Parental death from cardiovascular disease and dietary habits in an elderly group

BY ROSA M. ORTEGA¹, PEDRO ANDRES², MATILDE AZUELA¹, ALFONSO ENCINAS-SOTILLOS³ AND MARIA JESUS GASPAR⁴

¹ Departamento de Nutrición, Facultad de Farmacia, Universidad Complutense, 28040-Madrid, Spain

² Departamento de Nutrición y Bromatología II, Laboratorio de Técnicas Instrumentales, Facultad de Farmacia, Universidad Complutense, 28040-Madrid, Spain

⁴ Servicio de Análisis Clínicos, Hospital del INSALUD, Guadalajara, Spain

(Received 27 October 1993 – Revised 26 February 1993 – Accepted 1 April 1993)

The present study examines the influence of parental history of death from cardiovascular disease on dietary habits and nutritional status of a group of seventy-two Spanish elderly. Those with at least one parent who had died of cardiovascular disease (43.1% of the cases) had higher diastolic blood pressure (P < 0.05) and nutrient intakes less favourable from the cardiovascular risk point of view than those whose parents died of other causes. Descendants whose parents died of cardiovascular disease had higher total fat, animal fat, saturated fatty acids, myristic acid and palmitic acid intakes and a lower monounsaturated fatty acids:saturated fatty acids value than descendants of those who died from other causes (all P < 0.05).

Blood pressure: Cardiovascular disease: Dietary habits: Serum lipids: Elderly

Cardiovascular disease, the principal cause of death and invalidity in developed countries (Nikkila & Heikkinen, 1990), is a clear example of a diet-related pathology. The nutritional habits which increase the risk of these diseases are well known; high intakes of animal and total fat, dietary cholesterol, animal protein as well as total energy are positively correlated with cardiovascular mortality in cross-cultural comparisons, while polyunsaturated fatty acids (PUFA), fibre and vegetable protein are negatively correlated (Connor & Connor, 1986; Nissinen & Stanley, 1989; Rudman, 1989; Simopoulos, 1989; Katsouyanni *et al.* 1991).

A number of studies have investigated relationships between blood lipid and lipoprotein levels and family history of cardiovascular disease (Shear *et al.* 1985; Freedman *et al.* 1986). Fewer studies have examined the dietary intake of subjects whose parents had died of cardiovascular disease. It is possible that there may be genetic or cultural determinants of dietary habits, which may in turn determine plasma cholesterol concentrations and other cardiovascular risk factors (Cavalli-Sforza, 1990; Oliveria *et al.* 1992).

Therefore, the purpose of the present study was to assess the dietary and nutritional differences between two elderly groups, classified by parental cause of death (cardiovascular and other causes).

MATERIALS AND METHODS

The dietary habits and nutritional status of seventy-two non-institutionalized elderly (thirty-four males and thirty-eight females), aged 65–89 years (mean age 71 (se 1.4) years),

³ E. A. P. Rafael Alberti, Madrid, Spain

who attended the physician's general medicine surgery at the INSALUD (Spanish Social Security) in Madrid during October and November 1990 were studied. Subjects who voluntarily agreed to take part in the study and who were able to present the death certificate(s) of their parent(s) were included in the study; selected subjects comprised 70% of all the subjects aged 65–89 years who attended the physician's surgery during the 2-month period.

The study was approved by the Human Research Review Committee of the University Complutense of Madrid, Faculty of Pharmacy.

The elderly were grouped according to the cause of death of their parents: one group included those who had at least one parent who died of cardiovascular disease (C; coronary ischaemic cardiopathy or cerebrovascular ischaemic accident), and the other group included those whose parents died of other causes (NC).

For the present study dietary, anthropometric, biochemical and blood pressure data were recorded.

Diet survey

A prospective food record questionnaire was compiled during five consecutive days (including Sunday). A set of kitchen scales was provided for all the elderly to facilitate the food weighing. After the questionnaire was completed, the booklets were returned in person. A qualified nutritionist inspected the records to ensure that they were complete and that sufficient detail had been recorded. In the same interview a food frequency intake questionnaire was completed.

The energy and nutrient content of all the food ingested was determined using the *Spanish Food Composition Tables* (Institute of Nutrition, 1990*a*). Intakes were compared with dietary recommendations for the Spanish population made by the Institute of Nutrition (1990*b*).

Anthropometric survey

Weight and height were determined without shoes, using a digital electronic weighing scale (Seca alpha; Rue Lavoisier 91430, Igmy, France; range: 0.1-150 kg) and a digital stadiometer (Harpender Pfifter 450; Badem, Padum Aveny, Carlstadt, NJ, USA; range 0.70-2.05 m) respectively. Waist and hip diameter were also determined using a measuring tape (range 0-150 cms). From these data, body mass index (Quételet index; weight/height²; kg/m²) and waist:hip ratio were calculated.

Biochemical survey

Fifty-seven elderly (twenty-seven males and thirty females) agreed to the blood sampling. Blood was drawn without stasis by venepuncture (antecubital fossa) after an overnight 12 h fast. Serum was obtained by centrifugation at 1100 g for 10 min at 4°. Triacylglycerols were measured by enzymic hydrolysis (GPO/PAP method; Merck, Merck-Igoda, S. A. Apdo., 47 de Mollet del Vallés, Barcelona, Spain; coefficient of variation (CV) 3·1%; Bucolo & David, 1973). Total cholesterol (TC; Merck; CV 2·2%) and high-density-lipoprotein (HDL)-cholesterol oxidase (EC 1.1.3.6) colorimetry (Allain et al. 1984), the latter after precipitation of serum with phosphotungstic acid and Mg²⁺ (Burstein et al. 1970), automated in an autoanalyser (ERIS-6170; Merck-Olympus-Eppendorf). Low-density-lipoprotein (LDL)-cholesterol concentrations were calculated according to Friedewald et al. (1972) and very-low-density-lipoprotein (VLDL)-cholesterol was calculated according to Wilson et al. (1981).

PARENTAL DEATH AND NUTRITION OF DESCENDANTS

Blood pressure

Blood pressure was measured three times after a 5 min rest in the right arm of the seated participant by a trained technician using a Hawksley random-zero sphygmomanometer (W.A. Baum Co., Copaigue, NY, USA) and an appropriate-size cuff. First- and fifth-phase Korotkoff sounds were recorded (Expert Committee of the Spanish Ministry of Health, 1991) and the mean of the second and third blood pressure values was used in analysis.

Statistical methods

Means with their standard errors are presented for all the elderly by sex and parental cause of death.

Data were compared by parametric (Student's *t* test and analysis of variance) and nonparametric (Mann–Whitney and Kruskal–Wallis) tests to determine whether differences in variables existed with respect to parental cause of death. Differences were considered statistically significant at P < 0.05 (Wonnacott & Wonnacott, 1977).

RESULTS

Cardiovascular disease was the cause of death of one or both parents for 43.1% and 12.5% respectively of the elderly studied. Non-cardiovascular causes of death were: cancer (33%), cirrhosis of liver (5%), renal insufficiency (9%), grave respiratory insufficiency (4%), diabetes (5%), infection (22%), puerperium (5%), accidents and suicide (15%). The remaining 1% of the parents were still alive.

When at least one of the parents died of cardiovascular disease (group C), we observed a higher systolic and diastolic blood pressure in their descendants than that in the NC group, the difference being significant for diastolic blood pressure (P < 0.05; Table 1). If hypertension is defined as diastolic blood pressure ≥ 90 mmHg or systolic blood pressure ≥ 160 mmHg, 30.8% of group C elderly were hypertensive compared with 6.3% of group NC elderly.

There were no anthropometric differences between C and NC groups (Table 1). Group C elderly tended to have higher total serum cholesterol and LDL-cholesterol values than group NC elderly, although the difference was not significant (Table 1). The cholesterol concentrations of $86\cdot4\%$ of the group C elderly and $75\cdot8\%$ of the group NC elderly were higher than $5\cdot2 \text{ mmol/l}$ and $45\cdot5$ and $30\cdot3\%$ respectively had serum cholesterol concentrations greater than $6\cdot2 \text{ mmol/l}$.

Group C elderly tended to have higher intakes of most foodstuffs, except cereals, vegetables, legumes and fish, but the differences were not statistically significant (Table 2).

Table 3 shows the nutrient intakes and Fig. 1 shows the energy and nutrient intakes as a percentage of the recommended intakes (Institute of Nutrition, 1990*b*). Intakes of energy, Zn, Mg and vitamin D were lower than recommended values, and intakes of protein, niacin, vitamins B_{12} and C were greater than recommended values.

Intakes of fibre, Ca, Fe, Mg, thiamin and folate were lower than recommended intakes (Institute of Nutrition, 1990*b*) for 58.6, 34.5, 34.5, 79.3, 13.8 and 51.7% respectively, of C elderly, whereas for NC elderly, intakes of these nutrients were lower than recommended for 42.9, 28.6, 25.7, 34.3, 11.4 and 42.9% respectively, of this group.

Comparison of energy and nutrient intakes of groups C and NC (Table 3), revealed that the greatest differences were for fat intakes which were higher among group C elderly; differences were significant for total fat (g/d; P < 0.05), saturated fatty acids (SFA; g/d and % energy; P < 0.01 and P < 0.05 respectively) and animal fat (g/d; P < 0.05). Group

Study group		Sut	Subjects whose parents die of cardiovascular disease	e parents e ular disea	died ise			Sut	Subjects whose parents died of other causes	e parents (· causes	died	
	Total (n 31)	(<i>n</i> 31)	Males (n 13)	(<i>n</i> 13)	Females (n 18)	; (n 18)	Total (n 41)	(<i>n</i> 41)	Males (n 21)	(<i>n</i> 21)	Females (n 20)	; (n 20)
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Age (years)	71.4	Ē	71-3	2-0	71-4	4	70.7	1:0	71-2	1.3	70-1	l·S
Wt (kg)	64.1	2·1	68.0	3.6	61.8	2.5	67-5	2.0	69.5	2.6	65.8	3.0
Ht (m)	1-55	0.10	1-64	0.07	1-49	0.06	1-57	0.08	1-62	60-0	1.52	0-04
BMI (kg/m ²)	26.9	6.0	25-6	1.7	27-7	Ŀ	27-7	6.0	26.6	ŀI	28-6	1.4
Waist : hip	0-99	1·0	1-02	0-1	0.93	0-1	0.95	0.1	0.98	0.1	0-92	0·1
Systolic blood pressure (mmHg)	145.8	9.9	136-0	13-3	151.9	9.9	139-1	4·1	135-0	7-1	143·1	4.4
astolic blood pressure (mmHg)	92·3	4·8	0.86	11-1	88.8	4.0	80-0*	2.5	79-9	3:3	80.1	4·1
iacylglycerols (mmol/l)	1.13	l·0	1-03	0.2	1.18	0·I	1-24	0·1	1-29	0·1	1.19	0·1
Cholesterol (mmol/l)	6.11	0:2	6-07	0.4	6.13	0.3	5-82	0.2	5.63	0.2	5-99	0.3
HDL-cholesterol (mmol/l)	1.51	0.1	1.58	0-2	1-49	0·1	1-29	0-1	1-25	1·0	1-34	0·1
DL-cholesterol (mmol/l)	4·13	0·2	4-04	0.5	4.16	0.3	4.03	0.2	3.80	0.2	4·16	0 S
VLDL-cholesterol (mmol/l)	0-52	0.1	0-47	0-1	0-54	$0 \cdot 1$	0-57	1.0	0.59	0·1	0.55	0·1

Table 1. Physical and biochemical characteristics of Spanish elderly grouped according to parental cause of death (Mean values with their standard errors) BMI, body mass index (weight/height^{*}); HDL, high-density-lipoprotein; LDL, low-density-lipoprotein; VLDL, very-low-density-lipoprotein. Mean value was sig-inficantly from the total value for the group whose parents died of cardiovascular disease: *P < 0.05.

death*
5
l cause
to parental
to
according
grouped
elderly
Spanish
£
<i>o</i> .
a)
g/d
ood intakes (
Table 2. F

(Mean values with their standard errors)

Study group		Sub of	Subjects whose parents died of cardiovascular disease	e parents sular dise:	died ase			Sul	Subjects whose parents died of other causes	e parents r causes	died	
	Tota	-	Males	les	Fem	Females	Total	tal	Males	ules	Fem	Females
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Fotal foods	1509-7	73-9	1658-9	133.8	1404·3	6.92	1411.8	63.6	1391-2	87.1	1431-3	94.5
Cereals	157-8	13-4	198.5	25.1	129-1	10-3	160.6	6-11	175-3	14-2	146-6	18.6
viilk	350-4	42·1	331-4	72-8	363-8	51.8	317-3	34.0	255-7	43-7	375-5	49-0
Iggs	17-5	2.8	22-0	3-7	14-3	3.9	12.9	1-9	14.9	3.5	10-9	1.7
Dil	27·0	3·1	30-0	5.7	24.8	3.4	21-7	1-7	21-7	6·1	21-6	2.9
bugar	20.8	4·2	33.3	8.3	12.0	2.9	16.1	2.7	21-2	4.5	11-3	2.8
Vegetables	240-1	27.6	2164	32-9	256-9	41-4	256-9	4-4	218-7	32-7	293-0	83.4
egumes	18.1	4:5	18-4	8·1	18-0	5.4	22.5	3.5	18.3	4·2	26.4	5.6
Truits	351-9	33-0	361-1	48.4	345-4	45.9	329-4	30·8	344-0	44·8	315-7	43·4
Aeat	121-1	9.6	142·2	15.8	106.2	11-0	118-2	7.5	117-0	10.2	119-3	11-3
Tish	55-9	7.3	66-5	10-2	48.4	9.9	62.4	6.8	73-8	8:4	51-7	10.3
Alcoholic beverages	62·1	23.7	95.5	51-6	38.6	17-3	56.6	19.1	6.7	36-5	18.7	8.2
Von-alcoholic beverages	85.9	50·1	135-6	116-2	50-8	27·I	24.5	9.5	35.4	17-4	14.2	8.0
Water	1138-2	65.2	1204-3	119-7	1091-5	73.5	1009.4	48·8	1051-4	2.67	969-7	59.2

* For details of subjects and procedures, see Table 1 and pp. 259-260.

PARENTAL DEATH AND NUTRITION OF DESCENDANTS

		Sub	(Mean values with their Subjects whose parents died	ues with t	(Mean values with their standard errors) ects whose parents died	ard errors)		Sul	Subjects whose parents died	e parents	died	
of	of		of cardiovascular disease	cular dise	ase				of other	of other causes		,
Total	al		Males	les	Fen	Females	Total	tal	Ma	Males	Fen	Females
Mean SE	SE		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
m	399	1	9024 ^b	720	6599	238	6960	240	7218 ^h	310	6716	376
	3.0		86.5	4.6	70-9	3.0	77.1	2.4	76.8	2-9	77-3	4.0
	0·0		16.7	1·02	17.6	0·8	18-9	0.6	18.1	L·O	19-7	Ŀ
	18-4		271-6 ^b	21.5	211.5	26.3	212-4	7-9-7	219-9 ^b	11-7	205-3	15-3
	١·S		47.6	1:3	46-5	2.5	47.8	1 4	48·1	1-9	47.6	2.0
	5:0		82·1 ^b	9.2	61.6	4.4	57-9 ^a	3-0	58-6 ^h	3.4	57-2	5.0
33-9 1-3	1:3		33-6	1.6	34·1	2.0	31-3	Ē	30-9	1·5	31·8	1-7
	0.5		6-2	1-0	6-5	0.6	5.9	0-3	5.9	0·3	6-0	0.6
	0.2		3.2	0.2	3.7	0-3	3:2	0·1	3.1	0.2	3-3	0.2
	2·8		39-0	5:3	29-6	2.6	28·8	1.5	28-9)·(27-2	0.4
	0.8		15.7	١٠	16.6	1-0	15.1	0.5	15-2	0·7	15.1	0.8
	1·6		$24.8^{\rm b}$	2.9	18-9	1.6	15·7ª	ĿI	16·6 ^b	1.5	14-9	1-7
	0.5		10-3	6-0	10.6	0·0	8-6 ^a	0-0	8.7	0.8	8.5°	0.8
	0-03		0.34	0.04	0-37	0.04	0.41	0.03	0-41	0.04	0-41	0.04
1.62 ^a 0.09	000	~	1-63	0.16	1.61	0.10	1-88ª	60.0	1.88	0.12	1-88	0.15
	0.20		1-9 ^b	0-3	1-7	0.3	1·2ª	0·14	l·l ^b	0-2	1-3	0.2

Table 3. Energy and nutrient intakes of Spanish elderly grouped according to parental cause of death*

264

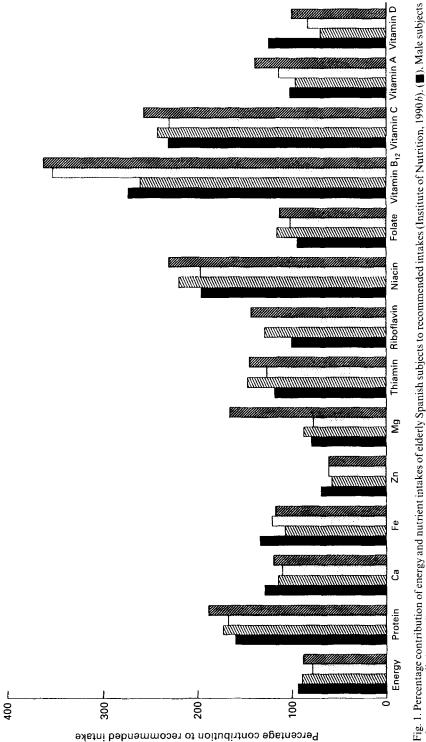
R. M. ORTEGA AND OTHERS

Palmitic acid (g/d)		0·8	13-9 ^b	<u> </u>	11	0·8	9.5ª	0-0	9.8	0·8	9.1	0·8
Stearic acid (g/d)		0.4	5.5	0-7	4.2	0.4	3.9	0.4	4·3	0.7	3.5	0-4
Oleic acid (g/d)		2-7	36-7	5.2	28-3	2.5	27-2	1:5	28-2	1.8	26·2	2:4
Linoleic acid (g/d)		0.5	6.7	6-0	5.5	0-5	5.2	0·4	5:3	0.5	5-0	0.6
Cholesterol: (mg/d)		17-9	283.6	18-0	219-2	26.4	229-9	13.6	245.8	20-4	215-0	18.1
(mg/MJ)		9-6	138-3	12.8	137.0	13-9	140-4	8.2	145.2	12·0	135.9	11-5
Animal fat (g/d)		3.0	36.3	5.7	29-8	3.2	24·4ª	2.0	27-5	2:7	21-4	2.8
Vegetable fat (g/d)		3.6	40·2	7-5	33-2	3:3	31-7	2.2	32-5	24	30.9	3.7
Dietary fibre (g/d)		1-4	20-3	1-9	21-1	2:2	22-5	1.5	21.9	2.2	23.0	2.4
Ca (mg/d)		52.9	792-9	95.9	9-662	62-6	752-2	43.0	662-9	57-3	836.6	58·1
Fe (mg/d)		0.5	13-3	0.7	10.7	C-0	11-9	0.5	12-1	0-8	11.7	0-7
Zn (mg/d)		0.4	10-3	0.6	8.6	0.5	9.1	0.4	9.1	0.5	9-1	0-5
Mg (mg/d)		11-4	275-0	19.0	262-8	14-5	274.0	13·1	269-3	18.5	278-4	18-9
Thiamin (mg/d)		0·1	ĿI	0-1	I·1	0·1	1:2	0.1	1·2	0·1	1.2	0·1
Riboflavin (mg/d)	14	0·1	14	0.2	1-4	0.1	1-5	0·1	l·3	0.1	1.6	0·1
Niacin (mg/d)		1:2	31.6	1·8	23·1	l 4	30-0	1·2	29-8	1-4	30·1	1.9
Folate $(\mu g/d)$		19-2	187.8	15.0	231-0	30-8	214-4	14·1	202-7	20-7	225-4	19-5
Vitamin B., (µg/d)		6-0	5:4	6-0	5.2	١·S	7·1	1.4	7-0	1-9	7-2	2.2
Vitamin C (mg/d)		11.1	138-3	13·4	145.2	16.6	146.2	13-6	137-9	16·2	154-0	21-9
Vitamin A $(\mu g/d)$		55.1	764.7	0-62	718-1	77-3	950.6	166.0	852-9	174-7	1042-9	281·1
Vitamin D $(\mu g/d)$		0·4	3.1	0-7	1.7	0-5	2:3	0-5	2·1	0.5	2.5	0.8
	10 million (10 mil											

^{a,b,c} Mean values in horizontal rows with the same superscript letter were significantly different: P < 0.05. PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids. * For details of subjects and procedures, see Table 1 and pp. 259–260.

PARENTAL DEATH AND NUTRITION OF DESCENDANTS

265



whose parents died of cardiovascular disease; (Z), female subjects whose parents died of cardiovascular disease; (Z), male subjects whose parents died of other causes; (S), female subjects whose parents died of other causes. For details of subjects and procedures, see Table 1 and pp. 259–260. For details of energy and nutrient intakes, see Table 1 and pp. 259–260. For details of energy and

C elderly had higher intakes of myristic (P < 0.05) and palmitic (P < 0.05) acids, slightly lower PUFA:SFA (not significant) and lower MUFA:SFA (P < 0.05) than group NC elderly.

Comparison within sex showed that group C males had higher energy (P < 0.05), carbohydrates (P < 0.05) and fat (P < 0.05) intakes than group NC males. There were no significant differences between females in groups C and NC.

DISCUSSION

In the elderly, hypertension is a major risk factor for cardiovascular disease (Nikkila & Heikkinen, 1990). Both the Framingham Study and the Chicago Stroke Study (Harris *et al.* 1988) have shown that untreated hypertension in older individuals is strongly associated with an increased risk of stroke and cardiovascular disorders (Tuck *et al.* 1988). Our finding that group C elderly had a higher mean diastolic blood pressure than group NC elderly (Table 1) is consistent with previous findings.

The results of the anthropometric study (Table 1) are similar to those found in other studies (Lemonnier *et al.* 1991). Although obesity has been considered to be a cardiovascular and hypertension risk factor (McCarron & Kotchen, 1983; Beilin, 1988), we did not find any difference in the Quetelet index between groups C and NC elderly. But the role of obesity as a cardiovascular risk factor is not as clear as that of some of the other factors (Alfin-Slater & Kritchevsky, 1990). To date obesity is seen as a cardiovascular risk factor, primarily because of its association with other risk factors such as hypertension, hypercholesterolaemia and diabetes (Harris *et al.* 1988).

Cholesterol and HDL-cholesterol levels were similar to those observed in other European elderly (De Groot *et al.* 1992), but were only slightly higher in group C elderly than in group NC elderly (Table 1).

The relationship between total energy intake and cardiovascular disease has been examined in several studies. Some authors have indicated that subjects who develop heart disease have a history of lower total energy intake, on average, compared with those who remain free of the disease (Lapidus *et al.* 1986). Other authors have found that total energy correlates positively with the mortality rates from coronary heart disease (Connor & Connor, 1986; Ginter, 1986; Williams *et al.* 1988; Nissinen & Stanley, 1989; Shah *et al.* 1990). In our study group C males had higher energy intakes than group NC males (Table 3). The results are similar to those obtained in several European countries by means of the EURONUT-SENECA study (De Groot *et al.* 1992). Also, in these populations men had higher energy intakes than women (De Groot *et al.* 1992).

The greatest differences in intakes between groups C and NC elderly were total fat, saturated fat and animal fat, which were higher among group C elderly (Table 3). Fat intake has been reported to be associated with hypercholesterolaemia and hypertension (McCarron & Kotchen, 1983; Beilin, 1988; Williams *et al.* 1988; Pietinen *et al.* 1989; Alfin-Slater & Kritchevsky, 1990) and also with a higher risk of mortality from coronary heart disease and stroke (Shimamoto *et al.* 1989).

Increasing PUFA:SFA has been suggested to reduce blood pressure (Puska *et al.* 1983). A negative association has also been demonstrated between increased consumption of MUFA and both systolic and diastolic blood pressure (Williams *et al.* 1987).

With regard to serum cholesterol, polyunsaturated and monounsaturated fats appear to be equally effective in reducing LDL-cholesterol levels when substituted for saturated fats in the diet. Their effect on HDL-cholesterol may differ, however. In metabolic ward studies using high-fat formula diets, polyunsaturated fats have reduced HDL-cholesterol, while monounsaturated fats have not (Dreon, 1990).

Based on these findings it is concluded that group NC elderly had a better status than

267

R. M. ORTEGA AND OTHERS

group C elderly, since their PUFA: SFA (P < 0.1) and MUFA: SFA (P < 0.05) values tended to be higher than those of group C elderly (Table 3).

All saturated fatty acids may not have the same effect on plasma cholesterol levels. Stearic acid is less hypercholesterolaemic than other saturated fatty acids (Cobbs, 1992). The major cholesterol-raising saturated fatty acids in the diet are palmitic acid and myristic acids (Ginter, 1986; Grundy & Denke, 1990). Group C elderly had significantly higher intakes of both fatty acids than group NC elderly (Table 3).

The energy intakes from saturated fat of 51.7% of group C and 34.2% of group NC were more than 10% of the total energy intake; 75.9% of group C and 62.9% of group NC had fat intake higher than 30% of total energy intake. In addition, 38% of group C and 20% of group NC had cholesterol intakes higher than 300 mg/d. These target values (saturated fat < 10% total energy intake, fat < 30% total energy intake, cholesterol < 300 mg/d) are based mainly on what is widely considered to be an ideal nutritional pattern for the prevention of cardiovascular diseases (National Cholesterol Education Program, 1989). From these findings the dietary intakes of group NC elderly appear to be closer to the recommendations designed to prevent or treat cardiovascular disease (Connor & Connor, 1986).

Nutrient and energy intakes were similar to those found in other elderly people (Moreiras-Varela et al. 1986), although saturated fat and cholesterol intakes were slightly lower than those in other studies (Nes et al. 1992).

In our study there were differences between the intakes of group C and NC males, but not between group C and NC females. Although we need more data to determine whether there are sex differences, some authors recognize that women enjoy a natural relative immunity to coronary atherosclerosis compared with their male counterparts because of their sex hormone status, especially in the premenopausal years. Thus, it may be more important for men to adopt a behaviour that reduces their risk of coronary artery disease (hypertension, hypercholesterolaemia; Hazzard, 1989). Other authors (Plaza et al. 1990) indicate that the influence of parental death from cardiovascular disease on the serum lipid levels of their descendants is different depending on whether it was the father or the mother who suffered from cardiovascular disease, observing a greater influence if it was the father who died from cardiovascular disease.

It is clear that there are dietary differences between group C and group NC elderly which can determine cardiovascular diseases (Connor & Connor, 1986). Even though our study deals with elderly who have survived over 65 years without cardiovascular diseases and other pathologies, their dietary habits show differences with respect to their parents' cause of death. It is possible that there may be genetic or cultural factors (dietary habits learned during childhood) which favour cardiovascular disease, predisposing the individuals to follow a diet which favours the development of these pathologies (Brunzell & Austin, 1990; Rozin, 1990).

This work was supported by grants of the Fondo de Investigaciones Sanitarias de la Seguridad Social (FISss, Spain).

REFERENCES

serum cholesterol. Clinical Chemistry 20, 470-475.

Beilin, L. J. (1988). The fifth Sir George Pickering Memorial Lecture: epitaph to essential hypertension - a preventable disorder of known aetiology? Hypertension 6, 85-96.

Alfin-Slater, R. B. & Kritchevsky, D. (1990). Nutrition and cardiovascular disease. In Geriatric Nutrition, pp. 269–280 [J. E. Morley, Z. Glick and L. Z. Rubinstein, editors]. New York: Raven Press. Allain, C. C., Poon, L. S., Chan, C. S. G., Richmond, W. & Fu, P. C. (1984). Enzymatic determination of total

- Brunzell, J. D. & Austin, M. A. (1990). Individuality, hyperlipidemia, and premature coronary artery disease. World Review of Nutrition and Dietetics 63, 72–83.
- Bucolo, D. & David, H. (1973). Quantitative determination of serum triglycerides by the use of enzymes. Clinical Chemistry 19, 476–482.
- Burstein, M., Scholnick, H. & Morfin, R. (1970). Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *Journal of Lipid Research* 11, 583–594.
- Cavalli-Sforza, L. L. (1990). Cultural transmission and nutrition. World Review of Nutrition and Dietetics 63, 35-48.
- Cobbs, T. K. (1992). Effects of dietary stearic acid on plasma cholesterol levels. Southern Medical Journal 85, 25–27.
- Connor, W. E. & Connor, S. L. (1986). Dietary cholesterol and fat and the prevention of coronary heart disease: risks and benefits of nutritional change. In *Diet and Prevention of Coronary Heart Disease and Cancers*, pp. 113–147 [B. Hallgren, O. Levin, S. Rössner and B. Vessby, editors]. New York: Raven Press.
- De Groot, L., Hautvast J. G. A. J. & van Staveren, W. A. (1992). Nutrition and health of elderly people in Europe: the EURONUT-SENECA Study. *Nutrition Reviews* 50, 185–194.
- Dreon, D. M. (1990). The effects of polyunsaturated fat vs monounsaturated fat on plasma lipoproteins. Journal of the American Medical Association 263, 2462–2466.
- Expert Committee of the Spanish Ministry of Health (1991). Consenso para el control de la hipertensión arterial en Espana (Consensus meeting for the control of arterial hypertension in Spain). Sistole 63, 1–14.
- Freedman, D. S., Srinivasan, S. R., Shear, C. L., Franklin, F. A., Webber, L. S. & Berenson, G. S. (1986). The relation of apolipoproteins A-I and B in children to parental myocardial infarction. *New England Journal of Medicine* 315, 721–726.
- Friedewald, W. T., Levy, R. J. & Fredrickson, D. S. (1972). Estimation of the concentration of the low-densitylipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clinical Chemistry* 18, 499–502.
- Ginter, E. (1986). Ernährung und atherosklerose (Nutrition and atherosclerosis). Ernährungsforschung 31, 49–52.
 Grundy, S. M. & Denke, M. A. (1990) Dietary influences on serum lipids and lipoproteins. Journal of Lipid Research 31, 1149–1172.
- Harris, T., Cook, E. F., Kannel, W. B. & Goldman, L. (1988). Proportional hazards analysis of risk factors for coronary heart disease in individuals aged 65 or older. *Journal of the American Geriatrics Society* **36**, 1023–1028.
- Hazzard, W. R. (1989). Why do women live longer than men? Biologic differences that influence longevity. Postgraduate Medicine 85, 271-278, 281-283.
- Institute of Nutrition (1990a). Spanish Food Composition Tables. Madrid: Institute of Nutrition.
- Institute of Nutrition (1990b). Tables of Recommended Intakes of Energy and Nutrients for the Spanish Population. Madrid: Institute of Nutrition.
- Katsouyanni, K., Skalkidis, Y., Petridou, E., Polychronopoulou-Trichopoulou, A., Willet, W. & Trichopoulos, D. (1991). Diet and peripheral arterial occlusive disease: the role of poly-, mono- and saturated fatty acids. *American Journal of Epidemiology* 133, 24–31.
- Lapidus, L., Anderson, H., Bengtsson, C. & Bosaeus, I. (1986). Dietary habits in relation to incidence of cardiovascular disease and death in women: a 12-year follow-up of participants in the population study of women in Gothenburg, Sweden. American Journal of Clinical Nutrition 44, 444-448.
- Lemonnier, D., Acher, S., Boukaïba, N., Flament, C., Doucet, C., Piau, A. & Chappuis, P. (1991). Discrepancy between anthropometry and biochemistry in the assessment of the nutritional status of the elderly. *European Journal of Clinical Nutrition* 45, 281–286.
- McCarron, D. A. & Kotchen, T. A. (1983). Nutrition and blood pressure control current status of dietary factors and hypertension. *Annals of Internal Medicine* **98**, 699–890.
- Moreiras-Varela, O., Ortega, R. M., Ruiz-Roso, B. & Varela, G. (1986). Nutritional status of an institutionalised elderly group in Segovia (Spain). *International Journal of Vitamin and Nutrition Research* 56, 109-117.
- National Cholesterol Education Program (1989). Report of the Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. DHHS Publication no. (NIH) 89–2925. Bethesda, MD: National Institutes of Health.
- Nes, M., Andersen, L. F., Solvoll, K., Sandstad, B., Hustvedt, B. E., Lovo, A. & Drevon, C. A. (1992). Accuracy of a quantitative food frequency questionnaire applied in elderly Norwegian women. *European Journal of Clinical Nutrition* 46, 809–821.
- Nikkila, M. & Heikkinen, J. (1990). Serum cholesterol, high-density lipoprotein cholesterol and five years survival in elderly people. *Age and Ageing* **19**, 403–408.
- Nissinen, A. & Stanley, K. (1989). Unbalanced diets as cause of chronic diseases. American Journal of Clinical Nutrition 49, 993–998.
- Oliveria, S. A., Ellison, R. C., Moore, L. L., Gillman, M. W., Garrahie, E. J. & Singer, M. R. (1992). Parent-child relationships in nutrient intake: The Framingham Children's Study. *American Journal of Clinical Nutrition* 56, 593–598.
- Pietinen, P., Vartiainen, E., Korhonen, J. J., Kartovaara, L., Uusitalo, U., Tuomilehto, J. & Puska, P. (1989). Nutrition as a component in community control of cardiovascular disease (The North Karelia Project). *American Journal of Clinical Nutrition* 49, 1017–1024.
- Plaza, I., Mariscal, R. P., Muñoz, M. T., Ross-Jellici, J., López, D., Madero, R., Hidalgo, I., Cenal, M. J., Baeza,

R. M. ORTEGA AND OTHERS

J., Cobaleda, A., Frutos, A., Ruiz-Jarebo, C., Dominguez, J., Puga, M., Otero, J., Asensio, J., Orellane, M. A., Sánchez, J. & Parre, M. I. (1990). Fuenlabrada Study: Association between lipids and lipoprotein levels in children and adolescents and the prevalence of coronary heart disease in their families. *Revista Española de Cardiología* 43, 212–218.

- Puska, P., Nissinen, A., Vartianinen, E., Dougherty, R., Mutanen, M., Iacono, J. W., Korhonen, H. J., Piennen, P., Leino, U., Moisio, S. & Huttunen, J. (1983). Controlled randomized trial of the effect of dietary fat on blood pressure. *Lancet* i, 1–5.
- Rozin, P. (1990). Acquisition of stable food preferences. Nutrition Reviews 48, 106-113.
- Rudman, D. (1989). Nutrition and fitness in elderly people. American Journal of Clinical Nutrition 49, 1090–1098.
- Shah, M., Jeffery, R. W., Laing, B., Savre, S. G., Natta, M. V. & Strickland, D. (1990). Hypertension prevention trial (HPT): food pattern changes resulting from intervention on sodium, potassium, and energy intake. *Journal* of the American Dietetic Association **90**, 69–76.
- Shear, C. L., Webber, L. S., Freedman, D. S., Srinivasan, S. R. & Berenson, G. S. (1985). The relationship between parental history of vascular disease and cardiovascular disease risk factors in children: The Bogalusa Heart Study. *American Journal of Epidemiology* 122, 762–771.
- Shimamoto, T., Komachi, Y., Inada, H., Doi, M., Iso, H., Sato, S., Kitamura, A., Iida, M., Konishi, M., Nakanishi, N., Terao, A., Naito, Y. & Kojima, S. (1989). Trends for coronary heart disease and stroke and their risk factors in Japan. *Circulation* 79, 503-515.
- Simopoulos, A. P. (1989). Introduction and conference resolutions of First International Conference on Nutrition and Fitness. American Journal of Clinical Nutrition 49, 917–927.
- Tuck, M. I., Griffiths, R. F., Johnson, L. E., Stern, N. & Morley, J. E. (1988). UCLA Geriatric Grand Rounds. Hypertension in the elderly. *Journal of the American Geriatrics Society* 36, 630–643.
- Williams, P. T., Fortmann, S. P., Terry, R. B., Garay, S. C., Vranizan, K. M., Ellsworth, N. & Wood, P. D. (1987). Associations of dietary fat, regional adiposity, and blood pressure in men. *Journal of the American Medical Association* 257, 3251–3256.
- Williams, R. R., Hunt, S. C., Hasstedt, S. J., Berry, T. D., Wu, L. L., Barlow, G. K., Stults, B. M. & Kuida, H. (1988). Definition of genetic factors in hypertension: a search for major genes, polygenes, and homogeneous subtypes. *Journal of Cardiovascular Pharmacology* 12, S7–S20.
- Wilson, P. W., Abbott, R. D., Garrison, R. J. & Castelli, W. P. (1981). Estimation of very low density lipoprotein cholesterol from data on triglyceride concentration in serum. *Clinical Chemistry* 27, 2008–2010.
- Wonnacott, H. W. & Wonnacott, R. J. (1977). Introductory Statistics, New York: John Wiley and Sons.