Nutrition News and Views

September/October 2003 Vol.7, No.5 For health professionals only

CHOLESTEROL, FATS, AND HEART ATTACKS – PART II

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One-fifth of Americans (about 59 million) have coronary heart disease (CHD). Many factors contribute to the problem, but attention has been focused on cholesterol and dietary fats. Part I of this article showed that neither cholesterol levels nor various lipoproteins (such as HDL, LDL) in the blood appear to be directly related to heart attacks; neither does cholesterol consumed in the diet. What about other types of fats? [i]

TRIGLYCERIDES

Triglycerides are the most prevalent fats in food and in the blood. They are essential for good health; tissues rely on them for energy. Currently, the normal blood range for triglycerides is between 100 to 200 milliliters per deciliter. The cutoff of 200 was selected for ease of memory – it is the same cutoff number used for cholesterol. The blood level of triglycerides depends on and is influenced by many factors including ingestion of food. The level after a meal can rise several hundred percent higher than that of the fasting state. Up to 12 hours must pass before the level returns to 'normal' (base). Anyone who eats three times a day, snacks, and has an occasional glass of wine will have values that are "too high" most of the time. Since triglyceride levels change throughout the day (rising and falling), a blood measurement is meaningless unless the person has been fasting for the previous 12 hours. Normal fasting levels are highly variable among individuals and analysis of blood levels is highly inaccurate. If a lab analysis finds 200 mg/dl, the true level may be anything between 100 and 300 mg/dl. For a more reliable measure of the normal level, the average of at least three measurements made at three different occasions (each preceded by 12 hours of fasting) needs to be calculated.

Some studies seem to incriminate high triglyceride levels as a CHD risk factor. One study found the risk of having a first heart attack was more than twice as high in those with the highest triglyceride levels as in those with the lowest levels. The Physicians' Health Study reported that, for every 100-point rise in triglycerides, the risk of a heart attack climbed 40%. Another study found that high triglycerides alone increased the risk of heart attack nearly three-fold. It has been suggested that the ratio of triglycerides to HDL is a stronger predictor of heart attack than the LDL/HDL ratio. Yet researchers admit there "still isn't proof that high triglycerides **cause** heart disease."

Overweight people have higher levels than thin people. Smokers have more than nonsmokers. Diabetics have higher levels than non-diabetics. People who lead a sedentary lifestyle have more than physically active people. Persons under stress have higher amounts than people at ease. Overweight, smoking, inactive, stressed, and diabetic people have more heart attacks than others. Is this due to higher triglyceride levels or are the elevated triglyerides a result of factors which contribute to CHD? It is easy to blame and medically treat triglycerides (with drugs such as niacin or Lopid). Weight loss, smoking cessation, exercise, and 'blood sugar control' are more difficult but the best ways to lower triglycerides. These actions also lower heart attack rates. But a direct relationship between lower triglycerides and fewer heart attacks has not been shown.

"Going overboard on carbohydrates," specifically refined carbohydrates, can raise triglycerides. High triglycerides are "clearly related to high insulin levels" brought on by confounding the pancreas with insults of refined carbohydrates – foods stripped of nutrients and other components that assist the body in handling sugars and starches properly. Refined carbohydrates not only lack nutrients, they deplete bodily nutrients. Unless the calories are immediately used for energy, the excess are stored as triglycerides. A person with elevated triglycerides who dramatically reduces consumption of refined carbohydrates will almost always lower his/her levels. A diet of whole natural foods and supportive supplements (to compensate for years of nonfood abuse) usually aids triglyceride levels fairly quickly. Triglycerides do not cause CHD; they reflect underlying factors that may do so.

Extra carbohydrate or fat calories are converted to fat (triglycerides) by the liver. Overweight persons with high insulin levels have higher fat formation rates than do lean persons with normal insulin levels. However, people with either high insulin levels OR normal insulin levels placed on a low-fat, high-carbohydrate diet with more than half of the carbohydrates in the form of **simple (refined) sugars**, have high fat formation and increased triglyceride concentrations. Meaning? "The low-fat, high-carbohydrate diet often recommended as a substitution for high-fat, low-carbohydrate diets may not be the best possible choice especially when most of the carbohydrate is in the form of simple sugars" as this can induce fat formation and insulin

resistance. Carbohydrate-containing whole foods are not harmful; refined, ravaged, robbed, ruined carbohydrates are the culprits. [ii]

DIETARY FATS

For several decades, reducing dietary fat intake has been promoted to decrease CHD risk. Yet despite all the studies, experience, and time, it is still a controversial issue whether dietary fat *per se* is an independent risk factor for CHD. Based on the premise that it "may" influence CHD, Americans are advised to reduce total and saturated fats to 30% and 10% (respectively) of daily calories while increasing their intake of complex carbohydrates.

When people decrease dietary fats they increase – not proteins (usually associated with fats) -- but carbohydrates, mostly REFINED carbohydrates. These raise insulin levels, lower HDL cholesterol, and elevate triglycerides (as much as 70%). "Research suggests that very low fat diets may trigger changes that increase risks of heart disease." Specifically, "high-carbohydrate, low-fat diets produce metabolic effects which would tend to increase the risk of heart disease " So "replacing saturated fat with carbohydrates will not reduce coronary heart disease risk."

A 1997 study challenged the notion that higher dietary fat *ipso facto* means increased CHD risk. A diet containing 42% fat did not cause any deterioration in heart rate, blood pressure, serum lipids, or exercise performance. Other research has had similar findings. "Several lines of evidence" indicate that the **types** of fats in the diet are more important in determining CHD risk than the total amount of fat. However, the "optimal mixture of different fatty acids" remains unsettled. "It has been increasingly recognized that the widely promoted low-fat concept is too simplistic and not compatible with available scientific data." Yet due to the vigorous campaign against fats, the belief that 'all fat is bad' is strongly imbedded and widespread.

In the prestigious journal, *Science*, Gary Taubes exposed the fact that, despite 50 years of mainstream research and hundreds of millions of research dollars, it has not been proved that eating a low-fat diet will help people live longer. Some people may benefit from lowering their consumption of some types of fat, he writes, but for people who eat a "reasonable" diet rich in whole foods such as fruits and vegetables, there is a question as to whether there are benefits sufficiently large to warrant concern. "It also questions whether all Americans will benefit from a low-fat diet." Decades of low-fat recommendations have "led many Americans to replace saturated fats with carbohydrates, not unsaturated fats." The data reveal that low-fat diets do not prevent deaths, and even if they seem to delay death, the effect is "marginal at best." [iii]

SATURATED FATS

Saturated fatty acids (SFAs) are often implicated in CHD risk, particularly long-chain SFAs. The claim is that saturated fats increase serum cholesterol levels and "harmful" LDL cholesterol, supposedly linking saturated fats, cholesterol, and CHD. SFAs are thought to raise blood cholesterol levels more than foods high in cholesterol. But the scientific basis for these assumptions is being questioned due to "large-scale misinterpretation and misrepresentation of the data." Foods containing high SFAs include animal fats like red meats, lard, dairy products (whole milk, cheese, ice cream, butter, etc.), chocolate, palm oil, palm kernel oil, and coconut oil. Hydrogenated vegetable oils could be included in this list, but they contain unnatural trans fatty acids, a different story.

One reason saturated fat is supposed to be bad is its high amount of calories. But other fats, including vegetables oils, are just as loaded with calories. Saturated fats like butter and coconut oil actually contain slightly lower levels of calories than polyunsaturated oils. Another reason for the saturated-fat attack is that it is easily converted by the liver into cholesterol. Eating saturated fats is assumed to raise blood cholesterol levels, which is believed to increase risk of CHD. But this has not been shown to be true, and neither saturated fat nor cholesterol has been shown to cause CHD. Besides, the liver converts other substances into cholesterol such as carbohydrates and other fats.

In one trial, a group of participants consumed a high-polyunsaturated, low-saturated fat diet, and the controls continued their high saturated-fat, low-polyunsaturated fat diet. The low-SFA group experienced eight deaths from heart attacks. The high-SFA group experienced no heart attack deaths. In numerous studies (such as the Roseto, Irish Brothers, and Malhotra studies) it was found that those with the "wrong" fat composition in their diet (high saturated fat) were having far fewer heart attack deaths than people on high polyunsaturated, low-saturated fat diets. A 1998 study in India found that the prevalence of CHD and "coronary risk factors" was higher in people with BOTH low AND high-saturated fat intake. The Fulani (a Nigerian seminomadic pastoral group) consume a diet rich in saturated fats, do not use tobacco, are lean, and have an active lifestyle. "Despite a diet high in saturated fat," the Fulani have low risks for CHD.

Stearic acid, a SFA, has been exonerated from upping plasma cholesterol concentrations because the body readily converts it to a "neutral" mono-unsaturated fatty acid, oleic acid. Some scientists

still consider stearic acid "thrombogenic" (causing blood clots). Other scientists say stearic acid may lower total and LDL cholesterol. Palm oil, a saturated fat previously viewed as virtual artery poison, also received a "bad rap." It does not raise blood cholesterol levels. Another misaligned fat, coconut oil, contains a type of saturated fat with "unique health properties," is no longer associated with arterial plaque buildup (atherosclerosis), and does not increase risk of heart disease. Ironically, over the past 25 years, coconut oil and palm oil in foods have been replaced by partially hydrogenated vegetable oils containing trans fatty acids -- fats conclusively shown to be dangerous.

Findings from a large study "do not support the strong association between intake of saturated fat and risk of coronary heart disease .Although a direct association between saturated fat intake and risk of coronary disease has been reported in several studies, those findings may have been confounded by fibre [fiber] intake .Benefits of reducing intakes of saturated fat and cholesterol are likely to be modest unless accompanied by an increased consumption of foods rich in fibre." A diet of whole, natural foods – unrefined and unaltered – would fit the bill. Biochemist Michael Gurr wrote " that whatever causes CHD, it is not primarily a high intake of SFAs. By the same token, major changes in SFA consumption are unlikely to lead to major benefits from CHD reduction." Research shows either a nonsignificant association between heart attacks and saturated fat intake or no association at all. Dr. William Castelli, director of the famous Framingham study, stated that " the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower the person's serum cholesterol." He conceded that "the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least, and were the most physically active."

Because saturated fats have no double carbon bonds – weak links that are easily broken – they are much more stable than polyunsaturated oils. Exposure to light, oxygen, and heat (normal cooking temperatures) does not result in an immediate or appreciable degree of oxidation – they are not easily altered or made rancid. These qualities make them preferential for use with food.

Still, Americans may consume excessive amounts of SFAs because they get inadequate amounts of other needed fatty acids. One reason for the imbalance is the manner in which meat animals are now raised. Ruminant animals, like cattle, are fed large amounts of grain, which is not natural for them. Their meat has four to six times more total fat and twice as much saturated fat than meat from totally grassfed cattle. Milk from grassfed cows contains up to five times (500%) more conjugated linoleic acid (CLA) than milk from standard, grainfed cows. Milk from grassfed animals contains more beneficial fats including well over twice as much omega-3 fatty acids as grainfed cattle. The same is true for chickens, turkeys, and eggs; free-range poultry have more beneficial fatty acids than commercially-raised. Free-range chickens have 21% less total fat, 30% less saturated fat, and 28% fewer calories. The meat has 50% more vitamin A and 100% more omega-3s. Free-range eggs contain almost 20 times more omega-3 fatty acids than supermarket eggs. People who traditionally and historically have eaten generous amounts of animal food were, in essence, eating different food. Is saturated fat "bad?" No. It is the distortions, deficiencies, and denaturing that takes its toll. Food raised in compliance with Nature's directions never contributes to CHD or any other problem. [iv]

TRANS FATTY ACIDS

Trans fatty acids (TFAs) have been conclusively linked with CHD. They raise LDL cholesterol levels, lower HDL cholesterol levels, increase lipoprotein (a) levels, raise triglyceride levels, **impair the ability of blood vessels to dilate**, and **interfere with essential fatty acids metabolism**. People consuming the most trans fats have a 50% to 66% higher risk of CHD than those consuming the least amount. A 14-year study of over 80,000 nurses showed that the women who consumed the largest amounts of trans fats had a 53% increased risk of suffering a heart attack than those at the low end of trans fat consumption. Total fat intake had little effect on the heart attack rate. Women with the largest consumption of total fat (46% of calories) had no greater risk of heart attack than those with the lowest consumption of total fat (29%).

TFAs occur in fats that have been "chemically altered by manufacturing processes" to hold a solid shape (as margarine) or thicken foods (as peanut butter). TFAs are formed when mono- or poly-unsaturated oils are heated to high temperatures, changing the fatty acid molecules. Heat converts normal unsaturated fatty acids with their natural *cis* structure into saturated fatty acids with a toxic, unnatural *trans* structure. Hydrogen is added to make the oils more solid and stable. Hydrogenation gives margarine, shortening, and pudding a creamy consistency, and prolongs the shelf life of cookies, chips, crackers, cakes, popcorn, chocolate, and other foods that contain these semi-solid oils. Partially hydrogenated oils stay solid at room temperature and do not become rancid as quickly as unaltered oils. Rancidity causes unpleasant tastes, so for sale-ability, trans fats are preferred.

TFAs are found in thousands of processed foods including cakes, cookies, French fries, corn chips, doughnuts, Danish pastries, biscuits, white bread,

pies, stick margarine, soft margarine, and vegetable shortening. Margarine may be the most common source in the US. But partially hydrogenated oils and shortenings added to processed foods contribute more to the overall consumption of trans fats than margarine. About 40% of ALL foods available in grocery stores contain TFAs. They can easily be avoided by eating unrefined, whole, unprocessed, non-fried, unaltered foods.

A study involving several European countries found that, in every country where there was low consumption of margarine and high consumption of **either olive oil or butter**, there was a very low incidence of death from heart disease. Belgium and Norway both consumed the same amount of margarine – a whopping 25 pounds per person per year. But Belgium consumes twice as much butter and twice as much olive oil as Norway. Norway is among the top countries experiencing deaths from CHD and Belgium is sixth from the bottom. This indicates that butter or olive oil (or both) is protective against the atherogenic effects of margarine. Spain and France are two countries with the lowest death rates from heart disease. Spain eats lots of olive oil but little butter; France has one of the highest butter-consumption rates. Consumption of olive oil or butter does not seem to matter. Avoidance of margarine and other trans fats makes the difference.

But, some may argue, trans fatty acids occur "naturally" in meat and dairy products. True, but "not all trans fatty acids may be harmful." The trans fats in meat do "not seem to increase the risk of myocardial infarction [heart attack]," **unlike** the trans fats in human-made partly hydrogenated fats. The former are natural, the latter are not. The body knows the difference. The artificial trans fatty acids created by technology are foreign to the body and cannot be used in a productive manner. Foods containing trans fats are promoted as containing "no cholesterol," yet they raise plasma cholesterol levels far more than natural saturated-fat and cholesterol-containing foods like butter, meat, or whole milk. Hydrogenated oils "may be the most destructive food additive currently in common use." They are used for deep-frying in fast-food restaurants. The fast-food industry switched from beef tallow to these trans-infested vegetable oils due to consumer demand for a "healthier" fat. They got just the opposite! Many researchers believe that TFAs have a greater influence on the development of CHD than any other dietary fat. Studies clearly show that trans fatty acids can contribute to atherosclerosis and heart disease – not because they are fats, but because they are altered, unnatural fats. "These are probably the most toxic fats ever known." CHD has multiple causes of both known and unknown origin. Trans fats are obviously one of them. [v]

POLYUNSATURATED FATS

The best known polyunsaturated fatty acids (PUFAs) are omega-6 fatty acids (arachidonic, linoleic, gamma[*y*]-linolenic) and omega-3 fatty acids (eicosapentaenoic [EPA], docosahexaenoic [DHA], alpha[*a*]-linolenic [ALA]). Polyunsaturated fats (such as corn, sunflower, safflower, and soy oils) seem to lower total blood cholesterol levels, particularly LDL (so-called "bad") cholesterol. They were hailed as the most healthful oils to replace saturated fats. However, researchers have found that PUFAs also lower HDL (so-called "good") cholesterol. Since a low HDL level is believed to be a risk factor for CHD, people are now nudged to use monounsaturated fats (such as olive or canola oils) since they leave HDL intact. Polyunsaturated fats evidently harm the lining of blood vessel walls (endothelial damage) that leads to atherosclerosis and CHD. Correlations have been found between PUFAs in the diet and adipose (body) fat, serum fats, and plaques in arteries.

Linoleic acid, an omega-6 and major PUFA, is the most abundant fatty acid in aortic plaque. The fat in plaques is derived from plasma LDL (vehicles that deliver fats to cells) in which there is more linoleic acid combined to cholesterol than any other fatty acid. The consumption of polyunsaturated fatty acids has steadily increased over the past 50 years or so. Now the long-term safety of diets rich in the omega-6 form is being questioned. But a recent study in Israel did not find a risk association of CHD with linoleic acid stored in bodily tissues. Yet the researchers said this is "not conclusive evidence for its safety." Other studies have been just as vague and inconclusive, whereas some find clear risks.

Linoleic acid is an essential fatty acid – it must be supplied by foods on a regular basis. Are people consuming too much polyunsaturated fats or not obtaining the correct ratio with other fatty acids? Omega-6 fatty acids like linoleic acid must be balanced with omega-3 fatty acids, for example. This concept of fatty acid ratios in the diet **alone** being the cause of problems is "overly simple." Research shows that high PUFA intake along with low antioxidant intake (from fruits and vegetables) is associated with increased CHD risk. Antioxidant portions of food complexes protect PUFAs and other fats from premature rancidity or breakdown. But this is not the entire solution either.

The processing methods for vegetable oils and the disturbed fatty-acid levels in commercially raised animals have created biochemical disruptions and imbalances that contribute to CHD and other problems. PUFAs are unstable, lacking hydrogen atoms needed for stability, and are highly susceptible to "oxidative modification" – rancidity. The more unsaturated it is, the more unstable it is.

In hundreds of studies, processed vegetable oils have been shown to harm the immune system and produce cancer in animals. In humans, excess consumption is associated with inflammatory and allergic diseases as well as cancers. Most fats in foods sold at supermarkets are processed PUFAs including cooking oils, salad dressings, baked goods, snack foods, and other items. Many are trans fats, polyunsaturated fats "spoiled by industrial hydrogenation."

When commercially extracted, the polyunsaturated oils are subjected to very high temperatures that promote rancidity and the formation of harmful breakdown products. They are then deodorized to remove the smell of rancidity. In other words, people consume large amounts of imbalanced, decomposed, putrid fats that are highly toxic. They consume inadequate amounts of fresh, natural (untampered-with) whole foods containing healthful PUFAs – vegetables, fruit, whole grains, raw nuts and seeds, legumes, fish, meat, eggs, and whole dairy products. Researchers recommend more polyunsaturated fats in the diet, "principally from cereals and vegetables," not from commercial oils and processed foods. Whole foods and their natural fatty components do not cause heart attacks. It is the altered, denatured, disrupted, fats that contribute to degeneration and disease. [vi]

OMEGA-3 FATTY ACIDS

Sufficient amounts of omega-3 fatty acids are grossly lacking in the typical American diet. People in the US consume far more omega-6 fatty acids than the omega-3 group. Many randomized trials have demonstrated that dietary or supplemental intake of omega-3 fatty acids significantly helps to lower risk of CHD, reduce risk of fatal heart attacks, prevent second heart attacks, prevent sudden death, and lower overall mortality. Data suggests that omega-3s may have an antiarrhythmic effect (assisting regular heart rhythm), may decrease heart rate variability, modestly reduce blood pressure, reduce elevated triglyceride levels (up to 30%), improve arterial compliance (flexibility) and endothelial (blood vessel lining) function. The "synergistic effects" of omega-6 and omega-3 fatty acids are inversely related to CHD prevalence.

Recent research indicates that inflammation occurs with CHD. Inflammation is the body's method of attempting repair. People given supplementary omega-3 fatty acids from fish have less inflammation and stronger arterial plaques (thicker with more fibrous caps) than controls. Since plaques are "patches" to support and reinforce weak or damaged areas in blood vessel walls, the stronger the "patches," the less need for continued inflammation and repair. Omega-3s reduce the rate of growth of atherosclerostic plaque because they help reduce the need for more "patches" and enhance plaque stability. The "vulnerability of plaque to rupture is the primary determinant of acute thrombosis-mediated cardiovascular events."

ALA (alpha-linolenic acid) is an essential fatty acid of the omega-3 group. ALA is precursor to the long-chain omega-3s EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid). Ingestion of ALA or EPA and DHA from foods or supplements elicits benefits. However, there is controversy regarding the extent of conversion of ALA into EPA and DHA when foods contain ALA and little or no EPA and DHA. Fish is one of the best sources of the end products EPA and DHA. ALA, primarily found in vegetable-source foods, must be converted to EPA and DHA by desaturase enzymes in the body. Trans fatty acids can or do inhibit the various enzyme conversions. Avoidance of manufactured trans fats would no doubt improve conversion capacity. Rich ALA sources include leafy greens (purslane also contains a small amount of EPA), flaxseeds, several types of nuts, canola and soy oils, avocado oil, and more. Plants grown in the wild or organically tend to contain more omega-3s than those conventionally-grown. Meat, poultry, eggs, and dairy products from animals raised entirely on pasture or free-range contain far more omega-3s than commercial types.

Supplemental fish sources of omega-3s might bring a quicker response, but vegetable sources "work" just as well though they may take a little longer for measurable results, perhaps six to 12 months. It takes time to incorporate nutrients into the biochemistry. Scientific studies do not usually last for months. Supplemental intake of isolated EPA and DHA may cause adverse reactions including gastrointestinal upset, clinical bleeding, a fishy aftertaste, worsening blood sugar metabolism, and a rise in LDL cholesterol. Whole food sources make more sense. Fish oil, such as cod liver oil, would seem to be an ideal supplement, but the "use of fish oil cannot be recommended in general" because it can contain significant levels of methlmercury (mercury), plychlorinated biphenyls (PCBs), dioxins, and other environmental contaminants. Processes that remove the contaminants often remove most of the nutrients too. But molecular separation (skimming off low molecular-weight components that hold the contaminants) leaves high molecular weight components and concentrates nutrients like vitamin A complex and fatty acids such as the omega-3s. [vii]

MONOUNSATURATED FATTY ACIDS

The most common monounsaturated fatty acid (MUFA) is oleic acid. The best known source of oleic acid is olive oil. Yet oils from foods such as avocado and nuts (like hazelnuts) have higher

levels of oleic acid than olive oil. Canola oil – a bioengineered low-erucic acid rapeseed oil – has become a popular source of oleic acid. And high-oleic varieties of sunflower and safflower oils are appearing. A new type of palm oil is being grown that yields high oleic oil. Peanut oil is a good source. But all these vegetable-source oils must be nonhydrogenated to be of value. Many animal fats including tallow, lard, butter, chicken fat, eggs, and human milk are also high in oleic acid.

Oleic acid evidently inhibits oxidation (rancidity) of LDL cholesterol. This is one reason why olive oil and a Mediterranean diet are accredited with preventing CHD. However, there are many sources of oleic acid, some oils containing more than olive oil. There is at least one other compound in olive oil (and another substance in whole olives) that may have protective effects on the cardiovascular system by a different mechanism. Olive oil has been researched far more than other oils and foods containing MUFAs which may also contain their own unique nutrients or other components that are just as or more protective.

MUFAs are much more stable and resistant to oxidation (rancidity) than PUFAs. The oils are less likely to be partially hydrogenated or subjected to high heats. Heat is less damaging to MUFAs than it is to PUFAs. So MUFAs are less likely to cause biochemical disturbances and more likely to retain nutrients. **Extra virgin** olive oil contains the highest concentrations of flavonoids, nutrients supportive to the integrity of the blood vessel walls. It should also be remembered that a Mediterranean-type diet characteristically provides plenty of whole grains, fresh vegetables, fruits, and legumes -- all supplying nutrients supportive to cardiovascular health – with little or no altered, denatured, toxic fats such as hydrogenated oils. While MUFAs may be beneficial to the cardiovascular system, it may also be the absence of manufactured, toxic fats and limited consumption of refined, overly-processed, contaminated foods that contribute to the healthful reputation of a Mediterranean diet.

People in Italy, Greece, and Spain who consume huge amounts of olive oil enjoy better health and longevity than Americans. Inhabitants of Sardinia, where cancer and CHD are rare, drink olive oil by the glassful. Not only are their traditional diets composed of whole, natural, locally-raised foods, but the olive oil they consume is different. Their oils are greenish, opaque, and thick, whereas the olive oil in the US is yellow and clear, an indication that nutrients have been filtered out to give the oil a 'pure' appearance. Humans should not tamper with Nature's creations; all fresh, complete, unchanged foods support health, repair, and balance. [viii]

To be concluded in Part III.

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