Monounsaturated fatty acid intake and lipid metabolism

Ingestão de ácidos graxos monoinsaturados e metabolismo lipídico

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Abstract

The objective of this review is to present and discuss the most recent findings related to the effects of monounsaturated fatty acids (MUFA) on plasma markers of lipid metabolism observed in postprandial studies and clinical nutritional intervention studies. Searches were conducted on several different databases for publications from 2010 to 2014 using the following keywords: MUFA, Lipemia, Lipid Metabolism, Triglycerides and Postprandial. High-MUFA meal has presented beneficial effect on postprandial lipidemia response, but it is not yet completely clear whether this response to MUFA intake may be different in people with excess weight and/or other chronic diseases. In general, cardiovascular risk factors were reduced and lipid profiles improved after interventions with MUFA. In conclusion, recent studies have demonstrated that consuming MUFA has beneficial effects at short and long time by increasing/maintaining HDL cholesterol concentrations and reducing levels of LDL cholesterol.

Keywords: monounsaturated fatty acids; triacylglycerol; oleic acid; cardiovascular diseases.

Resumo

Esta revisão teve como objetivo apresentar e discutir os achados mais recentes do efeito dos ácidos graxos monoinsaturados (AGMI) sobre marcadores plasmáticos do metabolismo lipídico em estudos pós-prandiais e de intervenção clínica nutricional. Realizou-se busca em diferentes bases de dados entre 2010 e 2014, usando os seguintes termos de indexação: *MUFA*, *Lipemia*, *Lipid Metabolism*, *Triglycerides* e *Postprandial*. O consumo de refeição com alto conteúdo de AGMI tem demonstrado efeito benéfico na resposta lipidêmica pós-prandial, mas se essa resposta pode ser alterada em indivíduos com excesso de peso e/ou outras doenças crônicas após consumo de AGMI, ainda não está totalmente elucidado. De modo geral, após a intervenção com AGMI, os fatores de risco cardiovascular diminuíram, além de haver melhora no perfil lipídico. Em conclusão, os estudos recentes têm demonstrado um efeito benéfico do consumo de AGMI em curto e longo prazos, mediante aumento/manutenção das concentrações de HDL colesterol e diminuição do LDL colesterol.

Palavras-chave: ácidos graxos monoinsaturados; triacilgliceróis; ácido oleico; doenças cardiovasculares.

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INTRODUCTION

Cardiovascular diseases (CVD) are notorious and are the subject of much discussion in clinical practice. However, a great deal of what is known about these diseases and their risk factors is based on the results of assaying markers of lipid metabolism in a fasting state. While these tests are undoubtedly important, we spend the majority of our lives in a non-fasting state and, as a result, there is constant variation in lipemia levels.[1]

In turn, postprandial metabolism has been linked with increases in inflammation and oxidation, interfering in vascular endothelial function and impacting on CVD risk.[2] Despite this, the postprandial response to lipid overload has not yet been well established and published findings remain controversial.

In this context, dietary lipids are important factors in modulation of postprandial lipemia, which is a possible early marker of metabolic abnormalities that are not observed in a fasting state.[2] When we eat a meal containing excessive quantities of lipids, the body is faced with an excess of triacylglycerols (TAG) and their removal becomes inefficient, resulting in a state of postprandial lipemia,[3] which is associated with CVD.[4]

One of the subgroups of fatty acids of greatest interest is the monounsaturated fatty acids (MUFA), of which oleic acid (OA) is the principal member. These fatty acids are found in olive oil, in canola oil, in olives, avocadoes and in oleaginous plants.[5] However, OA contained in olive oil (from 55 to 85%) can account for from 60 to 80% of the entire daily dietary intake of OA.[6]

The objective of this review is to present and discuss the most recent findings related to the effects of monounsaturated fatty acids (MUFA) on plasma markers of lipid metabolism observed in postprandial studies and clinical nutritional intervention studies.

METHODOLOGY

Searches were run on the MEDLINE/PubMed, SciELO and Web of Science databases for work published in Portuguese, English or Spanish from 2010 to 2014. The following keywords were used to identify articles of interest: MUFA, Lipemia, Lipid Metabolism, Triglycerides and Postprandial, plus combinations of these terms and expressions containing them. Searches were run using these keywords with the Boolean connectors AND, OR and NOT. The titles and abstracts of the studies identified by the electronic search were then selected or rejected according to the following inclusion criteria: MUFA intake during the study period, intervention studies or postprandial, studies with adult humans that assessed changes in the plasma lipid profile after consumption of MUFA.

Articles describing animal models and in vitro studies were excluded, as were any that did not assess the effects of MUFA intake on lipid metabolism after dietary intervention. Additionally, editorials, articles lacking sufficient data, summaries of presentations to meetings and studies that did not consider the association between consumption of a source of MUFA and markers of lipid metabolism were also excluded.

The full texts of potentially relevant articles were read in order to verify that they met the inclusion criteria. Additionally, articles from other sources were also included in the review with the objective of contextualizing and justifying the subject under discussion and to enrich that discussion.

RESULTS AND DISCUSSION

The search and selection process (Figure 1) resulted in 21 articles, which are described in detail in Tables 1 and 2.

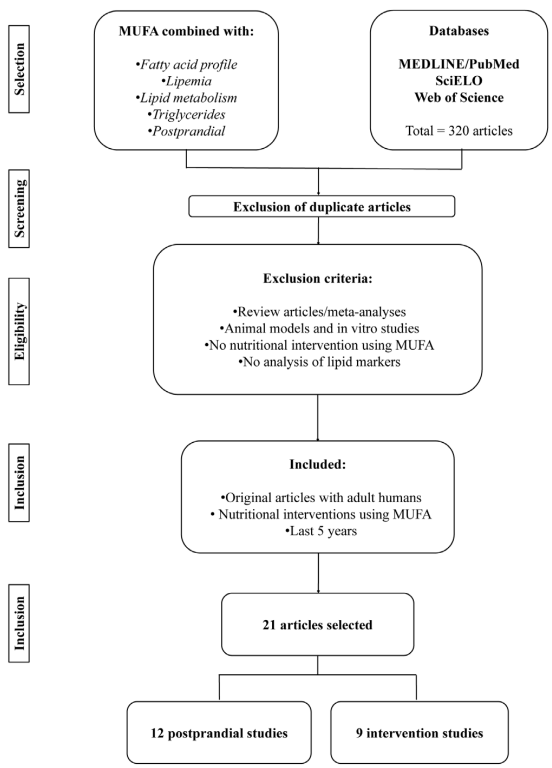

Figure 1. Flow diagram illustrating search and selection of articles. MUFA: monounsaturated fatty acids.

Table 1. Acute effects of monounsaturated fatty acid intake on markers of lipid metabolism.

Authors	Type of study	Sample	Duration of intervention	Test groups	Quantity of MUFA ingested	Main results (MUFA)
Jiménez-Gómez et al.[7]	Randomized	130 people with MS BMI = NR Age = NR	8 hours	Group 1: 38% SFA Group 2: 43% MUFA Group 3: low fat diet (28%) + omega 3 Group 4: low fat diet (28%)	43% of total lipids	accentuated ↑ in TAG, returning to baseline in a shorter time than other groups
Hartwich et al.[8]	Randomized	164 people with MS criteria BMI = 20 to 40 kg/m^2 Age = 35 to 70 years	8 hours	Group1: 16% SFA Group2: 20% MUFA Group 3: low fat diet + omega 3 Group 4: low fat diet	20% of TCV of the meal	accentuated ↑ in TAG, returning to baseline in a shorter time than other groups
Bouwens et al.[9]	Randomized Single-blind Crossover	21 healthy men BMI = 18 to 27 kg/m^2 Age 19 to 27 years	6 hours	Group1: 70% SFA Group 2: 80% MUFA Group 3: 65% PUFA	55 g (80% of total lipids)	Greater reduction in cholesterol after 6h compared to other groups
Perez-Martinez et al.[1]	Randomized Crossover	20 healthy men BMI = 24.5±2.7 kg/m^2 Age = 22±1.8 years	11 hours	Group 1: 35% SFA Group 2: 36% MUFA Group 3: 55% CHO+ 8% PUFA	36% of total lipids	↓ in total number and ↑ in size of TRL ↓ cardiovascular risk compared to other groups
Lopez et al.[10]	Crossover	14 hypertriglyceridemic men BMI = 24.2±5.1 kg/m^2 Age = 33±7 years	8 hours	Group1: 10 kcal of MUFA per kg of body weight Group2: 10 kcal of SFA per kg of body weight Group 3: Control (without lipids)	10 kcal/kg weight	↑ TAG, ↑ NEFA, ↑ insulin (Groups 1 and 2)
Teng et al.[11]	Randomized Single-blind Crossover	10 healthy men BMI = 21±1.6 kg/m^2 Age = 21.9±0.7 years	4 hours	Group 1: 50 g of MUFA Group 2: 50 g of SFA Group 3: 50 g of PUFA	50 g	↑ in TAG was greatest in Group 1, followed by Group 2
Lozano et al.[12]	Randomized Crossover	21 healthy men BMI > 26.18 kg/m^2 and BMI < 26.18 kg/m^2 Age = 23±1.5 years	11 hours	Group 1: 38% MUFA Group 2: 35% SFA Group 3: 20% SFA, 24% MUFA and 16% PUFA	38% of TCV of the meal	↓ TRL-TAG compared to other groups
Van Dijk et al.[13]	Randomized Crossover	42 men (healthy weight, obese and obese with type 2 diabetes) BMI = NR Age = 50 to 70 years	4 hours	Group 1: 51 g of SFA Group 2: 79 g of MUFA Group 3: 38 g of PUFA	79 g	↓ Free fatty acids over time (MUFA > SFA > PUFA)
Lozano et al.[2]	Randomized Crossover	21 men BMI = NR Age = 23±1.5 years	11 hours	Group 1: 38% MUFA Group 2: 35% SFA Group 3: 20% SFA, 24% MUFA and 16% PUFA	38% of TCV of the meal	No differences between groups
Raz et al.[14]	Crossover	54 individuals BMI = 25±0.9 kg/m^2 Age = 41.7±3.1 years	4 hours	Group 1: 51 g of MUFA Group 2: 51 g of SFA	51 g	↑ in TAG smaller after MUFA
Pietraszek et al.[15]	NR	17 healthy people 17 people with DM2 BMI= NR Age = NR	4 hours	Group 1: free from DM2, MUFA intake Group 2: with DM2, MUFA intake	67 g	Healthy people responded better to treatment
Cabello-Moruno et al.[16]	Randomized Crossover	10 healthy men BMI = 23.7±2 kg/m^2 Age = 26±4.3 years	6 hours	Group 1: 70 g pomace olive oil (MUFA) Group 2: 70 g refined olive oil (MUFA)	70 g	No differences in TAG between groups Larger TRL Particles in the pomace group

MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids; SFA: saturated fatty acids; DM2: type 2 Diabetes mellitus; TAG: triacylglycerols; NEFA: non-esterified fatty acids; TRL: triacylglycerol-rich lipoproteins; CHO: carbohydrates; iAUC: incremental area under the curve; ↑: increase/high quantity; ↓: decrease/low quantity; MS: metabolic syndrome; NR: not reported.

Table 2. Effect of dietary intervention containing monounsaturated fatty acids on markers of lipid metabolism.

Authors	Study type	Sample	Duration of intervention	Test groups	Quantity of MUFA ingested	Main results
Adams et al.[17]	Crossover	10 hypercholesterolemic men BMI = 26.8±1.1 kg/m^2 Age = 49.3±8.6 years	5 weeks 3-week washout	Group 1: 16.7 g of SFA Group 2: 20.2 g of MUFA	20.2 g	↓calorie intake, ↑HDL-C, ↑stearic, oleic and linoleic acids Positive correlation between TAG and VLDL-C, palmitic, palmitoleic and oleic acids Negative correlation between HDL-C and palmitic and palmitoleic acids; and between LDL-C diameter and palmitic, stearic and oleic acids, ↑ MUFA and ↓ palmitic acid
Gillingham et al.[18]	Randomized Controlled Crossover Single-blind	36 hypercholesterolemic people BMI =22 to 36 kg/m^2 Age = 18 to 65 years	28 days 4 to 8 weeks' washout	Group 1: 70% PUFA Group 2: 70% MUFA Group 3: Western diet (35% lipids)	70% of total lipids	↑ MUFA and ↓ SFA totals in blood ↓total cholesterol and LDL-C
Gilmore et al.[19]	Randomized Crossover	27 men with normal lipid profiles; BMI = NR Age 23 to 60 years	5 weeks 4-week washout	Group 1: 32±3 g/day MUFA Group 2: 31±4 g/day MUFA	32±3 g/day or 31±4 g/day	↑HDL-C and ↓ LDL: HDL-C ratio positively correlated with insulin
AlSaleh et al.[20]	Parallel design	367 men and women BMI = NR Age = 30 to 70 years	4 weeks 3 nutritional interventions	Group 1: 18% SFA Group 2: 20% MUFA Group 3: low fat diet (28% lipids)	18% of daily TCV	↓phospholipids, ↓ APO B ↓ Total cholesterol and ↓ LDL-C
Baxheinrich et al.[21]	Parallel design	81 people with MS BMI = NR Age = NR	26 weeks	Group 1: 30 mL of oil + 20g of margarine (PUFA) Group 2: 30 mL of oil + 20g of margarine (MUFA)	30 mL of oil + 20 g of margarine	↓ weight, total cholesterol, LDL-C and insulin ↓ MS indices
Bozzetto et al.[22]	Controlled	12 people with DM2 BMI = 28±1 kg/m^2 Age = 59±4 years	4 weeks	Group 1: 23% MUFA Group 2; 52% CHO with low GI	23% of daily TCV	↓TRL (CHO/Low GI > MUFA)
Phillips et al.[23]	Randomized Cohort study	486 men and women with MS BMI = 20 to 40 kg/m^2 Age = 35 to 70 years	12 weeks	Group 1: 12% SFA Group 2: 20% MUFA Group 3: ↑ CHO + MUFA Group 4: ↑ CHO + ω -3	20% of daily TCV	No changes in lipid profile in healthy weight or obese groups
Nishi et al.[24]	Randomized Crossover	27 people (LDL-C elevated/high) BMI = 25.7±3 kg/m^2 Age = 64±9 years	4 weeks	Group 1: 7.6% MUFA Group 2: PUFA (% NR) Group 3: Control (MUFA + PUFA - % NR)	7.6% of daily TCV	↑ oleic acid, ↑ MUFA and ↓ palmitic acid in MUFA and control groups Positive correlations between palmitic acid concentrations and TAG and cholesterol and negative correlation with HDL-C

MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids; SFA: saturated fatty acids; ω-3: omega 3 fatty acid; TAG: triacylglycerols; NEFA: non-esterified fatty acids; TRL: triacylglycerol-rich lipoproteins; CHO: carbohydrates; GI: glycemic index; ↑: increase/high quantity; ↓: decrease/low quantity; MS: metabolic syndrome; NR: not reported.

Table 2. Continued...

Authors	Study type	Sample	Duration of intervention	Test groups	Quantity of MUFA ingested	Main results
Bozzetto et al.[25]	Randomized Parallel	45 people with DM2 Overweight/obese BMI = NR Age = NR	8 weeks	Group 1: low GI CHO and fiber (with and without exercise) Group 2: 27±1% MUFA (with and without exercise)	27±1% of daily TCV	No changes to concentrations of total cholesterol and increased TAG in MUFA group compared with CHO group

MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids; SFA: saturated fatty acids; ω-3: omega 3 fatty acid; TAG: triacylglycerols; NEFA: non-esterified fatty acids; TRL: triacylglycerol-rich lipoproteins; CHO: carbohydrates; GI: glycemic index; ↑: increase/high quantity; ↓: decrease/low quantity; MS: metabolic syndrome; NR: not reported.

Markers of lipid metabolism

When discussing CVD, it is impossible not to consider their primary prevention, the traditional risk factors associated with them (systemic arterial hypertension, diabetes mellitus and dyslipidemia, among others) and the markers of risk and for diagnosing these events, the most often employed of which are total cholesterol (TC) and fractions, HDL cholesterol (HDL-C) and LDL cholesterol (LDL-C), TAG and arterial blood pressure.[26,27]

However, attempts have been made to identify new markers, with the objective of improving early diagnosis rates and treatment of cardiovascular events. The most interesting candidates are apolipoproteins, analysis of the size and diameter of lipoprotein particles and of the quantities of TAG present in these particles and free fatty acids in plasma.

On this basis lipoproteins can be divided into two groups: (1) those that are rich in TAG and are larger and less dense, including chylomicrons, which originate in the intestines, and very low density lipoproteins (VLDL-C) from the liver; and (2) those that are rich in cholesterol, including low density cholesterol (LDL-C) and high density cholesterol (HDL-C).[27]

Triglyceride-rich lipoproteins (TRL) are made up of proteins and lipids and their composition changes dynamically in response to a person's diet and lipid metabolism. This composition, in turn, determines the length of time they remain in circulation and their rate of absorption and transport through the endothelium, playing an important role in atherogenesis.[28] The size and number of TRL particles may be better predictors of atherosclerosis than simply assaying TAG,[1] since TAG are measured in a fasting state and atherosclerosis may be a postprandial phenomenon in which TRL play a dominant role.[29]

Triglyceride-rich lipoproteins consist of chylomicrons, originating from the small intestine and containing apo B-48 as a structural protein, and VLDL, which originate in the liver and contain apo B-100, as a structural protein.[16] Additionally, there is evidence that postprandial TRL elevation can lead to pancreatic beta cell dysfunction, suggesting that the plasma lipoprotein abnormalities observed in patients with obesity-linked metabolic syndrome (MS) may not be merely a consequence of the disease, but also a cause of it.[12] Triglyceride-rich lipoproteins can cross the endothelial barrier, enter the wall of the vessel and facilitate build-up of lipids in macrophages, forming foam cells.[16]

It is against this background that scientific interest in markers of postprandial lipemia has been increasing, since there could be other metabolites of lipid metabolism that could be identified more quickly after dietary intake, which could facilitate diagnosis of certain diseases or the risk of cardiovascular events. According to the studies identified, in general the concentration of the classic markers of CVD, such as TC and its fractions, do not change after dietary intake and so they may not be good markers for assessing acute effects.[1,7-9,12] In contrast, TAG concentrations have been shown to change after meals and may provide more plausible evidence as predictors of risk of CVD,[7-12] and the same is true of TRL.[1,12,16]

Acute effects of MUFA intake on lipid metabolism: postprandial studies

This review identified 12 studies published in the last 5 years that evaluated postprandial changes in the lipid profile after subjects had eaten a meal containing MUFA. Researchers investigated postprandial responses in men of healthy weight,[1,2,9,11,12,14,16,30] and in obese or healthy-weight people with type 2 diabetes mellitus (DM2),[15,31] hyperlipoproteinemia[10] or MS.[7,8] The body mass index (BMI) of participants varied from 20 to 40 kg/m^2 and their ages ranged from 19 to 70 years. The test foods used as sources of MUFA included drinks and shakes, muffins and meals with

the lipid under investigation added (sources: olive oil, sunflower oil and/or macadamia oil) and with MUFA contents varying from 20 to 80% of the total lipids in the meals given to the participants.

The duration of the postprandial lipemia cycle in healthy adults is from 6 to 8h,[32] contrasting with the results of the articles reviewed, in which the duration of the cycle ranged from 4 to 11h. The markers most often used to evaluate postprandial lipidemic responses were concentrations of TAG[1,2,7-12,14-16,31] and concentrations and size of TRL.[1,2,7,8,10,12,16,30]

The majority of these studies demonstrated that postprandial responses to meals with a high content of saturated fatty acids (SFA), MUFA or polyunsaturated fatty acids (PUFA) diverge from one another. Four studies observed a greater increase in TAG concentrations after consumption of a meal with a high MUFA content than after intake of sources of SFA or PUFA.[8,11,13] One explanation for this effect could be that the TRL particles supplied by the MUFA-rich meal could have a greater affinity for the hepatic receptor involved in metabolism, inducing more rapid and effective clearance of these TRL than of other types of lipids.[12] Concentrations of TAG could exhibit the same behavior when postprandial increase is balanced by more efficient clearance. Indeed, Jiménez-Gómez et al.[7] and Hartwich et al.[8] assessed people with MS and with symptoms of MS, respectively, finding that those who ate a meal containing MUFA (43% and 20% of the total lipid content of the meal, respectively), exhibited a faster rise in TAG concentrations (peaks at 2h and 4h after the meal, respectively) compared with those who were given other sources of lipids; although the return to baseline levels was more efficient (at around 8h after the meal).[7,8]

However, other studies with healthy people that determined the concentrations of TAG found similar postprandial results for different types of fats (MUFA, PUFA and SFA).[2,14,16] These differences in the results could be because of different quantities or sources of MUFA, the number of hours after the meal at which concentrations were tested, age, number of people tested and prior nutritional status of participants (normal weight versus overweight), or even due to metabolism of MUFA compared with other lipid sources. Considering that the sources of lipids eaten do not only contain SFA, MUFA and PUFA, but also a wide variety of other nutrients, such as carbohydrates, fiber, protein, and other compounds with biological activity, such as polyphenols and sterols, the postprandial results could also have been influenced by the presence of these nutrients.[2]

There is still no certainty about the postprandial lipemic responses to consumption of different lipid sources in people with excess weight and/or other chronic diseases. Notwithstanding, the effects of eating a meal with a high MUFA content have been shown to be beneficial for these people's postprandial lipidemic response. For example, overweight people who ate a meal containing 1g of olive oil per kg of body weight had lower TRL concentrations than after eating a meal rich in butter (SFA) or nuts (PUFA), whereas in a subset of people with healthy weights there was no difference between different lipid sources.[12]

As such, the results reported indicate that MUFA have beneficial effects on postprandial lipid metabolism, and this could be an important mechanism of this FA's cardioprotective action. Indeed, these findings suggest that the concentrations and size of postprandial TRL could be promising biomarkers of lipid metabolism as predictors of metabolic disorders and cardiovascular risk. However, recommendations for their use in clinical practice should still be cautious, since the results are not conclusive in terms of the dosage needed to obtain a response.

The long-term effects of MUFA intake on lipid metabolism: intervention studies

A total of nine studies were selected that evaluated markers of lipid metabolism as part of the follow-up of nutritional interventions based on diets with high MUFA content, varying from 7.6% to 28% of the total calorie value (TCV), or from 20.2g to 32g of MUFA. The duration of interventions varied from 4 to 26 weeks. The volunteers were men and women, the majority with excess weight, or DM2 or abnormalities of markers of lipid metabolism (abnormal TAG, TC and/or LDL-C). The age of participants ranged from 18 to 70 years. The markers most often assessed were TAG, TC and fractions, in addition to total lipids. Diets were calculated with dietary fat either supplemented or substituted by MUFA. The test foods most often used as sources of MUFA were muffins made with olive oil, nuts, fortified meats and extra virgin olive oil itself.

The studies found reductions in TC,[18,20,21,24] LDL-C,[17,18,20,21] and TAG[17,21] and increases in HDL-C.[17,19] In general, cardiovascular risk factors (TC, LDL-C and TAG) reduced after intervention with MUFA and there were improvements in the lipid profile, compared with the other diets tested. These results appear to be related to the FA profile of the bloodstream.

For example, Gilmore et al.[19] observed that a nutritional intervention with a high MUFA content lasting 5 weeks increased the concentration of HDL-C

and reduced the LDL-C:HDL-C ratio.[19] Plasma TAG concentrations were positively correlated with plasma insulin concentration and negatively correlated with HDL-C and stearic acid levels. These findings suggest that activity of hepatic Stearoyl-CoA Desaturase-1 (SCD1) can regulate plasma TAG concentrations.[19]

The proportion of palmitic acid (PA) was also positively associated with plasma concentrations of TAG and the TC:HDL-C ratio and inversely associated with HDL-C concentrations among hyperlipidemic individuals (high LDL-C plasma concentrations).[24] In turn, the 10-year risk of CVD was inversely associated with OA proportions. Saturated fatty acid proportions were negatively associated with HDL-C concentrations and positively associated with 10-year risk of CVD.[24]

Adams et al.[17] found that palmitoleic acid (PTA) was the fatty acid with the strongest correlation with changes in TAG, VLDL-C and HDL-C, followed by PA. The highest plasma PTA concentrations were observed at the end of the cycle after consumption of SFA and the lowest concentration was observed after consumption of MUFA. These results suggest that the high concentration of PTA after SFA intake is the result of increased stimulation of hepatic SCD1 activity, in contrast with MUFA intake.[17]

It is interesting that it was not only the concentrations of markers of lipid metabolism that were changed, since the size of particles was also altered. The diameters of LDL-C particles were reduced by a dietary intervention with a high SFA content and remained altered, even after a 3-week washout period and also after consumption of a diet rich in MUFA.[17]

In a similar manner, plasma PA was increased by a high SFA diet and remained elevated thereafter. Changes in LDL-C particle diameter are specific metabolic abnormalities that increase the atherogenicity of LDL-C. These small LDL-C particles are a risk factor for CVD, since they are more susceptible to oxidative damage and they also provoke vascular inflammation. Presence of PA in elevated concentrations may also be related to small LDL-C diameter that is maintained over time and a negative correlation has been detected between PA and the diameter of LDL-C particles.[17]

However, four studies did not detect effects on markers,[19,22,23,25] although these results may be linked with the fact that the studies did not compare a diet rich in MUFA with other sources of lipids[25] or may be due to differences between the study participants (age, sex, BMI, baseline values of markers of lipid metabolism).

However, the reductions in these indicators of cardiovascular risk factors and the components of MS observed in these studies may not be exclusively the result of different dietary sources of lipids, but may also be associated with the reductions in weight observed in some studies,[17,21] which can be considered a confounding factor, since weight loss itself improves the lipid profile.

A considerable proportion of the interest in the role of MUFA in prevention of CVD is because of the beneficial effects of the Mediterranean diet that have been observed. This diet has a high olive oil content (14-40% of daily energy intake) and, consequently, is rich in MUFA.[24] The protective effect of regular OA intake on parameters related to CVD is primarily related to the Mediterranean region, where the population's diet includes a high MUFA intake because of greater olive oil consumption. The reduction in cardiovascular risks may be linked to an improvement in the profile of dietary lipoproteins (increased HDL-C and reduced LDL-C), in addition to improved endothelial function due to an increase in vasodilation-related flow and reduced inflammation and oxidative stress.[33]

On the other hand, it is important to investigate the possible positive effects of MUFA in people with DM2, since DM2 is itself an independent risk factor for CVD. The search for strategies to manage postprandial dyslipidemia is therefore an issue of clinical relevance and nutritional changes achieved by adhering to a dietary plan are capable of influencing postprandial lipid response in patients at cardiometabolic risk.

The intervention studies indicate that there are beneficial effects on markers do of lipid metabolism from habitual consumption of MUFA (12-28% of total calorie value) contained in foods such as olive oil and nuts, in comparison with low fat diets or consumption of other sources of lipids. In fact, the intake levels that have shown positive results are higher than the level recommended for cardiovascular health, which is around 15% MUFA as a proportion of TCV.[26] The mechanisms through which MUFA act appear to be related to their effect on the concentrations and sizes of lipoprotein particles and, consequently, on their metabolism at the cellular level.

CONCLUSIONS

As shown by the findings discussed in this review, MUFA intake in the form of habitual consumption of olive oil and nuts is supported by the most up-to-date scientific literature. These results show that the effects of MUFA can be beneficial over the short term

(postprandial lipemia), primarily in relation to TAG metabolism, and over the long term in association with improvements in the plasma lipid profile, whether in terms of the concentrations or of the sizes of HDL-C and LDL-C particles, which are well-known as CVD protection and risk factors, respectively. These studies also emphasize the importance of determining the concentrations of fatty acids in plasma and the size of lipoproteins as biomarkers of lipid metabolism and the relevance of postprandial testing to increase knowledge of the mechanisms involved.

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REFERENCES

1. Perez-Martinez P, Ordovas JM, Garcia-Rios A, et al. Consumption of diets with different type of fat influences triacylglycerols-rich lipoproteins particle number and size during the postprandial state. Nutr Metab Cardiovasc Dis. 2011;21(1):39-45. http://dx.doi.org/10.1016/j.numecd.2009.07.008. PMid:19819118.
2. Lozano A, Perez-Martinez P, Marin C, et al. An acute intake of a walnut-enriched meal improves postprandial adiponectin response in healthy young adults. Nutr Res. 2013;33(12):1012-8. http://dx.doi.org/10.1016/j.nutres.2013.08.010. PMid:24267040.
3. Chan DC, Pang J, Romic G, Watts GF. Postprandial hypertriglyceridemia and cardiovascular disease: current and future therapies. Curr Atheroscler Rep. 2013;15(3):309. http://dx.doi.org/10.1007/s11883-013-0309-9. PMid:23345190.
4. Katsanos CS. Clinical considerations and mechanistic determinants of postprandial lipemia in older adults. Adv Nutr. 2014;5(3):226-34. http://dx.doi.org/10.3945/an.113.004903. PMid:24829469.
5. Bressan J, Hermsdorff HH, Zulet MA, Martínez JA. Hormonal and inflammatory impact of different dietetic composition: emphasis on dietary patterns and specific dietary factors. Arq Bras Endocrinol Metabol. 2009;53(5):572-81. http://dx.doi.org/10.1590/S0004-27302009000500010. PMid:19768248.
6. Oi-Kano Y, Kawada T, Watanabe T, et al. Extra virgin olive oil increases uncoupling protein 1 content in brown adipose tissue and enhances noradrenaline and adrenaline secretions in rats. J Nutr Biochem. 2007;18(10):685-92. http://dx.doi.org/10.1016/j.jnutbio.2006.11.009. PMid:17418557.
7. Jiménez-Gómez Y, Marín C, Peérez-Martínez P, et al. A low-fat, high-complex carbohydrate diet supplemented with long-chain (n-3) fatty acids alters the postprandial lipoprotein profile in patients with metabolic syndrome. J Nutr. 2010;140(9):1595-601. http://dx.doi.org/10.3945/jn.109.120816. PMid:20631323.
8. Hartwich J, Leszczynska-Golabek I, Kiec-Wilk B, et al. Lipoprotein profile, plasma ischemia modified albumin and LDL density change in the course of postprandial lipemia. Insights from the LIPGENE study. Scand J Clin Lab Invest. 2010;70(3):201-8. http://dx.doi.org/10.3109/00365511003663630. PMid:20233037.
9. Bouwens M, Grootte Bromhaar M, Jansen J, Müller M, Afman LA. Postprandial dietary lipid-specific effects on human peripheral blood mononuclear cell gene expression profiles. Am J Clin Nutr. 2010;91(1):208-17. http://dx.doi.org/10.3945/ajcn.2009.28586. PMid:19923369.
10. Lopez S, Bermudez B, Ortega A, et al. Effects of meals rich in either monounsaturated or saturated fat on lipid concentrations and on insulin secretion and action in subjects with high fasting triglyceride concentrations. Am J Clin Nutr. 2011;93(3):494-9. http://dx.doi.org/10.3945/ajcn.110.003251. PMid:21209225.
11. Teng KT, Nagapan G, Cheng HM, Nesaretnam K. Palm olein and olive oil cause a higher increase in postprandial lipemia compared with lard but had no effect on plasma glucose, insulin and adipocytokines. Lipids. 2011;46(4):381-8. http://dx.doi.org/10.1007/s11745-010-3516-y. PMid:21197586.
12. Lozano A, Perez-Martinez P, Delgado-Lista J, et al. Body mass interacts with fat quality to determine the postprandial lipoprotein response in healthy young adults. Nutr Metab Cardiovasc Dis. 2012;22(4):355-61. http://dx.doi.org/10.1016/j.numecd.2010.07.013. PMid:21093231.
13. Van Dijk S, Feskens E, Bos MB, et al. Consumption of a high monounsaturated fat diet reduces oxidative phosphorylation gene expression in peripheral blood mononuclear cells of abdominally overweight. J Nutr. 2012;142(7):1219-25. http://dx.doi.org/10.3945/jn.111.155283. PMid:22623392.
14. Raz O, Steinvil A, Berliner S, Rosenzweig T, Justo D, Shapira I. The effect of two iso-caloric meals containing equal amounts of fats with a different fat composition on the inflammatory and metabolic markers in apparently healthy volunteers. J Inflamm. 2013;10(1):3. http://dx.doi.org/10.1186/1476-9255-10-3. PMid:23369030.
15. Pietraszek A, Gregersen S, Pedersen SB, Holst JJ, Hermansen K. Acute effects of monounsaturated fat on postprandial lipemia and gene expression in first-degree relatives of subjects with type 2 diabetes. Eur J Clin Nutr. 2014;68(9):1022-8. http://dx.doi.org/10.1038/ejcn.2014.64. PMid:24824014.
16. Cabello-Moruno R, Martinez-Force E, Montero E, Perona JS. Minor components of olive oil facilitate the triglyceride clearance from postprandial lipoproteins in a polarity-dependent manner in healthy men. Nutr Res. 2014;34(1):40-7. http://dx.doi.org/10.1016/j.nutres.2013.10.003. PMid:24418245.
17. Adams TH, Walzem RL, Smith DR, Tseng S, Smith SB. Hamburger high in total, saturated and trans-fatty acids decreases HDL cholesterol and LDL particle diameter, and increases TAG, in mildly hypercholesterolaemic men. Br J Nutr. 2010;103(1):91-8. http://dx.doi.org/10.1017/S0007114509991516. PMid:19674491.
18. Gillingham LG, Gustafson JA, Han S-Y, Jassal DS, Jones PJH. High-oleic rapeseed (canola) and flaxseed oils modulate serum lipids and inflammatory biomarkers in hypercholesterolaemic subjects. Br J Nutr. 2011;105(3):417-27. http://dx.doi.org/10.1017/S0007114510003697. PMid:20875216.
19. Gilmore LA, Walzem RL, Crouse SF, et al. Consumption of high-oleic acid ground beef increases HDL-cholesterol concentration but both high- and low-oleic acid ground beef decrease HDL particle diameter in normocholesterolemic men. J Nutr. 2011;141(6):1188-94. http://dx.doi.org/10.3945/jn.110.136085. PMid:21525253.
20. AlSaleh A, O'Dell SD, Frost GS, et al. Interaction of PPARG Pro12Ala with dietary fat influences plasma lipids in subjects at cardiometabolic risk. J Lipid Res. 2011;52(12):2298-303. http://dx.doi.org/10.1194/jlr.P019281. PMid:21949049.
21. Baxheinrich A, Stratmann B, Lee-Barkey YH, Tschoepe D, Wahrburg U. Effects of a rapeseed oil-enriched hypoenergetic diet with a high content of α-linolenic acid on body weight and cardiovascular

risk profile in patients with the metabolic syndrome. Br J Nutr. 2012;108(4):682-91. http://dx.doi.org/10.1017/S0007114512002875. PMid:22894911.

22. Bozzetto L, De Natale C, Di Capua L, et al. The association of hs-CRP with fasting and postprandial plasma lipids in patients with type 2 diabetes is disrupted by dietary monounsaturated fatty acids. Acta Diabetol. 2013;50(2):273-6. http://dx.doi.org/10.1007/s00592-011-0318-5. PMid:21833777.
23. Phillips CM, Tierney AC, Perez-Martinez P, et al. Obesity and body fat classification in the metabolic syndrome: impact on cardiometabolic risk metabotype. Obesity. 2013;21(1):154-61. http://dx.doi.org/10.1002/oby.20263. PMid:23505198.
24. Nishi S, Kendall CWC, Gascoyne A, et al. Effect of almond consumption on the serum fatty acid profile : a dose – response study. Br J Nutr. 2014;112(7):1137-46. http://dx.doi.org/10.1017/S0007114514001640. PMid:25138064.
25. Bozzetto L, Annuzzi G, Costabile G, et al. A CHO/fibre diet reduces and a MUFA diet increases postprandial lipaemia in type 2 diabetes: no supplementary effects of low-volume physical training. Acta Diabetol. 2014;51(3):385-93. http://dx.doi.org/10.1007/s00592-013-0522-6. PMid:24132660.
26. Santos R, Gagliardi A, Xavier H, Magnoni C, Cassani R, Lottenberg AI. Diretriz sobre o consumo de gorduras e saúde cardiovascular. Arq Bras Cardiol. 2013;100(3):1-40. PMid:23598585.
27. Xavier HT, Izar MC, Faria JR No, et al. V Diretriz Brasileira de Dislipidemias e Prevenção da Aterosclerose. Arq Bras Cardiol. 2013;101(4, Supl 1):1-20. http://dx.doi.org/10.5935/abc.2013S010. PMid:24217493.
28. Wang YI, Bettaieb A, Sun C, et al. Triglyceride-rich lipoprotein modulates endothelial vascular cell adhesion molecule (VCAM)-1 expression via differential regulation of endoplasmic reticulum stress. PLoS One. 2013;8(10):1-13. PMid:24205197.
29. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA. 2007;298(3):299-308. http://dx.doi.org/10.1001/jama.298.3.299. PMid:17635890.
30. Varela LM, Ortega-Gomez A, Lopez S, Abia R, Muriana FJG, Bermudez B. The effects of dietary fatty acids on the postprandial triglyceride-rich lipoprotein/apoB48 receptor axis in human monocyte/macrophage cells. J Nutr Biochem. 2013;24(12):2031-9. http://dx.doi.org/10.1016/j.jnutbio.2013.07.004. PMid:24231096.
31. Van Dijk SJ, Mensink M, Esser D, Feskens EJM, Müller M, Afman LA. Responses to high-fat challenges varying in fat type in subjects with different metabolic risk phenotypes: a randomized trial. PLoS One. 2012;7(7):e41388. http://dx.doi.org/10.1371/journal.pone.0041388. PMid:22844471.
32. Petto J, Vasques LMR, Pinheiro RL, et al. Comparison of postprandial lipemia between women who are on oral contraceptive methods and those who are not. Arq Bras Cardiol. 2014;103(3):245-50. PMid:25317941.
33. Sales-Campos H, Souza PR, Peghini BC, Silva JS, Cardoso CR. An overview of the modulatory effects of oleic acid in health and disease. Mini Rev Med Chem. 2013;13(2):201-10. PMid:23278117.

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