Available online at www.sciencedirect.com
ScienceDirect
Journal of Nutritional Biochemistry 19 (2008) 200–205
Journal of Nutritional Biochemistry

A meal rich in oleic acid beneficially modulates postprandial sICAM-1 and sVCAM-1 in normotensive and hypertensive hypertriglyceridemic subjects☆

Yolanda M. Pacheco[a,1], Sergio López[a,1], Beatriz Bermúdez[a], Rocío Abia[a], José Villar[b], Francisco J.G. Muriana[a,*]

[a]Cellular and Molecular Nutrition, Instituto de la Grasa (CSIC), 41012 Seville, Spain
[b]Service of Internal Medicine, Hospitales Universitarios Virgen del Rocío, 41013 Seville, Spain

Received 28 November 2006; received in revised form 16 March 2007; accepted 27 March 2007

Abstract

This study investigated whether subjects with permanent activated endothelium have altered soluble forms of intercellular adhesion molecule 1 (sICAM-1) and vascular cell adhesion molecule 1 (sVCAM-1) postprandial response to a high-fat meal and whether this phenomenon is modulated by the nature of dietary fats. Twenty-eight hypertriglyceridemic (14 normotensives and 14 hypertensives) and 14 healthy male subjects were placed in a randomized and crossover design on diets enriched in refined olive oil (ROO) or high-palmitic sunflower oil (HPSO) for a 1-week lead-in period. Thereafter, subjects ate the corresponding fat-rich meal as a breakfast and underwent sampling hourly for 8 h. Plasma triglycerides (TG), sICAM-1 and sVCAM-1 were assayed. sICAM-1 and sVCAM-1 postprandial peak levels were significantly higher and occurred later in hypertriglyceridemic subjects (all $P<.001$) compared with healthy subjects. ROO meal resulted in smaller areas under the curve for sICAM-1 and sVCAM-1 in hypertriglyceridemic (normotensive and hypertensive) and healthy subjects compared to HPSO meal. Hypertension did not aggravate the postprandial response of TG, sICAM-1 and sVCAM-1. We conclude that the challenge of a meal with ROO appears to have a significant postprandial benefit on sICAM-1 and sVCAM-1 as surrogate markers of endothelial activation and vascular inflammation in healthy and more importantly in hypertriglyceridemic (normotensive and hypertensive) subjects.

Keywords: Postprandial metabolism; Adhesion molecules; Hypertriglyceridemia; Hypertension; Olive oil; High-palmitic sunflower oil

1. Introduction

Clinical studies have consistently shown that the increase in the postprandial triglyceride (TG) response constitutes an independent cardiovascular risk factor and may predict the incidence of coronary artery disease (CAD) [1]. Recently, postprandial hypertriglyceridemia has been reported to induce endothelial activation in hypertriglyceridemic subjects [2]. This fact suggests a role for dietary TG in the injury of the artery wall during the postprandial state, which could be of particular relevance in subjects with permanent activated endothelium.

Soluble forms of intercellular adhesion molecule 1 (sICAM-1) and vascular cell adhesion molecule 1 (sVCAM-1) are found in plasma as a result of ADAM (A Disintegrin And Metalloproteinase)-mediated shedding from the surface of activated endothelial cells [3,4]. They strongly mediate the recruitment of leukocytes to the endothelium, a crucial early step in the initiation of atherosclerosis. Enhanced levels of sICAM-1 and sVCAM-1 have been found in hypertension [5,6] and fasting hypertriglyceridemia [7,8], which are counteracted after therapeutic reductions of blood pressure [6] or plasma TG [7]. Despite hypertension and fasting hypertriglyceridemia

☆ This study was supported by grants from MCYT and MEC (AGL2001-0584 and AGL2004-04958) and in part by the Spanish G03/181 Network.
* Corresponding author. Tel.: +95 4611550; fax: +95 4616790.
E-mail address: muriana@ig.csic.es (F.J.G. Muriana).
[1] To be considered as equal first authors.

0955-2863/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.jnutbio.2007.03.002

being two common determinant abnormalities that frequently coexist [9], there are, however, no data available regarding postprandial levels of sICAM-1 and sVCAM-1 as surrogate markers of endothelial activation and vascular inflammation to a high-fat meal in hypertriglyceridemic subjects with coexisting hypertension. In fact, very little is known about the postprandial events in hypertension and fasting hypertriglyceridemia [10,11], and to the best of our knowledge, the role of different dietary fats in modulating the postprandial TG response in subjects affected with hypertension and fasting hypertriglyceridemia has never been assessed. Current dietary recommendations emphasize the role of monounsaturated fats (MUFA) in replacing saturated fats (SFA) to reduce the risk of CAD [12,13]. In this study, we compared the influence of two high-fat meals [refined olive oil (ROO) and high-palmitic sunflower oil (HPSO)] that differed in the content of MUFA and SFA on postprandial levels of TG, sICAM-1 and sVCAM-1 in healthy and hypertriglyceridemic (normotensive and hypertensive) subjects.

2. Subjects and methods

This study was conducted according to the guidelines of good clinical practice: prior to the beginning of the study, all subjects provided informed consent using Human Clinic Commission- and Ethic Committee-approved protocols. The investigation conforms with the principles outlined in the Declaration of Helsinki.

2.1. Subjects

Twenty-eight hypertriglyceridemic (14 normotensives and 14 hypertensives) and 14 healthy male subjects, aged between 21 and 38 years, were recruited by advertising. They were required to have no evidence of established coronary heart disease and excluded if any evidence of renal impairment, hypothyroidism or liver dysfunction, based on clinical chemistry testing. Hypertensive and normotensive subjects with Type IIb or IV hyperlipoproteinemia and without clinical evident target organ damage had a body mass index (BMI) lower than 27 kg/m^2. Hypertensive subjects displayed mild essential hypertension according to the World Health Organization–International Society of Hypertension criteria (i.e., diastolic blood pressure 90–99 mmHg and/or systolic blood pressure 140–159 mmHg) on repeated measurements [14]. Subjects with secondary forms of hypertension were excluded. The characteristics of subjects that were enrolled for the study are depicted in Table 1.

2.2. Diet protocol and blood sampling

The design of the study was randomized, within-subject crossover and blinded. Subjects had a lead-in baseline period of 1 week on a National Cholesterol Education Program (NCEP) Step I diet. Thereafter, the subjects were instructed to follow the same diet or they were switched to an NCEP diet supplemented with ROO or HPSO for a further period of 1 week. Fasting blood samples were then taken 12 h after the evening meal. Immediately afterwards, the subjects were administered a fat-rich meal consisting of the corresponding dietary fats (ROO or HPSO, 40 g/m^2 body surface area) along with a portion of plain pasta (50 g), one slice of brown bread (28 g) and one skimmed yogurt. The range of fat consumed per subject was 76.8–79.2 g. The average total energy provided by the meals was 3700 kJ (885 kcal) with a macronutrient profile of 72% fat, 22% carbohydrate and 6% protein. ROO and HPSO had no phenolic compounds. The fatty acid composition of the meals is shown in Table 2. Subjects were asked to consume the meal in less than 15 min, after which time blood was

Table 1
Subject characteristics at entry

	Healthy subjects	Hypertriglyceridemic normotensive subjects	Hypertriglyceridemic hypertensive subjects	*P* value
Subjects	14	14	14	
Males (%)	100	100	100	
Age (years)	27±7	33±7	32±3	NS
Weight (kg)	75±6	79±8	78±5	NS
BMI (kg/m^2)	23.9±1.9	24.2±5.1	26.0±3.4	NS
Blood pressure (mmHg)				
Systolic	123±9	135±9	155±5	<.001*
Diastolic	77±9	79±11	96±5	<.001*
Triglyceride (mmol/L)	0.86±0.27	4.49±0.89	4.57±0.70	<.001†
Cholesterol (mmol/L)	4.09±0.31	6.89±1.09	6.78±0.82	<.001†
LDL-C (mmol/L)	2.34±0.85	4.19±0.70	4.23±0.51	<.001†
HDL-C (mmol/L)	1.39±0.21	0.80±0.10	0.90±0.12	<.001†
sICAM-1 (ng/ml)	601±100	986±188	957±144	<.001†
sVCAM-1 (ng/ml)	847±128	1246±232	1302±216	<.001†

* Hypertriglyceridemic hypertensive subjects vs. healthy and hypertriglyceridemic normotensive subjects.

† Hypertriglyceridemic normotensive and hypertensive subjects vs. healthy subjects.

Table 2
Fatty acid composition of the meals

Fatty acid	ROO	HPSO
	% by weight of total fatty acids	
16:0	11.7	24.9
16:1n-7	1.0	6.8
18:0	2.8	2.0
18:1n-9	79.8	58.7
18:2n-6	3.6	6.9
Others	1.1	0.6
SFA	14.9	27.2
MUFA	81.0	65.8
PUFA	4.0	7.0
MUFA/SFA	5.43	2.42

PUFA, polyunsaturated fatty acids.

collected from a cubital vein catheterized with a small-bore extension set and a Smartsite needleless valve port. A blood sample was drawn every 1 h for a total of 8 h into precooled tubes containing sodium citrate (final concentration, 0.129 mmol/L). The plasma was separated immediately by centrifugation (2000 g, 4°C, 20 min), and the aliquots were transferred into sterile cryovials of 1 ml and stored at −70°C until further analysis. The test meals were well tolerated by the subjects without any unpleasant side effects. The subjects rested and read during the postprandial period. Only water was available on request.

Each 1-week cycle of the NCEP diet in between the corresponding test fat was considered as a washout and adaptation period to eliminate any carryover effects on the next diet being tested [15]. The diets were prepared by the subjects themselves under the direction of a registered dietician and consisted of whole foods according to calculated menus and standardized recipes. For subjects to maintain a constant body weight during the study, energy intake was periodically adjusted. None of the subjects consumed tobacco or special diets or took vitamins, antioxidants or medication. They were asked to maintain their usual exercise pattern and abstain from alcohol.

2.3. Analytical methods

TG and other plasma lipids were quantified by auto-analyzer using commercially available reagents (Roche Diagnostics GmbH). Plasma levels of sICAM-1 and sVCAM-1 were determined in duplicate with commercially available immunosorbent kits (ICAM-1 and VCAM-1, Elipair, Diaclone). The intra- and interassay coefficients of variation were below 5%. All the assays were standardized according to the Standardization Program of the Spanish Society of Chemical Chemistry and the International Federation of Chemical Chemistry.

2.4. Statistical analysis

Continuous variables are expressed as mean±S.D. Differences were assessed with repeated-measures ANOVA, with time of measurement as the within-factor and meal type as the grouping factor. If differences reached statistical significance, post hoc analyses with two-tailed paired t test was used, using Bonferroni correction for multiple comparisons. The "response to the meal" was analyzed by calculating the TG, sICAM-1 and sVCAM-1 net incremental area under the curve (netAUC), including the entire incremental area below the curve and the area below the fasting concentration by the trapezoidal rule. Statistical significance was defined as $P<.05$.

3. Results

The baseline values of TG, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), sICAM-1 and sVCAM-1 after the washout and adaptation periods on the NCEP Step I diet supplemented with ROO or HPSO are shown in Table 3. No significant differences were observed between diets in each group (lower baseline values with HPSO did not reach statistical significance by post hoc pairwise comparisons). However, the baseline values of TG, LDL-C, sICAM-1 and sVCAM-1 were significantly higher, whereas those of HDL-C were lower in hypertriglyceridemic (normotensive and hypertensive) compared to healthy subjects.

The effect of the nature of dietary fatty acids on the postprandial levels of TG, sICAM-1 and sVCAM-1 was evaluated by comparing baseline values with samples collected hourly from 1 to 8 h after the consumption of the test meals enriched in ROO or HPSO (Fig. 1). Values markedly increased and peaked postprandially. Postprandial peak levels of sICAM-1 and sVCAM-1 were significantly ($P<001$) higher and occurred later in hypertriglyceridemic (normotensive and hypertensive), compared with healthy subjects. In general, the three groups experienced parallel rises in plasma TG, sICAM-1 and sVCAM-1 until the postprandial peak. Thereafter, the levels of theses variables decreased fastest with the ROO

Table 3
Baseline values of TG, sICAM-1 and sVCAM-1 after the lead-in washout and adaptation period on ROO- and HPSO-enriched diets in healthy subjects and in hypertriglyceridemic subjects without and with coexisting hypertension

	TG (mmol/L)	sICAM-1 (ng/ml)	sVCAM-1 (ng/ml)
Healthy subjects			
ROO	0.68±0.19	535.4±90.9	758.3±110.6
HPSO	0.69±0.16	454.7±54.2	705.9±120.4
Hypertriglyceridemic normotensive subjects*			
ROO	4.10±0.58	1105.0±201.7	1342.8±238.7
HPSO	3.99±0.94	935.0±256.2	1208.7±201.1
Hypertriglyceridemic hypertensive subjects*			
ROO	4.17±0.66	1220.1±165.2	1383.1±190.3
HPSO	4.02±0.71	998.6±215.4	1257.6±206.9

Values are represented as mean±S.D. (n=14).
* $P<001$ vs. healthy subjects.

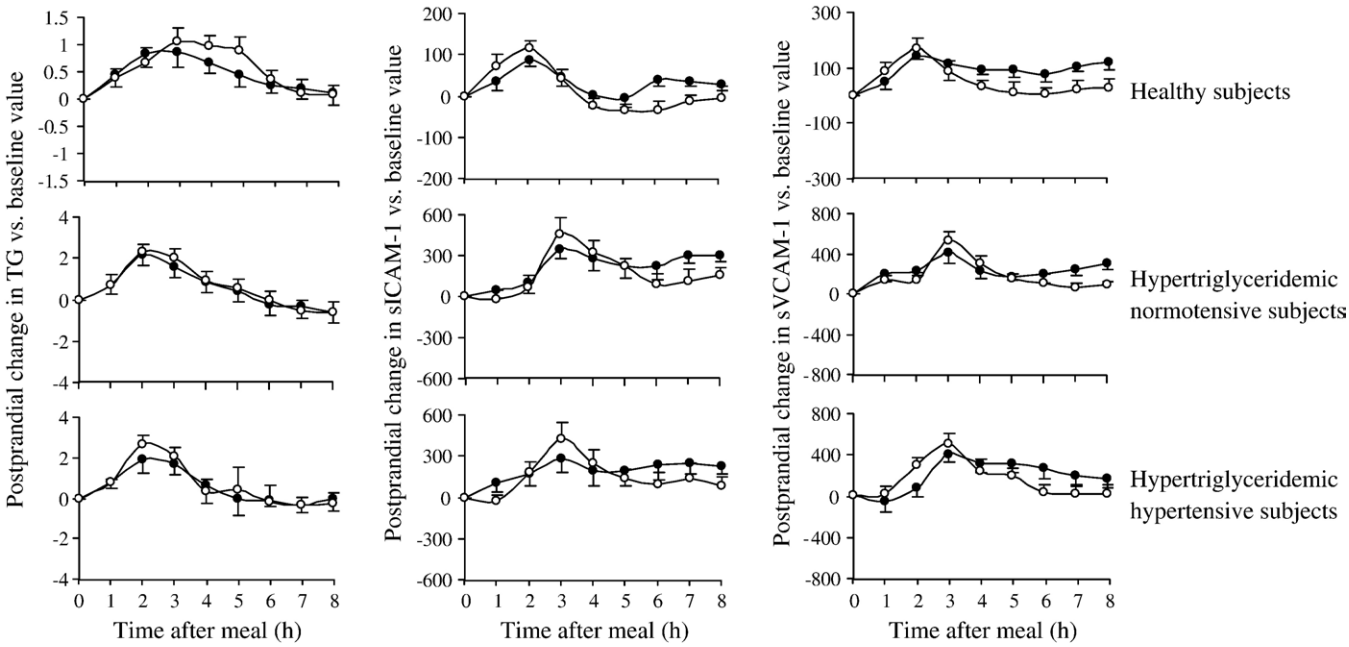

Fig. 1. Postprandial changes in plasma TG, sICAM-1 and sVCAM-1 vs. baseline value in healthy subjects and in hypertriglyceridemic subjects without and with coexisting hypertension after a meal enriched in ROO (open circles) or HPSO (closed circles). Values at corresponding time are represented as mean ±S.D. (n=14).

meal. No differences in the postprandial curves were found between normotensive and hypertensive hypertriglyceridemic subjects.

Postprandial net$AUC_{0-8\ h}$ for TG was marginally lower in healthy subjects after the HPSO meal (P=.046) and did not differ between groups (Table 4). However, the postprandial response for sICAM-1 and sVCAM-1 was higher with both meals in hypertriglyceridemic (normotensive and hypertensive) subjects. The ROO meal induced a decrease (P<001) in the postprandial net$AUC_{5-8\ h}$ for sICAM-1 and sVCAM-1 in all subjects.

4. Discussion

The up-regulation of sICAM-1 and sVCAM-1 is synonymous with endothelial activation and vascular inflammation [3,4], which is emphasized during the postprandial state in subjects with hypertension [10] or fasting hypertriglyceridemia [11]. Due to the importance of adopting healthy dietary habits, many controlled studies have focused on replacing dietary SFA by MUFA to reduce the risk of CAD [12,13]. Thus, studies addressing the role of MUFA and SFA on the postprandial levels of sICAM-1 and sVCAM-1 appear

Table 4
Postprandial responses for TG, sICAM-1 and sVCAM-1 to a meal enriched in ROO and HPSO in healthy subjects and in hypertriglyceridemic subjects without and with coexisting hypertension

	TG net$AUC_{0-8\ h}$ (mmol·h/L)	sICAM-1 net$AUC_{0-4\ h}$ (ng·h/ml)	sICAM-1 net$AUC_{5-8\ h}$ (ng·h/ml)	sVCAM-1 net$AUC_{0-4\ h}$ (ng·h/ml)	sVCAM-1 net$AUC_{5-8\ h}$ (ng·h/ml)
Healthy subjects					
ROO	4.45±0.67	185.1±40.0	−69.7±15.6	380.3±84.8	46.6±9.1
HPSO	3.75±0.51*	165.3±41.2	83.4±22.9†	450.5±72.3	284.0±75.8†
Hypertriglyceridemic normotensive subjects‡					
ROO	5.63±0.99	940.3±106.1	380.4±48.4	1177.3±130.1	283.0±53.2
HPSO	4.87±1.32	872.2±88.5	787.5±95.8†	1136.1±134.8	659.5±72.4†
Hypertriglyceridemic hypertensive subjects*					
ROO	5.48±1.12	893.4±122.4	335.8±40.4	1155.8±178.2	160.8±23.0
HPSO	4.74±0.90	844.0±116.1	698.0±108.3†	897.3±183.5§	706.9±59.4†

Values are represented as mean±S.D. (n=14).
* P=.046 vs. ROO.
† P<001 vs. ROO.
‡ P<001 vs. healthy subjects for sICAM-1 and sVCAM-1.
§ P<05 vs. ROO.

to be needed to identify the mechanisms by which dietary fatty acids might influence atherogenesis. The current study is the first demonstration that a MUFA-rich meal, when compared to a meal rich in SFA, reduces postprandial levels of sICAM-1 and sVCAM-1 in healthy subjects and in normotensive and hypertensive subjects with fasting hypertriglyceridemia.

Abundant evidence is now available that subjects with elevated fasting levels of TG or LDL-C have reduced HDL-C [16], which, per se, is considered a key factor in determining elevated levels of sICAM-1 and sVCAM-1 [17]. That low HDL-C results from hypertriglyceridemic states represents, at least in part, inefficient catabolism of TG-rich lipoproteins (TRL) and increased transfer of TG from the core of TRL to HDL. TG-enriched HDL particles are more rapidly catabolized by lipases, thereby resulting in cholesterol depleted HDL particles. It is interesting to note that sICAM-1 and sVCAM-1 are positively associated with low HDL-C concentrations in subjects with fasting hypertriglyceridemia [18] but not in subjects with normal levels of TG [19]. These observations highlight the importance of TG and TG enrichment of HDL particles secondary to fasting hypertriglyceridemia in the values of sICAM-1 and sVCAM-1. In our study, we did not find any statistical differences between normotensive and hypertensive hypertriglyceridemic subjects or diets (rich in SFA and MUFA) in the fasting values of TG, HDL-C, sICAM-1 and sVCAM-1. However, hypertriglyceridemic (normotensive and hypertensive) subjects had higher fasting TG, LDL-C, sICAM-1 and sVCAM-1 and lower HDL-C values than those found in healthy subjects, indicating a common unfavourable lipoprotein phenotype and similar systemic inflammation [20] between normotensive and hypertensive hypertriglyceridemic subjects. To our knowledge, sICAM-1 and sVCAM-1 have not yet been studied in hypertriglyceridemic subjects with coexisting hypertension and without other metabolic abnormalities, such as obesity or some degree of glucose intolerance, which could complicate the search for the independent effect of the hypertensive state on fasting (and postprandial) levels of sICAM-1 and sVCAM-1 in hypertriglyceridemic subjects.

High postprandial levels of TG may further induce elevated sICAM-1 and sVCAM-1 in subjects with fasting hypertriglyceridemia, but data are not available on hypertriglyceridemic subjects with hypertension, which is considered a classic and strong risk factor for CAD. This issue has been addressed here by determining the effect of high-fat meals rich in MUFA or SFA on postprandial levels of TG, sICAM-1 and sVCAM-1 in healthy and hypertriglyceridemic (normotensive and hypertensive) subjects. We found that the postprandial levels of sICAM-1 and sVCAM-1 increased as TG did, but the netAUC for sICAM-1 and sVCAM-1 was rather related to the nature of the fat included in the meal. The postprandial levels of sICAM-1 and sVCAM-1 severely increased in hypertriglyceridemic (normotensive and hypertensive), compared with healthy subjects. Interestingly, ROO meal (having a higher ratio of MUFA to SFA) induced lower netAUC for sICAM-1 and sVCAM-1 than HPSO meal (having a lower ratio of MUFA to SFA). These observations are fully consistent with the results of in vitro experiments showing that MUFA, but not SFA, acutely inhibited cytokine-induced expression of ICAM-1 and VCAM-1 [21]. The mechanism of the effect is uncertain, although it may be the result of fatty acid changes in lipid moieties (phospholipids and mainly TG) of nascent TRL that could be “transferred” to other lipoproteins as a result of postprandial TRL metabolism. This assumption agrees with recent studies, indicating that SFA adversely affected postprandial anti-inflammatory (antiadhesive) properties of HDL [22] and that postprandial LDL is more effective than postabsorptive LDL in up-regulating ICAM-1 on the surface of endothelium [23]. In addition, MUFA were described to have protective effects on vascular inflammation during the acute-phase response [24]. However, we do not exclude the possibility that other mechanisms unrelated to fatty acids could also be involved.

In a complementary finding, the present study also shows no differences in the postprandial levels of TG, sICAM-1 and sVCAM-1 between normotensive and hypertensive hypertriglyceridemic subjects after the ROO or HPSO meal. One possible explanation may be that a high-fat meal does not acutely impair endothelial function in resistance vessels and that the resistance vessels are resistant to the effects of postprandial TRL [25]. Our data suggest that although high-fat meal may further contribute to endothelial activation and vascular inflammation in fasting hypertriglyceridemia, it probably does not predispose to a higher risk in hypertensive with regard to normotensive hypertriglyceridemic subjects.

We conclude that the challenge of a meal with a high ratio in MUFA to SFA appears to have a significant postprandial benefit on sICAM-1 and sVCAM-1 as surrogate markers of endothelial activation and vascular inflammation in healthy and, more importantly, in hypertriglyceridemic (normotensive and hypertensive) subjects. Therefore, it is conceivable that diets rich in oleic acid (MUFA) may lead to the reduction of the risk of CAD in subjects with permanently activated endothelium by improving vascular inflammatory response postprandially.

References

[1] Hyson D, Rutledge JC, Berglund L. Postprandial lipemia and cardiovascular disease. Curr Atheroscler Rep 2003;5:437–44.
[2] Giannattasio C, Zoppo A, Gentile G, Failla M, Capra A, Maggi FM, et al. Acute effects of high-fat meal on endothelial function in moderately dyslipidemic subjects. Arterioscler Thromb Vasc Biol 2005;25:406–10.
[3] White JM. ADAMs: modulators of cell–cell and cell–matrix interactions. Curr Opin Cell Biol 2003;15:598–606.
[4] Wu JT, Wu LL. Association of soluble markers with various stages and major events of atherosclerosis. Ann Clin Lab Sci 2005;35:240–50.
[5] Boulbou MS, Koukoulis GN, Makri ED, Petinaki EA, Gourgoulianis KI, Germenis AE. Circulating adhesion molecules levels in type 2 diabetes mellitus and hypertension. Int J Cardiol 2005;98:39–44.

[6] Rosei EA, Rizzoni D, Muiesan ML, Sleiman I, Salvetti M, Monteduro C, et al. Effects of candesartan, cilexetil and enalapril on inflammatory markers of atherosclerosis in hypertensive patients with non-insulin-dependent diabetes mellitus. J Hypertens 2005;23:435–44.
[7] Abe Y, El-Masri B, Kimball KT, Pownall H, Reilly CF, Osmundsen K, et al. Soluble cell adhesion molecules in hypertriglyceridemia and potential significance on monocyte adhesion. Arterioscler Thromb Vasc Biol 1998;18:723–31.
[8] Lupattelli G, Lombardini R, Schillaci G, Ciuffetti G, Marchesi S, Siepi D, et al. Flow-mediated vasoactivity and circulating adhesion molecules in hypertriglyceridemia: association with small, dense LDL cholesterol particles. Am Heart J 2000;140:521–6.
[9] Haffner SM. The metabolic syndrome: inflammation, diabetes mellitus, and cardiovascular disease. Am J Cardiol 2006;97:3A–11A.
[10] Kolovou GD, Anagnostopoulou KK, Pavlidis AN, Salpea KD, Iraklianou SA, Tsarpalis K, et al. Postprandial lipemia in men with metabolic syndrome, hypertensives and healthy subjects. Lipids Health Dis 2005;4:21–8.
[11] Marchesi S, Lupattelli G, Lombardini R, Roscini AR, Siepi D, Vaudo G, et al. Effects of fenofibrate on endothelial function and cell adhesion molecules during postprandial lipemia in hypertriglyceridemia. J Clin Pharm Ther 2003;28:419–24.
[12] Kris-Etherton PM, Hecker KD, Binkoski AE. Polyunsaturated fatty acids and cardiovascular health. Nutr Rev 2004;62:414–26.
[13] Rivellese AA, Maffettone A, Vessby B, Uusitupa M, Hermansen K, Berglund L, et al. Effects of dietary saturated, monounsaturated and n-3 fatty acids on fasting lipoproteins, LDL size and postprandial lipid metabolism in healthy subjects. Atherosclerosis 2003;167:149–58.
[14] Guidelines Subcommittee. 1999 World Health Organization–International Society of Hypertension Guidelines for the Management of Hypertension. J Hypertens 1999;17:151–83.
[15] Pacheco YM, Bermudez B, Lopez S, Abia R, Villar J, Muriana FJG. Ratio of oleic to palmitic acid is a dietary determinant of thrombogenic and fibrinolytic factors during the postprandial state in men. Am J Clin Nutr 2006;84:342–9.
[16] Miller M, Langenberg P, Havas S. Impact of lowering triglycerides on raising HDL-C in hypertriglyceridemic and non-hypertriglyceridemic subjects. Int J Cardiol 2007;119:192–5.
[17] Calabresi L, Gomaraschi M, Villa B, Omoboni L, Dmitrieff C, Franceschini G. Elevated soluble cellular adhesion molecules in subjects with low HDL-cholesterol. Arterioscler Thromb Vasc Biol 2002;22:656–61.
[18] Lupattelli G, Marchesi S, Lombardini R, Siepi D, Bagaglia F, Pirro M, et al. Mechanisms of high-density lipoprotein cholesterol effects on the endothelial function in hyperlipemia. Metabolism 2003;52: 1191–5.
[19] Stanojevic NB, Ivanovic ZJ, Djurovic S, Kalimanovska VS, Spasic S, Ostric DK, et al. Lack of association between low HDL-cholesterol and elevated circulating cellular adhesion molecules in normolipidemic CAD patients and healthy subjects. Int Heart J 2005;46:593–600.
[20] Marz W, Scharnagl H, Winkler K, Tiran A, Nauck M, Boehm BO, et al. Low-density lipoprotein triglycerides associated with low-grade systemic inflammation, adhesion molecules, and angiographic coronary artery disease: the Ludwigshafen Risk and Cardiovascular Health study. Circulation 2004;110:3068–74.
[21] De Caterina R, Liao JK, Libby P. Fatty acid modulation of endothelial activation. Am J Clin Nutr 2000;71:213S–23S.
[22] Nicholls SJ, Lundman P, Harmer JA, Cutri B, Griffiths KA, Rye KA, et al. Consumption of saturated fat impairs the anti-inflammatory properties of high-density lipoproteins and endothelial function. J Am Coll Cardiol 2006;48:715–20.
[23] Marschang P, Gotsch C, Kirchmair R, Kaser S, Kahler CM, Patsch JR. Postprandial, but not postabsorptive low-density lipoproteins increase the expression of intercellular adhesion molecule-1 in human aortic endothelial cells. Atherosclerosis 2006;186:101–6.
[24] Bellido C, Lopez-Miranda J, Blanco-Colio LM, Perez-Martinez P, Muriana FJG, Martin-Ventura JL, et al. Butter and walnuts, but not olive oil, elicit postprandial activation of nuclear transcription factor kappaB in peripheral blood mononuclear cells from healthy men. Am J Clin Nutr 2004;80:1487–91.
[25] Gudmundsson GS, Sinkey CA, Chenard CA, Stumbo PJ, Haynes WG. Resistance vessel endothelial function in healthy humans during transient postprandial hypertriglyceridemia. Am J Cardiol 2000;85: 381–5.