O9 NUTRITION

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RED WINE CONSUMPTION DOES NOT AFFECT THE OXIDATION OF LOW-DENSITY LIPOPROTEINS IN HUMANS

Y.B. de Rijke, P.N.M. Demacker, N.A. Assen. L.M. Sloots, M.B. Katan, A.F.H. Stalenhoef
University Hospital Nijmegen, Division of General Internal Medicine, P.O. Box 9101, 6500 HB Nijmegen, the Netherlands

Red wine phenolics may protect low-density lipoproteins (LDL) against oxidative modification, thereby reducing the risk of cardiovascular morbidity. However, in vivo data are scarce. We have now assessed whether the consumption of red wine affects the susceptibility of LDL to Cu^{2+}-mediated oxidative modification and antioxidant concentrations in blood. Twentyfour healthy nonsmoking normolipidemic volunteers consumed a flavonoid-poor white wine for two weeks (baseline period). They were then randomly assigned to consumption of white or red wine (flavonoid-rich) (test period), with stratification for age, sex and plasma cholesterol concentration. Before consumption the alcohol content was reduced by evaporation. Subjects followed a flavonoid-poor diet and abstained from tea and red wine. The average flavonoid intake from foods was 8.1 ± 4.6 in the white and 10.0 ± 6.0 mg in the red wine group.
Two fasting blood samples were collected on separate days both at the end of the baseline period and the test period. Lag times, reflecting the resistance of LDL against oxidative modification, were similar in both wine groups and were not prolonged after consumption of 550 ml (about 4-5 glasses) of red wine for 4 weeks. Plasma lipids were unchanged as were aqueous and lipophilic antioxidants in blood or plasma. In conclusion, this study does not support the proposed beneficial effect of red wine consumption on LDL oxidation.

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EFFECT OF SATURED, MONOUNSATURED AND POLYUNSATURED N-6 AND N-3 FATTY ACID-RICH DIETS ON THE SUSCEPTIBILITY OF LDL TO OXIDATIVE MODIFICATION IN WOMEN

R. Alonso (*), P. Mata, C. Lahoz, R. Andrés, F. González, C. Garcés, E. Blazquez, J.M. Ordovás (**), M. de Oya
Unidad de Lípidos, Fundación Jimenez Díaz, Madrid, Spain. (*) Universidad de Valparaíso, Chile.(**) Lipid Metabolism Laboratory, HNRCA-Tufts University, Boston

The purpose of this study was to compare the effect of different dietary fat saturation on plasma LDL oxidation. 18 healthy women (mean age 45,8 ± 9,5 years, 10 were postmenopausic) were placed on four consecutive periods of diets prepared using natural foods: 1) saturated (SAT) fat-rich diet (17% saturated fat (S), 13% monounsaturated (M) fat and 4% polyunsaturated (P) fat); 2) monounsaturated (MONO)-rich diet (9% S. 22% M, 3,7% P); 3) polyunsaturated (POLY n-6)-rich diet; and 4) polyunsaturated (POLY n-3)-rich diet (both diets had 9% S. 12% M and 12,9% P). Each dietary period lasted 5 weeks. Lipid profile was measured as recommended by tbe Lipid Research Clinics using enzimatic methods. LDL susceptibility to oxidation was studied by measuring the lag time (minutes) and formation of conjugated dienes (nmol/mg LDL protein) after the incubation with copper ions, on an UV-spectrophotometer. Lipid peroxidation was judged by the thiobarbituric acid-reacting substances assay (nmol/mg LDL protein). All unsaturated diets lowered LDL cholesterol when compared to SAT diet, but the effect was higher in both POLY periods. LDL oxidation parameters are shown in the following table:

Diet	Conjugated Dienes	Lag time	TBARS
SAT	247±51 ab	46±4 a	1.08±O.4 ab
MONO	230±40 cd	55±7 abc	1.10±O.3 cd
POLYn-6	307±53 ac	47±5 b	1.33±O.3 ac
POLYn-3	323±42 bd	43±7 c	1.48±O.5 bd

Numbers with the same letter are significantly diffierent, $P<0.05$.
LDL vitamin E content was higher during both POLY diets compared only with SAT diet ($P<0.05$). Our data indicates that a MONO-rich diet protects LDL from "in vitro" oxidation when compared to SAT and POLY-rich diets.

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EFFECTS OF STEARIC ACID AND TRANS FATTY ACIDS ON SERUM LIPIDS, APOPROTEINS AND LIPOPROTEIN(a)

A. Aro, M. Jauhiainen, M. Mutanen
Departments of Nutrition and Biochemistry, National Public Health Institute and Department of Nutrition, University of Helsinki, Finland

We compared the effects of stearic acid (C18:0) and trans fatty acids (transFA) from hydrogenated vegetable oils in 80 healthy subjects. The diets included 37 en% fat, the sum of saturated fatty acids (SFA) and transFA was 17 en%, MUFA 14 en% and PUFA 4 en%. All subjects started on a diet with 17 en% SFA, 3.3 en% C18:0 and < 1 en% transFA for 5 weeks. Thereafter 40 subjects consumed a diet with 9.2 en% C18:0 and < 1 en% transFA and 40 subjects a diet with 2.5 en% C18:0 and 9.0 en% transFA for another 5 weeks. The fats were mixed into solid foods. Fasting serum samples were obtained at the end of each period. Lipoproteins were separated by ultracentrifugation.
Compared with the SFA diet, both C18:0 and transFA reduced total cholesterol (by 9.4% and 7.5%, respectively, $p<0.001$). C18:0 reduced LDL-cholesterol by 4% ($p=0.055$) and transFA increased it by 3% (ns). HDL_2- and HDL_3-cholesterol were reduced by both C18:0 and transFA ($p<0.01$). C18:0 decreased apoB and increased the apoA-I/apoB ratio compared to transFA ($p<0.05$ for differences). Lp(a) was increased by both C18:0 and transFA but more by transFA ($p<0.01$ for difference).
Compared with a high SFA diet, both C18:0 and transFA reduced serum HDL-cholesterol and apoA-I but C18:0 reduced also LDL-cholesterol and apoB levels in contrast to transFA. TransFA increased Lp(a) more than C18:0.

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TRANS FATTY ACIDS AND THE COMPOSITION OF HUMAN AORTIC PLAQUES

C.V. Felton, D. Crook, J.C. Stevenson.
Wynn Department of Metabolic Medicine, National Heart & Lung Institute, London, UK

The trans fatty acid (TFA) composition of human aortic plaques and adipose tissue were compared to determine whether plaque TFA content is influenced by dietary intake. Patients with coronary artery disease (CAD) have greater proportions of TFA in adipose tissue and serum compared with controls suggesting an association between TFA intake and CAD. It has been proposed that TFA interfere with endothelial repair, accumulate in vessel wall intima and thus promote atherosclerosis. As humans obtain TFA solely from the diet, and adipose tissue levels reflect dietary intake, we tested this hypothesis by comparing the percentages of trans(t)16:1, t18:1 and t18:2 isomers in plaques and adipose tissue. Greater proportions in plaques would provide evidence of accumulation. Samples were taken from 10 Caucasian men (<70 years) who died following an acute coronary event. TFA content was determined using capillary chromatography. The results suggest that different TFA influence plaque composition to different extents (table), t18:1 isomers accumulate in plaques and dietary t18:1 intake is positively associated with plaque content ($p=0.06$).

	Percentage of total fatty acids (±SD)	
Fatty acid	Plaque(n=10)	Adipose tissue(n=10)
1 trans 16:1	1.16 (0.4)	1.16 (0.3)
2 trans 18:1	5.20 (1.9)*	3.26 (1.0)
3 trans 18:2	0.24 (0 2)*	0.48 (0.1)

*$p<0.01$, paired t-test, plaque versus adipose tissue

Accumulation of TFA in plaques may reduce membrane fluidity and increase the susceptibility of plaques to fissuring. Impairment of prostaglandin synthesis may have implications for thrombus formation.