Hemp Oil
Composition and Health Benefits

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Introduction

Hemp (*Cannabis sativa)* has been grown for fibre and seed for the last 5,000 years, however there are indications that it was cultivated 20,000 years ago in China. Hemp was produced in North America until 1937 when the Marijuana Tax Act made it a forbidden crop, although the import of hemp seeds for use in bird feed was permitted. During the Second World War hemp production was revitalized as access to imported fibre had been curtailed. Prior to 1937, an active seed breeding program was on-going at the USDA. After this point, such programs continued in only a few European countries. Only China continued the production of hemp seed to any extent.

Hemp seed and oil are increasingly used in natural food products, such as snacks, nutrition bars, hummus, nondairy milks, breads, cereals, prepared foods and numerous other applications. The oil is available in capsules or bottles. The market for hemp oil as a topical ingredient in natural body care and cosmetic products is also growing.

Hemp seed composition is quite interesting in that unlike other oilseeds, it has near equivalent levels of oil, protein and carbohydrates. Intact hemp seed contains approximately 24% crude protein, 30% crude fat, and 32% neutral detergent fibre (a reflection of total fibre content), and 5% ash (a reflection of the total mineral content)[1].

Table 1: Composition of Hemp Seed (%)

Component	Whole Seed	Dehulled Seed
Hull[a]	20 - 35	-
Protein	20 - 25	30 - 35
Oil	25 - 34	42 - 47
Carbohydrates	32 - 36	10 - 12
Dietary Fibre	30 - 34	3 - 10
Soluble Fibre	5 - 15	3 - 7
Ash	6	6

a - Seed cover, husk, external part of the seed.

1.0 Fatty Acids

Dietary fat fulfills certain roles in the body including as a concentrated source of energy. In North America, dietary fat accounts for approximately 33-40% of total caloric intake. It serves as a source of essential fatty acids and as a carrier of fat soluble vitamins. Fat contributes to the palatability of foods and to overall feeling of satiety.

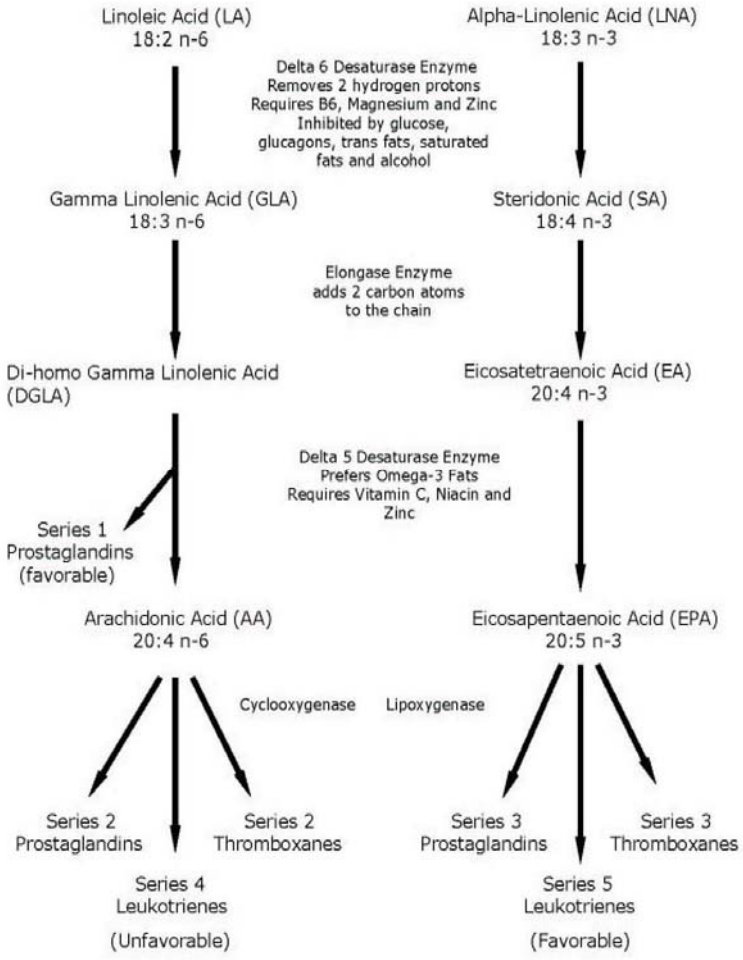
Fatty acids are the nutritional components found in dietary fats and oils. Polyunsaturated fatty acids (PUFAs) are categorized as either omega-6 or omega-3 depending upon the position of their first double bond from the terminal end of the fatty acid. The most important PUFAs for human nutrition in each omega series are:

Omega-6 (n-6) Fatty Acids		
Linoleic Acid	C18:2n-6	LA
Gamma Linolenic Acid	C18:3n-6	GLA
Dihomogamma Linolenic Acid	C20:3n-6	DGLA
Arachidonic Acid	C20:4n-6	AA
Omega -3 (n-3) Fatty Acids		
Alpha Linolenic Acid	C18:3n-3	ALA
Steardonic Acid	C18:4n-3	SDA
Eicosapentaenoic Acid	C20:5n-3	EPA
Docosahexaenoic Acid	C22:6n-3	DHA

1.1 Essential Fatty Acids (EFAs)

EFAs are required in the diet as they cannot be synthesized by humans from the shorter chain fatty acid, oleic acid (C18:1n-9). The two foundation EFAs are linoleic acid (C18:2n-6, LA) and alpha-linolenic acid (C18:3n-3, ALA)[2]. Many of the health benefits attributable to oilseeds are due to their levels of EFAs, in particular ALA.

LA and ALA are components of cellular membranes and act to increase membrane fluidity. The proper functioning of all body cells depends upon healthy membranes as they act as “gate-keepers” in the cells. The physical properties of the cell including its contents; the shape and “fluidity” of the membrane; and the permeability of substances into and out of the cell are greatly influenced by the EFA. In some membranes, such as in the skin and the layer around the nerves, specific PUFAs provide a moisture barrier and an insulating layer, respectively.



As noted in the Figure to the left, the biosynthesis of PUFAs requires a sequence of chain elongation and desaturation steps. The first step of desaturation at the six carbon position is believed to be rate limiting and involves the introduction of a double bond to form GLA and SDA of the omega-6 and omega-3 families, respectively. Genetic, dietary and environmental factors are believed to impair the human Δ -6 desaturase enzyme including aging, stress, diabetes, alcohol, smoking, cholesterol, trans- and saturated fatty acid consumption, n-3 fatty acid deficiency and vitamin and mineral deficiencies.

LA and ALA through their conversion to the longer chain PUFAs are the dietary starting point for the production of a number of important, very active, hormone-like compounds called “eicosanoids”. Eicosanoids contain twenty carbons and include compounds called prostaglandins (PGE), prostacyclins (PGI), thromboxanes (TXB) and leukotrienes (LTB).

The omega-6 and the omega-3 fatty acid families form different eicosanoids with very different activities. The different eicosanoids compete with one another for cyclooxygenase and lipoxygenase which catalyze the release of the EFA from the cell membranes.

An excess of one family of fatty acids can interfere with the metabolism of the other, reducing its incorporation into tissue lipids and altering biological effects. A proper balance of the EFAs in the diet is important for the maintenance of good health[3].

Omega 6 DGLA is the precursor of the physiologically important 'Series 1' eicosanoids:

- PGE1 inhibits platelet aggregation and inflammation, produces vasodilation, inhibits cholesterol biosynthesis, regulates immune responses and reduces blood pressure.
- 15-OH-DGLA inhibits 5- and 12-lipoxygenases, therefore inhibiting the formation of proinflammatory compounds from arachidonic acid (C20:4n-6 or ARA) such as PGE2 and 4-series leukotrienes.

Omega 3 EPA is converted to 'Series 3' eicosanoids:

- TXA3 is a weak platelet aggregator and vasoconstrictor.
- PGI3 stimulates vasodilation and inhibits platelet aggregation.
- LTB5 is a weak inducer of inflammation and a weak chemotaxic agent.

Omega 6 AA is the substrate for the "Series 2" prostaglandin:

- PGE2 exhibits pro-inflammatory and vasoconstrictive properties. It also induces inflammation, which includes redness and heat due to arteriolar vasodilation as well as swelling and localized edema resulting from increased capillary permeability.
- PGI2 acts a potent platelet anti-aggregatory agent and vasodilator.
- TXB2 causes vasoconstriction and platelet aggregation.
- LTB4 is a potent pro-inflammatory agent and a powerful inducer of neutrophil chemotaxis and adherence.

When diets are too high in omega-6 fatty acids, AA and its potent eicosanoids are produced in abundance, resulting in an over-active immune system that may contribute to chronic diseases like cancer, stroke, diabetes and coronary heart disease.

When humans consume GLA, ALA, EPA and/or DHA, these fatty acids are incorporated into cell membranes within and around the cells of the body. They partially replace AA and also compete with AA for cyclooxygenase and lipoxygenase activities. Hence the ingestion of these fatty acids can result in decreased production of PGE2, TXB2 and LTB4. Counteracting these decreases in AA derived eicosanoids are increases in the eicosanoids produced from DGLA and EPA. These alterations in the levels of eicosanoids influence the metabolic processes involved in a number of metabolic functions.

2.0 Hempseed Fatty Acids

As shown in Table 2, hempseed oil has a unique fatty acid profile especially when compared to other oils. Hempseed oil is rich in EFAs[4]. Hemp oil is rich in LA (over 55%) and contains, depending on plant variety, 15-20% ALA. Hempseed oil is low in saturated fat which constitutes only 9% of total fatty acids.

Hemp oil is a rare plant source of an omega-6 EFA called gamma linolenic acid or GLA (GLA, C18:3, omega 6). GLA may be as high as 5%, but is usually between 2 – 3%. The presence of upwards of 3% of another unique fatty acid called stearidonic acid (SDA, C18:4 omega 3) is a distinctive property of the oil. Newer varieties of hemp have fatty acid compositions that are skewed towards higher levels of the important GLA, ALA and SDA[5].

Table 2: Composition of Hemp and Selected Oils

Fatty acids (%)	Hemp	Flax	Soybean	Canola
C16:0	4 - 9	6 - 8	10 - 13	3 - 6
C18:0	2 - 4	1 - 3	3 - 5	1 - 2
Total Saturated	**6 - 13**	**7 - 11**	**13-18**	**4-8**
C18:1	8 - 15	18 - 20	18 - 28	52 - 63
Total Monounsaturated	**8 - 15**	**18 - 20**	**18-28**	**52-63**
C18:2 (LA)	53 - 60	16 - 18	50 - 58	16 - 25
C18:3 alpha (ALA)	15 - 25	53 – 57	6 - 13	9-13
C18:3 gamma (GLA)	0 - 5	-	-	-
C18:4 (SDA)	0 - 3	-	-	-
Total Polyunsaturated	**68 - 90**	**69-75**	**56-71**	**25-38**

The following chart and figure clearly identifies the limited dietary sources of ALA, GLA and SDA in fats and oils. Hempseed oil is more readily available than many of the specialty oils noted in this figure and is much more versatile for home and institutional food use.

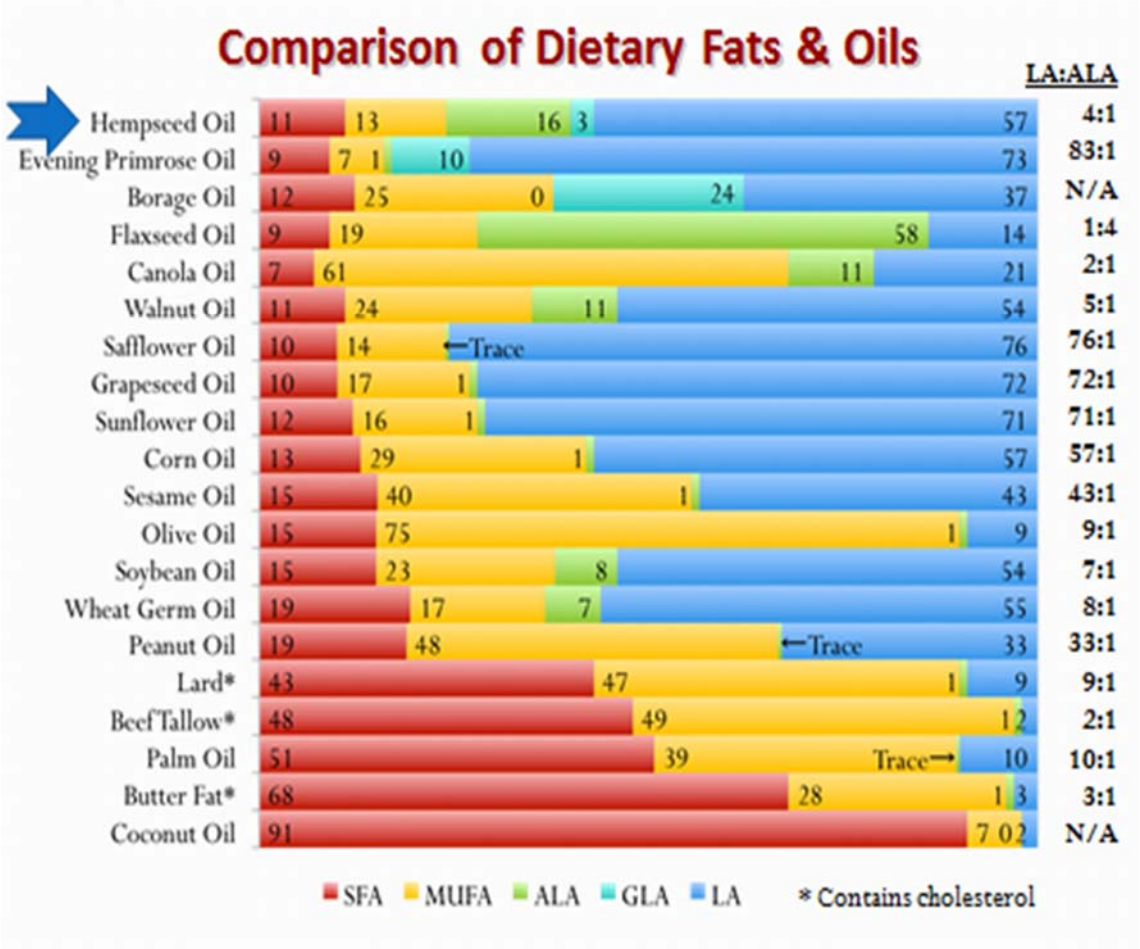
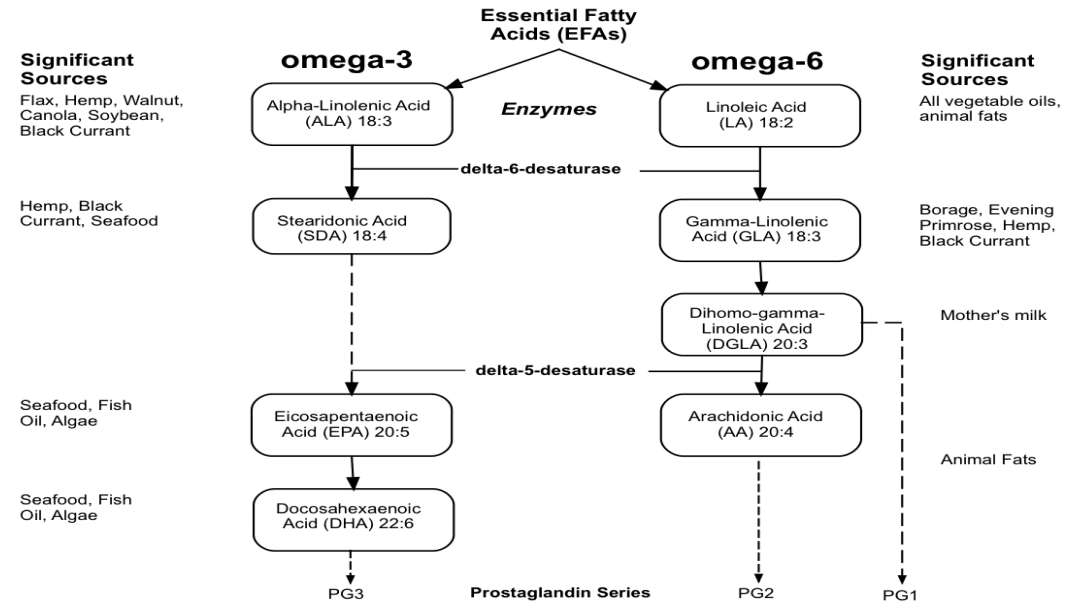


Figure 1. Metabolism of Omega-3 and -6 Fatty Acids in Humans



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3.0 Health Effects of Fatty Acids

3.1 Alpha-linolenic acid (ALA)

Certain fats and oils can significantly reduce the risk of several diseases and even contribute to the treatment of chronic conditions. Clinical research suggests that the **ALA** shows promise in the prevention of cardiovascular disease, inflammation, diabetes and hypertension.

ALA is an important contributor to total omega 3 intake and the body's requirements for EPA, commonly found in fish and fish oil. More than 25% of the North American population will not eat fish or consume fish oil. In the Health Professional Follow-up Study, which began in 1986 with a cohort of 45,772 health professionals, in men who consumed <100 mg of EPA+DHA, each 1g/day ALA intake was associated with a 58% lower risk of non-fatal heart attack and a 47% lower risk of CVD[6].

A meta-analysis of 5 cohort studies; 3 clinical trials and 9 cohort and case-control studies suggested that increasing the intake of ALA by 1.2 g/day decreases the risk of fatal CHD by at least 20%[7].

The mechanisms by which ALA can reduce CVD risk remain under investigation. However, ALA appears to:

- **Lower risk of mortality from heart disease.** ALA appears to be able to reduce the risk of uncontrolled heart rhythms and developing fatal blood clots, two major causes of heart disease deaths. EFAs, therefore, lower the chances that heart attacks will be fatal.

- **Improved heart rhythms.** Dangerously fast heartbeats or disordered heart rhythms can be fatal. EFAs help maintain stable heart rhythms by affecting the electrical activity of the heart. Thus, it is more difficult for uncontrolled heart rhythms to develop. Unstable and uncontrolled heart rhythms underlie sudden death, the cause of nearly half of all cardiac mortality. By stabilizing heart rhythms, Omega3s reduce the chance of dying suddenly.

- **Improved heart rate.** Heart rate is the number of beats a minute that the heart pumps at rest. It accelerates to increase the amount of blood ejected by the heart. Heart rate is lower in people who are physically fit and higher in the obese. Usual heart rate is between 60 and 80 beats per minute and usually increases with age. Heart rate adapts to changing conditions and generally speaking, the greater adaptability of the heart, the better its condition. Consumption of Omega3s is associated with lower heart rate and with greater heart rate variability, conditions that reflect better heart health and lower the chance of heart attack.

- **Less chance of having a first heart attack.** There is evidence that people who do not have signs of heart disease may be able to avoid having a first heart attack if they consume

Omega3s often. For people who have had a heart attack, risk of another is also substantially reduced. Consuming these fatty acids regularly improves heart rhythms, reduces the likelihood of blood clots forming, reduces the low-grade inflammation that accompanies heart disease, and improves blood lipid patterns - all effects that discourage heart failure.

- **Less chance of stroke.** A stroke results from a blood clot blocking an artery in the brain. Clots may develop there or be carried to the brain from elsewhere. Non-fatal strokes can cause serious disability. In most western countries, ischemic strokes, the kind caused by blood clots or lack of oxygen, are the most common type. Omega3s have been shown to lower the chance of having a stroke.

- **Improving blood lipids.** The blood carries different types of lipids, including fats and cholesterol, throughout the body. High levels of blood lipids lead to deposit in the walls of arteries called plaques. These reduce blood flow and supply of oxygen to the heart. ALA, EPA and DHA improve blood lipids by lowering the amount of LDL-cholesterol and triglycerides, respectively, in the blood, these improvements in blood lipids are especially important in people with type 2 diabetes who have high triglycerides and low HDL.

- **Reduced blood clotting.** Some blood clotting is essential for life, but an excessive tendency towards clotting increases the risk of blocked arteries. These can be fatal when a clot completely closes a blood vessel in the heart, lungs, or brain. Omega3s reduce platelet clumping and affect certain clotting factors reducing the tendency for blood to clot. Omega3s also improve blood flow and make red blood cells more flexible so they pass through tiny blood vessels more easily.

3.2 ALA Conversion

The conversion of ALA to EPA which is important for the generation of eicosanoids which are protective against cardiovascular and other diseases, is usually described as limited and is the subject of a great deal of controversy. Estimates of the amount of ALA converted to EPA range from 0.2% to 8%[8] with young women showing a conversion rate as high as 21%[9].

Conversion of ALA to DPA is estimated to range from 0.13% to 6%[10]. Conversion of ALA to DHA appears to be limited in humans, with most studies showing a conversion rate of about 0.05%[5]. A conversion rate of 9% was reported in young women[4]. The large differences in the rates of ALA conversion reflect major differences in study methodologies.

EPA conversion from ALA is important to meet dietary long chain PUFA requirements. If we assume a conservative rate of 2-5% conversion[11], intakes of the following Canadian crops have the potential to contribute significantly to EPA levels in the body.

Table 3: EPA synthesis from ALA

ALA Source	ALA Amount (g)	2% conversion – amount of EPA (mg)	5% conversion – amount of EPA (mg)
Canola oil, 1 T.	1.3	26	65
Flaxseed oil, 1 T.	8.0	160	400
Flaxseed, 1 T.	1.8	36	90
Hemp oil, 1 T.	**2.7**	**54**	**135**
Hempseed*, 1 T.	**1.2**	**24**	**60**

* Dehulled basis

The importance of ALA in contributing to EPA requirements is obvious when considering that the average EPA+DHA intake is roughly 100 – 135 mg/day[12]. These intake levels have not changed over the last decade despite an abundance of consumer encouraging the consumption of fish and fish oils for numerous health conditions, especially CVD.

The recommended amounts of EPA and DHA for the diet range from 200 mg-500 mg/day. Recognizing that 25% or so of the North American population do not consume fish, plant based ALA containing hemp oil provide a very critical source of omega 3 fatty acids in the Western diet[13].

3.3 Importance of ALA in Vegetarian diets

Growing evidence though is suggesting that in those people who do not consume a “typical” Western style diet high in omega 6 in combination with no fatty fish or supplements – that is vegans and vegetarians – the need for supplemental EPA and DHA may not be necessary.

At the 6th International Congress on Vegetarian Nutrition at Loma Linda University held in February 2013, numerous speakers noted that vegans and lacto-ovo vegetarians are significantly less likely than their non-vegetarian counterparts to develop heart disease, despite their low - or zero - intakes of EPA and DHA. There is also no evidence that vegans and vegetarians are at higher risk of depression, Alzheimer’s disease or other cognitive problems.

There are clearly independent health benefits for ALA which often times get overlooked.

The cardio benefits of a vegan or vegetarian diet could be attributed to the fact that they typically eat more fibre, less saturated fat, and fewer calories as well as consuming more cardio-protective phytochemicals, plant-based healthy fats.

Dr Tom Sanders, a professor in the Department of Medicine at King's College in London reported that meta-analysis of prospective cohort studies indicate a lower risk of CHD associated with increasing intakes of ALA as well as long-chain n-3 PUFA. Vegetarians and vegans have virtually no long-chain n-3 fatty acid intake and although proportions of DHA are lower in the blood and tissues of omnivores especially in vegans, the levels are in a steady

state. The cardiovascular risk profile of vegetarians and vegans is much more favourable than omnivores because of substantially lower LDL cholesterol concentrations, lower body mass index and moderately lower blood pressure. Arterial ageing as measured by arterial stiffness appears lower in vegans than in omnivores. A recent UK study has found lower incident CHD in vegetarians compared with omnivores.

Present evidence does not justify advice to vegetarians and vegans to consume long-chain n-3 fatty acids. Dietary advice should focus on ensuring that intakes of ALA are not less than 0.5% energy in all dietary groups.

4.0 Stearidonic Acid (SDA)

SDA has been the subject of limited clinical assessment. In a double-blind, parallel design, encapsulated SDA (from modified canola oil), ALA, or EPA was ingested daily in doses of 0.75 g and then 1.5 g for periods of 3 weeks each by healthy male and postmenopausal female subjects (*n* = 15/group)[14]. Dietary SDA increased EPA and docosapentaenoic acid (DPA) concentrations but not DHA concentrations in erythrocyte and in plasma phospholipids. At A 0.75-g daily dose (0–3 wk), the effectiveness of SDA was 3.1- to 5.0-fold that of ALA, and the effectiveness of EPA was 3.1- to 3.9-fold that of SDA. At a1.5-g daily dose (3–6 wk), the effectiveness of SDA was 3.7- to 4.1-fold that of ALA, and the effectiveness of EPA was 3.1- to 3.6-fold that of SDA.

5.0 Gamma Linolenic Acid (GLA)

The physiological and clinical effects of GLA have been studied for several decades. In general, healthy individuals are able to synthesize GLA from dietary LA. The conversion of LA to GLA is a slow step. It can be further inhibited by a variety of factors including diabetes, aging, zinc deficiency, excess alcohol consumption, high levels of cholesterol, certain viral infections and catecholamines released during stress. People who have inherited an atopic disposition (atopies), which makes them liable to develop eczema, asthma, allergic rhinitis or other allergies, are less able to convert LA to GLA than other people. Thus, insufficient amounts of GLA may be produced by the body even in the presence of adequate LA.

Diabetics require higher amounts of EFA because of impairments in both the delta 6 – (D6D) and delta 5-desaturase enzymes. Insulin stimulates the activity of the D6D enzyme and therefore, this enzyme's activity is reduced in diabetes. Reduced D6D activity affects the omega−6 products significantly more than the omega−3 products. Fatty acid abnormalities have been demonstrated in diabetics, particularly low DGLA and ARA in nerve membranes and red blood cell membranes. Low DGLA levels result in reduced levels of PGE1 which impairs circulation and also increases phospholipase A2 (PLA2) activity, resulting in the release of ARA

from membranes and increasing membrane stiffness. Free AA forms vasoconstrictors, restricting circulation and, over time, a deterioration of motor and sensory nerves results.

In a number of diabetic rat studies, it has been hypothesized that through its conversion from DGLA, PGE1 can prevent and reverse deficits in nerve conduction velocity (NCV) and blood flow[15]. Following GLA treatment, although diabetes develops in alloxan induced diabetic animals, the severity is much less. GLA also restores the antioxidant status to normal range in various tissues and thus can attenuate the oxidant stress prevalent in diabetic tissues[16].

In humans, GLA improves established diabetic neuropathy symptoms through an improvement in nerve flow[17]. The results of a large scale trial involving 400 patients supplemented with 480 mg/day GLA increased phospholipid GLA and DGLA in cholesterol esters; plasma and red blood cells. GLA supplementation improved a number of parameters of neuropathophysiology and clinical measurements of thermal threshold. These improvements increased over time from 3 to 12 months. In contrast, in the placebo treated group, all parameters were negatively affected. After treatment was continued for a second year, further improvements were noted in the GLA treated patients. The mechanism responsible for the positive effects of GLA may be due to a restoration of normal sciatic NCV.

6.0 Hempseed Antioxidants

In 2000, the Institute of Medicine (IOM) defined a dietary antioxidant as follows: *"A dietary antioxidant is a substance in foods that significantly decreases the adverse effects of reactive species, such as reactive oxygen and nitrogen species, on normal physiological function in humans."*

Well-known antioxidants include a number of phytochemicals, vitamin C, vitamin E and beta carotene (which is converted to vitamin A) that are capable of counteracting the damaging effects of oxidation. Antioxidants are also commonly added to food products like vegetable oils and prepared foods to prevent or delay their deterioration from the action of air.

Normal cellular processes produce free radicals – primarily reactive oxygen and reactive nitrogen molecules. Antioxidants are capable of stabilizing free radicals before they can react and cause harm. Antioxidants "neutralize" free radicals and offer protection against oxidative damage[18]. These compounds protect structural and enzymatic proteins, and prevent mutation formation that could occur when DNA and RNA are attacked by free radicals. They also preserve cell structure by keeping cell membranes fluid and functional by protecting membrane and other cellular lipids.

Hempseed oil contains tocopherols (natural Vitamin E) a fat-soluble vitamin that exists in eight different forms. Each form has its own biological activity, which is the measure of potency or functional use in the body. Alpha-tocopherol (α-tocopherol) is the most active form of vitamin E in humans and is also a powerful biological antioxidant[19].

Vitamin E may help prevent or delay coronary heart disease. Oxidative modification of LDL-cholesterol promotes blockages in coronary arteries that may lead to atherosclerosis and heart attacks[20]. Vitamin E may help prevent or delay coronary heart disease by limiting the oxidation of LDL-cholesterol. Vitamin E also may help prevent the formation of blood clots (thrombosis), which could lead to a heart attack. Observational studies have associated lower rates of heart disease with higher vitamin E intake. A study of approximately 90,000 nurses suggested that the incidence of heart disease was 30% to 40% lower among nurses with the highest intake of vitamin E from diet and supplements. The range of intakes from both diet and supplements in this group was 21.6 to 1,000 IU (32 to 1,500 mg), with the median intake being 208 IU (139 mg)[21].

Results of two national surveys, the National Health and Nutrition Examination Survey (NHANES III 1988-94)[22] and the Continuing Survey of Food Intakes by Individuals (1994-96 CSFII)[23] indicated that diets of most Americans do not provide the recommended intake for vitamin E. Caution was noted that low fat diets can result in a significant decrease in vitamin E intake if food choices are not carefully made to enhance α-tocopherol intakes.

As noted, hempseed oil contains tocopherols at higher levels than other oils containing GLA, such as borage, blackcurrant and evening primrose. The composition of tocopherols in hemp oil is similar to soybean oil where the gamma isomer is dominant with smaller levels of the alpha form. Gamma tocopherol has the strongest antioxidant capacity in food systems such as oils[24].

Table 4: Tocopherol Content in Hempseed and other Oils

Tocopherols (%)	Hemp	Borage	Black-Current	Evening Primrose	Soybean	Canola
Alpha	10	10	10	40	10	30
Beta	1	-	2	-	5	6
Gamma	85	20	80	55	65	60
Delta	4	70	8	5	20	4
Total Tocopherols (ppm)	**600-1200**	**500-800**	**400-800**	**200-600**	**1200-2200**	**500-1200**

7.0 Hempseed Phytosterols

Phytosterols, also known as plant sterols, are a naturally occurring class of compounds found in the cells and membranes of grains, fruits and vegetables. There are approximately 250 different sterols and related lipid-like compounds with the most common ones being beta-sitosterol, stigmasterol, and campesterol. The phytosterols available to the food industry are mostly derived from vegetable oils such as corn and soy, or from tall oil. Plant sterols are naturally present in Western diets in amounts similar to that of dietary cholesterol (~170–358 mg/d)[25].

Phytosterols have a similar molecular structure to dietary and endogenously secreted cholesterol. The most abundant phytosterols (sitosterol, campesterol, and stigmasterol) differ from cholesterol only in the identity of one side chain or the presence of an extra double bond.

Because cholesterol and phytosterol molecules are similar, phytosterols can compete with cholesterol for absorption in the small intestine. Phytosterols block the absorption of dietary cholesterol into the bloodstream and inhibit the re-absorption of cholesterol from bile acids in the digestive process, thus reducing the amount of cholesterol entering the bloodstream[26].

More than 40 years of investigation in animals and humans has shown that plant sterols can reduce total and LDL-cholesterol concentrations. Several investigations of normocholesterolemic to mildly hypercholesterolemic subjects have shown that consumption of margarine enriched with esterified plant sterols effectively lowers plasma total and LDL-cholesterol concentrations by 10% and 15%, respectively [15]. LDL-cholesterol was also lowered with reduced-fat spreads containing plant sterol esters consumed as part of an NCEP Step I diet by men and women with mild-to-moderate primary hypercholesterolemia. Additionally, total cholesterol values were lowered 6 %, the ratio of total to HDL cholesterol decreased 8.1% and triacylglycerol concentrations decreased by 10.4%[27].

In September, 2000, the U.S. FDA issued an interim final rule on plant stanol/sterol esters, allowing labelling on food to state that they have been proven to lower cholesterol and may lower the risk of heart disease when part of a diet low in saturated fat and cholesterol. Scientific studies conducted indicated that 1.3 grams per day of plant sterol esters or 3.4 g per day of plant stanol esters in the diet are needed to show a significant cholesterol lowering effect. In order to qualify for this health claim, a food must contain at least 0.65 g of plant sterol esters per serving or at least 1.7 g of plant stanol esters per serving.

In addition to receiving approval by the FDA, phytosterol products have been endorsed by a coalition of major health organizations in the US. In 2001, the NCEP (National Cholesterol Education Program) published a major report (the Adult Treatment Panel III, ATP III), that recommended the use of phytosterols for the treatment of elevated cholesterol[28]. The guidelines support plant sterols and stanols as “therapeutic dietary options to enhance lowering of LDL (low density lipoprotein) cholesterol”; 2 grams of phytosterols or phytostanols per day, along with 10-25 grams of soluble fibre, was recommended for significant cholesterol reduction.

Plant oils are the most common sources of dietary phytosterols. The levels and types of phytosterols differ among oilseeds.

Table 5: Phytosterols in Various Oilseeds (ppm)84F[29]

Oil	Brassicasterol	Campesterol	Sitosterol	Stigmasterol	Total Sterols
Canola	8.8-13.6	30-37	48-64	<0.2	3300-8000
Hemp		15-17	55-80	13-18	800-1200
Soybean	<0.2	17.6-24.0	50-65	<4	2600-4500
Corn	<0.1	18.0-21.5	64-73	<0.6	7000-11000
Sunflower		8.0-10.0	58-68	10-18	2600-4000
Cotton		7.0-8.0	80-85	0.2-0.3	3800
Peanut	<0.1	12.8-18	80	0.2-0.3	1000-1800
Sesame	<0.1	16-20	68	0.3-0.5	3300-5800
Safflower	<0.1	13.8-16.1	62	4.0-18	3400-4100
Hazelnut	<0.16	3.8-5.8	87-94	0.9-3.7	1000-2000
Almond		2.8-4.0	83	0.1-0.7	1800-2700

The phytosterols found in hempseed oil may contribute to its positive effects on blood lipids.

8.0 Health Benefits of Hempseed and Hemp Oil

Schwab and colleagues conducted a study comparing hempseed oil and flaxseed oil on several biomarkers of lipid function in healthy humans.[30] Fourteen subjects consumed hempseed oil and flaxseed oil (30 ml/day) for 4 weeks each in a randomized, double-blind crossover design. The periods were separated by a 4-week washout period. The total-to-HDL (good) cholesterol ratio, which has been suggested to better predict the risk of coronary heart disease than LDL cholesterol concentration alone decreased significantly during the hempseed oil period compared with the flaxseed oil period.

Recent animal data suggests that hemp oil can impact CVD through reductions in thrombosis (lowering of platelet aggregation or clotting).[31] The supplementation for 12 weeks, of either 5% or 10% (wt/wt) hempseed in the diets of male rats significantly inhibited platelet aggregation in comparison to a control chow or palm oil rich diet. Platelet aggregation was approximately 35% lower following the hempseed diets. Decreasing blood clotting can reduce the risk of a heart attack or a stroke.

In a more recent study by the same researchers, hempseed ingestion over an 8-week period protected rabbits against the negative effects associated with dietary cholesterol supplementation.[32] Rabbits were fed one of six diets: control (RG), the control diet then supplemented with (wt/wt) 5% coconut oil (CO), or 10% hempseed (HP), or 0.5% cholesterol (OL), or with both 10% hempseed and 0.5% cholesterol (OLHP) or with 10% hempseed that was partially de-oiled (SC). Cholesterol supplementation to the diet induced significant aortic plaque development. The addition of hempseed to the diet had a protective effect on the cholesterol-induced defects in relaxation. The data demonstrate that dietary hempseed provides mildly beneficial effects against contractile dysfunction associated with atherosclerotic vessels in the cholesterol-fed rabbit.

Hempseed and hemp oil appear to have beneficial effects against cardiovascular disease markers.

9.0 Dietary Ratio of Omega-6 to Omega-3 Fatty Acids

The dietary n-6/n-3 ratio affects inflammation and gene expression, thus influencing the development of chronic disease. The n-6/n-3 ratio recommended by international agencies and some European countries ranges from 4:1 to 10:1.[33]. The Institute of Medicine supports a ratio of 5:1 for the U.S. and Canadian populations.[34]. However, the ratio may be as high as 17:1 in some Western diets.[35] and is estimated to be 10:1 in the U.S. diet.[36]. In the Women's Health Study, participants had an average dietary ratio of ~8:1, although some women ate diets with a low ratio of about 1:1 while others ate diets with a ratio as high as 33:1.[37].

A dietary imbalance between n-6 and n-3 fats leads to a high ratio of n-6/n-3 fatty acids in cell membranes.[38], which can have adverse effects, including the overproduction of pro-inflammatory eicosanoids, many of which are derived from arachidonic acid, which in turn, stimulate the release of inflammatory cytokines and acute phase proteins. The end result is low-grade chronic inflammation that contributes to health problems such as atherosclerosis, Alzheimer disease, cancer, cardiovascular disease, metabolic syndrome, obesity, osteoporosis, type 2 diabetes and periodontitis.[39,40]

Hemp oil has a ratio of n-6:n-3 of 4:1. Flax seed oil contains more than three times as much omega-3 fatty acids than omega-6 fatty acids, with a ratio of 0.3:1. When comparing other plant based oils, corn oil has an omega-6:omega-3 ratio of 58:1 (data not shown), soybean oil 7:1, and canola oil 2:1.

Table 6: Omega-6 to Omega-3 Ratio in Selected Oil

Fatty acids	Hemp	Flax	Soybean	Canola
C18:2 (LA)	53 - 60	16 - 18	50 - 58	16 - 25
C18:3 alpha (ALA)	15 - 25	53 – 57	6 - 13	9 - 13
Ratio	**4:1**	**0.3:1**	**7:1**	**2:1**

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