



AURUM OIL

4 Pillars - Essential Fatty Acids & Cofactors



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AURUM OIL

4th Pillar

Ingredients:

Cannabis sativa (Hemp) seed oil (Linoleic acid, alpha linolenic acid, oleic acid, Palmitic acid, gamma linoleic acid (GLA), behenic acid, arachidic acid) Carthamus tinctorius (Safflower) oil (high linoleic variety yields conjugated linoleic acid (CLA), oleic acid, palmitic acid, stearic acid) Punica granatum (Pomegranate) seed oil (conjugated linolenic acid (CLnA) punicic acid, linoleic acid, oleic acid, palmitic acid, stearic acid), Turmeric (Curcuma longa) rhizome oil (Turmerone accounts for approx. 50% of the essential oil, Alpha Phellandrene, Delat 3 carene, Eucalyptol, Beta Caryophellene, Beta Farnesene, Curcumene, Beta Bisabolene, Beta sesqui Phellandrene) Moroccan Rosemary (Rosmarinus officinalis) flowering aerial parts oil (1,8-cineol, camphor, α -pinene, α -terpineol, camphene, borneol, limonene and p-cymene).

ESSENTIAL FATTY ACIDS

We must eat food to survive. Food selection and food intake are determined by physiological needs as well as psychological and behavioural (including motivational and emotional) variables.

***“There is no love sincerer than the love of food”* George Bernard Shaw**

However, severe and sometimes critical medical syndromes can result from excessive or deficient nutritional or food intake (overeating, malnutrition, undernutrition). Such conditions may include cardiovascular, endocrine, neurological, and psychiatric symptoms.



THE OMEGA-3 (ALPHA-LINOLENIC ACID) AND OMEGA-6 (LINOLEIC ACID) OILS

Linoleic acid (LA; omega-6; 18:2n6) is the ‘parent’ fatty acid of the omega-6 group. The human body does not synthesize LA, and therefore must be supplied in the diet. All other members of the omega-6 group are derivatives of LA, which is why it is the parent omega-6. Similarly, the parent compound of the omega-3 group is alpha-linolenic acid (ALA, omega-3; 18:3n3). All other members of the omega-3 group are derived from ALA, and together they form the PUFAs. In each group, the derivative fatty acids can convert to longer chain fatty acids through two mechanisms: desaturation and elongation. Because the enzymes that are involved in these mechanisms have the same functions in the two fatty acid groups, the omega-6 and omega-3 fatty acids compete for the same enzymes.

WHAT IS THE DIFFERENCE BETWEEN THE OMEGA-3 AND OMEGA-6 OILS?

While the differences between omega-6 and omega-3 fatty acids are minimal (and may appear insignificant) from a chemical point of view, they exert different and sometimes even opposite biological effects. These opposing effects are not easily explained. It was recently suggested that the distinction between omega-6 and omega-3 PUFAs is based on the differential capacity of proteins, and of large and membrane-bound proteins, in particular, to recognize various PUFAs.

THE IMPORTANCE OF THE RATIO OF OMEGA-6 TO OMEGA-3 FATTY ACIDS

There are several aspects to the issue of the optimal recommended ratio of omega-6 fatty acids to omega-3 fatty acids. One point is the total daily dietary intake recommended in various phases of life (e.g., infancy, pregnancy, adulthood, and old age). Another element is the optimal ratio of PUFAs for dietary supplementation or medical treatment of specific conditions. PUFAs are used in the body in a variety of conditions, including dermatological diseases and cardiovascular disorders. One particular area of interest is the role of PUFAs in the brain and the utility of PUFAs for the protection and stabilization of the neuronal membrane in health and disease.



Delta 6 desaturase	Stimulate	Inhibit
	Low EPA, DHA, GLA High dietary linoleic, linolenic or arachidonic acid Cold exposure Insulin Insulin resistance Obesity Abdominal obesity Elevated BMI Cardiovascular disease CRP Elevated triglycerides Elevated blood sugar Elevated blood cholesterol Adiponectin Cancer (lung and melanoma) Estrogen	High EPA, DHA, GLA Low dietary linoleic, linolenic or arachidonic acid Deficiency zinc, magnesium, selenium, vitamin B3, B6, B12, folate, vitamin C Melatonin deficiency Trans fats Hydrogenated oils Stress Alcohol Radiation Norflurazon Glucagon Adrenaline (epinephrine) Testosterone Trans fats Hydrogenated oils
Elongase	Elevated blood sugar Insulin resistance Cancer (breast) Cold exposure Alcohol	Elevated fasting insulin
Delta 5 desaturase	Stimulate	Inhibit
	Insulin Obesity Abdominal obesity Elevated BMI Cardiovascular disease Inflammation CRP Elevated triglycerides Elevated blood sugar Elevated blood cholesterol Polyphenols turmeric Resveratrol Elevated blood sugar Insulin resistance Cancer (breast) Cold exposure	Stress Abdominal obesity Quinoline Quinazoline Naphthalene Insulin resistance Deficiency of vitamin A, C, E, B3, zinc, melatonin Turmeric (inhibits delta 5 desaturation of omega 6 but not omega 3)
Delta 4 desaturase	Cold exposure	Stress



OMEGA 3 FAMILY



ALPHA-LINOLENIC ACID

- Hemp seed
- Flax (linseed)
- Chia seed
 - Walnut
 - Almonds
 - Pistachio
 - Soybean
 - Canola
- Pumpkin seed
- Black current seed
 - Algae
- Dark green vegetables
 - Pasture grasses
 - Plankton

STEARODONIC ACID

- Black currant seed

EICOSATETRAENOIC ACID EICOSAPENTAENOIC ACID (EPA)

- Fish
- Krill

DOCOSAPENTAENOIC ACID (DPA) DOCOSAHEXAENOIC ACID (DHA)

- Fish
- Krill
- Breast milk

OMEGA 6 FAMILY

LINOLEIC ACID

- Avocado
- Safflower
- Sunflower
- Olive oil
- Sesame
- Canola
- Grape seed
- Corn
- Soybean
- Chia
- Pumpkin seed
- Walnut
- Wheat germ
- Rice bran
- Cotton

Y-LINOLENIC ACID (GLA)

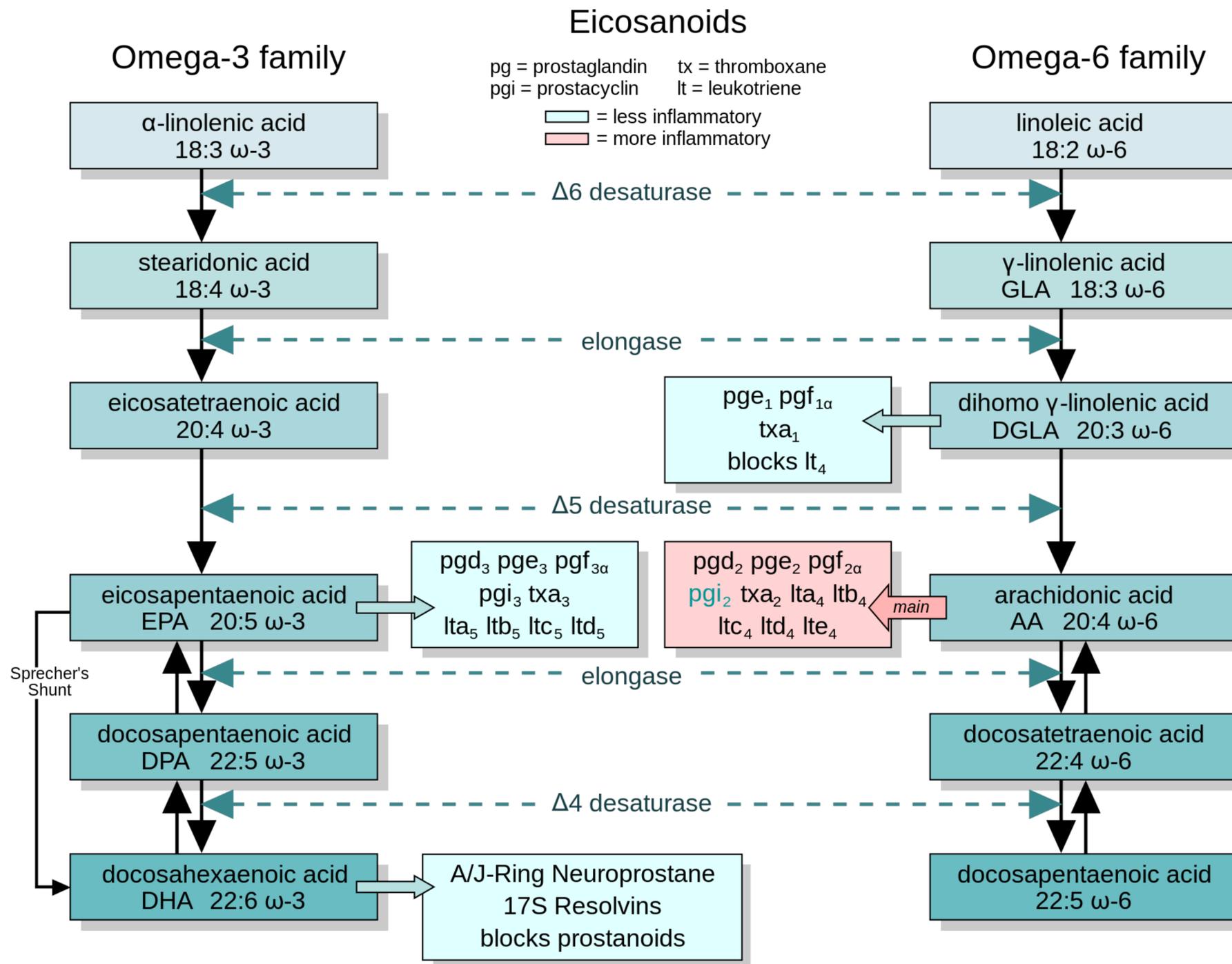
- evening primrose oil
- borage seed
- black currant seed
- hemp seed

DIHOMO Y-LINOLENIC ACID ARACHIDONIC ACID

- egg yolk
- red meat
- poultry
- organ meat

DOCOSATETRAENOIC ACID DOCOSAPENTAENOIC ACID





Created by David R. Throop, vectorized by Fvasconcellos - w:Image:EFA to Eicosanoid.JPG, Public Domain, <https://commons.wikimedia.org/w/index.php?curid=1630962>
 Figure 1 This diagram summarises some of the factors influencing our fatty acid metabolism

	Fatty Acid	Benefits
Omega-3	Alpha-Linolenic Acid	Reduce inflammation, arthritis, heart disease and depression Lowers triglycerides Improves skin disorder (eczema, psoriasis, acne) Reduce macular degeneration Reduces occurrence of asthma Essential for foetal and brain development Hypertension Lowers high cholesterol Diabetes ADHD, bipolar, schizophrenia Osteoporosis Inflammatory Bowel Disease (IBS) Premenstrual Syndrome Breast and Prostate Cancers
Omega-6	Linoleic Acid & Conjugated linoleic Acid	IBS Diabetes Rheumatoid arthritis Allergies ADHA and cognitive functioning Breast cancer Skin disorder Hypertension Menopausal symptoms Premenstrual syndrome (PMS)
Omega-5	Punicic Acid Conjugated Linolenic Acid (CLnA)	Powerful antioxidant anti-inflammatory Repairs damaged cells Regulates glucose transport into cell membrane Treating metabolic diseases such as PCOS, Diabetes type II, atherosclerosis, obesity and serum lipid dysregulation Inhibit fat storage Enhance fatty acid oxidation
Omega-7	Palmitoleic Acid	Improve insulin sensitivity Polycystic Ovarian Syndrome (PCOS) Metabolic Disease Diabetes Weight Loss Anti-inflammatory
Omega-9	Oleic Acid	Reduce risk of cardiovascular disease Boost energy and mood Decrease cognitive decline Anti-inflammatory IBS

THE FULL PICTURE WITH NON-ESSENTIAL FATTY ACIDS AND INTER-CONVERSION IS MUCH MORE COMPLEX

Public health initiatives emphasize low saturated fat, high monounsaturated fat, and high polyunsaturated fat with a lower omega-6 to omega-3 ratio as a general rule for good health. Such universal recommendations on what component of a pathway can be dangerous if we do not acknowledge the need for other essential dietary vitamins and minerals such as B complex vitamins, vitamin C, zinc and magnesium that control the activity of the oils and regulate ratios via interconversion from one form of oil to another to create the delicate balance we need. We can't separate essential nutrients and believe one is more important than the other. That word essential means we must consume it. We can't make it.

As mentioned previously, the essential fatty acid building blocks must come from our diet as we cannot synthesize them ourselves. So, we need to ensure we do consume them in adequate quantity and with sufficient quality.

But that doesn't mean we must avoid and can ignore other fats and oils. As we can undo our good work with omega 3 and 6 ratios by neglecting the big picture. Although we can make our own non-essential fatty acids, they are considered conditionally essential and they influence the metabolism of the essential fatty acids.

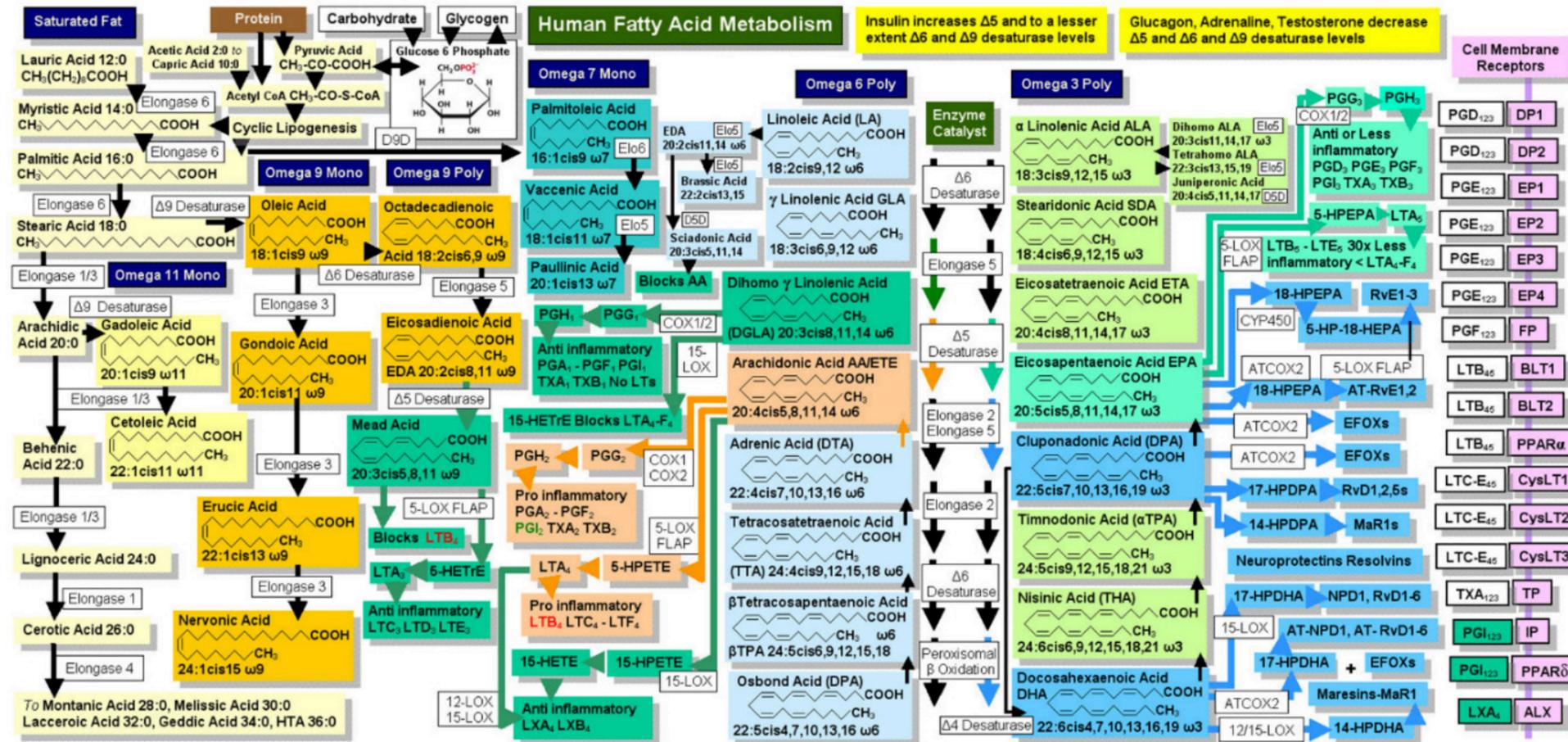
For example

- Oleic acid can alter cellular fatty acid composition in our tissues and organs.
- Saturated and unsaturated fatty acids are both essential components in;
 - cell membranes
 - coordinating lipid synthesis, transport, storage, degradation, and elimination
 - ligands of nuclear and cell-surface receptors and thus maintain cellular homeostasis by sensing cellular lipid levels and regulating gene expression to control lipid overload.
 - regulation of energy and glucose homeostasis through a feedback regulation between the gut and brain
 - Palmitic, lauric, and stearic acids stimulate the expression of mitochondrial uncoupling proteins, UCP2 and UCP3, which enhance fatty acid oxidation and also help to reduce oxidative stress

CONVERSION OF OILS

Imbalanced intake and poor-quality oils or defective enzymes can create imbalances in your oil / fat balance and contribute to chronic disease. ^{IV}

Refer to the following diagram to see all of the different forms of oils and the arrows showing how they can convert between each other.



All credit for this amazing biochemical chart goes to Gordon Ritchie.

These arrows indicate enzyme conversion pathways. They are turned on and off according to other your fatty acid balance, quality and quantity as well as the availability of the essential vitamins and minerals, may be modulated by hormones and immune activity priorities or enhanced or blocked with botanical compounds or drugs.

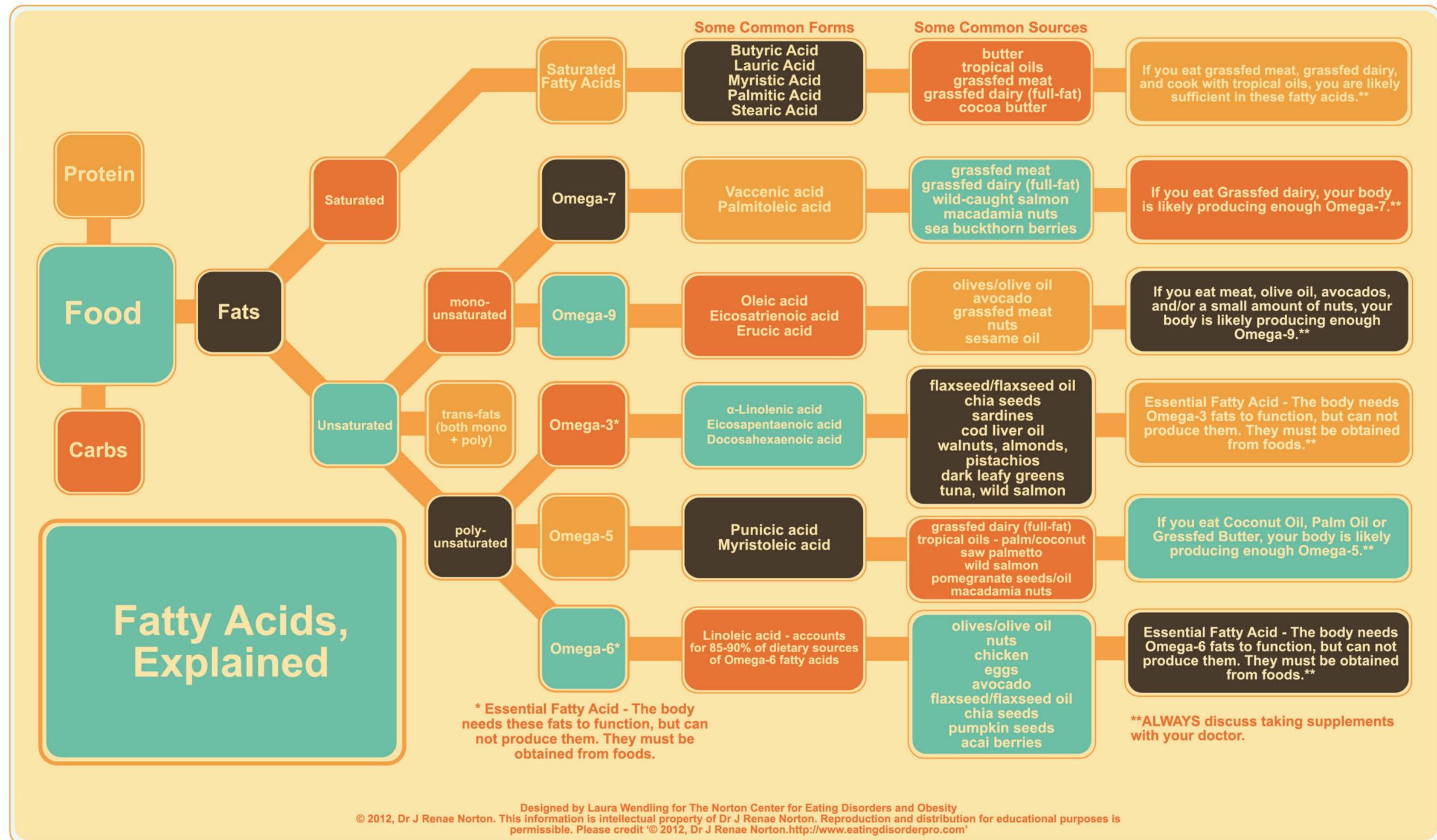
So, having grand statements such as omega 3 and omega 6 ratios are the only thing to consider or worse still when told saturated fat is bad and unsaturated fat is good is lazy.

Quantitative imbalances (ratios and amounts of the different forms from the diet) and / or qualitative imbalances (quality of the oil, is it damaged or converted or is it biologically active) may create imbalances in your body oil ratios even when eating as recommended by government policies.

For example, linoleic acid (LA) is an unsaturated vegetable oil that we may eat an excess of as it is a “good oil” creating a quantitative imbalance by eating too much of that compared to other oils. If the quality is not maintained during processing, cooking, storage or if our diet and inside of our bodies we lack antioxidants than LA is oxidized to form “OXLAMs” (oxidized LA metabolites) that fills up our LDL cholesterol with oxidized oils that in turn oxidise our LDL cholesterol and contribute to hardening of the arteries worse than normal bad cholesterol.

So, how should I balance my oils?

Eat a balanced fatty acid diet. The following diagram provides a nice summary of the food sources of the various fats and oils we need.



The key is to supply a good balance of different forms of oils and ensure other essential nutritional cofactors are available and then work with each individual to balance their physiology to ensure there are no major inflammatory triggers and hormonal imbalances driving our oils down the wrong pathway.

“...a dietary program should also take into consideration the necessity of saturated, monounsaturated, and polyunsaturated fatty acids, phytochemicals, antioxidants, and minerals, such that the body tissues would have access to all the required raw chemicals/ ingredients to form the beneficial bioactive compounds to optimize health.”^v

AURUM OIL

Plant based dietary oils supplying a combination of essential fatty acids and essential oils.

Omega 3, 5, 6, 7, 9, 11, GLA and CLA and CLnA preserved, protected and enhanced with rosemary and turmeric essential oils.

Take 2.5ml daily as part of the 4 pillars stack and a healthy balanced diet.

Works synergistically with;

Multifood - plant-based, natural and standardised essential vitamins for conversion pathways to be able to function.

Resilience – turmeric extract with curcuminoids for conversion pathway support. Helps with systemic inflammation and antioxidant support. Modulates priorities away from inflammation.

Gutright – polyphenols and rosemary extract with carnasoic acid to work synergistically with essential fatty acids. Helps with gut derived inflammation. Modulates gut flora regulation of fatty acid conversion pathways. Modulates priorities away from inflammation.



Ingredients:

Cannabis sativa (Hemp) seed oil (Linoleic acid, alpha linolenic acid, oleic acid, Palmitic acid, gamma linoleic acid (GLA), behenic acid, arachidic acid)

Carthamus tinctorius (Safflower) oil (high linoleic variety yields conjugated linoleic acid (CLA), oleic acid, palmitic acid, stearic acid)

Punica granatum (Pomegranate) seed oil (conjugated linolenic acid (CLnA) punicic acid, linoleic acid, oleic acid, palmitic acid, stearic acid)

Turmeric (Curcuma longa) rhizome oil (Turmerone accounts for approx. 50% of the essential oil, Alpha Phellandrene, Delta 3 carene, Eucalyptol, Beta Caryophellene, Beta Farnesene, Curcumene, Beta Bisabolene, Beta sesqui Phellandrene)

Moroccan Rosemary (Rosmarinus officinalis) flowering aerial parts oil (1,8-cineol, camphor, α -pinene, α -terpineol, camphene, borneol, limonene and p-cymene)

Why Hemp seed oil?

Hemp seed oil is a rich source of polyunsaturated fatty acids (PUFAs), which are present mainly as linoleic acid and alpha-linolenic acid. These two fatty acids are known as essential fatty acids (EFAs) because they are necessary for humans and other animals for good health, but they cannot synthesize them.

As well as these beneficial oils, hempseed oil contains tocopherols, which may reduce the risk of cardiovascular diseases, cancer, and age-related macular degeneration. Tocopherols act as antioxidants and prevent the oxidation of unsaturated fatty acids.

The composition of oil in Hemp seed oil?

For hemp seed oil, the most abundant fatty acids were linoleic (omega-6), α -linolenic (omega-3), and oleic acids (omega-9), which together ranged between 84.93 and 88.61% of the total fatty acid composition. The linoleic acid content in different samples of hemp oil ranged between 56 and 59%. Researchers found the same material of linoleic acid as found in walnut oils. All oils presented a high amount of α -linolenic acid which makes them very interesting for nutritional implications and beneficial physiological effects in the prevention of coronary heart disease and cancer.

The ideal omega-6 to 3 ratio and saturated fat levels is found in hemp oil

All oils presented the ideal omega-6/omega-3 ratio 3:1 which is much higher than most vegetable oils, and the recommended high ratio of polyunsaturated (P) to saturated (S) fatty acids (>10), considered to be very important to reduce the risk of arteriosclerosis and coronary heart disease. Hemp seed oil also contains Gamma-linolenic (GLA) acid is used for medical treatment of PMS, and psoriasis.



What is so good about having a variety of oils?

In nature, we eat millions of 'chemicals' daily. Before you freak out, 'chemicals' can be both beneficial and harmful and makeup everything is known to humanity. We need a wide variety of chemicals, and this includes the entire spectrum of essential fatty acids. It is imperative that we present the body with these oils so as the body is capable of making the many essential fatty acids from the fats found in hemp oil.

Other health benefits of hemp seed oil

Apart from the obvious cardiovascular listed earlier, hemp seed oil also is an excellent supplement for the health of your skin. A study completed back in 2005 compared hemp seed oil with olive oil and despite olive oil also is good for the surface, the study found that hemp seed oil was excellent for the cover. On top of that, when they measured the blood lipid methods, there were significant increases in omega-3 and beneficial omega-6 levels.^{vii}

Hemp seed oil for the Brain

It is no shock that healthy oils are good for the brain. It may come as a surprise that the right fats can even benefit one of the most horrible brain diseases, multiple sclerosis (MS). A few years ago, a study was compiled to see whether the combination of a special diet, evening primrose oil, and hemp oil could benefit MS. The great news is that the study has found that MS sufferers could benefit from a combination of things, including hemp seed oil.^{viii}



SAFFLOWER OIL

Cold pressed safflower seed oil contains 70-75% linoleic acid as conjugated linoleic acid and about 15% oleic acid along with palmitoleic acid, palmitic acid, and less than 1% of lauric acid, myristic acid, arachidic acid, eicosenoic acid, behenic acid and stearic acid.



CLA

- AMPK activator
- PPAR modulator
- Stimulates lipolysis
- Enhances fatty acid oxidation
- Inhibits fat storage
- Anti-inflammatory
- Adipocyte apoptosis (self-destruct gene in fat cells)

Conjugated linoleic acid (CLA) reduces adiposity in adipocytes (flash way of saying fatness in fat cells) by increasing energy expenditure and fatty acid oxidation (fat burning), AMP-K activation, increase the carnitine-palmitoil-transferase-1 (CAT-1) activity, its interaction with PPAR γ , and to raise the expression of UCP-1, reduced inflammation, and can help to induce apoptosis in defective cells (selfdestruct).

Apoptosis is a process researched heavily in regards to cancer treatment and prevention. But other cells that are abnormal, defective or obsolete also undergo apoptosis, including adipocytes (fat cells). CLA has been studied and shown to induce apoptosis in the fat cells of pigs.

CLA interacts with the Co-activator complex PPAR increasing lipolysis (β -oxidation), mitochondrial biogenesis (cellular energy warehouse production) and insulin sensitivity, and these actions can help to explain the fat loss reputation for CLA.

Oleic acid – The one that made olive oil famous is found in Hemp and Safflower

Since ancient times, physicians such as Galen, Hippocrates, and Dioscorides have acknowledged the therapeutic effects of olive oil/oleic acid. One of the beneficial properties of the oleic acid in olive oil is a reduced risk of coronary artery disease (CAD) through many different mechanisms, including decreases plasma cholesterol, a possible increase of HDL, and decreased LDL susceptibility to oxidation during fasting. The Seven Countries Study showed for the first time that the Greek population from Crete had very low morbidity and mortality rates from CAD when compared to people from other countries.

Additionally, Cretans were found to have lower plasma cholesterol levels than Americans, and this difference was speculated to contribute to the observed differences in the prevalence of CAD in these two countries. Differences were also observed between the American and Cretan cohorts in adipose tissue fatty acid composition. In particular, the adipose tissue of the Cretans had a higher concentration of oleic acid than the adipose tissue of the American cohorts, indicating that the traditional Cretan diet was high in oleic acid, which comes primarily from olive oil consumption.

Influence of oleic acid on insulin release and action

The loss of beta-cell function and insulin sensitivity is known to contribute to the development of diabetes, a metabolic disorder that develops throughout months to years. It has been hypothesized that insulin resistance syndromes might be a postprandial phenomenon linked to acute dietary fat metabolism. Exaggerated postprandial hypertriglyceridemia is indeed an inherent feature of diabetic dyslipidaemia and is frequently found even in diabetic patients with normal fasting triglycerides.

Dietary fats, particularly those containing SFA, are known to promote insulin secretion and resistance. Indeed, beta-cells are particularly sensitive to the degree of unsaturation of fatty acids. It is likely that oleic acid and palmitic acid could compete at the level of the beta-cell, which is in line with a previous model that explains the ability of fatty acids to trigger insulin secretion by glucoseresponsive triglyceride/free fatty acid cycling.

The islet tissue, which expresses LPL, could access postprandial triglycerides as a source of free fatty acids, in which case the type and concentration of the free fatty acid near the beta-cells are likely to be dependent on the nature of the dietary fats. This system could be linked to the local promotion of both intracellular triglyceride lipolysis and fatty acid esterification. Free fatty acid deprivation in islet tissue has indeed been reported to impede glucose-stimulated insulin secretion, a process rapidly reversed by replacement with exogenous free fatty acids. Oleic acid was found to elicit half the insulinotropic potency of palmitic or stearic acids. Thus, it has been hypothesized that, in comparison with saturated fats, the oleic acid in olive oil might moderate the postprandial hyperactivity of beta-cells.^{ix}

Influence of oleic acid on postprandial inflammatory processes

Inflammation plays an instrumental role in all stages of atherosclerosis, with several inflammatory mediators, including adhesive and signaling mechanisms, being involved in early atherosclerotic lesion formation. These mediators participate in the early stages of the atherothrombotic plaque, including recruitment of monocyte-leukocytes from circulating blood by vascular endothelial cells, migration of leukocytes into the intima, and transition of monocytes into macrophages and eventually into lipid-laden foam cells. Inflammatory processes involving activation of cytokines, proteolytic enzymes, tissue factor, and growth factors continue to play a role in the subsequent development of atherosclerotic lesions into complicated plaques.^{xxi}

POMEGRANATE SEED OIL

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CLnA

- Modulates PPAR
- Enhances fatty acid oxidation
- Converts to CLA in the body
- Anti-inflammatory



WHAT IS PPAR?

This can get really complicated and long so I will attempt to summarise their functions and give a basic overview.

PPARs are receptors involved in the regulation of lipid and energy balance. PPARs function as sensors for fatty acids and their derivatives, and control metabolic pathways involved in energy homeostasis. The three PPAR isoforms exhibit distinct and non-interchangeable functional roles in energy metabolism.

The main role of PPARs is to regulate the expression of genes involved in lipid and glucose metabolism. Disruption of this pathway contributes to disease progression in obesity, diabetes, and cancers. This occurs through regulation of growth and migration, apoptosis, fatty acid (FA) metabolism pathways, and oxidative stress responses. PPARs are also known to regulate inflammatory processes that are linked to metabolic homeostasis in tissues, such as liver, adipose tissue, intestine, skeletal muscle, and cardiovascular system.

Peroxisome proliferator-activated receptors (PPARs) comprise three different isoforms, PPAR- α , PPAR- β/δ , and PPAR- γ .

PPAR - ALPHA

- PPAR- α predominantly regulates lipid catabolism
- Activated to stimulate fat burning during fasting and exercise
- Breaks down triglycerides to release free fatty acids
- Increases fatty acid transport into mitochondria
- Increases beta fatty acid oxidation
- Increases ketogenesis
- Anti-inflammatory
- Browning of white fat

PPAR- α predominantly regulates the breakdown, the release and the burning of fat. PPAR- α regulates mitochondrial, peroxisomal, and microsomal fatty acid oxidation, as well as senses the influx of fatty acids during fasting to enhance the fatty acid burning capacity. Many studies have shown that PPAR α is activated by omega 3 linolenic acids, CLnA, and CLA.

The PPAR- α gene functions as a major regulator of fatty acid homeostasis. PPAR- α is a major regulator of fatty acid oxidation pathways and delivery of fats into the mitochondria. PPAR- α activation inhibits pro-inflammatory gene expression in vascular smooth muscle cells (VSMCs) and attenuates development of atherosclerosis. PPAR- α agonists decrease triglyceride-rich lipoproteins through an increase in the gene expression of fatty acid oxidation (increased fat burning).

PPAR - BETA/DELTA

- PPAR β / δ insulin sensitizer
- Increases fatty acid oxidation in skeletal muscle
- Enhances insulin sensitivity and glucose uptake by skeletal muscle
- Increases glycogen synthesis
- Anti-inflammatory
- Wound healing

Activation of PPAR- β/δ also induces expression of genes required for fatty acid oxidation and energy dissipation in skeletal muscle and adipose tissue, which in turn lead to improved lipid profiles and reduced adiposity. In the liver, PPAR- β/δ can be activated by plasma free fatty acids influxed during fasting, during exercise and with strategic fatty acid supplementation. The PPAR- β/δ gene is known to increase lipid catabolism in adipose tissue, skeletal muscle, and the heart and has been shown to improve the plasma high density lipoprotein- (HDL-) cholesterol levels and insulin resistance. Additionally, activation has been shown to induce cell proliferation and differentiation and to limit weight gain with anti-inflammatory effects in the vessel wall.

PPAR - GAMMA

- Regulates glucose balance and lipid storage
- Insulin sensitivity
- Browning of white fat
- Ant-inflammatory
- Reduced gluconeogenesis
- Fatty acid oxidation
- Inhibit lipogenesis

PPAR- γ is an essential regulator of adipogenesis and fat storage in adipocytes. The PPAR- γ gene plays an essential regulatory role in glucose metabolism, adipocyte differentiation, and lipid storage by controlling the transcription of a number of genes involved in these metabolic processes. PPAR- γ also regulates genes involved in insulin signalling and the expression of proinflammatory cytokines.

PPAR- γ functions as a master switch in controlling adipocyte differentiation and development, and its activation plays an important role in glucose metabolism by enhancing insulin sensitivity. PPAR- γ agonists improve glucose and insulin parameters and increase whole body insulin sensitivity.

Therefore, they are called insulin-sensitizers and are used in the treatment of diabetes.

THE ATP PROJECT PODCAST

POMEGRANATE PULLED APART



Welcome to the ATP Project, Episode 159 – Pomegranate Pulled Apart.

In today's podcast Jeff and Matt discuss the benefits of consuming pomegranate. From the fruit, peel, flowers and leaves, the pomegranate has amazing health benefits that can help with everything from gut health to hardened arteries.

RELATED PODCASTS



▶ THE BRAIN



▶ THE CRITICAL IMPORTANCE OF FATS AND OILS



▶ PPAR MODULATORS

References

- ⁱ Arch Med Sci. 2015 Aug 12; 11(4): 807–818.
Published online 2015 Aug 11. doi: 10.5114/aoms.2015.53302
PMCID: PMC4548034
PMID: 26322094
Potential role of dietary lipids in the prophylaxis of some clinical conditions
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- ⁱⁱ Arch Med Sci. 2015 Aug 12; 11(4): 807–818.
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- ⁱⁱⁱ J Nutr Sci Vitaminol (Tokyo). 1992 Aug;38(4):353-63.
Effects of sesamin and curcumin on delta 5-desaturation and chain elongation of polyunsaturated fatty acid metabolism in primary cultured rat hepatocytes.
Fujiyama-Fujiwara Y1, Umeda R, Igarashi O.
- ^{iv} Potential role of dietary lipids in the prophylaxis of some clinical conditions
Urvashi Bhagat corresponding author and Undurti N. Das corresponding author Arch Med Sci. 2015 Aug 12;11(4): 807–818.
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PMID: 26322094
- ^v PMCID: PMC4548034
PMID: 26322094
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Urvashi Bhagat corresponding author and Undurti N. Das corresponding author
- ^{vi} Carla Da Porto, Deborah Decorti, & Andrea Natolino. Journal of Dietary Supplements, 12(1):1–10, 2015.
Potential Oil Yield, Fatty Acid Composition, and Oxidation Stability of the Hempseed Oil from Four Cannabis sativa L. Cultivars.
- ^{vii} Callaway J1, Schwab U, Harvima I, Halonen P, Mykkänen O, Hyvönen P, Järvinen T. J Dermatolog Treat. 2005 Apr;16(2):87-94. Efficacy of dietary hempseed oil in patients with atopic dermatitis.
- ^{viii} Rezapour-Firouzi S1, Arefhosseini SR2, Ebrahimi-Mamaghani M3, Baradaran B4, Sadeghihokmabad E5, Mostafaei S6, Torbati M7, Chehreh M8. Alteration of delta-6-desaturase (FADS2), secretory phospholipase-A2 (sPLA2) enzymes by Hot-nature diet with co-supplemented hemp seed, evening primrose oils intervention in multiple sclerosis patients. Complement Ther Med. 2015 Oct;23(5):652-7.
- ^{ix} Curr Pharm Des. 2011;17(8):831-43. Oleic acid in olive oil: from a metabolic framework toward a clinical perspective. Bermudez B1, Lopez S, Ortega A, Varela LM, Pacheco YM, Abia R, Muriana FJ.
- ^x Curr Pharm Des. 2011;17(8):831-43. Oleic acid in olive oil: from a metabolic framework toward a clinical perspective. Bermudez B1, Lopez S, Ortega A, Varela LM, Pacheco YM, Abia R, Muriana FJ.
- ^{xi} Carla Da Porto, Deborah Decorti, & Andrea Natolino. Journal of Dietary Supplements, 12(1):1–10, 2015.
Potential Oil Yield, Fatty Acid Composition, and Oxidation Stability of the Hempseed Oil from Four Cannabis sativa L. Cultivars.
- ^{xii} Food Chem. 2018 Mar 1;242:9-15. doi: 10.1016/j.foodchem.2017.09.042. Epub 2017 Sep 9.
Antioxidant activity of rosemary essential oil fractions obtained by molecular distillation and their effect on oxidative stability of sunflower oil.
Mezza GN1, Borgarello AV1, Grosso NR2, Fernandez H3, Pramparo MC1, Gayol MF4.
- ^{xiii} Meat Sci. 2018 Sep;143:153-158. doi: 10.1016/j.meatsci.2018.04.035. Epub 2018 May 4.
Consumer profile and acceptability of cooked beef steaks with edible and active coating containing oregano and rosemary essential oils.
Vital ACP1, Guerrero A2, Kempinski EMBC3, Monteschio JO4, Sary C4, Ramos TR4, Campo MDM5, Prado IND6.
- ^{xiv} BMC Complement Altern Med. 2014 Jul 7;14:225. doi: 10.1186/1472-6882-14-225.
Antioxidant activity of rosemary (Rosmarinus officinalis L.) essential oil and its hepatoprotective potential.
Rašković A, Milanović I, Pavlović N1, Čebović T, Vukmirović S, Mikov M.
- ^{xv} J Food Sci. 2007 Nov;72(9):C504-8.

Oregano and rosemary extracts inhibit oxidation of long-chain n-3 fatty acids in menhaden oil.

Bhale SD1, Xu Z, Prinyawiwatkul W, King JM, Godber JS.

^{xvi} Turk J Med Sci. 2018 Jun 14;48(3):644-652. doi: 10.3906/sag-1704-166.

Effect of cineole, alpha-pinene, and camphor on survivability of skin flaps

İnce B, Dadacı M, Kılınç İ, Oltulu P, Yazar S, Uyar M.

^{xvii} Anti-inflammatory and antinociceptive effects of *Rosmarinus officinalis* L. essential oil in experimental animal models.

Takaki I, Bersani-Amado LE, Vendruscolo A, Sartoretto SM, Diniz SP, Bersani-Amado CA, Cuman RK.

J Med Food. 2008 Dec;11(4):741-6. doi: 10.1089/jmf.2007.0524.

^{xviii} Eur J Nutr. 2012 Feb;51(1):57-68. doi: 10.1007/s00394-011-0182-7. Epub 2011 Mar 29.

Dietary d-limonene alleviates insulin resistance and oxidative stress-induced liver injury in high-fat diet and LNAME-treated rats.

Victor Antony Santiago J1, Jayachitra J, Shenbagam M, Nalini N.

^{xix} J Basic Clin Physiol Pharmacol. 2015 Jan;26(1):95-103. doi: 10.1515/jbcpp-2013-0165.

Gastroprotective activity of essential oils from turmeric and ginger.

Liju VB, Jeena K, Kuttan R.

^{xx} An evaluation of antioxidant, anti-inflammatory, and antinociceptive activities of essential oil from *Curcuma longa*. L.

Liju VB, Jeena K, Kuttan R.

Indian J Pharmacol. 2011 Sep;43(5):526-31. doi: 10.4103/0253-7613.84961.

PMID: 22021994

^{xxi} Aromatic-Turmerone Attenuates LPS-Induced Neuroinflammation and Consequent Memory Impairment by Targeting TLR4-Dependent Signaling Pathway.

Chen M, Chang YY, Huang S, Xiao LH, Zhou W, Zhang LY, Li C, Zhou RP, Tang J, Lin L, Du ZY, Zhang K.

Mol Nutr Food Res. 2018 Jan;62(2). doi: 10.1002/mnfr.201700281. Epub 2018 Jan 8.

PMID: 28849618

^{xxii} Asian Pac J Cancer Prev. 2014;15(16):6575-80.

Chemopreventive activity of turmeric essential oil and possible mechanisms of action.

Liju VB1, Jeena K, Kuttan R.