Soy Connection Fact Sheet

SOYBEAN OIL FOR HEALTH

COMPOSITION

Oil from crushed soybeans, after the meal (protein) has been separated, is commonly labeled in grocery stores as "vegetable" oil. For this reason, despite soybean oil being the most commonly consumed vegetable oil in the United States, many consumers are unaware they are consuming it.

Soybean oil is primarily comprised of polyunsaturated fatty acids (PUFA) as it is 12 to 15% saturated fat, 22 to 30% monounsaturated fat (oleic acid) and 55 to 58% PUFA (table 1).[1,2] The PUFA is comprised of 5 to 7% alpha-linolenic acid (ALA), the essential omega-3 fatty acid, and about 50% linoleic acid (LA), the essential omega-6 fatty acid. The composition of soybean oil varies slightly among soybean varieties and is affected by growing conditions.[1] In the U.S., soybean oil accounts for over 40 percent of the intake of LA and ALA.[3]

Fatty Acid Composition of Soybean Oil

FATTY ACID	G/100 g[1]
Total Polyunsaturated	**57.740**
Linoleic (n-6, 18:2)	50.414
Linolenic (n-3, 18:3)	6.780
Total Monounsaturated	**22.783**
Oleic Acid (C18:1)	22.550
Gondoic Acid (20:1)	0.223
Total Saturated	**15.650**
Palmitic Acid (C16)	10.455
Margaric Acid (C17)	0.340
Stearic Acid (C18)	4.435
Arachidic Acid (C20)	0.361
Behenic Acid (C22)	0.366

[1]Source: USDA Nutrient Database

In the U.S., soybean oil accounts for over 40 percent of the intake of LA and ALA.[3]

The ratio of LA to ALA in soybeans is about 8–9 to–1. The rise in U.S. consumption of vegetable oils such as com and soybean oil has led to an increase in the dietary ratio of omega–6 to omega–3 PUFA. Blasburg et al.[3] estimated that this ratio increased from 6–to–7–to–1 in 1909 to be 10–to–0–to–1 in 1999. The health implications of this ratio are discussed in a later section.

NEW: Soybean Oil Achieves FDA's Qualified Health Claim

"Supportive but not conclusive scientific evidence suggests that eating about 1½ tablespoons (20.5 grams) daily of soybean oil, which contains unsaturated fat, may reduce the risk of coronary heart disease. To achieve this possible benefit, soybean oil is to replace saturated fat and not increase the total number of calories you eat in a day. One serving of this product contains 20.5 grams* of soybean oil."

*U.S. Food and Drug Administration. "Soybean Oil and Reduced Risk of Coronary Heart Disease." July 31, 2017.

PHYTOSTEROLS

CHOLESTEROL REDUCTION

Phytosterols are a group of lipophilic steroid alcohols found in plants that have some chemical similarities to cholesterol. They come in two forms: sterols and stanols. All plant foods contain phytosterols, but the most concentrated sources are vegetable oils with the exception of palm oil. Soybean oil contains approximately 300 milligram (mg) per 100 gram (g).

Phytosterols are best known for their hypocholesterolemic effects – therapeutic doses have been shown to reduce low–density–lipoprotein cholesterol (LDL–C) by about 10 to 12 percent.[4] In 2000, the U.S. Food and Drug Administration (FDA) approved a coronary heart disease (CHD) health claim for a daily intake of only 1.3 g phytosterol esters and 1.7 g phytostanol esters.

Although 2 grams per day (g/d) is the amount of phytosterols typically used therapeutically to lower elevated cholesterol, some evidence

indicates that much lower amounts that can be obtained via the diet, are also efficacious.[5] For example, in a study where participants received a test breakfast containing 30–35 g conventional corn oil or one stripped of phytosterols, cholesterol absorption was 38 percent higher when consuming the sterol-free corn oil. When corn oil phytosterols were added back to sterol-free corn oil at a concentration of 150 mg/test meal, cholesterol absorption was reduced by 12.1 percent.

After inclusion of 300 mg phytosterols, it was reduced by 27.9 percent.[6] Similar results have been demonstrated by removing phytosterols from wheat germ.[5] Additionally, in the Netherlands cohort of the European Prospective Investigation into Cancer study, higher phytosterol intake was associated with lower total cholesterol and LDL-C, particularly among men.[7] These results agree with the findings from several other observational studies.[8-12] Phytosterols, found in soybean oil, have been shown to lower LDL cholesterol by 10–12%.

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CANCER

In-vitro phytosterols have been shown to mediate cell cycle arrest, inducing cellular apoptosis,[13] and in rats they have been shown to reduce cellular oxidative stress.[14] Epidemiological studies have reported that phytosterol consumption is associated with lower risk of developing cancer of the lung,[15] breast,[16] esophagus,[17] stomach[18] and ovaries.[19]

Nair et al. found that Seventh-day Adventists who consumed high levels of phytosterols in their diets had a decreased risk of developing colorectal cancer, compared to the general population.[20] On the other hand, a Dutch cohort study found no reduction in colorectal cancer risk in relation to a high intake of any phytosterol.[21] Most recently, Huang et al. found in a Chinese observational study involving 1,802 colorectal cancer cases and 1,813 controls that after adjusting for various confounders, the risk of cancer was reduced by 50 percent when comparing the fourth phytosterol intake quartile with the first.[22] An inverse association was also found between the consumption of individual phytosterols and colorectal cancer risk including beta-sitosterol, campesterol and campestanol.

VITAMIN-E

Naturally occurring vitamin E exists in eight chemical forms that have varying levels of biological activity. The most relevant forms of vitamin E are alpha-and gamma-tocopherol (y-tocopherol). The current recommended dietary allowance (RDA) is 15 mg/d of a-tocopherol, which is based on the amount required to adequately protect red blood cells when exposed to hydrogen peroxide. Surveys suggest nearly 90 percent of American adults do not meet the estimated average requirement (EAR) of 12 mg/d of a-tocopherol.[23] This inadequate intake is potentially an important public health issue given the aging population and recent research, which found that in older people, Alzheimer's disease is associated with low serum vitamin E concentrations.[24] On the other hand, it is possible that vitamin E intake is much higher than estimates indicate, because surveys often do not account for the amounts and types of fat added during cooking.[25]

Serum concentrations of vitamin E (a-tocopherol) depend on the liver, which takes up the nutrient after the various forms are absorbed from the small intestine. The liver preferentially re-secretes only a-tocopherol via the hepatic a-tocopherol transfer protein and metabolizes and excretes the other vitamin E forms.[14] As a result, blood and cellular concentrations of other forms of vitamin E are lower than those of a-tocopherol and have been the subject of less research.[15] Soybean oil is the principal source of vitamin E in the U.S. diet.[26]

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Plasma and tissue concentrations of y-tocopherol are generally significantly lower than those of a-tocopherol, but it's actually the most common form of vitamin E in the U.S. diet.[27,28] Many vegetable oils-soybean oil in particular-are especially rich sources of y-tocopherol (table 2). Because of the widespread use of these plant products, y-tocopherol represents ~70 percent of the vitamin E consumed by Americans.[27] Interestingly, research shows that y-tocopherol but not a-tocopherol scavenges reactive nitrogen oxide species to produce 5-nitro y-tocopherol from nitrogen dioxide[29,30] or from the highly reactive peroxynitrite radicals generated in vivo from phagocytes during inflammation.[31,32] Also, y-tocopherol, but not a-tocopherol, acts as an anti-inflammatory agent by inhibiting cyclooxygenase-catalyzed prostaglandin E2 formation,[27,33] inhibiting protein kinase C activity and aiding in cell signaling, and is metabolized to a natriuretic factor.[34]

Vitamin E Content of Selected Oils

FOOD	USDA DATABASE NUMBER	TOCOPHEROL FORM			
		ALPHA	BETA	GAMMA	DELTA
Soybean	04044	8.18	0.90	64.26	21.30
Corn	04518	14.30	N/A	N/A	N/A
Olive	04053	14.35	0.11	0.83	0.00
Canola	04582	17.46	0.01	27.34	0.99
Sunflower	04506	41.06	N/A	N/A	N/A
Sesame	04058	1.40	N/A	N/A	N/A
Peanut	04042	15.69	0.46	15.91	1.37
Palm	04055	15.94	0.00	0.00	0.00
Coconut	04047	0.11	0.60	0.00	0.10

Source: National Nutrient Database for Standard Reference Release 28 Slightly revised May, 2016 So ware v.2.6.1.

DIETARY FAT, CORONARY HEART DISEASE AND LA TO ALA RATIO

Reduced saturated fat intake has been recommended for decades to protect against CHD.[35,36] The basis for this recommendation is the well-established hypercholesterolemic effect of saturated fat and to a lesser extent, the hypocholesterolemic effect of PUFA.[37] Not surprisingly given its composition, soybean oil has been shown to lower LDL-C.[38,39] Clinical research demonstrating the cholesterol-lowering effect of soybean oil when replacing fat higher in saturated fat has been underway for decades.[38]

The hypocholesterolemic effect of soybean oil was recently formally recognized by the FDA in the form of a health claim.[40] It is notable that the language recommended to be used when referencing the qualified health claim for soybean oil is much stronger than the language recommended to be used for com oil, canola oil or olive oil. Despite the years of research into the health effects of saturated fat and PUFA, in recent years a controversy has arisen over the strength of the evidence implicating the role of saturated fat.[41-43] Certainly, it is recognized that not all dietary saturated fatty acids exert the same effect on serum LDL-C.[37] Additionally, the impact of dietary saturated fatty acids on serum LDL-C may depend upon the type and composition of food in which the saturated fat is consumed. For example, saturated fat in butter raises LDL-C to a much greater extent than saturated fat in cheese.[44] This difference has been attributed to the high calcium content of cheese, which can form insoluble salts with the saturated fatty acids, preventing them from being absorbed.[45] Replacing 5 percent of energy from saturated fat with equivalent energy from PUFA was associatewith a 25 percent reduction in CHD risk.

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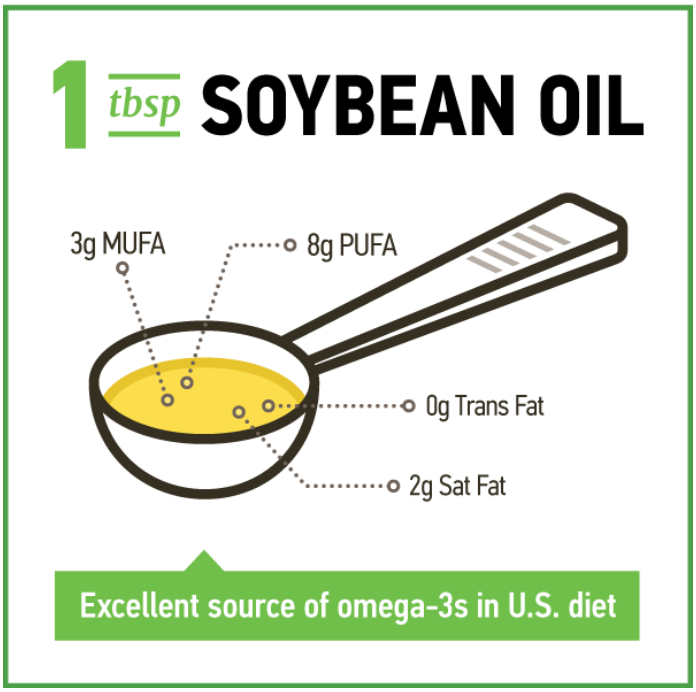
More importantly, the failure of some observational studies to show saturated fat intake is associated with an increased risk of cardiovascular disease doesn't appear to be because saturated fat doesn't raise risk, but rather, because the impact of saturated fat is dependent upon the macronutrient that replaces it.[46] To this point, a combined analysis of the Nurses' Health Study (1980 to 2010, n=84,628) and the Health Professionals Follow-up Study (1986 to 2010, n= 42,908 men) found that replacing 5% of energy intake from saturated fat with equivalent energy intake from PUFA, monounsaturated fat or carbohydrates from whole grains was associated with a 25%, 15%, and 9% lower risk of CHD, respectively. On the other hand, replacing saturated fat with carbohydrates from refined starches/added sugars was not significantly associated with CHD risk.[47] Similar findings exist for total mortality and for CHO-specific mortality.[48] With respect to individual fatty acids, replacing just 1% of energy from palmitic acid (the most abundant saturated fat in the diet) with 1% of energy from PUFA lowered CHD risk by a statistically significant 120%.[49]

Despite its cholesterol-lowering effect, some concerns have arisen that too much omega-6 PUFA and in particular, LA, may increase CHO risk by increasing inflammation. However, the American Heart Association (AHA) has rejected concerns about the pro-inflammatory properties of LA and concluded that omega-6 PUFA play a critical role in heart-healthful diets.[50] This position is supported by a comprehensive review by Johnson and Fritsche,[51] published in 2012, which concluded that "virtually no evidence is available from randomized, controlled intervention studies among healthy, non-infant human beings to show that addition of LA to the diet increases the concentration of inflammatory markers." More recently, in the Kuopio Ischaemic Heart Disease Risk Factor Study, Virtanen et al.[52] found that serum LA levels were strongly inversely related to serum C-reactive protein, which is a general marker of inflammation. This study involved 1,287 generally healthy men aged 42-60 years.

The evidence indicates that diets high in omega-6P UFA are not pro-inflammatory.



Despite common belief, there are several reasons why omega-6 PUFA are not pro-inflammatory. One is that although LA is converted in vivo to arachidonic acid (AA), the fatty acid from which a number of pro-inflammatory eicosanoids are produced, tissue levels of AA don't substantially increase in response to LA intake because they are tightly regulated.[53] Another reason is that it is now recognized that not all of the eicosanoids produced from AA are pro-inflammatory; some in fact may be anti-inflammatory.[54]

There is no rationale to recommend a specific dietary ratio of omega-3 and omega-6 fatty acids.

Given these data, it is not surprising that the Food and Agricultural Organization (FAO) of the United Nations concluded that that there is no rationale for a specific recommendation regarding the ratio of omega-6 to omega-3 fatty acids. This is as long as the omega-6 fatty acid intake is between 2.5% and 9% of energy, and omega-3 fatty acid intake is between 0.5% and 2% of energy.[55] The National Academy of Medicine does not recommend a specific ratio of omega-6

to omega-3 PUFA either. According to U.S. food disappearance data, LA and ALA intakes fall within the United Nations' recommendations: these fatty acids represent 7.21% and 0.72% of energy intake, respectively.[3] Increasingly, evidence suggests that both LA and ALA have a role in reducing the risk of CHD and that the absolute amount consumed of these essential fatty acids should be emphasized rather than the ratio.[56]

One key remaining issue is the inhibitory effect of LA intake on the conversion of ALA to the longer chain omega-3 PUFA (such as eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]).[57] However, according to the AHA, because this conversion is already low,[58] it isn't clear that additional small changes would have net effects on CHO risk after the other benefits of LA consumption are taken into account. In a review of this issue, internationally recognized CHD expert William S. Harris concluded that the focus should not be on dietary ratios but rather on intake levels of each type of essential fat.[59] Harris et al.[50] noted that decreasing LA intake as a means of increasing the dietary ratio of LA to ALA, as some have called for,[60-62] could very well have the opposite effect of that intended.

Furthermore, it is important to recognize that the evidence in support of the coronary benefits of EPA and DHA has weakened quite substantially in recent years. In fact, in 2018, the largest evaluation of the clinical data concluded that, "Increasing EPA and DHA has little or no effect on all-cause deaths and cardiovascular events (high-quality evidence) and probably makes little or no difference to cardiovascular death, coronary deaths or events, stroke, or heart irregularities (moderate-quality evidence, coronary events are illnesses of the arteries which supply the heart)."[63] In contrast, there was some support for the coronary benefits of ALA.[63]

Recently, Ramsden et al.[42] have argued that diets high in LA are harmful because LA is easily oxidized. They cited six references in support of this contention.[64-69] Of these six, two involved the measurement of lipid changes in oils in response to heat;[65,67] one evaluated the effects of manipulating LA intake (12.5 to 25-fold increases) on nociceptive lipid autacoid content in rats;[66] one reported on the absorption of lipid oxidation products in women;[68] another reported on the effect of consuming foods deep fried in sunflower oil with or without antioxidants on eicosanoid production in obese individuals;[69] and the sixth showed that lowering LA intake from 6.74 to 2.42 percent kcal reduces oxidized LA metabolites in humans.[64] Overall, the evidence cited in support of the harmful effects of LA is unimpressive.

DIETARY FAT AND CHD RISK INDEPENDENT OF EFFECTS ON LIPID LEVELS

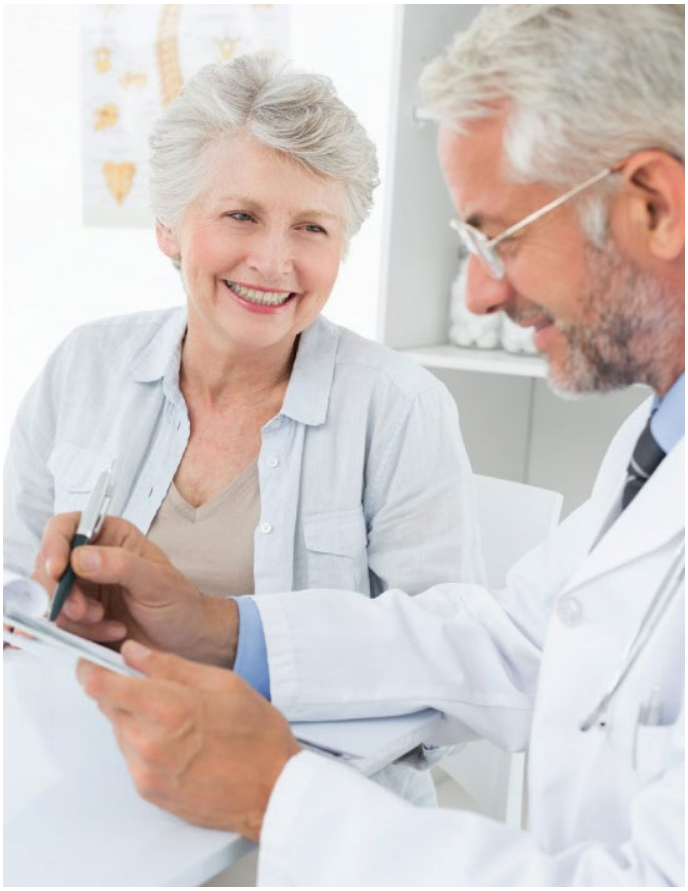
Many studies have evaluated the impact of dietary fat on lipid levels, whereas relatively few studies have focused on the impact of fat on other physiologic processes potentially related to CHD risk, such as insulin resistance (IR). IR refers to the diminished ability of cells to respond to the effects of insulin in transporting glucose from the circulation into muscle and other tissues.

Until recently, there were conflicting views on the impact of fatty acids on IR. In 2009, after an extensive review of the data, Riserus et al.[70] concluded that the clinical data indicate replacing saturated fat and trans fat with PUFA or monounsaturated improves insulin sensitivity. In contrast, one year later, a report by the FAO concluded the data were inconsistent.[55]

However, the largest systematic review and meta-analysis of clinical trials to examine the effects of fatty acid type on IR, which included 102 trials, 239 diet arms and 4,220 adults, found clear benefits of PUFA.[71] In this 2016 publication, energy intake substitution with PUFA was linked to lower fasting glucose, lower glycosylated hemoglobin levels, improved homeostatic model assessment of insulin resistance (HOMA-IR) and improved insulin secretion capacity. These effects were generally seen whether PUFA replaced carbohydrate or saturated fat. Furthermore, insulin secretion capacity also improved when PUFA replaced monounsaturated fat (MUFA). In comparison, MUFA consumption did not appear to significantly influence fasting glucose compared to other macronutrients but was seen to reduce glycosylated hemoglobin and improve HOMA-IR, in comparison to either carbohydrate or saturated fat.

The findings of this meta-analysis have significant implications for reducing risk of diabetes and CHO. Its authors estimated that because for each 5% energy of increased MUFA or PUFA, glycosylated hemoglobin improved by approximately 0.1%, this type of dietary change would reduce risk of type 2 diabetes by 22.0%[72] and CVD by 6.8%.[73]

Finally, recent evidence shows that in comparison to saturated fat, unsaturated fat can enhance cholesterol efflux capacity.[74] Efflux capacity, which refers to the ability to remove cholesterol from lipid-laden macrophages, is inversely related to CHD risk.[75] Additionally, in 2018, British researchers found that when compared to saturated fat, unsaturated fat increased the number of endothelial progenitor cells, which are cells that play a role in regenerating the lining of the endothelium. This cross-over trial had men and women consume three diets containing different amounts of saturated and unsaturated fat for 16-week periods.[76] Enhancing endothelial function and efflux capacity may represent two ways in which unsaturated fat can lower risk of CHD, independent of their favorable effects on lipid levels.



HIGHLY REFINED SOYBEAN OIL IS NOT ALLERGENIC

The U.S. Food Allergen Labeling & Consumer Protection Act (FALCPA) mandates labeling of all ingredients derived from commonly allergenic foods. In the U.S., eight foods (Big 8) have been identified as the most frequent human food allergens, accounting for 90 percent of food allergies. These foods are milk, eggs, fish, crustacea, wheat, peanuts, tree nuts and soy.[78] However, these foods are not equally allergenic – in fact, soy protein allergies are relatively uncommon.[79] The most recent nationwide survey, which was conducted by the FDA, found only one out of 1,000 adults are allergic to soy protein.[79] The prevalence of soybean allergy is the same as the prevalence of allergy to pea protein and to chocolate, neither of which have to be labeled as an allergen. Allergy to soy protein is much less common than allergy to milk/dairy and peanuts; in fact, allergy to these foods is approximately 20-to-40 and 6-to-9 times more common, respectively, than soy protein allergy.[79] Highly refined soybean oil does not cause allergic reactions in soy-allergic individuals.

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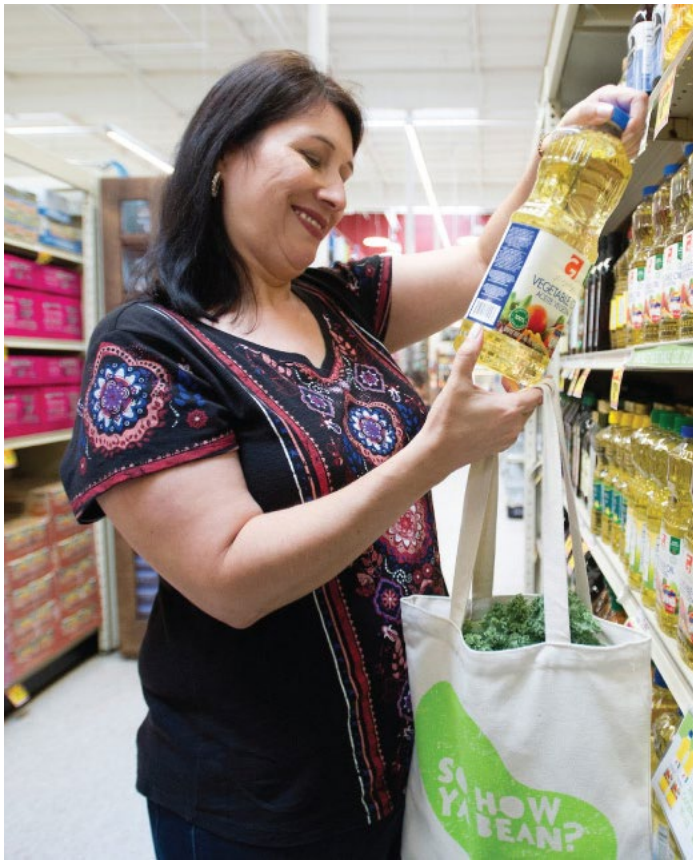

Importantly, the FALCPA exempts highly refined oils from these labeling provisions because highly refined soybean, peanut and sunflower seed oils have been clinically documented to be safe for consumption by individuals allergic to the source food.[80-83] Soy is viewed similarly in Europe as it is in the U.S., in that soy protein is classified as one of the 14 most common foods that induce allergic reactions, yet fully refined soybean oil is exempt from labeling.[84]

The process of commercially refining soybean oil involves extraction with hot solvents, bleaching and deodorization, which serves to eliminate almost all soy protein – and thus allergens – from the oil.[85] However, it is extremely difficult to quantify the protein content of oil. Attempts to do so indicate that crude oils contain about 100-300 mg/kg, whereas fully refined oils contain at least 100 times less.[85] This difference explains the lack of reaction observed in response to ingesting highly refined oils, unlike ingesting unrefined or partially refined culinary oils, which have been found to elicit allergic reactions in sensitized individuals.[86]

While highly refined soybean oil does contain residual soy protein, the residue levels are extremely low – too low to elicit an allergic response in nearly all cases.[85,57–59] Analytical data from Rigby et al.[90] on cumulative threshold doses for soy protein suggests that even the most sensitive individuals would need to consume at least 50 g of highly refined oil to experience subjective symptoms.

There have been a few cases where soybean oil elicited an allergic response, but these followed intravenous infusion of an emulsion containing soybean oil, which seems far removed from typical means of consumption.[88,91,92] There is also one unusual case of a possible soybean oil–induced allergy after an infant was fed exclusively on an amino acid–based formula containing a soybean oil–based component.[93] The circumstances of exposure in this exceptional case are unusual and the association with the soybean oil component of the formula was somewhat speculative.

In addition to the clinical studies cited here showing that highly refined soybean does not elicit an allergic response, circumstantial evidence supporting the clinical results comes from the work of the Swedish National Food Administration. Since 1994, this group has been recording and investigating all cases of fatal and severe reactions to foods.[94,95] While soy protein featured in about 25 percent of the reported cases (compared to ~33 percent for peanuts), none implicated soybean oil, or a product containing soybean oil, as the only source of soy.

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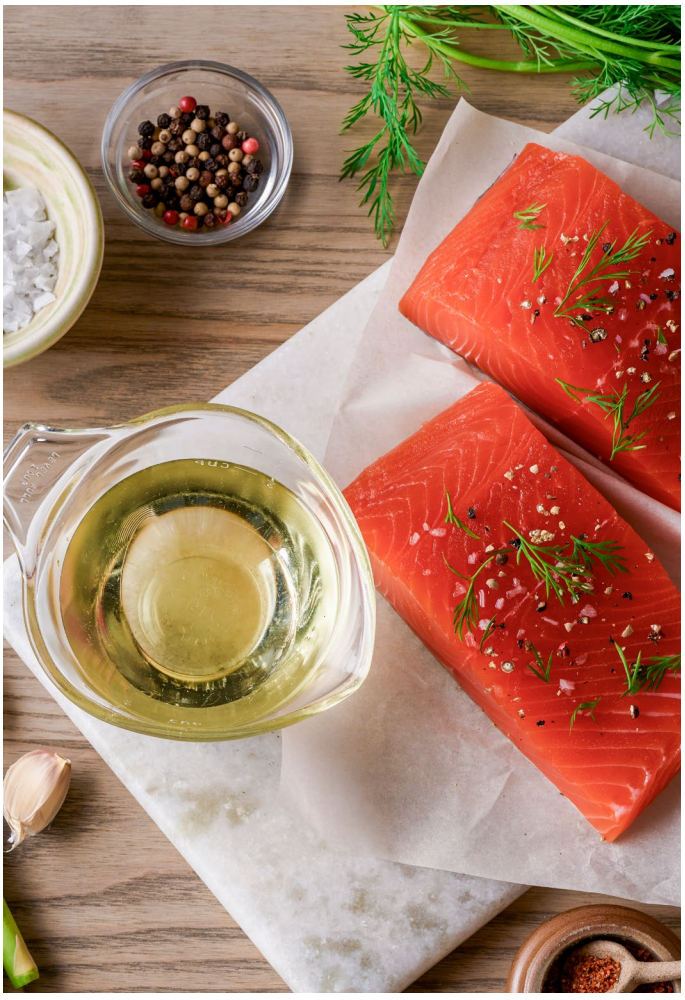

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