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To: Committee, Health (REPS)
Cc: Freelander, Mike (MP); James Muecke
Subject: ADG evidence submission: seed oils
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Attachments: ADG evidence submission on seed oils .docx
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Dear Committee,

Further to my comments on Friday regarding seed oils, and the request for evidence submitted recently by our team for the public consultation step of the revision of *ADG 2013*, I have attached a document outlining concerns regarding seed oils and margarines.

I would also ask you to please consider Submission 381 by Dr Chris Knobbe, a world leading expert on the dangers of seed oils and author of the recently published and well-researched book *The Ancestral Diet Revolution*.

Best wishes

James

ADG evidence submission: seed oils

"…demonising one major food group (or type of dietary fat) is a mistake. Foods contain a wide range of saturated, mono- and poly- unsaturated fatty acids in varying proportions, and the different fatty acids never exist in isolation, meaning fats in food can have contrasting good and bad effects on the different functionality of lipoprotein particles… We need critics and debate more than we need outdated inflexible guidelines or eatwell plates." Professor Tim Spector, 2018.[i]

Introduction

A consequence of the diet-heart hypothesis – the concept that dietary saturated fats and cholesterol increase blood cholesterol leading to increased risk of coronary heart disease – is the endorsement by dietary guidelines for replacement of dietary saturated fats with carbohydrates and/or seed and bean oils as a means of reducing risk of cardiovascular disease (Hamilton et al).[1] Thus a cornerstone of the *ADG 2013* advice to reduce saturated fats is increased consumption of carbohydrates, and/or and plant-based oils. While the latter are commonly called vegetable oils, they are more accurately described as seed oils and bean oils. They include corn, rapeseed ("canola"), cottonseed, soybean, sunflower, safflower, grapeseed, and rice brain oils. Their entry into the human food supply is very recent, and correlates closely with the rise of non-communicable and chronic diseases in the last 100 years.

Seed oils have been promoted as 'heart healthy' while saturated fats continue to be vilified. However, there is substantial evidence that excessive consumption of seed oils high in linoleic acid (LA), the most common omega-6 polyunsaturated fatty acid (PUFA) in the Modern Western diet (MWD) is likely to contribute to many modern chronic diseases. A previous section has examined the evidence that exonerates saturated fats.

This section of the submission will examine lines of evidence that challenge the widespread belief that seed oils are healthy and preferable to dietary saturated fats:

- Historical, including evolutionary perspectives
- Studies of the physiology of PUFAs and LA in humans and other animals, and randomised trials and meta-analyses.

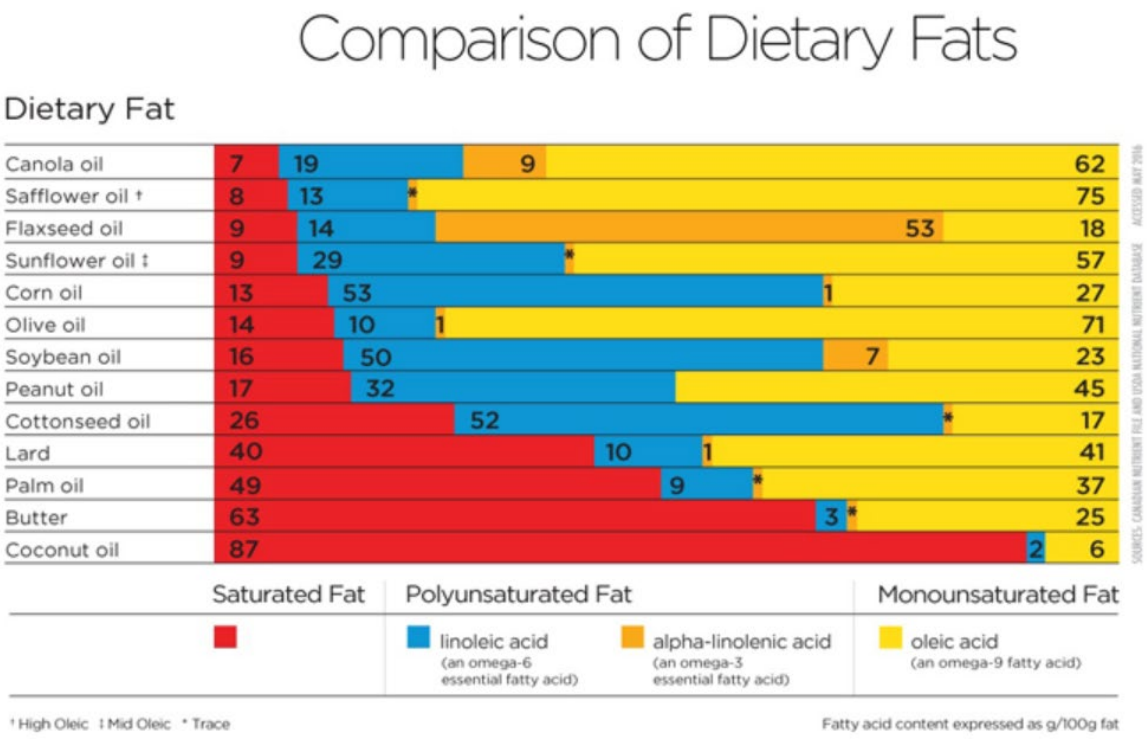
The history

Human intake of Linoleic Acid (LA) has risen dramatically over the past century, due to a substantial increase in consumption of seed and bean oils (Blasbalg et al, Guyenet et al , Burns, Knobbe and Alexander).[2,3,4,5] While LA is essential for humans, having roles in normal growth and development, cellular function and signaling, and immune response (Burns 2018),[4] the amounts of LA in Modern Western Diets (MWD) is far in excess of what is required, raising the possibility of LA toxicity (Knobbe and Alexander, Choque et al 2015, Taha).[5, 6,7]

Both human history and evolution indicate that humans are biologically adapted to a diet that includes meat and the associated animal fats (Leroy and Cofnas).[8] Animal fats contain a

range of fatty acids but are generally not high in PUFAs such as LA. (see Fig 1 Comparison of dietary fats). Similarly, evolutionary data indicates that hunter-gather diets are low in LA (Knobbe and Alexander, Speth and Spielman.[5,9] For the duration of human history, low levels of dietary LA were clearly enough for essential physiological functions. The high LA-content seed oils were not available in large quantities until the latter half of the 19th century. (Knobbe and Alexander).[5] However, seed oils have gradually displaced the natural animal fats such as lard, tallow and dairy fat, and LA now constitutes between 8 and 12% of daily energy from dietary fats in the US. (Knobbe and Alexander, Taha, DiNicolantio and O'Keefe).[5,7,10]

Hydrogenated seeds oils such as *Crisco*, originally manufactured from cottonseed oil, were first marketed in the US in 1911 (*Crisco* website).[11] By the middle of the 20th century, the diet-heart hypothesis had taken hold and the *American Heart Association* (AHA) received significant funding from *Procter & Gamble*, the manufacturer of *Crisco* (Hamilton, Teicholz).[1,8] From 1961, the AHA recommended that all people reduce their consumption of saturated fats, replacing animal fats with seed- and bean-derived PUFA oils as a means of preventing atherosclerosis. Despite the obvious conflict of interest and lack of evidence for the diet-heart hypothesis (Harcombe 2017, Dinicolantonio),[13,14] this advice was enshrined in the *US Dietary Guidelines* in 1980. These guidelines have had considerable influence upon dietary guidelines around the globe, including Australia, where we continue to have dietary guidelines and peak bodies that recommend reductions in dietary saturated fat and replacement of saturated fats with PUFA, despite the lack of evidence for these recommendations, as noted in previous sections of this evidence submission.



The studies

As noted above, the dramatic rise in PUFA and LA consumption has coincided with the rapid rise in chronic metabolic disease including obesity and type 2 diabetes. This section will outline evidence from animal and human studies that suggest that PUFA and LA have deleterious effects on animal and human physiology.

The primary concerns about omega-6 PUFAs such as LA are that they are present in historically unprecedented levels in Western diets, and they oxidise readily into toxic metabolites known as oxidized linoleic acid metabolites (OXLAMs) (Taha).[7] Humans evolved consuming low levels of omega-6 fatty acids, in an approximate ratio of 1:1 with omega-3 fatty acids (Simopoulos).[15] The ratio is now estimated at 20:1 or greater. Fatty acids such as LA consumed in the habitual diet accumulate in and correlate with human adipose tissue composition (Garaulet et al).[16] Historically high levels of omega-6 fatty acids have been found human red cell membrane phospholipids and adipose tissues (Simopoulos, Dinicol/o'K 2018).[15,10] Central obesity is positively associated with omega-6 PUFAs in adipose tissue (Garaulet).[16]

Lipids and their metabolites are not simply molecules that supply energy; they are also needed for cell membrane integrity, signal transduction pathways, immune functions, and gene expression regulation (Rochling).[17] Lipid metabolites from omega-6 fatty acids are distinct from the omega-3 metabolites, with different biological functions (Choque 2015).[6] Omega-6 fatty acid metabolites are well known as precursors to potent mediators of inflammation, thrombosis and vasoconstriction (Simopoulos).[15] Thus the accumulation of LA and its metabolites has significant consequences for human physiology, and excessive consumption of LA in MWD raises many questions about their role in human and disease (Choque et al).[6]

For example, high LA intake interferes with desaturation and elongation of the omega 3 fatty acid, alpha linoleic acid (ALA), thus inhibiting ALA metabolism to long chain eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), omega 3 fatty acids with anti-thrombotic and anti-inflammatory metabolites. Western diets high in LA shift the physiological state to one that is proinflammatory, prothrombotic and pro-aggregatory, with increase in blood viscosity, vasospasm, vasoconstriction and cell proliferation (Simopoulos).[15] The latter are factors associated with modern diseases including cardiovascular disease.

In contrast to the diet-heart hypothesis, the oxidised linoleic acid hypothesis proposes that numerous lines of evidence show that high levels of dietary LA promote oxidative stress, oxidised LDL, chronic low grade inflammation and atherosclerosis, and is likely a causative factor for cardiovascular disease (Dinicol/O'K 2018).[10] The multiple lines of evidence are summarised in Box 1 of Dinicolantonio and O'Keefe.[10] Of note, OXLAMs including hydroxyoctadecadienoic acids (HODEs) are in higher concentrations in the LDL of humans with diseases characterised by inflammation, compared with LDL from healthy people (Spiteller et al).[18] HODEs are found in LDL from patients with atherosclerosis (Jira et al),[19] and are implicated in the conversion of macrophages into foam cells characteristic of atherosclerotic plaque (Nagy).[20]

Additionally, randomised controlled trials substituting seed oils for saturated fats failed to demonstrate mortality benefits. Re-analyses of the *Sydney Diet Heart Study* (SDHS) and *Minnesota Coronary Survey* (MCS) challenge the diet-heart hypothesis (Ramsden et al 2013, Ramsden et al 2016).[21,22] In the SDHS, substitution of saturated fats with safflower oil rich in LA significantly reduced total cholesterol but was associated with significant increases in all-cause mortality, cardiovascular disease and coronary heart disease deaths. The authors note the mechanistic links between OXLAMs and atherosclerosis and suggest that diets high in omega-6 LA may be particularly detrimental in the context of oxidative stress induced by smoking or alcohol (Ramsden et al 2013).[21]

Likewise, the re-analysis of the MCS showed that while substitution of dietary saturated fat with LA-rich corn oil lowered cholesterol, this did not translate into improved survival in the intervention group. Participants who had the greater reductions in serum cholesterol also had higher risk of death. (Ramsden et al 2016).[22] The authors speculate that complete publication of data from RCTs such as the SDHS and MCS could have altered key public policy recommendations, such as those to replace saturated fats with omega 6 seed and bean oils, recommendations that continue to be enshrined in dietary guidelines around the world.

A range of research since these crucial trials has confirmed that LA-derived HODEs are abundant in oxidised LDL, a precondition for atherosclerosis. For example, 9-HODE is much more abundant in oxidised LDL than other lipid peroxidation products, and 9-HODE increases as people age and develop more atherosclerosis (Jira).[19] A 2017 study showed that high LA from soy oil in the diet was demonstrated to increase apolipoprotein B (apo B) and oxidised LDL in healthy adults in only 8 weeks. (Kim et al).[23] While increasing dietary LA may decrease serum Total Cholesterol and LDL, the other effects, such as the increased susceptibility of LDL and other lipoproteins to oxidation, and increase in small dense LDL, reduction in HDL and increases in triglycerides from consumption of excess LA suggest that LA is likely to increase rather than reduce cardiovascular risk (Dinic/O'K 2015).[10]

LA is also implicated in the global epidemic of obesity, with studies showing that nutrition transitions are typified by increasing use of cheap and abundant seed oils high in LA (Naughton et al).[24] Mechanisms include the inflammatory OXLAMs derived from LA, and the endocannabinoid system. Endocannabinoid mediators include anandamide and 2-arachidonyl glycerol (2-AG), molecules which are derived from dietary LA, and which have a role in stimulating appetite. (Naughton, Alvheim 2014).[24,25] Animal studies confirm that increasing the percentage of LA in the diet from 1% (ie similar to historical levels of LA in human diets) to 8% (ie equivalent to LA content of MWDs) increases endocannabinoid LA derivatives, increasing the risk of obesity for animals even when consuming a low fat diet (Alvheim 2014).[25] Similarly, despite isocaloric feeding, weight gain in rats was less in animals given a saturated fat based diet, in comparison with animals fed an LA-rich safflower oil based diet (Pan and Storlien).[26]

Various studies have confirmed correlations between LA consumption and obesity. For example, in obese subjects, habitual diet correlated with adipose tissue composition, and central obesity was associated with omega-6 PUFAs tissue concentration (Garaulet).[16] LA accumulates in breast milk, which is the primary source of nutrition for infants (Taha).[7]

Maternal plasma omega-6 PUFA concentration in late pregnancy was shown to predict children's fat mass at 4 and 6 years (Moon et al).[27]

A 2014 intervention study comparing control oils (soybean and safflower ie high LA) with canola oil and olive oil in Asian men with fatty liver found that lower LA consumption was associated with greater weight loss and improvements in fatty liver (Nigam et al).[28] Leptin receptor polymorphisms have been shown to interact with diets high in omega 6 and low in omega 3 PUFA to increase the risk of leptin resistance and metabolic syndrome in humans (Phillips 2010).[29]

There are multiple other concerns about the role of LA and other omega-6 rich oils in Modern Western Diets. Concern has been raised that high LA intake may compromise critical regulatory mechanisms in glucose control, promoting insulin resistance and reducing pancreatic beta cell insulin secretion (Hamilton).[1] Excess dietary LA increases the brain's vulnerability to inflammation, while reducing dietary LA maybe neuroprotective and merits further study (Taha).[7]

Another potentially pathological mechanism of LA toxicity is the production of aldehyde metabolites both by heating PUFA oils (Han),[30] and by enzymic lipid peroxidation reactions. These aldehydes, which include HNE, have been extensively studied and implicated in tissue damage, potentially mediating a range of human disease pathological states such as Alzheimer's disease, diabetes, cardiovascular and inflammatory diseases (Shoeb.)[31] HNE is both a marker of oxidative stress and acts as a secondary messenger on a number of cell signalling pathways. HNE is implicated in both cancer and type 2 diabetes, and may be a key molecule linking these conditions (Jaganjac).[32]

Many researchers agree that the imbalance in the ratio of omega-6 to omega-3 fatty acids is due to the dramatic increase in dietary LA in western diets during the last century. Some argue that the most practical remedy is to increase the intake of long chain marine omega-3 oils (Fabian et al),[33] with omega 3 fatty acids apparently offering some protective against some adverse effects of high LA consumption (Sanders et al 1997).[34] However, given that lowering dietary LA reduces the production and accumulation of OXLAMs such as HODEs and HNE, it makes sense to lower LA while also increasing omega-3 fatty acids (Taha, Ramsden et al 2015, Dinicolantonio/O'K 2018).[7,35,36] Avoiding dietary seed and bean oils by substituting good quality appropriately raised and produced fats such as butter, lard and tallow, or coconut oil is ancestrally appropriate (Knobbe and Alexander).[4] To this end, the next iteration of the Australian Dietary Guidelines should recommend against the consumption of seed oils, margarines and ultra-processed foods, and promote or at least permit the use of stable natural fats with low potential for oxidative damage.

Studies of reducing LA consumption provide powerful counterexamples to the studies that link dietary LA with pathological conditions. Lowering LA in human diets results in reduction of plasma LA, and decreased oxidised LDL and apolipoprotein B compared to a high LA diet (Kim).[23] A low LA diet intervention also reduced both LA and bioactive oxidised LA metabolites in humans, with broad implications for human chronic disease (Ramsden et al 2012).[37] For example, omega 6 PUFA and oxidised LA metabolites promote upregulation of nociceptive processing, potentially exacerbating chronic pain conditions (Sanders 2022,

Ramsden 2012.[37,38] A randomised trial demonstrated that lowering dietary omega-6 and increasing omega 3 oils resulted in significantly less headaches and improved quality of life in a group of people with chronic daily headache. (Ramsden et al 2013).[39]

Conclusions

The range of scientific literature covering the topic of seed oil toxicity is vast and this section has touched only on a small proportion. We recommend the ADG review process consider the many references in the book *The Ancestral Diet Revolution* (Knobbe and Alexander)[4] when considering future population recommendations.

There is a clear disconnect between the recommendations from various peak bodies on the benefits of omega-6 PUFAs, and the wide range of evidence that shows that LA and its derivatives are recent additions to the human diet and clearly linked to modern western diseases. Given the many questions about the excessive amount of LA in MWD, the precautionary principle suggests that the human requirement for LA should be reconsidered (Choque 2015),[6] especially in the context of population-wide dietary recommendations (Dinicol 2014).[14]

We recommend that the Expert Committee carefully consider all the evidence as well as all that is unknown about these recent industrial additions to the human food supply. Evidence for benefits of PUFAs is at best weak, based mostly on epidemiological cohort studies and thus highly debatable. The ADG should promote real, whole foods and recommend against consumption of ultra-processed foods, including seed oils and margarines.

References

1. Hamilton JS, Klett EL. Linoleic acid and the regulation of glucose homeostasis: A review of the evidence. Prostaglandins, Leukotrienes and Essential Fatty Acids. 2021 Dec 1;175:102366
2. Blasbalg TL, Hibbeln JR, Ramsden CE, Majchrzak SF, Rawlings RR. Changes in consumption of omega-3 and omega-6 fatty acids in the United States during the 20th century. The American Journal of Clinical Nutrition. 2011 May 1;93(5):950-62.
3. Guyenet SJ, Carlson SE. Increase in adipose tissue linoleic acid of US adults in the last half century. Advances in Nutrition. 2015 Nov;6(6):660-4.
4. Burns JL, Nakamura MT, Ma DW. Differentiating the biological effects of linoleic acid from arachidonic acid in health and disease. Prostaglandins, Leukotrienes and Essential Fatty Acids. 2018 Aug 1;135:1-4
5. Knobbe, C., and Alexander, S., The Ancestral Diet Revolution: How Vegetable Oils and Processed Foods Destroy Our Health - and How to Recover. 2023
6. Choque B, Catheline D, Delplanque B, Guesnet P, Legrand P. Dietary linoleic acid requirements in the presence of α-linolenic acid are lower than the historical 2% of energy intake value, study in rats. British Journal of Nutrition. 2015 Apr;113(7):1056-68.
7. Taha AY. Linoleic acid–good or bad for the brain?. NPJ science of food. 2020 Jan 2;4(1):1.

8. Leroy F, Cofnas N. Should dietary guidelines recommend low red meat intake?. Critical Reviews in Food Science and Nutrition. 2020 Sep 7;60(16):2763-72.
9. Speth JD, Spielmann KA. Energy source, protein metabolism, and hunter-gatherer subsistence strategies. Journal of Anthropological Archaeology. 1983 Mar 1;2(1):1-31.
10. DiNicolantonio JJ, O'Keefe JH. Omega-6 vegetable oils as a driver of coronary heart disease: the oxidized linoleic acid hypothesis. Open Heart. 2018;5(2).
11. https://crisco.com/about-crisco/
12. Teicholz N. A short history of saturated fat: the making and unmaking of a scientific consensus. Current Opinion in Endocrinology & Diabetes and Obesity. 2023 Feb 1;30(1):65-71.
13. Harcombe, Z., 2017. Dietary fat guidelines have no evidence base: where next for public health nutritional advice?. *British Journal of Sports Medicine*, *51*(10), pp.769-774.
14. DiNicolantonio JJ. The cardiometabolic consequences of replacing saturated fats with carbohydrates or Ω-6 polyunsaturated fats: do the dietary guidelines have it wrong? Open Heart. 2014 Feb 1;1(1):e000032.
15. Simopoulos AP. An increase in the omega-6/omega-3 fatty acid ratio increases the risk for obesity. Nutrients. 2016 Mar 2;8(3):128.
16. Garaulet M, Pérez-Llamas F, Pérez-Ayala M, Martínez P, de Medina FS, Tebar FJ, Zamora S. Site-specific differences in the fatty acid composition of abdominal adipose tissue in an obese population from a Mediterranean area: relation with dietary fatty acids, plasma lipid profile, serum insulin, and central obesity. The American Journal of Clinical Nutrition. 2001 Nov 1;74(5):585-91.
17. Rochling FA. Intravenous lipid emulsions in the prevention and treatment of liver disease in intestinal failure. Nutrients. 2021 Mar 10;13(3):895.
18. Spiteller G. Linoleic acid peroxidation--the dominant lipid peroxidation process in low density lipoprotein--and its relationship to chronic diseases. Chemistry and Physics of Lipids. 1998 Oct;95(2):105-62.
19. Jira W, Spiteller G, Carson W, Schramm A. Strong increase in hydroxy fatty acids derived from linoleic acid in human low density lipoproteins of atherosclerotic patients. Chemistry and physics of lipids. 1998 Jan;91(1):1-1.
20. Nagy L, Tontonoz P, Alvarez JG, Chen H, Evans RM. Oxidized LDL regulates macrophage gene expression through ligand activation of PPARgamma. Cell. 1998 Apr 17;93(2):229-40.
21. Ramsden CE, Zamora D, Leelarthaepin B, Majchrzak-Hong SF, Faurot KR, Suchindran CM, Ringel A, Davis JM, Hibbeln JR. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. BMJ. 2013 Feb 5;346:e8707.
22. Ramsden CE, Zamora D, Majchrzak-Hong S, Faurot KR, Broste SK, Frantz RP, Davis JM, Ringel A, Suchindran CM, Hibbeln JR. Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73). BMJ. 2016 Apr 12;353.
23. Kim M, Kim M, Lee A, Yoo HJ, Her JS, Jee SH, Lee JH. Impact of 8-week linoleic acid intake in soy oil on Lp-PLA2 activity in healthy adults. Nutrition & metabolism. 2017 Dec;14(1):1-9.

24. Naughton SS, Mathai ML, Hryciw DH, McAinch AJ. Linoleic acid and the pathogenesis of obesity. Prostaglandins & other lipid mediators. 2016 Sep 1;125:90-9.
25. Alvheim AR, Torstensen BE, Lin YH, Lillefosse HH, Lock EJ, Madsen L, Frøyland L, Hibbeln JR, Malde MK. Dietary linoleic acid elevates the endocannabinoids 2-AG and anandamide and promotes weight gain in mice fed a low fat diet. Lipids. 2014 Jan;49:59-69.
26. Pan DA, Storlien LH. Dietary lipid profile is a determinant of tissue phospholipid fatty acid composition and rate of weight gain in rats. The Journal of nutrition. 1993 Mar 1;123(3):512-9.
27. Moon RJ, Harvey NC, Robinson SM, Ntani G, Davies JH, Inskip HM, Godfrey KM, Dennison EM, Calder PC, Cooper C, SWS Study Group. Maternal plasma polyunsaturated fatty acid status in late pregnancy is associated with offspring body composition in childhood. The Journal of Clinical Endocrinology & Metabolism. 2013 Jan 1;98(1):299-307
28. Nigam P, Bhatt S, Misra A, Chadha DS, Vaidya M, Dasgupta J, Pasha QM. Effect of a 6-month intervention with cooking oils containing a high concentration of monounsaturated fatty acids (olive and canola oils) compared with control oil in male Asian Indians with nonalcoholic fatty liver disease. Diabetes technology & therapeutics. 2014 Apr 1;16(4):255-61.
29. Phillips CM, Goumidi L, Bertrais S, Field MR, Ordovas JM, Cupples LA, Defoort C, Lovegrove JA, Drevon CA, Blaak EE, Gibney MJ. Leptin receptor polymorphisms interact with polyunsaturated fatty acids to augment risk of insulin resistance and metabolic syndrome in adults. The Journal of nutrition. 2010 Feb 1;140(2):238-44.
30. Han IH, Csallany AS. Formation of toxic α, β-unsaturated 4-hydroxy-aldehydes in thermally oxidized fatty acid methyl esters. Journal of the American Oil Chemists' Society. 2009 Mar;86:253-60.
31. Shoeb M, H Ansari N, K Srivastava S, V Ramana K. 4-Hydroxynonenal in the pathogenesis and progression of human diseases. Current medicinal chemistry. 2014 Jan 1;21(2):230-7.
32. Jaganjac M, Zarkovic N. Lipid Peroxidation Linking Diabetes and Cancer: The Importance of 4-Hydroxynonenal. Antioxidants & Redox Signaling. 2022 Dec 1;37(16):1222-33.
33. Fabian CJ, Kimler BF, Hursting SD. Omega-3 fatty acids for breast cancer prevention and survivorship. Breast cancer research. 2015 Dec;17(1):1-1.
34. Sanders TA, Oakley FR, Miller GJ, Mitropoulos KA, Crook D, Oliver MF. Influence of n-6 versus n-3 polyunsaturated fatty acids in diets low in saturated fatty acids on plasma lipoproteins and hemostatic factors. Arteriosclerosis, thrombosis, and vascular biology. 1997 Dec;17(12):3449-60.
35. Ramsden CE, Zamora D, Makriyannis A, Wood JT, Mann JD, Faurot KR, MacIntosh BA, Majchrzak-Hong SF, Gross JR, Courville AB, Davis JM. Diet-induced changes in n-3- and n-6-derived endocannabinoids and reductions in headache pain and psychological distress. The Journal of Pain. 2015 Aug 1;16(8):707-16.
36. DiNicolantonio JJ, O'Keefe JH. Importance of maintaining a low omega–6/omega–3 ratio for reducing inflammation. Open heart. 2018 Nov 1;5(2):e000946.
37. Ramsden CE, Ringel A, Feldstein AE, Taha AY, MacIntosh BA, Hibbeln JR, Majchrzak-Hong SF, Faurot KR, Rapoport SI, Cheon Y, Chung YM. Lowering dietary linoleic acid

reduces bioactive oxidized linoleic acid metabolites in humans. Prostaglandins, Leukotrienes and Essential Fatty Acids. 2012 Oct 1;87(4-5):135-41.
38. Sanders AE, Weatherspoon ED, Ehrmann BM, Soma PS, Shaikh SR, Preisser JS, Ohrbach R, Fillingim RB, Slade GD. Circulating polyunsaturated fatty acids, pressure pain thresholds, and nociplastic pain conditions. Prostaglandins, Leukotrienes and Essential Fatty Acids. 2022 Sep 1;184:102476
39. Ramsden CE, Faurot KR, Zamora D, Suchindran CM, MacIntosh BA, Gaylord S, Ringel A, Hibbeln JR, Feldstein AE, Mori TA, Barden A. Targeted alteration of dietary n-3 and n-6 fatty acids for the treatment of chronic headaches: a randomized trial. PAIN®. 2013 Nov 1;154(11):2441-51.

Name	% Linoleic acid†
Safflower oil	78%
Grape seed oil	73%
Poppyseed oil	70%
Sunflower oil	68%
Hemp oil	60%
Corn oil	59%
Wheat germ oil	55%
Cottonseed oil	54%
Soybean oil	51%
Walnut oil	51%
Sesame oil	45%
Rice bran oil	39%
Pistachio oil	32.7%
Peanut oil	32%
Canola oil	21%
Egg yolk	16%
Linseed oil	15%
Lard	10%
Olive oil	10%
Palm oil	10%
Cocoa butter	3%
Macadamia oil	2%
Butter	2%
Coconut oil	2%
	†average val

https://twitter.com/RDValerie/status/1659595658494459915

[i] Tim Spector: Butter or margarine? Food religion challenged - The BMJ