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Tobacco and alcohol consumption: impact on other cardiovascular and cancer risk factors in a southern European Mediterranean population

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Tobacco and alcohol consumption are strongly related to other cardiovascular and cancer risk factors. The aim of the present study was to analyse the association of nutrient intake, blood lipid variables and leisure-time physical activity with tobacco and alcohol consumption status. Participants were recruited in a cross-sectional population-based survey, including cardiovascular risk factor measurements and evaluation of physical activity and diet intake in a Mediterranean population (*n* 1748). Multiple linear regression analysis, adjusted for several confounders, showed a direct association of saturated fatty acids (g and % total energy intake), dietary cholesterol intakes and serum triacylglycerol with smoking. An inverse association was observed for smoking and unsaturated fatty acids (% energy intake), vitamin C, α-tocopherol and β-carotene intakes, leisure-time physical activity and HDL-cholesterol. These associations were not observed for alcohol drinking. After adjusting for the confounders earlier mentioned, low dietary intakes of vitamin C and dietary fibre were more likely in heavy-smokers as compared with non-smokers (odds ratio 1·74 (95 % CI 1·07, 2·73) and 1·94 (95 % CI 1·29, 2·92) of low vitamin C (< 60 mg/d) and dietary fibre intakes (< 10 g/d) respectively). Alcohol consumption was directly associated with HDL-cholesterol and triacylglycerol, and attenuated the effects of smoking on HDL-cholesterol. These results suggest that the dietary intake of fibre and several antioxidant components of the Mediterranean diet is reduced in smokers, who also show an adverse lipid profile. However, the worst triacylglycerol levels are associated with the combination of heavy smoking and heavy alcohol drinking. Moderate alcohol consumption was not associated with an unhealthy diet pattern or adverse lipid profile. The health benefits of the Mediterranean diet appear to be strongly counteracted by smoking.

Cancer: Cardiovascular risk factors: Diet

Smoking is strongly associated with development and progression of CHD (Gensini *et al.* 1998), various types of cancer and increased mortality (Giovannucci & Martinez, 1996; Kabat, 1996; Zeegers *et al.* 2000). Cancer induction mechanisms (Duthie *et al.* 1995), lipoprotein metabolism (Craig, 1993) and lipid peroxidation (Morrow *et al.* 1995) are affected by tobacco smoke components. However, some association between these diseases and smoking can be confounded by unhealthy lifestyles. Smoking has been shown to be associated with dietary habits that may contribute to the higher risk for these diseases in smokers compared with non-smokers (Midgette *et al.* 1993; McPhillips *et al.* 1994).

Moderate alcohol intake seems to reduce the risk of CHD by increasing HDL concentrations (Rimm *et al.* 1999). However, heavy drinkers have increased coronary mortality rates, often accompanied by high HDL-cholesterol concentrations (Paunio *et al.* 1996). Furthermore, higher intakes of alcohol and lower intakes of antioxidant vitamins and of dietary fibre observed in smokers might increase their risk of cancer induction (Dallongville *et al.* 1998; Lloveras *et al.* 2001; Palaniappan *et al.* 2001).

The Mediterranean diet, which is characterized by high consumption of monounsaturated fat, vegetables, pulses and fruits (Trichopoulou & Lagiou, 1997) is one of the main reasons for the lower incidence of cancer and CHD observed in southern European Mediterranean regions as compared with northern and middle Europe and the USA (Verschuren *et al.* 1995; Menotti *et al.* 1999; Trichopoulou *et al.* 2000). However, the extent to which smoking and alcohol consumption influence the healthy dietary habits of the Mediterranean region remains unknown.

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The aim of the present study was to analyse the effect of tobacco smoking and alcohol consumption on dietary habits and their association with cardiovascular risk factors in a Mediterranean population.

Subjects and methods

Subjects

The methods for the 1994–1996 cardiovascular heart risk population-based cross-sectional survey conducted in Gerona, Spain, have been described in detail elsewhere (Masiá *et al.* 1998). In brief, free-living Spanish men and women from the province of Gerona, 25–74-years-old, participated in this study from September 1994 – January 1996. Subjects (*n* 3000) were randomly selected from the general population of Gerona according to the 1991 census, with a two-stage sampling stratified by five age groups and sex. After excluding census errors, 2404 eligible subjects were left, of whom 1748 (72·7 %) agree to participate. All participants were duly informed and consented to be entered in a computer database and cede their biological samples for the necessary analyses. The protocol was approved by an Ethics Committee and the results sent to participants.

Measurements

BMI was calculated as weight divided by height squared (kg/m^2), and obesity was defined as BMI ≥ 30. Venous blood samples were taken from a superficial forearm vein. Blood extractions (25 ml) were made after a 14 h fast without venous compression (or less than 60 s duration when strictly necessary) using a syringe with a holder and vacuum tubes with separating gel. Samples were centrifuged at 2500 rpm for 15 min between 30 and 60 min post-extraction and the sera immediately frozen at $-120\,^{\circ}C$ in liquid N_2, and stored long-term at $-80\,^{\circ}C$ within 7 d. Analyses were performed at an interval of 3–4 months post-extraction in groups of 600 samples. Total cholesterol and triacylglycerol levels were determined enzymatically (Roche Diagnostica, Basel, Switzerland). HDL-cholesterol was measured as cholesterol after precipitation of apoprotein B-containing lipoproteins with phosphotungstic Mg^{2+} (Boehringher Mannheim, Mannheim, Germany). Inter-assay CV were 2·47, 4·46 and 3·20 % for total cholesterol, HDL-cholesterol and triacylglycerol respectively. External quality assessment was performed with EQA-WHO (World Health Organization, Prague, Czech Republic). LDL-cholesterol was calculated by the Friedwald equation. Hpercholesterolaemia and hypertriacylglycerolaemia were defined as total cholesterol $\geq 5{\cdot}69$ mmol/l and triacylglycerol $\geq 2{\cdot}27$ mmol/l respectively.

Information on lifestyle, health and eating and drinking habits of the participants was obtained by a structured interview carried out by trained personnel. Participants were categorized as those who had never smoked, former smokers or current smokers. The latter were classified according to the number of cigarettes smoked per d (less than two or twenty or more per d). The mean daily nutrient intake and energy consumption were measured by a validated structured 72 h recall (Schröder *et al.* 2001). This recall was administrated by a trained interviewer. During the dietary recall interviews, participants were requested to describe precisely their food and non-alcoholic beverage intake during the previous 3 d. Each of the foods listed was characterized by a full description of the usual serving size. Energy, fat, carbohydrate and protein intakes were calculated from the 72 h recalls with the software Diet Analysis Nutritionist IV (N Squared Computing, San Bruno, CA, USA). This software includes a database of 9879 food items supplemented with Spanish food items. In brief, validity of the questionnaire (i.e its ability to classify study subjects according to rank of nutrient intake) was reflected by Pearsons's correlation coefficient between nutrient intake reported in the reference method (3 d record) and the 72 h recall (r 0·42), and intraclass correlation coefficients between nutrient intake reported in the reference method and the 72 h recall (r 0·55). Furthermore, 37 % of subjects were in the same quartile with the 72 h recall and with the reference method, while only 5·3 % were found in extreme quartiles (Schröder *et al.* 2001).

Current alcohol intake was recorded separately by asking participants how many glasses of wine, bottles of beer and drinks or shots of brandy or similar beverages were consumed during the previous 1 week. Mean daily alcohol intake was calculated and participants classified as non-drinkers, drinkers within the recommended amount (men ≤ 40 g alcohol/d and women ≤ 25 g alcohol/d) and excessive drinkers. Energy intake derived from consumption of alcoholic beverages (kJ) was calculated as follows: alcohol content (g) of beverage $\times 30$.

Socio-economic status was characterized by the following levels of education: illiterate, primary school, secondary school, university studies.

Leisure-time physical activity was measured by the Minnesota physical leisure-time questionnaire which has been validated for Spanish men and women (Elosua *et al.* 1994, 2000)

Statistical analysis

ANOVA by a general linear model (GLM) was used to estimate dietary intakes according to smoking and alcohol drinking status. A *post-hoc* Bonferroni correction for multiple comparisons was carried out to determine differences in nutrient intake among groups. Analysis of co-variance from a general linear model was used to perform a linear test for trends of dietary intake and serum cholesterol, HDL-cholesterol, LDL-cholesterol and triacylglycol in defined smoking and alcohol consumption groups.

The odds ratio of low dietary fibre (<10 g/d), low vitamin C (<60 mg/d) intake and/or prudent nutrient intake for smoking- and alcohol consumption-defined groups was analysed with logistic regression adjusting for potential confounders.

Linear regression analysis, adjusted for sex, age, BMI, educational level and physical activity, was carried out to analyse the association of total cholesterol, HDL-cholesterol, LDL-cholesterol, triacylglycerol and the dietary components (vitamin C, β-carotene, α-tocopherol, fibre,

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Table 1. Characteristics of the study participants
(Mean values with their standard errors)

	Males (*n* 765)		Females (*n* 812)	
	Mean	SEM	Mean	SEM
Age (years)	50·8	13·9	50·2	13·7
BMI (kg/m^2)	26·6	4·1	26·5	4·8
Total cholesterol (mmol/l)	5·79	1·13	5·76	1·19
LDL-cholesterol (mmol/l)	3·93	1·13	3·80	1·07
HDL-cholesterol (mmol/l)	1·23	0·36	1·48	0·37
Triacylglycerol (mmol/l)	1·43	1·16	1·09	0·56
Leisure-time physical activity (kJ/d)	1497	1449	886	879
Current smokers (%)	29·2		16·0	
Former smokers (%)	33·6		5·2	
Never smokers (%)	37·2		78·8	
Cigarette consumption of current smokers (*n*/d)	17·7	12·2	13·0	9·2
Hypercholesterolaemia (%)*	53·1		48·6	
LDL-cholesterol risk (%)†	40·9		33·8	
HDL-cholesterol risk (%)‡	16·4		14·1	
Hypertriacylglycerolaemia (%)§	11·1		3·6	
Current alcohol consumers (%)	78·1		41·3	
Alcohol intake of consumers (g/d)	50·9	40·4	22·5	18·5

* Serum total cholesterol ≥5·69 mmol/l.
† Serum LDL-cholesterol >4·15 mmol/l.
‡ Serum HDL-cholesterol for men <0·91 and women <1·09 mmol/l.
§ Serum triacylglycerol ≥2·27 mmol/l.

cholesterol and unsaturated and saturated fatty acids) with the number of cigarettes smoked and amount of alcohol ingested. Associations between cardiovascular risk factors and smoking or alcohol drinking were further adjusted for alcohol drinking or smoking respectively. Analysis of the data was conducted using SPSS for Windows (version 9.0) statistical software package (SPSS Inc., Chicago, IL, USA). In all statistical tests performed *P* values of $<0·05$ were considered significant.

Results

Characteristics of the participants are presented in Tables 1 and 2. Age and BMI were similar between non-smokers and former smokers (Table 2), with no difference between moderate and heavy former smokers (results not shown). However, current smokers were younger than non-smokers. Non-smokers had the highest mean BMI, followed by heavy smokers and moderate smokers.

Table 2. Main characteristics of non-smokers, former smokers (ex-smokers) and moderate smokers (more than twenty cigarettes per d) and heavy smokers (twenty or more cigarettes per d) of both sexes
(Mean values with their standard errors)

	Non-smokers		Ex-smokers		Moderate-smokers		Heavy-smokers	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Age (years)	52·3	13·3	52·9	13·9	43·6	13·5	42·3	11·4
BMI (kg/m^2)	26·9	4·5	26·8	3·6	24·8	4·1	25·7	5·2
Men								
n	260		271		110		118	
%	28·9		86·3		57·3		71·5	
Women (*n*)	638		43		82		47	
Total	898		314		192		165	
Educational status								
University								
n	46		23		14		13	
% men	34·8		82·6		42·9		46·2	
Secondary school								
n	128		51		51		50	
% men	28·9		74·5		47·1		72·0	
Primary school								
n	654		210		119		95	
% men	30·1		88·6		6·2		74·7	
Illiterate								
n	46		14		4		3	
% men	8·7		92·9		50·0		33·3	

Table 3. Daily energy consumption and nutrient intake of non-smokers, former smokers (ex-smokers) moderate smokers (more than twenty cigarettes per d) and heavy smokers (twenty or more cigarettes per d) of both sexes, adjusted for age, sex and BMI‡

(Adjusted mean values)

Nutrient	Non-smokers (*n* 893)	Ex-smokers (*n* 310)	Moderate smokers (*n* 189)	Heavy smokers (*n* 165)	*P* value for trend
Energy (MJ)§	9·77	9·87	9·90	10·38	NS
Carbohydrates (g)	237·4	234·7	233·7	240·3	NS
Protein (g)	118·2	121·8	119·9	123·0	NS
Fat (g)	86·6	86·7	87·6	87·3	NS
Carbohydrates (% total energy intake)	43·1	42·4	42·3	42·8	NS
Protein (% total energy intake)	21·7*	22·3	22·1	22·2	NS
Fat (% total energy intake)	35·2*	35·3	35·6	35·0	NS
Saturated fatty acids (g)	26·6	26·7	26·8	27·9	NS
Unsaturated fatty acids (g)	53·9	55·4	57·8	54·7	NS
Saturated fatty acids (% total energy intake)	11·8	11·7	11·7	12·1	NS
Unsaturated fatty acids (% total energy intake)	23·4	23·5	23·5	23·1	NS
Cholesterol (mg)	408·2	413·6	406·1	432·5	NS
Fibre (g)	17·8†	17·8†	16·7	15·3	0·0001
Vitamin C (mg)	162·3†	170·8†	154·1	135·2	0·001
β-Carotene (μg)	1521·1	1655·6†	1618·8†	1349·7	0·02
α-Tocopherol (mg)	4·7†	4·7†	4·5	4·1	0·001
Alcohol (g/d)	21·6†	23·9†	26·6†	36·4	0·0001

Mean value was significantly different from that of ex-smokers (corrected for multiple comparisons (twelve) by Bonferroni method): *$P<0·05$.
Mean values were significantly different from those of heavy smokers (corrected for multiple comparisons (twelve) by Bonferroni method): †$P<0·05$.
‡ For details of subjects and procedures, See Tables 1 and 2 and p. 274.
§ Energy intake includes alcoholic beverages.

Nutrient intake according to smoking and alcohol drinking status was similar between men and women. Therefore, results are presented together and data were pooled for analysis. Mean dietary intakes according to smoking and alcohol drinking status are shown in Tables 3 and 4. We observed slightly higher total energy consumption in heavy smokers, which was accompanied by higher intakes of macronutrients (Table 3). However, macronutrient intake expressed as % total energy intake was similar among all groups. Intake of saturated fatty acids (g/kg fat intake) and dietary cholesterol were higher in heavy smokers in comparison with all other groups, although

Table 4. Daily energy consumption and nutrient intake by level of alcohol, consumption; non drinker (non-drinkers), moderate drinkers (<40 g alcohol/d for men, <25 g alcohol/d for women) and heavy drinkers (>40 g alcohol/d for men, >25 g alcohol/d for women) of both sexes, adjusted for age, sex and BMI‡

(Adjusted mean values)

Nutrient	Non-drinkers (*n* 630)	Moderate drinkers (*n* 543)	Heavy drinkers (*n* 372)	*P* value for trend
Energy (MJ)§	8·99	9·28	9·25	NS
Carbohydrates (g)	234·9	241·1	233·1	NS
Protein (g)	117·0†	120·8	122·4	0·003
Fat (g)	84·5†	87·7	89·4	0·003
Saturated fatty acids (g)	26·7	26·9	26·9	NS
Unsaturated fatty acids (g)	51·9*	54·6†	57·8	0·0001
Carbohydrates (% total energy intake)	43·2†	43·0†	41·8	0·0001
Protein (% total energy intake)	21·9	21·8	22·2	NS
Fat (% total energy intake)	34·9†	35·1†	35·9	0·001
Saturated fatty acids (% total energy intake)	12·1*	11·8	11·5	0·0001
Unsaturated fatty acids (% total energy intake)	23·2*	23·5†	23·7	0·0001
Cholesterol (mg)	401·9	419·2	415·3	NS
Fibre (g)	17·7†	17·7†	16·6	0·02
Vitamin C (mg)	164·5	159·5	151·6	NS
β-Carotene (mg)	1582·7	1547·1	1473·3	NS
α-Tocopherol (mg)	4·7	4·7	4·6	NS
Cigarettes (smokers only *n* per d)	16·4*	12·7†	18·4	NS
Smokers (%)	22·5†	22·1†	30·2	0·006

Mean values were significantly different from those of the moderate drinkers (corrected for multiple comparisons (six) by Bonferroni method): *$P<0·05$.
Mean values were significantly different from those of the heavy drinkers (corrected for multiple comparisons (six) by Bonferroni method): †$P<0·05$.
‡ For details of subjects and procedures, see Tables 1 and 2 and p. 274.
§ Energy intake excludes alcoholic beverages.

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not reaching statistical significance. A statistically significant negative trends for dietary fibre, vitamin C, vitamin E, α-tocopherol and β-carotene across groups of smoking status were observed, with the lowest intakes for heavy smokers. There was a statistically significant positive linear trend with the highest alcohol intake for heavy smokers (Table 3). Furthermore, statistically significant lower dietary fibre, vitamin C and α-tocopherol intakes were found in heavy smokers in comparison with non-smokers and former smokers, whereas alcohol intake was significantly greater in heavy smokers when compared with al other groups.

The somewhat higher energy intake (excluding consumption of alcoholic beverages) of heavy drinkers was accompanied by a statistically significant higher intake of protein and fat compared with non-drinkers ($P<0{\cdot}05$, Table 4). The carbohydrate intake, expressed as % total energy consumption, was significantly lower in heavy drinkers when compared with non-drinkers and moderate drinkers, and showed a significant decreasing linear trend across groups of alcohol drinking status ($P<0{\cdot}05$, Table 4). Intake of saturated fatty acids, expressed as % total energy intake, of non-drinkers was significantly higher that that of moderate and heavy drinkers ($P<0{\cdot}05$). Furthermore, a significant decreasing linear trend of saturated fatty acids intake, expressed as % energy consumption, was observed across groups of alcohol drinking status, whereas an increasing trend was observed for unsaturated fatty acids intake.

Concentrations of serum total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol, according to smoking and drinking status and respective linear trends adjusted for sex, BMI and age among groups, are shown in Fig. 1. Serum triacylglycerol increased significantly across types of smoking status ($P<0{\cdot}0001$, Fig. 1(d)), whereas an inverse linear trend was observed for HDL-cholesterol ($P < 0{\cdot}0001$, Fig. 1(c)). The highest triacylglycerol concentrations were found in heavy smokers when compared with all other smoking status groups. In contrast, HDL-cholesterol was significantly lower in this group as compared with non-smokers ($P<0{\cdot}02$).

Serum concentration of total cholesterol, HDL-cholesterol and triacylglycerol increased significantly across the levels of alcohol consumption ($P<0{\cdot}001$, $P<0{\cdot}01$, $P<0{\cdot}001$, Fig. 1(a), (c) and (d) respectively). Heavy drinkers had significantly higher concentrations of these blood lipid variables compared to non drinkers and moderate drinker ($P<0{\cdot}01$).

Heavy smoking, in combination with levels of alcohol consumption, showed that alcohol counteracts the effect of smoking on HDL-cholesterol concentrations (Fig. 1(c)). Interactions were tested but failed to be statistically significant. Furthermore, the highest triacylglycerol concentration was found in the heavy smoker–heavy drinker group (*n* 59), which significantly differed ($P<0{\cdot}02$) from the other groups.

Relative risks of low vitamin C, low dietary fibre and low prudent nutrient intake, according to smoking and alcohol consumption status, are presented in Table 5. After adjusting for sex, age, physical activity, BMI and educational status, heavy smokers had the highest risk for a low vitamin C and dietary fibre intake, and the lowest risk for prudent nutrient intake. On the other hand, alcohol consumption did not increase the risk of a lower ingestion of these dietary components.

Associations of alcohol intake and cigarette consumption with dietary compounds and cardiovascular risk factors were calculated by multiple linear regression adjusted for potential confounders (Tables 6 and 7). Total cholesterol and HDL-cholesterol significantly increased with alcohol intake, and triacylglycerol also increased with the number of cigarettes smoked. We observed a

Table 5. Association between non-smokers, former smokers (ex-smokers) moderate smokers and heavy smokers, and non-drinkers, moderate drinkers and heavy-drinkers of both sexes and the prevalence of low vitamin C intake, low dietary fibre intake and healthy dietary habits*†‡

(Multi-adjusted odds ratios and 95% confidence intervals)

	Low vitamin C intake§ OR 95 % CI	Low fibre intake‖ OR 95 % CI	Prudent nutrient intake¶ OR 95 % CI
Smoking status			
Non-smokers	1	1	1
Ex-smokers	0·82 0·52, 1·28	1·17 0·82, 1·66	1·02 0·55, 1·89
Moderate smokers	1·14 0·7, 1·81	1·51 1·03, 2·19	0·40 0·14, 1·20
Heavy smokers	1·71 1·07, 2·73	1·94 1·29, 2·92	0·26 0·61, 1·14
Drinking status			
Non-drinkers	1	1	1
Moderate drinkers	1·02 0·83, 1·54	1·10 0·82, 1·47	1·54 0·90, 2·65
Heavy drinkers	0·99 0·59, 1·39	1·13 0·81, 1·57	0·90 0·44, 1·84
Heavy drinking and heavy smoking (*n* 59)	1·62 0·78, 3·32	1·73 0·93, 3·22	0·36 0·04, 2·89

OR, odds ratio.

* For details of subjects and procedures, see Tables 1, 2 and p. 274.

† Moderate smokers, less than twenty cigarettes per d; heavy smokers, twenty or more cigarettes per d; moderate drinkers *n* < 40 g alcohol/d, women < 25 g alcohol/d; heavy drinkers, men > 40 g alcohol/d women > 25 g alcohol/d.

‡ The relative risk was adjusted for age, sex, BMI, leisure-time physical activity, alcohol intake and educational level.

§ Low vitamin C intake < 60 mg vitamin C/d.

‖ Low dietary fibre intake < 10 g/d.

¶ Prudent dietary habits: Mean daily intake of dietary fibre (≥ 20 g, vitamin C > 60 mg, saturated fatty acids ≤ 10 % total energy intake and monounsaturated fatty acids ≥ 12 % total energy intake.

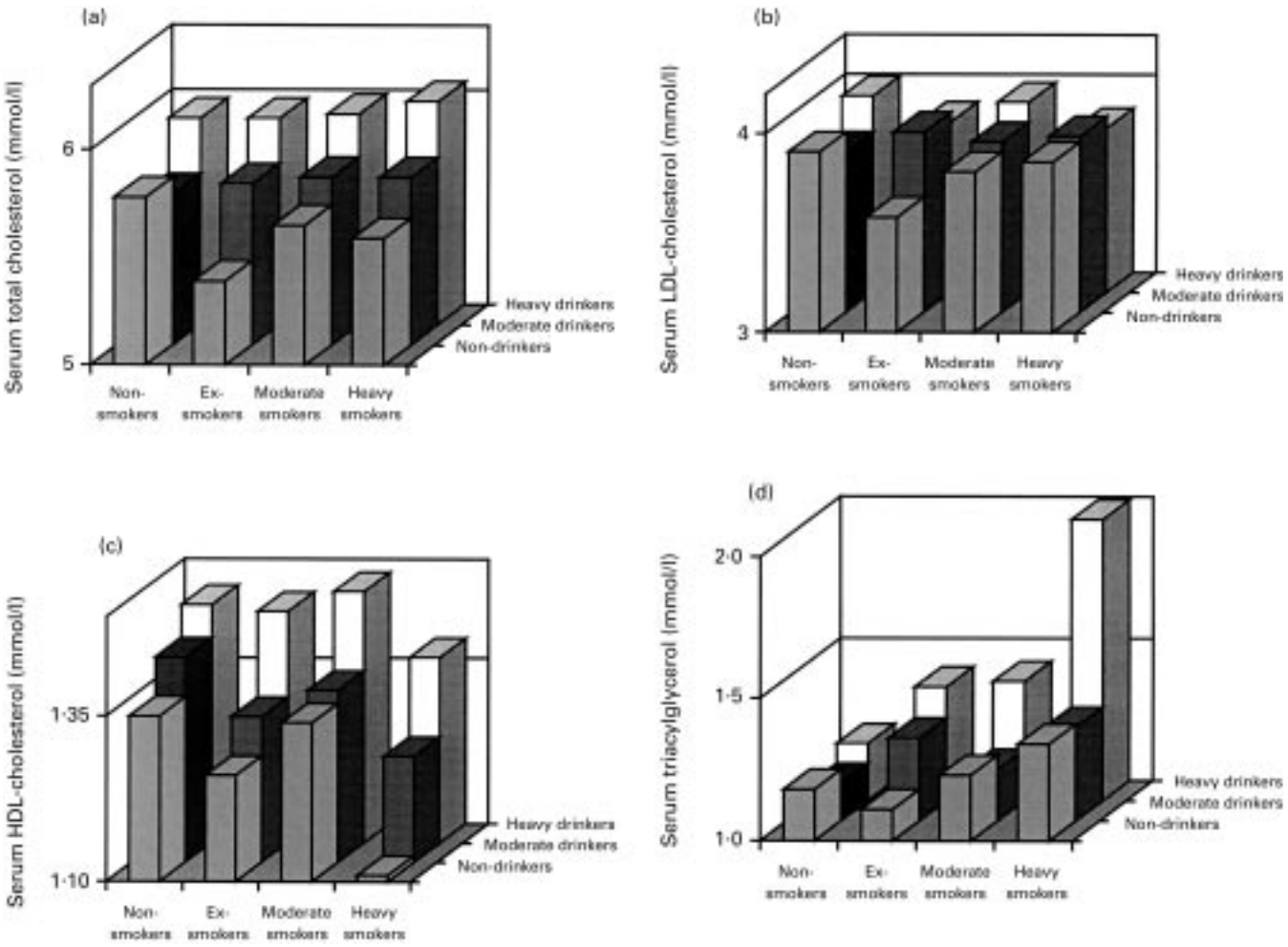

Fig. 1. Serum concentrations of (a) total cholesterol (*P* value for linear trends across drinking status $<0{\cdot}001$), (b) LDL-cholesterol, (c) HDL-cholesterol (*P* value for linear trends across drinking status $<0{\cdot}01$ and across smoking status $<0{\cdot}0001$), (d) triacylglycerol (*P* value for linear trends across drinking status $<0{\cdot}001$ and across smoking status $<0{\cdot}0001$). For details of subjects and procedures, see Tables 1, 2 and p. 274. Values were adjusted for age, sex, BMI, leisure-time physical activity and educational status.

strong inverse relationship between HDL-cholesterol and triacylglyceol (r 0·38, $P<0{\cdot}0001$). Since alcohol is a strong determinant of HDL-cholesterol, and high HDL-cholesterol is associated with lower triacylglycerol, the association between alcohol and triacylglycerol will be counterbalanced by the strong association with HDL-cholesterol. After further adjustment for HDL-cholesterol, we found that the amount of alcohol was directly associated with triacylglycerol. Cigarette smoking showed a significant inverse relationship with protective cardiovascular

Table 6. Multiple linear regression models of blood lipid variables and leisure time physical activity for cigarette smoking and alcohol drinking adjusted for sex, age, leisure-time physical activity, BMI and educational status*

	Smoking (ten cigarettes)			Alcohol (10 g alcohol)		
Dependent variables	B	SEM	*P*	B	SEM	*P*
Leisure-time PA (kJ) (r^2 0·092)†‡	−0·0302	0·0623	0·0440	0·0002	0·0025	0·7670
Total cholesterol (mmol/l) (r^2 0·096)	0·0122	0·2200	0·5800	0·0035	0·0009	<0·0001
LDL-cholesterol (mmol/l) (r^2 0·082)	0·0037	0·0355	0·9160	0·0019	0·0014	0·1840
HDL-cholesterol (mmol/l) (r^2 0·164)	−0·0439	0·0123	<0·0001	0·0034	0·0005	<0·0001
Triacylglycerol (10 mmol/l) (r^2 0·172)†	0·3610	0·1000	<0·0001	0·0010	0·0030	<0·0001
Triacylglycerol (10 mmol/l) (r^2 0·297)†§	0·2780	0·0610	<0·0001	0·0080	0·0030	<0·0001

PA, physical activity.
* For details of subjects and procedures, see Tables 1, 2, p. 274 and Table 7.
† Values were log transformed.
‡ Values were not adjusted for leisure-time physical activity.
§ Values were further adjusted for HDL-cholesterol.

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Table 7. Multiple linear regression models of dietary components for cigarette smoking and alcohol drinking adjusted for sex, age, leisure-time physical activity, BMI and educational status*

	Smoking (ten cigarettes)				Alcohol (10/g alcohol)		
Dependent variables	r^2	B	SEM	*P*	B	SEM	*P*
Saturated fatty acids (g)	0·014	0·800	0·311	0·015	−0·002	0·012	0·850
Unsaturated fatty acids (g)	0·058	0·309	0·580	0·594	0·065	0·020	0·003
Dietary cholesterol (mg)	0·088	1·162	4·340	0·008	−0·004	0·172	0·813
Saturated fatty acids (% total energy intake)	0·069	0·164	0·063	0·009	−0·009	0·003	<0·0001
Unsaturated fatty acids (% total energy intake)	0·069	−0·168	0·063	0·008	0·009	0·020	<0·0001
Vitamin C (mg)	0·028	−6·570	3·348	0·050	−0·162	0·133	0·224
β-Carotene (μg)†	0·050	−0·066	0·020	<0·0001	−0·001	0·001	0·271
α-Tocopherol (mg)	0·036	−0·174	0·064	<0·0001	−0·002	0·003	0·375

* For details of subjects and procedures, see Tables 1, 2 and p. 274.
† Values were log transformed.

risk factors HDL-cholesterol and leisure-time physical activity.

Smoking was directly related to saturated fatty acids, expressed in g and % energy intake, and dietary cholesterol intake, whereas alcohol consumption was associated with unsaturated fatty acids intake (g). The dietary antioxidant components vitamin C, α-tocopherol and β-carotene showed a significant inverse association with smoking.

Discussion

The present study sought to investigate the complex relationship of smoking and alcohol consumption with nutrient intake and cardiovascular risk factors in a representative southern European Mediterranean population. The results indicate that the intrinsic potentially deleterious effect of smoking, and particularly heavy smoking, may be enhanced by unhealthy dietary habits. In contrast, moderate alcohol drinking, and dietary habits that accompanied moderate drinking, might exert a protective effect, particularly on CHD.

In the present study, non-smokers had the highest mean BMI followed by heavy smokers and moderate smokers. Interestingly, a similar association between smoking and BMI was found 20 years ago by Jacobs *et al.* (1981) and recently by Primatesta *et al.* (2001).

Smoking and alcohol consumption have been shown to be associated with the progression of cardiovascular disease and cancer (Gaziano & Buring, 1998; Rehm & Bondy, 1998; Ringborg, 1998), and other environmental factors, including dietary habits, might exacerbate or counteract these progressions.

Evidence indicates that high intakes of dietary saturated fatty acids and cholesterol increase the risk of cardiovascular disease (Klör *et al.* 1997; Gaziano & Buring, 1998), whereas physical activity exerts a protective effect on this disease (Thune *et al.* 1998). In the present study, smoking was independently and directly associated with the ingestion of these nutrients and inversely related to physical activity. These findings might partially account for increased cardiovascular risk, especially for heavy smokers.

Low consumption of dietary fibre, β-carotene and the antioxidant vitamins C and E have been associated with higher risk for various cancers (Byers & Guerrero, 1995; Yong *et al.* 1997) and also with an elevated risk of cardiovascular diseases (Todd *et al.* 1999). In accordance with a previously published meta-analysis (Dallongeville *et al.* 1998), we observed lower intakes of vitamin C, vitamin E, β-carotene and fibre in smokers, and particularly in heavy smokers. Interestingly, former smokers were shown to have the highest vitamin C, vitamin E, β-carotene and fibre intakes, a fact that might reflect dietary habit changes accompanying cessation of smoking. In general, part of the effect of smoking on poor dietary habits may be related to the changes in the taste of food for smokers. The higher relative risk of insufficient vitamin C and dietary fibre intake for heavy smokers shown in the present study, and the independent association of smoking with low intake of potentially cancer-protecting antioxidant dietary components, might potentiate the 'per se' higher cancer risk for heavy smokers. Furthermore, antioxidant dietary components are also protective against cardiovascular disease (Todd *et al.* 1999). The lower intake of these nutrients by smokers, in combination with the atherogenic and carcinogenic effects of smoking, might be responsible for the high mortality rate observed in this group by other authors (Doll *et al.* 1994; Cosin-Aguilar *et al.* 1995). In accordance with the findings of other authors (Ballestros Pomar *et al.* 2000), we observed a vitamin C intake at least two-fold above the recommended daily allowance. Although heavy smokers consumed significantly lower amounts of vitamin C than non-or ex-smokers, the amount was greater than the recommended daily allowance. In this population, this finding may explain part of the low CHD incidence and mortality observed in Gerona despite high prevalence of CHD risk factors (Masiá *et al.* 1998). The overall pattern of nutrient intake, and the higher alcohol consumption by heavy smokers, might reflect a less healthy lifestyle when compared with the other smoking categories, including moderate smoking. This is in contrast with a study conducted in British men and women that showed similar nutrient intakes between heavy and moderate smokers (Margetts & Jackson, 1993).

Fat intake was significantly higher in heavy drinkers than in abstainers, as shown previously (D'Avanzo *et al.* 1997). However, higher fat intake resulted from a higher unsaturated fatty acids consumption in heavy drinkers when compared with non-drinkers. Furthermore, the

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amount of antioxidant vitamin intake was not different across categories of alcohol consumption, which is in accordance with results obtained in a study conducted in Italian women (D'Avanzo *et al.* 1997). Only dietary fibre intake was lower in heavy drinkers than in moderate drinkers and non-drinkers. Interestingly, although alcohol consumption is a strong risk factor for various diseases, the direct and inverse association of alcohol drinking with unsaturated fatty acids (g) and saturated fatty acids (% total energy) intakes respectively, show that alcohol ingestion was accompanied by less unhealthy dietary habits than smoking in this Mediterranean population.

The effect of smoking on cardiovascular disease is partially mediated by changes in lipid metabolism (Craig, 1993). In the present study, we observed a strong, alcohol independent, dose–response association between smoking and HDL-cholesterol. This association has been previously reported (Freeman *et al.* 1993). Moreover, heavy smoking was also associated with a significant increase in serum triacylglycerol that has been previously reported (Castelli *et al.* 1977).

Alcohol is known to also influence lipid metabolism. Several observational and experimental studies have reported strong relationships between alcohol and HDL-cholesterol, and, although to a lesser extent, with serum triacylglycerol (Castelli *et al.* 1977; Hulley & Gordon, 1981). In the present study, we observed a significant inverse dose–response association between smoking and HDL-cholesterol, after adjusting for several confounders including alcohol intake. However, analysing total lipoproteins, and LDL- and HDL-cholesterol, only the latter was directly related to alcohol drinking. Evidence indicates that low serum HDL-cholesterol concentration was associated with high mortality from CHD (Yano *et al.* 1977). Several observational studies have found a protective association between CHD and moderate alcohol intake that appears to be mediated partially by alcohol-induced increases of HDL-cholesterol (Goldbourt *et al.* 1985; Jacobs *et al.* 1990). In contrast to the study by Walmsley *et al.* (1998), we found higher serum triacylglycerol concentrations in heavy drinkers compared with non-drinkers and moderate drinkers. Furthermore, the observed strong relationship between alcohol intake and HDL-cholesterol counterbalanced the triacylglycerol response to alcohol consumption. This was clearly demonstrated by the positive association between alcohol intake and triacylglycerol after further adjustment with HDL-cholesterol.

We have examined the strong relationship between alcohol intake, smoking and blood lipids. Our present results indicated that alcohol drinking counteracts the effect of smoking on HDL-cholesterol.

We found the highest serum triacylglycerol concentration in the heavy smokers, heavy drinkers after adjusting for sex, age and BMI. This concentration was 19·0 and 34·0 % higher than that found in heavy smokers and heavy drinkers respectively, indicating an additional effect of heavy smoking and heavy drinking on serum triacylglycerol concentrations. Raised serum triacylglycerol levels are atherogenic in themselves (Rumpler *et al.* 1999), and the strong higher serum triacylglycerol concentrations found in heavy smokers, heavy drinkers and in heavy smokers, heavy drinkers might increase the risk of cardiovascular disease in these groups.

In conclusion, the dietary intake of fibre and several antioxidant components of the Mediterranean diet is reduced in smokers, who also show an adverse lipid profile. However, the worst triacylglycerol levels are associated with combination of the heavy smoking and heavy alcohol drinking. Moderate alcohol consumption was not associated with an unhealthy diet pattern or adverse lipid profile. The health benefits of the Mediterranean diet appear to be strongly counteracted by smoking.

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