A Diet For Long-Term Weight Control And Optimal Health

Part 6 – Essential Fatty Acids And Metabolic Disruption

Facts are the air of scientists. Without them you can never fly.
-Linus Pauling

As the human body lacks the ability to synthesize them, certain polyunsaturated fatty acids of the omega-6 and omega-3 classes must be supplied from the diet, and are thus deemed "essential fatty acids (EFAs)." This moniker, however, belies the fact that, though essential in small quantities, some essential fatty acids are often present in gross excess in our modern food supply.

The widespread use of vegetable oils in commercial food preparation, as well as misguided supplementation with essential fatty acid supplements is largely responsible for an unprecedented intake of these "essential" polyunsaturated fats. Ultimately, the metabolic disruption caused by an excess of polyunsaturated fatty acids (PUFAs) extends across every facet of human biology. Immune dysfunction, hormonal imbalances, gastrointestinal disorders, mood disorders, fatigue and related disorders of energy production, weight gain, and a general tendency towards premature aging can all result from the high-PUFA intake typical of the American diet.

The fact that the American diet contains a notable excess of omega-6 fatty acids relative to omega-3 fatty acids has led many to proclaim that omega-3s are widely "deficient." This claim has created a market for omega-3 essential fatty acid supplements, but, in fact, true deficiencies of omega-6 or omega-3 EFAs have never been noted outside of laboratory or hospital settings involving poorly-constructed artificial diets. Traditional cultures, eating various diets, likely never encountered essential-fatty-acid deficiency of any sort, even though most traditional diets contained far less polyunsaturated fatty acids than our industrialized diet today. As we've seen in previous Integrated Supplements Newsletters, many healthy

cultures consumed relatively low levels of both omega-6 and omega-3 fatty acids.

In the previous Integrated Supplements Newsletter, we examined how a dramatic increase in the consumption of polyunsaturated fatty acids in recent decades may be a major contributing factor in many uniquely modern diet-related disorders. In particular, we saw how an excess of the omega-6 polyunsaturated fat known as linoleic acid may lead to increased risk of heart disease and cancer. We also saw how the unique inflammatory and proliferative nature of linoleic acid may be a significant factor responsible for the modern epidemic of obesity.

In addition, many animal and human studies show that a dramatic ***reduction*** in the essential fatty acid content of the diet is associated with numerous health benefits including a remarkable increase in metabolism, weight loss, improved liver function, and increased resilience in the face of various toxins and pathogens.

Ultimately, the optimal dietary dose of omega-6 and omega-3 essential fatty acids may be far lower than is often recognized. Minimizing or reversing the damage caused by excess PUFA requires an understanding of their biological effects, and a diet geared towards the avoidance of all but trace amounts of these fatty acids.

Constructing a Low-PUFA Diet

Before looking further at the metabolic disruption cased by polyunsaturated fats, it's important to gain some practical perspective on how to construct a low-PUFA diet. If our goal is to build diets with ~2% of calories coming from omega-6 linoleic acid, for example, how do we calculate this, and how does the incorporation of different foods affect the diet's overall fatty acid levels and ratios?

Rather than spending an inordinate amount of time researching the nutritional composition of food and manually calculating the fatty acid breakdown of the diet, the task of constructing a low-PUFA diet can be made infinitely easier with the use of diet-analyzing software available free online.

For example, CRON-O-METER.

CRON-O-METER downloadable version

CRON-O-METER web version

CRON-O-METER uses food composition information from the USDA and custom databases to break down the calorie, macronutrient, vitamin, mineral, fiber, and fatty acid content of individual foods and overall diets. Simply enter the amount of various foods consumed, and the software automatically calculates totals for the day.

The CRON-O-METER calculates the fatty acid composition of the diet including omega-6 and omega-3 fatty acids. Where linoleic acid is by far the most prevalent dietary omega-6 fatty acid, the omega-6 figure gives us a fairly good indication of the linoleic acid content of the diet. To figure what percentage of dietary calories are coming from linoleic acid, we simply take the amount of linoleic acid in the daily diet, multiply by 9 (fats contain 9 calories per gram), and divide this number by the total daily calories.

The Percentage of Fatty Acids in the Diet

$$\frac{\text{Fatty Acid Total} \times 9}{\text{Total Calories}} \times 100$$

Fatty Acid Total (in grams) (e.g., linoleic acid); 9 (Fats Contain ~9 Calories Per Gram)

Now, let's take a look at a hypothetical day's diet, geared specifically towards reducing overall PUFA and linoleic acid intake:

A Hypothetical Low-PUFA Diet

Breakfast
Whole Milk 16 oz
Integrated Supplements CFM® Whey Protein Isolate Vanilla 1 serving
Integrated Supplements Fiber Balance® 1 serving

Lunch
Seasoned Ground Beef 8 oz
Romaine Lettuce Salad 100 grams
Raw Carrot

Snack
Medium Apple
Gelatin 9 grams

Pre-Workout
Integrated Supplements CFM® Whey Protein Isolate Vanilla 1 serving
Orange Juice 16 oz

Dinner / Evening Meal
2 eggs
(cooked in) Coconut Oil 1 tsp
Mixed Vegetables 1 cup
Butter 1 tbsp

Totals:

Total Calories: 2068
Omega-6: 4.5 grams
Omega-3: 1.3 grams
Percentage Omega-6: 1.6%
Percentage Omega-3: 0.6%
Omega-6-to-Omega-3 Ratio: ~ 3:1

In this example of a day's diet, we can see that even low-PUFA foods still contain some omega-6 and omega-3 fatty acids. When the overall PUFA intake is low, the omega-6-to-omega-3 ratio of the diet is naturally lowered to a healthier level.

For illustrative purposes only - not to be construed as dietary advice.

We can see that even when common sources of polyunsaturated fats are avoided, the diet still contains some omega-6 and omega-3 fatty acids. In all likelihood, the amounts of omega-6 and omega-3 fatty acids in the above hypothetical diet are sufficient to prevent EFA deficiency – and the omega-6 to omega-3 ratio of the above diet (~3:1) is certain to be healthier than the average American ratio which is often estimated to be greater than 10:1.

The above diet contains 1.6% of it's calories from omega-6 fatty acids. Let's now look at how this percentage changes on a similar diet which incorporates a bit more polyunsaturated fat. Say, for example, that we added 2 tablespoons of peanut butter to our morning protein shake. Then we added 1 tablespoon of salad dressing to our lunchtime salad; and we used olive oil to cook our eggs in the evening rather than coconut oil.

A Slightly Higher-PUFA Diet

Breakfast
Whole Milk 16 oz
Integrated Supplements CFM® Whey Protein Isolate Vanilla 1 serving
Integrated Supplements Fiber Balance® 1 serving
Peanut Butter 2 tbs.

Lunch
Seasoned Ground Beef 8 oz
Romaine Lettuce Salad 100 grams (w/ 1tbs salad dressing)
Raw Carrot

Snack
Medium Apple
Gelatin 9 grams

Pre-Workout
Integrated Supplements CFM® Whey Protein Isolate Vanilla 1 serving
Orange Juice 16 oz

Dinner / Evening Meal
2 eggs
(cooked in) Olive Oil 1 tsp
Mixed Vegetables 1 cup
Butter 1 tbsp

Totals:

Total Calories: 2300
Omega-6: 11 grams
Omega-3: 1.6 grams
Percentage Omega-6: 5%
Percentage Omega-3: 0.6%
Omega-6-to-Omega-3 Ratio: ~ 7:1

Even slight changes to the diet can significantly increase the diet's omega-6 content. Although this diet still contains a lower-than-average amount of omega-6, just a small amount of peanut butter and vegetable / olive oil more than doubled the omega-6 content relative to the previous diet.

For illustrative purposes only - not to be construed as dietary advice.

Even with these subtle changes, the omega-6 percentage of the diet more than doubles from 1.6% to nearly 5% - still a relatively low amount, but one at which animal studies indicate the metabolic problems associated with linoleic acid may begin to manifest.

For the person not familiar with the fatty acid composition of foods, CRON-O-METER, or similar diet software, is a valuable tool in beginning to build a long-term diet centered on low-PUFA foods. Over time, however, long-term diet analysis is likely to be unnecessary. Once some core low-PUFA foods are chosen (e.g., dairy, meats, fruits, most vegetables, coconut oil, etc.) such a diet actually requires very little ongoing effort (i.e., no calorie counting or arbitrary rules to follow).

In fact, the presence of linoleic acid in the diet provides a perfect example of why calorie-counting diets are often frustratingly ineffective. Many people know that all fats contain 9 calories per gram – but as we've seen, fats are far more than just sources of caloric energy. Even though, chemically-speaking, all fats can provide 9 calories of energy per gram, an excess of fats such as linoleic acid can fundamentally interfere with every aspect of metabolic efficiency. This inhibition of metabolism is why, in the long-run, polyunsaturated fats are likely to be significantly more "fattening" than saturated fats (or an equal amount of calories as carbohydrates). As evidence, though the metabolic disruption caused by polyunsaturated fats extends across every aspect of human biology, perhaps the most fundamental disruption they cause has to do with the thyroid – the master regulator of all aspects of metabolism.

Linoleic Acid, Metabolism, and The Thyroid

In the previous edition of the Integrated Supplements Newsletter, we noted linoleic acid's ability to stimulate the production of fat-building cytokines. In addition, linoleic acid may also lead to weight gain and obesity by inhibiting thyroid function and/or the cellular action of thyroid hormones. The proper functioning of thyroid hormone ensures a robust metabolic rate, and well-known (though not the only) symptoms of impaired thyroid function include weight gain, fatigue and lethargy. Proper thyroid-hormone functioning is essential to maintaining cellular defenses against various toxins – and where thyroid plays a major role in the proper metabolism of carbohydrates and fats, it's no surprise that hypothyroidism (i.e., low thyroid functioning) is associated with diabetes and diabetes-related symptoms such as insulin resistance and elevated blood glucose:

Study Link – Insulin Action in Adipose Tissue and Muscle in Hypothyroidism.

Quote from the above study:

> *In hypothyroidism…glucose uptake in muscle and adipose tissue is resistant to insulin.*

An excess of fats such as linoleic acid can fundamentally interfere with every aspect of metabolic efficiency.

Interestingly, animals fed extremely low levels of unsaturated fatty acids are known to experience remarkable elevations in their metabolic rate and increased resistance to toxic threats. These animals eat large amounts of food without gaining fat, and they seem to be resistant to the negative effects of various toxins, including the liver toxicity of alcohol:

Study Link - The effect of essential fatty acid deficiency on basal respiration and function of liver mitochondria in rats.

Quote from the above study:

> *Basal respiration in relation to the body weight is significantly increased by EFA deficiency; it is unchanged when related to total animals under the employed experimental conditions.*

The liver-toxicity of alcohol, in fact, has been shown to directly parallel the level of linoleic acid in the diet:

Study Link - Beef fat prevents alcoholic liver disease in the rat.

Quote from the above study:

> *Rats fed tallow and ethanol developed none of the features of [alcoholic liver disease], those fed lard and ethanol developed minimal to moderate disease, rats fed corn oil and ethanol developed the most severe pathology. The degree of histopathological abnormality correlated with the linoleic acid content of fat in the diet (tallow 0.7%, lard 2.5%, corn oil 56.6%)… The much lower weight gain in tallow fed animals is likely a result of essential fatty acid deficiency.*

One of the most notable anti-thyroid effects of PUFA is their ability to inhibit the activity of cytochrome oxidase – a mitochondrial enzyme essential to energy production. Conversely, animal studies have found that the increase in metabolic rate associated with a diet free of the polyunsaturated fats may be due to an increase in the activity of cytochrome oxidase:

Study Link – The Effects of Fat Deficiency Upon Enzyme Activity in the Rat.

Quote from the above study:

> *The activity of the cytochrome oxidase…is markedly increased in fat deficiency. Results obtained with the adult animals and with the weanling animals are identical. In each case the activity of livers from rats fed the basal diet was 38 per cent greater than from the linoleate-supplemented animals or from the animals receiving corn oil. This is particularly interesting in view of the observation of Burr and Beeber and Wesson and Burr that fat-deficient rats had a markedly increased metabolic rate. The latter authors reported that the basal and assimilatory metabolic rates of fat-deficient animals were 25 per cent greater than the rates of the control animals.*

As a frame of reference, known cellular toxins such as cyanide and carbon monoxide also interfere with the activity of cytochrome oxidase. By contrast, thyroid hormone's ability to increase the activity of cytochrome oxidase largely explains thyroid's role in enhancing the metabolic rate and offering protection against various toxins and carcinogens. It's likely that polyunsaturated fats inhibit cytochrome oxidase in humans as they do in animals, and the gross excess of PUFAs in the modern diet gives reason to believe that the vast majority of Americans may suffer from a greatly reduced metabolic rate and increased susceptibility to toxic threats (e.g., carcinogens) as a result.

Note: Some researchers – noting animal studies in which implanted and chemically-stimulated cancers grew rapidly when linoleic acid was added to the diet – have speculated that linoleic acid may be required for cancer growth, but that the fatty acid may not necessarily ***cause*** cancer itself. The inhibition of cytochrome oxidase, however, is a clear example of an overtly toxic and carcinogenic effect ***caused*** by linoleic acid.

Though reducing the PUFA content of the diet is the most physiologically-sound way to improve metabolism and foster fat loss, interventions which stimulate the activity of cytochrome oxidase can nevertheless be illustrative. Interesting research involving cytochrome oxidase has recently been conducted on non-invasive cosmetic procedures for bodyfat reduction. One such procedure uses laser light in the red spectrum (635nm) – a wavelength capable of stimulating the activity of cytochrome oxidase, and thus, cellular metabolism and fat loss.

Study Link - Low-level laser therapy as a non-invasive approach for body contouring: A randomized, controlled study.

Quote from the above study:

It is suggested that laser irradiation increases the rate at which cytochrome c oxidase transfers electrons from cytochrome c to dioxygen. Moreover, it has been proposed that laser irradiation reduces the catalytic center of cytochrome c oxidase, making more electrons available for the reduction of dioxygen.

As a related aside, sunlight contains similar therapeutic wavelengths of light in addition to wavelengths which may be harmful under certain conditions. But though modern dermatologists implore us to avoid sun exposure as much as possible to reduce the risk of skin cancer, it may be the presence of chemically-reactive and easily-damaged polyunsaturated oils in the diet (and hence, the skin) which are uniquely carcinogenic – not ultraviolet light *per se*.

Studies have found, for example, that the presence of unsaturated fats in the skin increases the carcinogenic activity of UV rays:

Study Link - Relation of Antioxidants and Level of Dietary Lipid to Epidermal Lipid Peroxidation and Ultraviolet Carcinogenesis.

Quote from the above study:

…we observed that the degree of saturation of dietary lipids markedly influenced the UV carcinogenic response. Unsaturated lipid enhanced UV carcinogenesis both with respect to latency and multiplicity and it was suggested that unsaturated lipid might be required in photocarcinogenesis just as is the case for certain chemically induced cancers.

In all, polyunsaturated fats inhibit thyroid function and thyroid hormone activity by almost every conceivable mechanism.

It's no coincidence that traditional cultures with the highest degree of sun exposure didn't often succumb to cancers of the skin. A higher amount of melanin was certain to offer protection, but, perhaps, so too did the stable tropical fats in their diets. We've already seen previously how polyunsaturated fatty acids can stimulate the development of cancer, but tropical fats like those found in coconut, for example, contain very few polyunsaturated lipids. Coconut oil contains among the most chemically stable lipids, and is well known to reduce skin aging both when eaten and when applied externally.

In addition to their effects on cytochrome oxidase, polyunsaturated fatty acids are also able to impair the activity of thyroid hormone in numerous other ways.

Unsaturated oils inhibit the binding of thyroid hormone to carrier proteins:

Study Link - Effect of long-chain fatty acids on the binding of thyroxine and triiodothyronine to human thyroxine-binding globulin.

Quote from the above study:

The results indicate that the unsaturated long-chain fatty acids are potent inhibitors of thyroxine binding to thyroxine-binding globulin, whereas the saturated fatty acids have little or no effect on thyroxine binding.

Unsaturated oils also inhibit the cellular activity of thyroid hormone:

Study Link – Inhibition of nuclear T3 binding by fatty acids.

Quote from the above study:

Unsaturated fatty acids were potent inhibitors of the binding of [125I] T3 to isolated rat liver nuclei. Doses (in mumol/L) causing a 50% inhibition of nuclear T3 binding were 10 for palmitoleic acid, 11 for linoleic acid, 22 for oleic acid, 24 for arachidonic acid, and 37 for linolenic acid.

In all, polyunsaturated fats inhibit thyroid function and thyroid hormone activity by almost every conceivable mechanism. Thyroid activity is so integral to all aspects of health and metabolism, that any diet geared towards weight loss and overall health likely ***must***, therefore, contain low levels of the polyunsaturated fatty acids.

Unsaturated Fatty Acids and Diabetes

Free fatty acids (and unsaturated fatty acids in particular) inhibit the utilization of glucose by several mechanisms in addition to the disruption of thyroid hormone activity. There is reason to believe that linoleic acid may be particularly problematic in this regard. Safflower oil (which is among the richest sources of linoleic acid) has been shown to induce hyperglycemia (high blood sugar) and obesity in laboratory studies:

Study Link – High fat diet-induced hyperglycemia: Prevention by low level expression of a glucose transporter (GLUT4) minigene in transgenic mice.

Quote from the above study:

…mice fed a high-fat (safflower oil) diet develop defective glycemic control, hyperglycemia, and obesity.

As relates to human nutrition, researchers have noted that an excess of linoleic acid in the modern food supply may be a connecting link between insulin resistance and atherosclerosis:

Study Link - Polyunsaturated Fatty acids, insulin resistance, and atherosclerosis: is inflammation the connecting link?

In all, polyunsaturated fats inhibit thyroid function and thyroid hormone activity by almost every conceivable mechanism.

Quote from the above study:

Linoleic acid, the major n-6 fatty acid, is metabolized into pro-inflammatory arachidonic acid, which, in turn, gives rise to leukotrienes and protaglandins. N-3 fatty acids, found in plants and in fish, reduce the levels of arachidonic acid, thereby lowering inflammatory mediator concentrations and increasing insulin sensitization. We discuss these findings and their implications for insulin resistance and their possible effect on coronary heart disease.

Similarly, researchers have noted that high-PUFA diets – though once widely recommended – are likely to be contributing factors to obesity, hyperinsulinemia, and coronary artery disease:

Study Link - Dietary fatty acids in the management of diabetes mellitus.

Quote from the above study:

In the past it was generally accepted that high-PUFA diets were without side effects. However, recent data may suggest otherwise with regard to obesity, hyperinsulinemia, and coronary artery disease.

But, where an excess of essential fatty acids often leads to such diabetes-related symptoms, diets lacking essential fatty acids may reverse these trends. In animal studies, essential-fatty-acid-deficient diets are associated with lowered blood glucose:

Study Link - Pancreatic function in the essential fatty acid deficient rat.

Quote from the above study:

The EFA-deficient (EFAD) rats showed higher basal plasma insulin concentrations and lower basal glucose levels than control rats.

And animals fed essential-fatty-acid-deficient diets are also remarkably resistant to chemically-induced diabetes:

Study Link - Essential fatty acid deficiency prevents multiple low-dose streptozotocin-induced diabetes in CD-1 mice.

Quote from the above study:

Streptozotocin-treated mice on the control diet uniformly developed diabetes (19/19). Essential fatty acid-deficient mice treated with streptozotocin did not develop diabetes (1/13). Mean plasma glucose levels for the control and essential fatty acid-deficient mice were 384.5 +/- 23.6 and 129.1 +/- 15.5 mg/dl, respectively, at the end of 1 month.

Study Link – Prevention of Diabetes in the BB Rat by Essential Fatty Acid Deficiency.

The above study involved the administration of a chemical which destroys the insulin-producing beta cells of the pancreas – a model likely to mimic the autoimmune aspects of type I diabetes. In laboratory studies, essential fatty acid deficiency has produced remarkably protective effects against numerous autoimmune disorders. As autoimmune conditions such as lupus, multiple sclerosis, thyroiditis, rheumatoid arthritis, and celiac disease are hallmark disorders of industrialized societies, there's reason to suspect that an excess of PUFA may play a major role in the development of these disorders as well.

Essential Fatty Acid Restriction in Humans

Though feeding essential fatty acid-deficient diets to laboratory animals gives important clues regarding the biological effects of polyunsaturated fats, a state of essential fatty acid deficiency is almost impossible to achieve in humans. All whole foods – even foods which aren't known for their fat content, like fruits and green leafy vegetables – will contain trace amounts of the essential fats sufficient to prevent deficiency. Even short of an overt essential fatty acid deficiency, however, diets containing extremely low amounts of the essential fats have been shown to cause numerous metabolic improvements in human subjects.

In 1934, a researcher from the University of Minnesota volunteered to consume an extremely low-fat diet for six months. His oddly-constructed diet was engineered to supply particularly low levels of the polyunsaturated essential fatty acids, and consisted of sucrose (table sugar), potato starch, defatted milk (and cottage cheese made from this milk), orange juice, salt, carotene, vitamin D, and mineral oil (to help avoid constipation). This diet certainly didn't constitute optimal human nutrition, but the study offers one of the few scientific investigations into the requirements for, and metabolic effects of, unsaturated fats in humans.

On what was deemed to be a maintenance caloric intake of 2500 calories per day, the researcher's weight declined from 152 to 138 pounds. The researcher's previously-elevated blood pressure normalized, and migraine headaches, with which he had suffered since childhood, ceased completely. It was observed that the researcher's skin and mucous membranes were "clear and soft," and that he remained free of even a single common cold during the duration of the diet. The most remarkable subjective effect of the diet, however, was said to be a "marked absence of fatigue." The study reported: "the somewhat tired feeling usually experienced after a day's work in the laboratory disappeared within a few days from institution of the diet." In all, no negative effects from the low-fat diet were noted in the study.

Study Link – Effects of Prolonged Use of Extremely Low-Fat Diet on an Adult Human Subject.

Quote from the above study:

> *The only objective clinical effects of the diet on the adult human subject were a) a gradual loss of body weight during the first 3 months from 69 to 62 kg. and b) a coincident reduction to normal of a slightly elevated arterial blood pressure… Subjectively the most striking effects were, a) disappearance of a previously experienced feeling of fatigue at the end of the day's work and b) disappearance of recurrent attacks of migraine-like headache from which the subject had suffered for some years prior to the time of the present experiment.*

Where the mere absence of essential fats from the diet was associated with weight loss and numerous other beneficial outcomes, this study seems to be further evidence of the ability of the unsaturated fats to profoundly impair metabolism. There's no guarantee, of course that such a diet wouldn't have led to an outright essential fatty acid deficiency eventually, but even a whole food diet designed to avoid PUFAs will still contain significantly more PUFA than the above researcher's specially-formulated diet.

Earlier in the 1930's, another study illustrated that the relatively long-term consumption of low-PUFA foods was without negative effect in a human subject. This study, however, was not conducted specifically to test the effects of low-PUFA consumption. In response to a public outcry at the time questioning the quality and healthfulness of ground beef, Billy Ingram, then owner of the White Castle hamburger franchise, convinced researcher Jesse McLendon Ph.D of the University of Minnesota to study the effect of a 13-week hamburger-only diet in one of the University's students.

The student ate only White Castle hamburgers – including the bun, onions, and pickles – and water for 13 weeks. Up to two dozen hamburgers were consumed

It may take 2 to 3 years for the body's lipid stores to reflect even a dramatically lowered level of PUFAs in the diet.

each day, and the diet was without discernible negative health effects:

Article Link – Well Done. An unusual hamburger experiment is part of the University of Minnesota's dietary research annals.

It's interesting to compare this fast-food-only diet to the one documented in Morgan Spurlock's 2004 documentary, *Super Size Me*. Mr. Spurlock's more-recent 30 day fast-food binge resulted in numerous detrimental health effects (e.g., obesity, liver disorders, mood disorders). Despite the film's implied contention that beef and animal fat were largely responsible, Mr. Spulock's diet, unlike the hamburger-only diet from the 1930's, was sure to include a particularly high amount of polyunsaturated fats from numerous deep-fried fast foods.

If it is possible to induce an essential fatty acid deficiency in humans, the process would undoubtedly take quite a while. Lipids liberated from storage in adipose tissue can supply fatty acids for some time, as the half-life of the lipids stored in the body's tissues has been estimated to be in the neighborhood of 600 days:

Study Link - A mathematical relationship between the fatty acid composition of the diet and that of the adipose tissue in man.

Quote from the above study:

> *Based on literature data, the hypothesis is advanced that in human subjects a direct mathematical relationship exists between the average fatty acid composition of the habitual diet and that of the lipid stores of subcutaneous adipose tissue. Since the half-life of adipose tissue fatty acids in man is in the order of 600 days, the fatty acid pattern of depot fat provides a qualitative measure of the fat intake over a period of 2 to 3 years.*

In other words, it may take 2 to 3 years for the body's lipid stores to reflect even a dramatically lowered level of PUFAs in

the diet. This is evidence that an essential fatty acid deficiency is practically impossible to achieve in humans, but it's also evidence that the full health benefits of a low-PUFA diet may take some time to fully manifest.

Requirements For Essential Fatty Acids

For as much publicity as essential fatty acids have received in recent years, there's still very little research to indicate what doses of EFAs are actually needed by humans. Researchers conducting animal studies in the early part of the 20th Century cured essential fatty acid deficiency in growing rats by incorporating a mere 0.4% of calories as PUFA supplied by lard. The deficiency was also cured when 0.1% of calories as PUFA were supplied by liver. In adult animals (including adult humans) with lower growth requirements, the dose of EFA needed to prevent deficiency is likely to be significantly lower than these levels. Even still, in a 2000 calorie diet, both of these doses translate into less than a gram of PUFA per day – an amount simply impossible to avoid on any whole food diet (the researchers conducting the following landmark study had to use a specially-prepared diet of sucrose and casein to induce EFA deficiency):

Study Link – A New Deficiency Disease Caused By The Rigid Exclusion Of Fat From The Diet.

In all likelihood, the optimal amount of linoleic acid in the diet may be as close to zero as possible if pre-formed sources of arachidonic acid (i.e., animal-based fats) are consumed.

As the above study indicates, the requirement for essential fats is dependent upon several factors involved in fatty acid metabolism. Beef liver, which contains pre-formed arachidonic acid and vitamins needed by the desaturase enzymes, was more effective in rectifying the deficiency relative to lard.

In all likelihood, the optimal amount of linoleic acid in the diet may be as close to zero as possible if pre-formed sources of arachidonic acid (i.e., animal-based fats) are consumed. Remember that, in the production of arachidonic acid *in vivo*, linoleic acid competes for enzymes which would otherwise metabolize omega-3 fatty acids. The consumption of pre-formed arachidonic acid, however, does not inhibit these enzymes (nor does it inhibit the incorporation of omega-3s into tissue like linoleic acid does).

Though many of the harmful effects of excess PUFA are attributable to linoleic acid, the marketing of omega-3 supplements in recent years has led many people to consume historically-unprecedented and potentially-harmful amounts of omega-3 fatty acids. As we've seen, the mere reduction of dietary linoleic acid allows trace amounts of dietary omega-3s to function optimally without producing harm. In the next edition of the Integrated Supplements Newsletter, we'll look at some of the unique health risks created by the recent fad of omega-3 supplementation.