Chapter

The Effects of Linoleic Acid Consumption on Lipid Risk Markers for Cardiovascular Disease

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Abstract

Cardiovascular disease (CVD) is the number one contributor to death in the United States and worldwide. Lipid risk markers for CVD include high serum concentrations of total cholesterol, low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), lipoprotein (a), and triglycerides, as well as low serum concentrations of high-density lipoprotein cholesterol (HDL-C). Additional factors to assess CVD risk include apolipoprotein A (associated with HDL) and apolipoprotein B (associated with LDL). A suggested dietary strategy to decrease these risk factors is to replace a portion of saturated fatty acids with unsaturated fatty acids – especially polyunsaturated fatty acids (PUFAs). One PUFA, in particular, is the essential omega-6 PUFA linoleic acid, which has been demonstrated to affect these CVD risk markers. Therefore, this chapter will discuss the effects of linoleic acid consumption on lipid risk markers for CVD in healthy individuals, the associated mechanisms, and dietary recommendations to decrease CVD risk.

Keywords: linoleic acid, fatty acids, lipid risk markers, cardiovascular disease, humans

1. Introduction

Cardiovascular disease (CVD) (includes heart disease and stroke) is the leading cause of death in the United States [1] and worldwide [2]. In the United States, heart disease is the number one contributor to death, causing 647,457 deaths (23% of total deaths), while stroke is the fifth leading cause of death, contributing to 146,383 deaths (5.2% of total deaths) in 2017 [1]. Worldwide, heart disease is the leading cause of death, leading to 8.9 million deaths, or 16% of the total deaths globally in 2019. Stroke is the second leading contributor to deaths worldwide, causing more than 6 million deaths, or 11% of the deaths, worldwide [2].

A suggested dietary strategy to decrease the risk factors for CVD is to replace a portion of saturated fatty acids (SFAs) with monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs) [3–10]. For example, the Nurses' Health Study [11] demonstrated that replacing 5% of energy from SFAs with equivalent

energy from MUFAs, PUFAs, or carbohydrates from whole grains, decreased the risk for coronary heart disease (CHD). A meta-analysis of randomized controlled trials reported that replacing saturated fat with polyunsaturated fat reduced CHD events [12].

However, certain authors and publications are not in agreement with these recommendations to decrease the risk factors for CVD, cardiovascular events, and/or mortality [13–22]. The PURE prospective cohort study concluded that intakes of total, saturated, and unsaturated fats were not significantly associated with the risk of myocardial infarction or CVD mortality [23]. Meta-analyses of prospective cohort studies demonstrated that consumption of saturated fat was not associated with an increased risk of CVD [24]. Interestingly, there was an inverse association between saturated fat intake and the risk of stroke [25]. Additionally, a meta-analysis of randomized controlled trials reported that replacing saturated fat with primarily polyunsaturated fat is "unlikely" to reduce CVD events or mortality [26]. Hooper et al. [27], in a review of randomized controlled trials, stated that there is "little or no effect of reducing saturated fat on all-cause mortality or cardiovascular mortality."

As noted, there is controversy regarding the effects of the consumption of fatty acids on CVD risk. One such controversy is the recommendation of linoleic acid, which is the essential omega-6 (or n-6) PUFA [28–30]. For example, it has been found that replacing saturated fat with linoleic acid lowers serum cholesterol, but does not lower the risk of death from CHD [21, 22]. Furthermore, there is concern regarding whether linoleic acid increases the risk for inflammation [31].

An analysis of prospective observational studies demonstrated that higher tissue and serum concentrations of linoleic acid decreased the risk for cardiovascular events [32]. The Cardiovascular Health Study, a prospective cohort study, discovered that higher circulating linoleic acid concentrations reduced total and CHD mortality [33]. A meta-analysis of prospective cohort studies found that decreased consumption of omega-6 PUFAs and increased intakes of saturated and trans-fatty acids increased CHD mortality [34]. Linoleic acid consumption reduced the risk of CHD events and death, according to another meta-analysis of prospective cohort studies [35]. A systematic review of randomized controlled trials, in which there was a replacement of dietary saturated and monounsaturated fatty acids with omega-6 fatty acids, concluded that omega-6 fatty acids lowered the risk of myocardial infarction. Additionally, the intake of omega-6 fatty acids reduced total serum cholesterol, but not "other blood fat fractions". It was also highlighted that "the benefits of omega-6 fats remain to be proven" [36].

According to the diet-heart hypothesis, a high consumption of saturated fat and cholesterol – and a low intake of polyunsaturated fat – increase the build-up of cholesterol and plaques in artery walls; these developments, therefore, increase the risks for atherosclerosis, cardiovascular disease, and myocardial infarction [18, 21, 37]. However, the diet-heart hypothesis has been evolving, and thus, some individuals recommend focusing more on overall dietary patterns, rather than individual fatty acids [37]. Moreover, there are a variety of factors that contribute to increasing the risk for CVD, such as high blood pressure, arrhythmia, inflammation, thrombosis, insulin resistance, endothelial dysfunction, obesity, cigarette smoke, genetics, the microbiome, a lack of exercise, a high alcohol consumption, and overall dietary patterns [14, 18, 37–42].

Lipid levels have also been proposed to be "strong" risk factors for CVD and mortality. These lipid risk factors include the following: high serum concentrations of total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), lipoprotein(a), and very-low-density lipoprotein cholesterol (VLDL-C), as well

as low serum concentrations of high-density lipoprotein cholesterol (HDL-C). In addition, apolipoprotein A1 (associated with HDL) and apolipoprotein B (associated with LDL) have been used as CVD risk markers [18, 43]. Interestingly, LDL particle size has also been utilized as a risk marker for CVD. The small, dense LDL subclass, compared to large, buoyant LDL particles, has been reported to be more atherogenic [44–50].

Therefore, this chapter will focus on the consumption of linoleic acid on lipid risk markers for CVD in healthy individuals, such as total cholesterol, triglycerides, LDL-C, LDL particle size, lipoprotein(a), VLDL-C, HDL-C, apolipoprotein A1, and apolipoprotein B. The associated mechanisms of action will also be covered. The chapter will conclude with recommendations to decrease the risk factors for CVD. Significant dietary sources of linoleic acid are presented in **Tables 1** and **2**. The chemical structure of linoleic acid is illustrated in **Figure 1**.

Oils	Linoleic acid (grams)
Corn oil	53.5
Cottonseed oil	51.9
Grapeseed oil	69.6
Peanut oil	32.0
Safflower oil	12.7
Sesame oil	41.3
Soybean oil	51.0
Sunflower oil	65.7
Walnut oil	52.9

Table 1.
Oil sources of linoleic acid (per 100 grams) [51, 52].

Nuts and seeds	Linoleic acid (grams)
Almonds	3.49
Brazil nuts	6.82
Pecans	5.85
Pine nuts	9.4
Pistachios	4.0
Pumpkin seeds	5.55
Sesame seeds	5.78
Sunflower seeds	9.29
Walnuts	10.8

Table 2.
Linoleic acid content in nuts and seeds (per 1 ounce or 28.3495 grams) [51, 52].

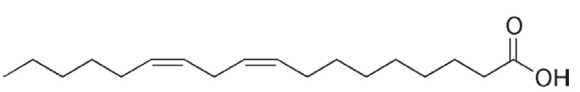

Figure 1.
The chemical structure of linoleic acid [53].

2. The effects of linoleic acid consumption on lipid risk markers for cardiovascular disease in healthy individuals

The consumption of linoleic acid has been demonstrated to affect lipid risk markers for cardiovascular disease. The discussed studies include intervention trials that investigated the effects of linoleic acid consumption, in grams or percentage of energy, on CVD lipid risk markers in healthy individuals. Therefore, epidemiological, postprandial, and animal studies are not covered. The results are organized by the respective CVD lipid risk marker.

2.1 Total cholesterol

The consumption of linoleic acid decreased total cholesterol compared to a usual U.S. diet (high in saturated fat and cholesterol) [54], and diets high in SFAs [55] (including stearic acid [56] and palmitic acid [57]), MUFAs [58], or medium-chain fatty acids [59]. A high intake of alpha-linolenic acid, the essential omega-3 fatty acid, decreased cholesterol concentrations compared to the control diet with the same percentage of linoleic acid [60]. In contrast, no significant differences in total cholesterol were observed after linoleic acid consumption compared to diets containing eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (omega-3 fatty acids) [61, 62], alpha-linolenic acid [63], high and low amounts of linoleic acid [64], oleic acid (a MUFA) [65, 66], or stearic acid [66]. Interestingly, intakes of SFA- or linoleic acid-rich diets – both supplemented with EPA and DHA – produced no significant differences in cholesterol concentrations [67].

2.2 Triglycerides

The consumption of linoleic acid decreased triglycerides compared to diets with significant amounts of oleic acid [58], stearic acid [56], or medium-chain fatty acids [59]. Intakes of linoleic acid, supplemented with EPA and DHA, reduced triglyceride concentrations versus a linoleic acid diet rich in oleic acid [62]. A SFA-rich diet and a diet high in linoleic acid, both with added EPA and DHA, lowered triglyceride concentrations, with no significant differences between diets [67]. In contrast, diets supplemented with EPA and DHA decreased triglycerides compared to linoleic acid intakes [55, 61]. No significant differences were observed regarding triglyceride concentrations between lower and higher linoleic acid intakes [54, 60, 64], and diets rich in linoleic acid versus diets high in alpha-linolenic acid [63], oleic acid [65], or stearic acid [66].

2.3 Low-density lipoprotein cholesterol (LDL-C)

Intakes of linoleic acid decreased LDL-C versus diets rich in oleic acid [58], SFAs [55], palmitic acid [57], stearic acid [56, 68], trans-fatty acids [68], or medium-chain fatty acids [59]. Furthermore, higher amounts of linoleic acid more significantly lowered LDL-C concentrations [54, 57]. There were mixed results or no significant differences in comparison to oleic acid [62, 65, 66, 68]. Additionally, there were no significant differences when comparing linoleic acid consumption to alpha-linolenic acid [63] or stearic acid [66]. Consuming low and high amounts of linoleic acid – along with significant amounts of alpha-linolenic acid [64] or EPA and DHA [62] – also did not differ. Moreover, no significant differences were observed with respect to LDL-C after following a SFA-rich diet or a diet high in linoleic acid – both supplemented with EPA and DHA [67].

2.4 LDL particle size

There were no significant differences in LDL particle size after consumption of low and high amounts of linoleic acid [60, 69], and intakes of linoleic acid compared to oleic acid or stearic acid [66]. Interestingly, there were decreases in large and small LDL particle concentrations after 10 days of a linoleic acid-rich diet compared to a diet high in SFAs, with both diets supplemented with EPA and DHA [70]. In contrast, no significant differences were observed in LDL particle size after 6 weeks of a linoleic acid-rich diet comparted to a SFA-rich diet (both supplemented with EPA and DHA) [67].

2.5 Very-low-density lipoprotein cholesterol (VLDL-C)

Following consumption of diets rich in linoleic acid, there were decreases in VLDL-C concentrations compared to diets containing significant amounts of oleic acid [58] or medium-chain fatty acids [59]. In contrast, intakes of a SFA- or linoleic acid-rich diet (both containing significant amounts of EPA and DHA) resulted in no significant differences between diets; however, both diets decreased VLDL-C concentrations [67].

2.6 High-density lipoprotein cholesterol (HDL-C)

HDL-C increased following consumption of linoleic acid compared to stearic acid [56]. In contrast, intakes of linoleic acid decreased HDL-C compared to EPA and DHA [61] or palmitic acid [57]. However, most studies noticed no significant differences regarding HDL-C concentrations after consuming low and high amounts of linoleic acid [54, 60, 62, 64], and linoleic acid compared to oleic acid [58, 65, 66], alpha-linolenic acid [60, 63], or stearic acid [66]. Interestingly, intakes of SFAs or linoleic acid (both diets supplemented with EPA and DHA) displayed no significant differences in HDL-C concentrations [67].

2.7 Lipoprotein(a)

Linoleic acid consumption reduced lipoprotein(a) concentrations compared with a diet high in trans-fatty acids [68]. However, linoleic acid intake increased lipoprotein(a) compared to a diet rich in SFAs [55]. In contrast, no significant differences were found after consuming linoleic acid compared to SFAs, oleic acid, or stearic acid [68].

2.8 Apolipoproteins A1, A2, and B

The consumption of linoleic acid increased apolipoprotein A1 compared to a typical U.S. diet [54]. Additionally, linoleic acid increased apolipoprotein A2 compared to EPA and DHA [55]. In contrast, apolipoproteins A1 and A2 decreased after following a diet rich in linoleic acid compared to a diet high in oleic acid [65]. Apolipoprotein A1 concentrations did not differ when comparing low and high linoleic acid intakes [60], and consumption of linoleic acid compared to diets containing high amounts of stearic acid or oleic acid [66]. There were decreases in apolipoprotein B concentrations after linoleic consumption compared to a typical U.S. diet [54], stearic acid, elaidic acid (a trans-fatty acid) [56], or SFAs [55]. There were no significant differences with respect to apolipoprotein B after intakes of linoleic acid compared with oleic acid [65, 66] or stearic acid [66].

3. The mechanisms by which linoleic acid affects lipid risk markers for cardiovascular disease

Linoleic acid (or PUFAs) has been demonstrated to affect CVD lipid risk markers. The mechanisms involved in altering these risk markers will be discussed in this section.

3.1 Total cholesterol

PUFAs have been shown to increase liver X receptor alpha (LXRα) gene expression [71, 72] via peroxisome proliferator activated receptors (PPARs) [71]. LXRα stimulates the expression of cholesterol 7 α-hydroxylase (CYP7), thereby converting cholesterol to bile acids. Therefore, by increasing CYP7 activity, PUFAs participate in cholesterol catabolism [73].

3.2 Triglycerides

PUFAs interact more strongly with PPARα compared to SFAs [74]. PPARα binds to peroxisome proliferator response elements (PPREs) located in the promotor regions of genes, such as apoC-III and lipoprotein lipase (LPL) [75]. It has been proposed that LPL may demonstrate increased activity towards VLDL triglycerides containing polyunsaturated fatty acids, thereby leading to increased breakdown of triglyceride-rich lipoproteins (chylomicrons and VLDL particles) [73, 76, 77]. LPL activity is inhibited by apoC-III, and thus, increases triglyceride concentrations [78]. It has been reported that PUFAs decrease apoC-III, thereby increasing LPL activity and, indeed, VLDL catabolism [73]. Moreover, omega-3 PUFAs have been shown to reduce triglycerides by lowering diacylglycerol acyltransferase, fatty acid synthase, and acetyl coenzyme A (CoA) carboxylase [79–84].

3.3 LDL-C

Intakes of linoleic acid [85] or PUFAs [73] have been demonstrated to increase LDL receptor activity, protein, and mRNA compared to SFAs. Furthermore, PUFAs increase membrane fluidity [73, 85, 86], which increases LDL receptor activity, and thus, increases LDL catabolism [87–89].

3.4 LDL particle size

It has been reported that consumption of SFAs increases large, buoyant LDL particles compared to lower SFA-containing diets [69, 90], whereas consumption of diets rich in PUFAs decreases large, buoyant LDL particles versus diets high in SFAs [70, 91]. It has been suggested that SFAs increase LPL and hepatic lipase activities [92, 93]. As such, LPL increases large, buoyant LDL particles, whereas hepatic lipase may stimulate the catabolism of triglyceride-rich lipoprotein remnants [92]. However, additional research is needed in this area regarding the mechanisms by which individual fatty acids affect LDL particle size.

3.5 VLDL-C

The sterol regulatory element-binding protein-1 (SREBP-1) is associated with lipogenesis and cholesterol synthesis in the liver [94, 95]. PUFAs have been shown to inhibit SREBP-1 gene transcription and/or protein [96], thereby lowering VLDL secretion from the liver [73, 96]. In addition, intakes of PUFAs increase VLDL catabolism and uptake [59, 81].

3.6 HDL-C

Replacing SFAs with MUFAs and/or PUFAs generates lower total cholesterol and LDL-C concentrations, with modest HDL-C reductions; however, a lower total cholesterol: HDL-C ratio results [4, 97]. It is thought that dietary fat increases the "transport rate" and decreases the "fractional catabolic rate" of HDL cholesterol ester and apolipoprotein A1 [98]. However, more research is needed to describe the mechanisms by which individual fatty acids impact HDL-C.

3.7 Lipoprotein(a)

Lipoprotein(a) is synthesized in the liver and contains apolipoprotein A, which is bound to apolipoprotein B-100 [99–101]. The biological activity of lipoprotein(a) is unknown [102]; however, high concentrations have been associated with CVD [101, 103, 104]. Genetics seem to be the primary determinant of lipoprotein(a) [105]. Hence, diet and exercise do not appear to be significant contributors to lipoprotein(a) concentrations. There have also been inconsistent findings of fatty acid consumption (including PUFAs) on lipoprotein(a) concentrations [106]. However, it has been suggested that fatty acids may affect liver apolipoprotein(a) synthesis, thereby impacting lipoprotein(a) [106–108]. As such, more research is needed to determine the effects of dietary composition on lipoprotein(a) concentrations.

3.8 Apolipoprotein A1

HDL particles contain apolipoprotein A1, which interacts with the ATP-binding cassette transporter on the surface of cells. Furthermore, apolipoprotein A1 is a cofactor for lecithin cholesterol acyl transferase, which generates mature HDL particles [43, 109]. Plasma apolipoprotein A1 concentration typically coincide with HDL-C concentrations [43]. The significance of apolipoprotein A2 is less clear [110]. Interestingly, PPARα also interacts with PPREs in the promoter region of the apolipoprotein A1 gene in the liver [75]. Hence, PUFAs may exert their effects on apolipoprotein A1 via PPARα [73].

3.9 Apolipoprotein B

Apolipoprotein B also occurs in two forms: apolipoprotein B-48 and apolipoprotein B-100. The intestine synthesizes apolipoprotein B-48, which is a component of chylomicrons. The liver produces apolipoprotein B-100, which is associated with VLDL and LDL particles. Apolipoprotein B is necessary for the binding of lipoproteins to the LDL receptor. Apolipoprotein B plasma concentrations are significantly associated with LDL-C concentrations [43, 111]. It has been reported that high apolipoprotein B concentrations increase the risk for CVD, whereas apolipoprotein A1 concentrations decrease CVD risk [43, 112]. As mentioned previously, PUFAs increase LDL catabolism, thereby reducing apolipoprotein B [87].

4. Linoleic acid recommendations

The adequate intake (AI) values for linoleic acid for males and females (19-50 years) are 17 grams/day and 12 grams/day, respectively. Regarding males and females ages 51-70 years, the AI values for linoleic acid are 14 grams/day and 11 grams/day, respectively. The American Heart Association recommends consuming 5 to 10% of energy as

linoleic acid to decrease CVD risk [29, 31]. Additionally, the World Health Organization recommends consuming 2.5 to 9% of energy from linoleic acid to decrease LDL and total cholesterol concentrations, and thus, lower the risk for CVD [113]. It is not recommended to consume more than 10% of energy as linoleic acid due to limited research.

5. Fat recommendations

In addition to linoleic acid recommendations, there are fat recommendations to reduce CVD risk, cardiovascular events, and/or mortality. For example, it has been suggested to replace approximately 5% of energy from SFAs with MUFAs and/or PUFAs [4, 8–12, 38, 113], and to consume less than 10% of energy as SFAs [113, 114]. On the other hand, certain studies do not coincide with these recommendations [14–17, 21–27, 115]. In addition, low-fat, and in-turn, high-carbohydrate diets, decrease LDL-C; however, there is also a reduction in HDL-C and increased concentrations of VLDLs or triglycerides [37, 97, 116–118], which may produce higher amounts of small, dense LDL particles [41, 119, 120]. It has been reported that higher-fat versus lower-fat diets increase large, buoyant LDL and/or decrease small, dense LDL particles [69, 92, 121–123]. Interestingly, higher SFA intakes also increase large LDL and/or decrease small LDL particles [69, 90, 124–126]. These small, dense LDL particles may increase the risk for CVD in the following ways: 1) increased transport into arterial walls [127]; 2) increased attachment to proteoglycans [128]; 3) increased oxidation [129, 130]; and 4) reduced binding to the LDL receptor [127, 131, 132].

Various organizations have published dietary recommendations to decrease the risk factors for CVD. The American College of Cardiology/American Heart Association Task Force suggests consuming a diet rich in fruits, vegetables, whole grains, nuts, legumes, lean animal or plant protein sources, and fish. Additionally, it is recommended to decrease the consumption of red and processed meats, refined carbohydrates, trans-fatty acids, sodium, cholesterol, and sugar-sweetened drinks [38]. The Dietary Guidelines for Americans suggest consuming vegetable oils to replace sources rich in SFAs, such as butter, shortening, lard, palm oil, palm kernel oil, coconut oil, full-fat dairy products, and high-fat meats [114]. The World Health Organization also recommends replacing SFAs with unsaturated fatty acids, such as sunflower, safflower, corn, soybean, canola, and olive oils, as well as nuts, avocado, and fish [113].

It has been recently proposed, however, that guidelines to lower the risk for CVD should focus on overall dietary patterns, rather than individual fatty acids [14, 133]. The consumption of low-fat diets, for example, did not reduce CVD risk [134, 135]. Furthermore, certain individuals with higher intakes of saturated fat and cholesterol do not possess high CVD mortality rates, as they have an increased consumption of plant foods – in addition to MUFAs and PUFAs [15]. Furthermore, some foods that are higher in SFAs have not been demonstrated to increase the risk for CVD. A proposed explanation for these outcomes is the food matrix of these items, such as macro- and micronutrients, phytochemicals, and probiotics [14, 37].

6. Conclusions

This chapter focused on the effects of linoleic acid consumption on lipid risk markers for CVD in healthy individuals. Interestingly, linoleic acid reduced total cholesterol and LDL-C compared to diets that were lower in PUFAs and/or higher in

SFAs. In contrast, linoleic acid generated inconsistent outcomes regarding triglycerides, whereas EPA and DHA more significantly reduced triglyceride concentrations. In limited studies, linoleic acid decreased VLDL-C compared to diets containing oleic acid or medium-chain fatty acids, and decreased HDL-C compared to palmitic acid or EPA and DHA; however, linoleic acid increased HDL-C compared with stearic acid. Additionally, linoleic acid reduced apolipoprotein B in comparison to a typical U.S. diet, SFAs, or trans-fatty acids. Interestingly, there were inconsistent

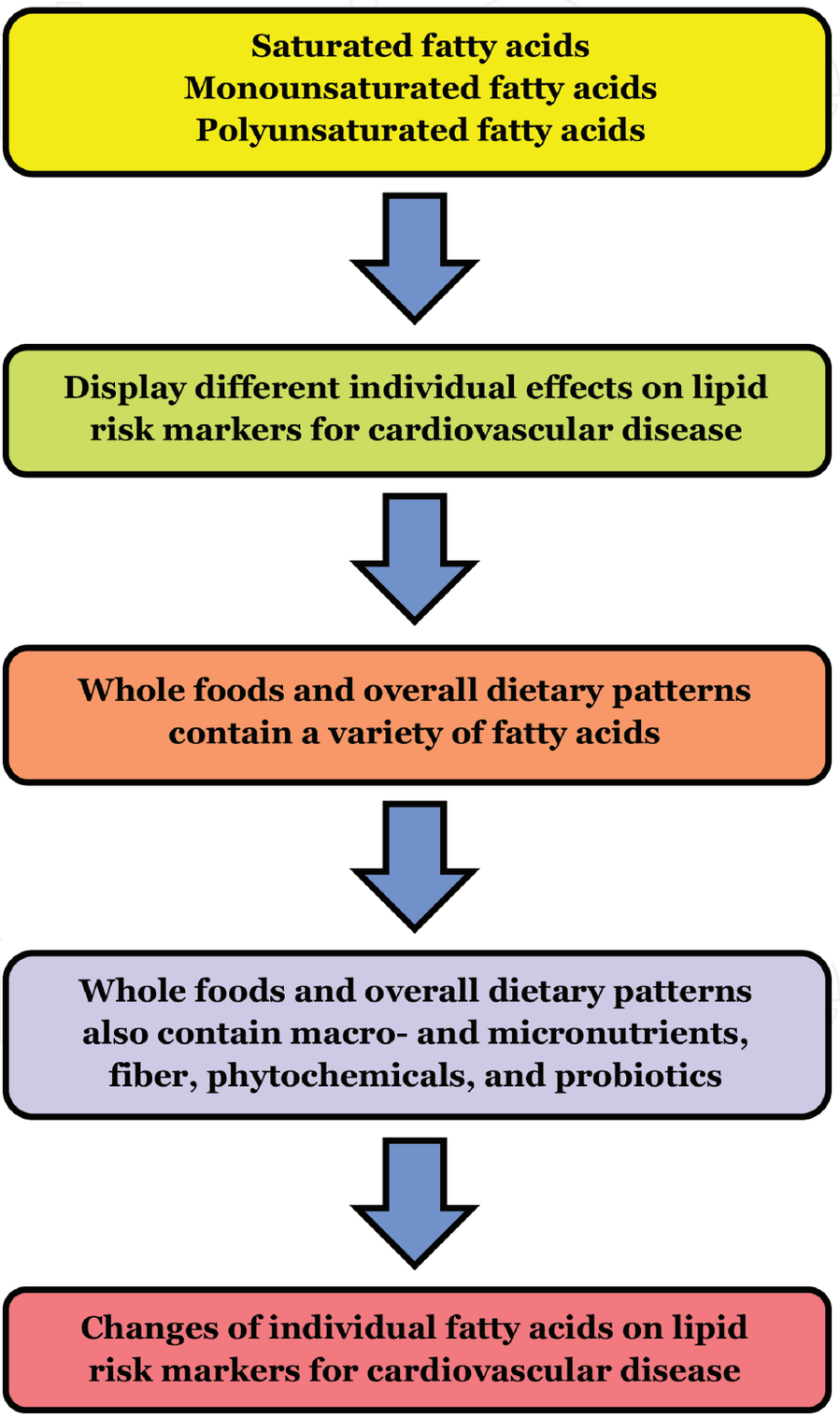

Figure 2.
The progression from individual fatty acids to whole foods and overall dietary patterns on lipid risk markers for cardiovascular disease.

results or no significant differences for selected CVD lipid risk markers – particularly when comparing linoleic acid to oleic acid. Therefore, additional research is needed regarding the effects of fatty acids on markers that increase the risk for CVD – in addition to the associated mechanisms.

The development of CVD is a complex process which involves many factors that influence the discussed lipid risk markers, such as exercise patterns, overweight/obesity, cigarette smoke, hypertension, high alcohol consumption, and genetics. To add to this complexity is our dietary patterns. As discussed, there are mixed results regarding the consumption of linoleic acid on CVD lipid risk markers. One such dietary explanation is the complex food matrices of these items, which may, therefore, influence CVD risk markers. In other words, we do not consume individual fatty acids; we consume food. For example, individual saturated and unsaturated fatty acids have differing effects on CVD risk markers; however, these individual effects may be diminished when these fatty acids are components of whole food items. This attribute may explain, in part, for the differing outcomes of saturated and unsaturated fat on CVD risk, events, and/or mortality. Perhaps, therefore, we should focus on whole foods and overall dietary patterns when providing guidelines to reduce the risk for CVD.

It is recommended that future studies investigate the effects of various dietary patterns on CVD risk markers, such as lower-carbohydrate versus higher-carbohydrate diets, lower-fat versus higher-fat diets, and plant-based versus meat-based diets. Based on the heterogeneity of the reviewed studies on the effects of linoleic acid consumption on lipid risk markers for CVD, future studies should be longer in duration – with more participants. Moreover, it should be clarified, in future publications, whether the discussed CVD lipid risk markers exist as strong and independent risk factors for CVD.

It is clear that the consumption of fat is a critical component to a healthy diet; consuming too much or too little can have detrimental effects on one's health. Therefore, moderation is an important factor to keep in mind regarding fat consumption. It seems, however, that certain dietary recommendations focus on decreasing the intakes of saturated fatty acids, and increasing the consumption of monounsaturated and polyunsaturated fatty acids. These recommendations may not be optimal in the following ways: 1) foods consist of individual fatty acids, which have different effects on CVD lipid risk markers; 2) overall dietary patterns and food components may offset the effects of specific fatty acids; and 3) individuals may not be familiar with significant food sources of saturated, monounsaturated, and polyunsaturated fatty acids. Therefore, it seems that dietary guidelines to lower the risk for CVD should focus on overall dietary patterns, rather than individual fatty acids (**Figure 2**).

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Conflict of interest

The author declares no conflict of interest.

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