrophils, followed swiftly by monocytes that mature into Inflammatory macrophages, that subsequently proliferate and thereby affect the functions of resident tissue macrophages.

This process causes the cardinal signs of acute inflammation: rubor (redness), calor (heat), tumor (swelling) and dolor (pain). Once the initiating noxious stimulus is removed via phagocytosis, the inflammatory reaction can decrease and resolve. During the resolution of inflammation, granulocytes are eliminated and macrophages and lymphocytes return to normal pre-inflammatory numbers and phenotypes.

The usual outcome of the acute inflammatory program is successful resolution and repair of tissue damage, rather than persistence and dysfunction of the inflammatory response, which can lead to scarring and loss of organ function. It may be anticipated, therefore, that failure of acute inflammation to resolve may predispose to auto-immunity, chronic dysplastic inflammation and excessive tissue damage.

Prostaglandins play a key role in the generation of the inflammatory response. Their biosynthesis is significantly increased in inflamed tissue and they contribute to the development of the cardinal signs of acute inflammation. While the pro-inflammatory properties of individual prostaglandins during the acute inflammatory response are well established, their role in the resolution of inflammation is more controversial.

There are four principal bioactive prostaglandins generated *in vivo*: prostaglandin (PG) E₂ (PGE₂), prostacyclin (PGI₂), prostaglandin D₂ (PGD₂) and prostaglandin F_{2α} (PGF_{2α}). They are ubiquitously produced – usually each cell type generates one or two dominant products - and act as autocrine and paracrine lipid mediators to maintain local homeostasis in the body. During an inflammatory response, both the level and the profile of prostaglandin production changes dramatically. Prostaglandin production is generally very low in un-inflamed tissues, but increases immediately in acute inflammation prior to the recruitment of leukocytes and the infiltration of immune cells.

Prostaglandin E2 and inflammation

PGE₂ is one of the most abundant PGs produced in the body, is most widely characterized in animal species, and exhibits versatile biological activities. Under physiological conditions, PGE₂ is an important mediator of many biological functions, such as regulation of immune responses, blood pressure, gastrointestinal integrity and fertility.

Dysregulated PGE₂ synthesis or degradation has been associated with a wide range of pathological conditions. In inflammation, PGE₂ is of particular interest because it is involved in all processes leading to the classic signs of inflammation: redness, swelling and pain. Redness and edema result from increased blood flow into the inflamed tissue through PGE₂-mediated augmentation of arterial dilatation and increased microvascular permeability.

Arachidonic acid (AA)

AA is one of the most abundant fatty acids in the brain, and is present in similar quantities to docosahexaenoic acid (DHA). The two account for approximately 20% of its fatty acid content. Like DHA, neurological health is reliant upon sufficient levels of AA. Among other things, AA helps to maintain hippocampal cell membrane fluidity. It also helps protect the brain from oxidative stress by activating peroxisome proliferator-activated receptor gamma. AA also activates syntaxin-3 (STX-3), a protein involved in the growth and repair of neurons.

AA is also involved in early neurological development. In one study funded by the U.S. *National Institute of Child Health and Human Development*, infants (18 months) given supplemental AA for 17 weeks demonstrated significant improvements in intelligence, as measured by the Mental Development Index.

This effect is further enhanced by the simultaneous supplementation of AA with DHA.

In adults, the disturbed metabolism of AA contributes to neurological disorders such as Alzheimer's disease and Bipolar disorder. This involves significant alterations in the conversion of AA to other bioactive molecules (overexpression or disturbances in the AA enzyme cascade). Dietary sources of AA are not available from plant sources but are almost exclusively from meat, eggs and dairy.

A meta-analysis by Cambridge University looking for associations between heart disease risk and individual fatty acids reported a significantly reduced risk of heart disease with higher levels of EPA and DHA (Omega-3 fats), as well as the AA (Omega 6 fat). A scientific advisory from the American Heart Association has also favorably evaluated the health impact of dietary Omega-6 fats, including AA. The group does not recommend limiting this essential fatty acid. In fact, the paper recommends individuals follow a diet that consists of at least 5–10% of calories coming from omega-6 fats, including AA. It suggests dietary AA is not a risk factor for heart disease, and may play a role in maintaining optimal metabolism and reduced heart disease risk.

Dihomo- γ -linolenic acid (DGLA)

DGLA is a carboxylic acid with a 20-carbon chain with three cis double bonds; DGLA is the elongation product of γ -linolenic acid, by an efficient enzyme which does not appear to suffer from any form of (dietary) inhibition. DGLA, the precursor to prostaglandin series 1 and 2, is an extremely uncommon fatty acid found only in trace amounts in animal products.

Gamma Linolenic Acid (GLA)

GLA is the precursor to DGLA and AA and the essential Series 1 and Series 2 Prostaglandins: hormone like substances which are involved in a myriad of physiological functions including; reducing platelet aggregation, lowering blood pressure, cholesterol and triglycerides, decreasing inflammation, balancing various hormone functions and maintaining a healthy immune system.

Docosahexaenoic Acid (DHA)

Docosahexaenoic acid (DHA) is essential for the growth and functional development of the brain in infants. DHA is also required for maintenance of normal brain function in adults. The inclusion of plentiful DHA in the diet improves learning ability, whereas deficiencies of DHA are associated with deficits in learning. DHA is taken up by the brain in preference to other fatty acids. The turnover of DHA in the brain is very fast, more so than is generally realized. The visual acuity of healthy, full-term, formula-fed infants is increased when their formula includes DHA. During the last 50 years, many infants have been fed formula diets lacking DHA and other omega-3 fatty acids. DHA deficiencies are associated with foetal alcohol syndrome, attention deficit hyperactivity disorder, cystic fibrosis, phenylketonuria, unipolar depression, aggressive hostility, and adrenoleukodystrophy.

Decreases in DHA in the brain are associated with cognitive decline during ageing and with onset of sporadic Alzheimer disease. The leading cause of death in western nations is cardiovascular disease. Epidemiological studies have shown a strong correlation between fish consumption and reduction in sudden death from myocardial infarction. The reduction is approximately 50% with 200 mg day⁻¹ of DHA from fish. DHA is the active component in fish. Not only does fish oil reduce triglycerides in the blood and decrease thrombosis, but it also prevents cardiac arrhythmias. The association of DHA

deficiency with depression is the reason for the robust positive correlation between depression and myocardial infarction.

DHA is present in fatty fish (salmon, tuna, mackerel), algae (*Schizochytrium aggregatum*) and mother's milk. DHA is present at low levels in meat and eggs, but is not usually present in infant formulas. EPA, another long-chain n-3 fatty acid, is also present in fatty fish. The shorter chain n-3 fatty acid, alpha-linolenic acid, is not converted very well to DHA in man (<0.1% conversion efficiency in humans).

These long chain n-3 fatty acids (also known as Omega-3 fatty acids) are now becoming available in Europe and Japan. Fish oil decreases the proliferation of tumor cells, whereas excess arachidonic acid, a long chain n-6 fatty acid increases their proliferation. These opposite effects are also seen with inflammation, particularly with rheumatoid arthritis and with asthma. DHA has a positive effect on diseases such as hypertension, arthritis, atherosclerosis, depression, adult onset diabetes mellitus, myocardial infarction, thrombosis, and some cancers.

ALA does not increase plasma DHA in humans (<0.1% conversion efficiency in humans) in fact, neither ALA nor EPA is an effective source of DHA due to minimal in vivo production of DHA from these precursors in humans, indication that preformed DHA is most effective in maintaining sufficient tissue stores.

Health Risks When the Natural Ratio between Omega 6 & 3 is altered.

A large scale prospective study, published in the *Journal of the National Cancer Institute*, follows up findings from a 2011 study published in the *American Journal of Epidemiology* which suggested that a high Omega-3 status may be linked to prostate cancer. It was also found that high blood concentrations of Omega-3 polyunsaturated fatty acids (EPA & DHA) were linked to a 71% higher risk of developing high grade prostate cancer. The study also found a 44% increase of low-grade prostate cancer and an overall 43% increase in risk for all prostate cancers.

These studies on the prostate cancer risk of higher consumption of fish oils in relation to Omega 6 oils are never referred to in the advertising of fish oil supplements which only refer to the well know cardiovascular benefits of EPA/DHA and infer that more and higher potency fish oil capsules is desirable

Balanced Ratio of GLA/DHA

In our dietary goal to provide these essential fatty acids during growth, pregnancy, lactation, or for therapeutic approach in the management of specific disease conditions, a balance of the Omega 6 and Omega 3 fatty acids should be ensured. The correct balance is important to maintain normal cellular and other functions. The ratio of Omega 6 to Omega 3 fatty acids in all cellular lipids is approximately 4:1 except in the central nervous system, where the ratio is nearer 1:1. Human milk samples from nine different countries showed a remarkable uniformity in the ratio of 5:1 in favour of the Omega 6 fatty acids in the milk lipids in spite of the wide variations in the mother's diet in the different countries. Human red blood cell membranes also show much the same ratio.

While there is yet no clear-cut answer to what the correct balance should be, we can look at Nature to obtain guidelines on this important question. All the comparative data from various studies show a predominance of the Omega 6 fatty acids over the Omega 3. Since the Omega 3 fatty acids are preferentially metabolized in the body, a ratio of 4:1 in favour of the Omega 6 acids will ensure a balanced composition at the cellular level. Such a ratio recommendation would be applicable when the parent acids, linoleic (LA) and alpha-linolenic (ALA) are the predominant constituents in the diet.

On the other hand, the longer chain derivatives such as gamma-linolenic acid (GLA), dihomo-gamma-linolenic acid (DGLA), arachidonic acid (AA), eicosapentaenoic acid (EPA) and docosapentaenoic acid

(DHA) are biologically more active and are incorporated into cell structures more efficiently. Also, the Omega 3 acid, EPA, is preferentially incorporated into cell membranes at the expense of AA.

In situations where these longer chain polyunsaturated fatty acids are provided in the diet, a ratio of 1:1 between GLA & DHA would be desirable to ensure a correct balance at the cellular level.

In many people, the ability to convert the essential fatty acids LA (Omega 6) & ALA (Omega 3) into GLA and DHA is deficient. Saturated fats, cholesterol, trans (twisted) fatty acids and sugar interfere with the conversion; and lack of certain vitamins such as niacin (B3) and pyridoxine (B6), as well as a deficiency of the minerals magnesium, zinc and copper make conversion slow down or stop. Diabetics are poor converters; virus infections interfere with conversion and ageing slows down the rate of essential fatty acid conversion.

Only in disease states where metabolic disorders have developed from years of faulty diet can dietary GLA and DHA help restore the natural balance and exert therapeutic benefits. Where there are conversion issues, dietary flax oil or soy oil will do little to provide the active DGLA or DHA necessary for prostaglandin production. In a healthy person, only about 1% of ALA is converted to EPA and only about 9% of EPA is converted to DHA. In other words, only 0.01% of ALA is converted to DHA.

Almost everyone can benefit from supplementing their diet with balanced levels of GLA and DHA, from which the hormone-like prostaglandins are made.

Evening Primrose Oil (GLA)

Like vitamins, Essential Fatty Acids (EFAs) are required for life. They must come from foods or supplements, because the human body cannot make them. Linoleic Acid (LA) is an EFA but the production of GLA from dietary Linoleic Acid occurs only slowly, or may be blocked entirely in many people. Yet GLA is a necessary precursor to DGLA and AA and the production of prostaglandins series 1 & 2 which, like hormones, regulate vital cell activities and determine the state of health of all cells, tissues and organs.

Clinical studies have demonstrated the value of dietary GLA (Evening Primrose Oil) in managing a variety of disorders including:

Atherosclerosis; Diabetes Mellitus; Eczema; Multiple Sclerosis; Pre-menstrual Syndrome (PMS); ADD (Attention Deficit Disorder);

Most Clinical studies that support the beneficial effects of GLA therapy have been conducted with Evening Primrose Oil (EPO). Until studies of GLA therapy using GLA sources other than EPO have been conducted, EPO remains the only proven biologically effective source of GLA.

One study (*Lawson L.D. & Hughes, B. G., Lipids, Volume 23, No. 4, 1988*) indicates that when linoleic acid occupies a middle position on triglyceride molecules, it enhances absorption of fatty acids on outside positions. Evening Primrose Oil has linoleic acid on most of its middle positions, far more often than other GLA-containing oils, & most of its GLA is on outside positions. The researchers conclude: "GLA in Evening Primrose Oil may be more efficiently absorbed than from other known GLA oils". The other study (*Manku et al, Journal of the American Oil Chemists Society, Volume 65, No.14, 1988*) shows that Evening Primrose Oil forms DGLA, the precursor of the beneficial prostaglandin 1 series, in higher concentrations than other GLA-containing oils (borage, black currant, fungus).

Schizochytrium aggregatum (DHA)

Schizochytrium aggregatum is a genus of unicellular protists, one of several that make up the thraustochytrids. These organisms were once thought of as a kind of fungus, but are now assigned to the stramenopiles, a group that also contains kelp and an array of micro-algae. Members of the genus produce significant amounts of docosahexaenoic acid and are now grown commercially as a source of this oil for nutrition supplementation, bio feeds, biomass and as the basis of biofuels.

This algal source of DHA is a recently available high concentration source of DHA without the potential risk of contamination from heavy metals or toxins which can come from fish sources of EPA/DHA and is a preferred source as it conserves fish stocks and helps preserve the delicate balance of marine aqua culture.

NOTE: Many companies refer to Oleic acid (Omega 9) as being in their formulas and by inference suggesting it has therapeutic value. Oleic acid (OA) is non-essential in humans. Virtually all animal and vegetable oils, including Evening Primrose Oil contain Oleic acid in varying degrees: there is no known deficiency in humans and no known therapeutic benefit in managing health conditions.

Summary:

- 1) A healthy Vegetarian Diet is optimum to maintain ideal levels of essential fatty acids necessary for production of prostaglandins series 1, 2, 3 which regulate all cell activity in the human body.
- 2) When lifestyle factors or disease cause breakdown in the natural conversion (desaturation) process a supplemental source of GLA and DHA is recommended.
- 3) **Vegan Omega 3-6-9** capsules offer the ideal ratio of GLA from Evening Primrose Oil and DHA from Algae (*Schizochytrium aggregatum*) in the optimum amounts and should be taken with optimum levels of all other essential vitamins, minerals and enzymes as provided in **SOURCE Optimum**