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ORIGINAL ARTICLE

Dietary fat intake and the risk of osteoporotic fractures in the elderly

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Objective: To explore the association between fat intake, serum lipids and the risk of osteoporotic fractures in the elderly.
Design: A hospital-based case–control study.
Setting: The study was conducted at a tertiary centre and referral hospital for the province of Jaén (Spain).
Subjects: Cases ($n=167$) were patients aged 65 years or more with a low-energy fracture selected from the population attended at the hospital. Controls (patients without antecedents of any fracture) were 1:1 matched to cases by sex and age ($n=167$).
Methods: Diet was assessed by a semiquantitative food frequency questionnaire. Serum total cholesterol and high-density lipoprotein (HDL) cholesterol were also measured.
Results: Participants in the two upper quartiles of polyunsaturated fat (PUFA) intake showed an increased risk of fracture, with statistically significant differences with respect to the first quartile in the adjusted model (odds ratio (OR) = 3.59; 95% confidence interval (CI) = 1.06–12.1 and OR = 5.88; 95% CI = 1.38–25.02); $P=0.01$ for the trend test). A higher ratio of monounsaturated fat (MUFA) to PUFA was associated with a reduced risk of fracture (OR = 0.20; 95% CI = 0.07–0.60 for the fourth quartile; $P=0.002$ for the trend test). The intake of omega-6 fatty acids was associated with an elevated risk of fracture (OR = 3.41; 95% CI = 1.05–11.15 for the fourth quartile; $P=0.01$ for the trend test). HDL-cholesterol levels were inversely associated with the risk of fracture (test for trend $P=0.03$ across quartiles).
Conclusions: PUFA intake was associated with an increased risk of osteoporotic fractures in the elderly, whereas a high ratio of MUFA:PUFA was associated with decreased risk.
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Keywords: osteoporosis; fractures; monounsaturated fat

Introduction

Osteoporotic fractures are a major public health problem in Western societies. The worldwide number of hip fractures per annum will increase between 1990 and 2050 from 1.6 million to 6 million (Keen, 2003). This growup is related to an increase in the number of elderly people in the population, improved survival and an increase in the age-specific fracture rates. The rising number of osteoporotic fractures and their associated morbidity place a heavy burden on the welfare of the elderly population as well as on the health-care resources. Efficient preventive approaches are needed.

It is worth noting that two major chronic diseases of the elderly, namely osteoporosis and coronary heart disease (CHD), may share similar determinants. Thus, there is evidence available to support that: (a) smoking is associated with a higher risk of fractures (Kanis *et al*., 2005); (b) an adverse serum lipid profile for CHD (high low-density

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lipoprotein-cholesterol level) is also associated with a higher risk of osteoporotic fractures (Hsu *et al*., 2006); (c) homocysteine concentration is a risk factor for hip fracture in older persons (McLean *et al*., 2004); (d) fat mass is related to a lower bone density and to a higher risk of fractures (Hsu *et al*., 2006).

In addition, intriguing evidence suggests that common genetic determinants may exist for both cardiovascular and skeletal diseases (Qi *et al*., 2003). These genes code for several key factors involved in the metabolism of nutrients, such as lipids and folate, that are under dietary modulation. In this context, nutritional factors, and specifically the dietary intake of fats, are very good candidates to represent modifiable determinants of the risk of osteoporotic fractures.

Several mechanistic explanations lead to the expectation that fat intake may exert an important effect on bone density and remodelling through changes in lipid oxidation (Parhami *et al*., 1999, 2000), metabolism of prostaglandins, induction of cytokines, and regulation of calcium and insulin-like growth factor I (Miyamoto *et al*., 2003).

However, the available epidemiological literature about the association between the intake of fat and the risk of osteoporotic fractures is scarce. Total fat intake (Kato *et al*., 2000; Macdonald *et al*., 2004) and, more specifically, saturated fat intake (Corwin *et al*., 2006) have been reported to be associated with a higher risk of bone loss and osteoporotic fractures. However, a previous study conducted in a Mediterranean country has reported a *beneficial* role for monounsaturated fat (MUFA) (Trichopoulou *et al*., 1997), whereas some studies have also found associations between high polyunsaturated fatty acids (PUFA) intake and lower bone density (Macdonald *et al*., 2004; Weiss *et al*., 2005). We evaluated the association between fat intake, serum lipids and the risk of osteoporotic fractures using a case–control design. Our hypothesis was that the type of fat intake would be influential in determining the risk of osteoporotic fractures in the elderly. We expected to find a lower risk of fractures associated with a MUFA-rich diet, whereas a PUFA-rich or saturated fatty acid-rich diet would be detrimental.

Methods

A hospital-based case–control study was conducted at the Hospital of Jaén, tertiary centre and referral hospital for the whole province of Jaén (Southern Spain). The study was approved by the Ethics Committee of the hospital. The study population was recruited from January 2002 to December 2003.

Cases were defined as patients aged 65 years or more with a low-energy fracture occurring in the period 6–24 months before inclusion in the study. We chose only elderly patients with a 'low-energy fracture' in order to obtain a higher specificity for osteoporotic fractures in our case series. For the present study, a low-energy fracture was defined as the fracture produced by a same-level fall. The exclusion criteria were the following: fracture in the previous 6 months, neoplasm (any site), infection after the fracture, complicated course requiring hospitalisation for a stay longer than 3 weeks, clinical vitamin C deficit, inability to answer the questionnaire and a score of <16 in the mini-mental state test (Folstein *et al*., 1975). Cases were selected from the population first attended to at the same hospital. A total of 431 patients potentially eligible as cases were contacted by ordinary mail, of whom 163 did not answer, 63 reported their inability to attend the study and 205 agreed to participate. Thirty-eight out of these 205 patients were excluded: 21 because of an inadequate cognitive level, 10 because of complications after fracture, four having cancer and three because of deafness. The final case series was constituted by 167 patients with fractures (35 hip, 19 pelvis, 14 vertebral, eight humerus, 44 wrist, 30 ankle and 17 other fractures).

Controls were outpatients attending the service of otolaryngology, without antecedents of any fracture in the previous 5 years, and coming from the same reference area as cases. Identical exclusion criteria were applied for the controls. Controls were 1:1 matched to cases by sex and age (± 5 years). A total of 243 potentially eligible controls were contacted by mail: 47 refused to participate or did not attend the appointment and 196 agreed to participate. Twenty-nine out of these 196 were excluded (12 because of inadequate cognitive level, eight having deafness, five with cancer and four because of a hospital stay during the last 6 months). A total of 167 controls were included in the study.

Data were collected by two trained interviewers. Information was gathered on sociodemographic characteristics (occupation, marital status, level of education), cognitive level (mini mental state test; Folstein *et al*., 1975), alcohol consumption and smoking. Smoking was assessed according to self-reports and it was categorised in three levels ('never smoker' for those who had smoked <100 cigarettes during their lifetime; 'ex-smoker' for those who had quit at least 6 months before, and 'current smokers'). Information on medical conditions (underlying disease, visual disability or eye disability) was gathered through a medical examination at the hospital. We enquired about 17 activities to quantify the amount of physical activity and a metabolic equivalent index (METs-h/week) was computed using a questionnaire previously validated in Spain (Martínez-González *et al*., 2005). The number of steps and stairs to access home was assessed.

Functional status was evaluated by Katz's index of activities of daily living (Katz *et al*., 1963, 1970). Diet was assessed by a 136-item semiquantitative food frequency questionnaire, previously validated for the Spanish population (Martín Moreno *et al*., 1993; Martínez-González *et al*., 2002), based on the Harvard–Willett questionnaire (Willett *et al*., 1985). Intake of vitamin supplements was also ascertained. Frequencies of consumption for each item were measured in nine categories (6 + per day, 4–6 per day, 2–3 per day, 1 per day, 5–6 per week, 2–4 per week, 1 per week, 1–3

per month, never or almost never for each food item). Nutrient intake scores were computed using an *ad hoc* computer program specifically developed for this aim. Nutrient scores were calculated as frequency × nutrient composition of specified portion size. A set of colour pictures was also shown to the participants to better establish the portion size of each food. A trained dietician updated the nutrient data bank using the latest available information included in food composition tables for Spain (Mataix, 2003; Moreiras, 2003). All dietary intakes were adjusted for total energy intake using the residuals method (Willett and Stampfer, 1998).

A blood sample was drawn from every participant in standard conditions after 10 h of fasting. Total cholesterol and high-density lipoprotein (HDL) cholesterol were measured in a fasting blood sample enzymatically with a Synchron LX20pro (Beckman Coulter, Fullerton, CA, USA) analyser.

In the statistical analysis, we first verified the matching conditions. Despite matching, a statistically significant difference of age between cases and controls was found (73.2 vs 71.2 years; $P< 0.05$). Therefore, all the analyses were adjusted for age. Means and standard deviations were estimated for continuous variables (nutrient intake, blood levels) in cases and controls. Age-adjusted means were compared using analysis of covariance. Fat intake and the intake of the different types of fat were categorised in quartiles according to the observed distribution in controls. The lowest quartile of intake was taken as the reference and ORs, and their 95% CIs for each of the three upper quartiles were estimated after adjusting for age by conditional logistic regression. Trend analyses across quartiles were performed introducing the median intake observed for each quartile as a continuous variable in the logistic regression model.

Multivariable analyses were also fitted to adjust for confounding, using analysis of covariance (for the comparison of adjusted means) and conditional logistic regression (for estimating adjusted ORs). We took into account previous knowledge and available evidence about known causes (Hernán *et al*., 2002) of osteoporotic fractures and low bone density to select the variables to be introduced in the multivariable models. All nutrients were adjusted for energy using the residuals method.

All the statistical analyses have been performed with the program Stata 8-SE (College Station, TX, USA).

Results

The study population consisted mostly of women (80%). There were no differences between cases and controls according to marital status (married 71.3% cases vs 67.1% controls; $P=0.477$), alcohol consumption (mean ± s.e.m.; 3.0 ± 0.6 g/d in controls vs 2.8 ± 0.7 in controls; $P=0.858$) and physical activity (5.4 ± 0.8 METS in cases vs 5.2 ± 0.7 in controls; $P=0.858$). Cases had a worse vision (29.9 vs 18.5% with visual disability; $P=0.024$) and worse audition (58.1 vs 39.5% with ear disability; $P=0.001$). Controls were more frequently independent in respect of their activities of daily living, 5.4% showing dependence on at least one activity vs 16.2% of cases ($P=0.004$). There were more controls dwelling at homes without steps in their access (19.8 vs 9.6%; $P=0.022$).

Table 1 Adjusted means of nutrient intake and serum lipids in cases and controls

	Mean ± s.e.m.		P
	Cases	*Controls*	
Daily intake			
Total energy (kJ)[a]	10858 ± 226 (2594 ± 54 kcal)	9950 ± 222 (2377 ± 53 kcal)	0.006
Carbohydrates (g)[b]	296 ± 3.5	288 ± 3.5	0.128
Proteins (g)[b]	104 ± 1.3	107 ± 1.3	0.140
Total fat (g)[b]	97 ± 1.5	99 ± 1.5	0.453
MUFA (g)[a]	44 ± 1.0	47 ± 1.0	0.050
PUFA (g)[a]	16 ± 0.3	14 ± 0.3	0.012
Ratio MUFA/PUFA[a]	3.0 ± 0.1	3.4 ± 0.1	0.001
SFA (g)[a]	28 ± 0.6	29 ± 0.6	0.046
Ratio of non-SFA/SFA[a]	2.1 ± 0.1	2.2 ± 0.1	0.650
Serum lipids (mg/dl)			
Total cholesterol[b]	215.2 ± 2.9	216.9 ± 2.9	0.700
HDL cholesterol[a]	52.9 ± 1.1	54.6 ± 1.1	0.290
Total cholesterol / HDL[b]	4.3 ± 0.1	4.2 ± 0.1	0.687

Abbreviations: HDL, high-density lipoprotein; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.
[a]Adjusted for age, calcium intake, vitamin C serum level, physical activity (METS-h/week), underlying disease, number of stairs at home, Katz's index and ear disability.
[b]Adjusted for age, total energy intake, MUFA and PUFA intake, calcium intake, vitamin C serum level, physical activity (METS-h/week), underlying disease, number of stairs at home, Katz's index and ear disability.

The adjusted means of macronutrients, type of fats and serum lipids are displayed in Table 1. Energy intake was higher among cases than controls, with a higher carbohydrate and PUFA intake.

The relationship between macronutrient intake and the risk of fracture is shown in Table 2. No significant association was observed with either carbohydrates, proteins or total fat intake.

In Table 3, we show the observed associations between the intake of each type of fat and the risk of fracture. The point estimates of the OR for the three upper quartiles of MUFA intake compared with the first quartile were lower than one, suggesting a threshold, that is the suboptimal intake of MUFA (first quartile) was associated with a higher risk of fracture, but the results were statistically significant only for the comparison between the first and the second quartile and the trend test did not show evidence of a significant decreasing trend. Contrarily, participants in the two upper quartiles of PUFA intake (⩾15 g/day) showed an increased risk of fracture, with statistically significant differences with respect to the first quartile (<10 g/d) in the fully adjusted

Table 2 Association between macronutrient intake and the risk of low-energy fractures

Variable (daily intake)	*Cases* n *(%)*	*Controls* n *(%)*	*OR (CI 95%)*[a]
Carbohydrates (g)			
<258	34 (20.4)	42 (25.2)	1 (reference)
258–289	34 (20.4)	42 (25.2)	2.14 (0.78–5.91)
290–317	38 (22.7)	41 (24.6)	2.35 (0.79–6.94)
⩾317	61 (36.5)	42 (25.2)	2.12 (0.71–6.28)
Linear trend			$P=0.182$
Proteins (g)			
<95	43 (25.7)	42 (25.2)	1 (reference)
95–105	34 (20.4)	42 (25.2)	0.74 (0.28–1.96)
106–117	33 (19.8)	41 (24.6)	0.74 (0.30–1.80)
⩾118	57 (34.1)	42 (25.2)	0.92 (0.31–2.70)
Linear trend			$P=0.722$
Fats (g)			
<87	35 (21)	42 (25.2)	1 (reference)
87–97	34 (20.4)	42 (25.2)	1.20 (0.48–3.03)
98–112	53 (31.7)	41 (24.6)	0.59 (0.23–1.50)
⩾112	45 (26.9)	42 (25.2)	1.32 (0.50–3.52)
Linear trend			$P=0.690$

[a]Adjusted for total energy intake, age, calcium intake, vitamin C serum level, physical activity (METS-h/week), underlying disease, number of stairs at home, Katz's index and ear disability.

model (OR = 3.59; 95% CI = 1.06–12.1 for the third quartile and OR = 5.88; 95% CI = 1.38–25.02 for the fourth quartile); in addition, the test for linear trend was statistically significant ($P=0.01$). A higher ratio of MUFA:PUFA was associated with a substantially reduced risk of fracture (OR = 0.20; 95% CI = 0.07–0.60 for the fourth quartile; $P=0.002$ for the trend test). We found no relationship of osteoporotic fractures with the intake of saturated fat, with the intake of omega 3 fatty acids or with the ratio of omega 3 to omega-6 fatty acids. However, the intake of omega-6 fatty acids was positively associated with an elevated risk of fracture (OR = 3.41; 95% CI = 1.05–11.15 for the fourth quartile; $P=0.01$ for the trend test).

The association between serum lipids and the risk of fracture was also analysed (Table 4). Total cholesterol was unrelated to the risk of fracture and no trend was apparent. HDL cholesterol was inversely associated with the risk of fracture. We found marginally significant results for those in the two upper quartiles of HDL (OR = 0.38; 95% CI:0.08–1.21 and OR = 0.29; 95% CI = 0.08–1.09 for the third and fourth quartiles, respectively); however, the test for trend was statistically significant ($P=0.03$). The ratio of total to HDL cholesterol showed an association with a higher risk of fracture for the two upper quartiles, although the trend test was not statistically significant.

Discussion

In this case–control study assessing an elderly Mediterranean population, we found that a high intake of PUFA (particularly *n*-6) was associated with a higher risk of osteoporotic fractures. However, a high MUFA intake was not associated with higher risk, and the ratio of MUFA:PUFA exhibited a significant inverse association. It is likely that the MUFA:PUFA ratio possibly reflects the effects of *n*-6 PUFA more than those of MUFA, but the point estimates of relative risk for higher levels of MUFA intake were lower than 1, suggesting a protection. In addition, these results are consistent with a previous cross-sectional study conducted in Greece, which reported that MUFA intake was associated with a higher bone mineral density (Trichopoulou *et al*., 1997). However, to our knowledge, no previous study has specifically assessed

Table 3 Association between the intake of specific fatty acids and the risk of low-energy fractures. Multivariable analysis

Variable (daily intake)	*Cases* n *(%)*	*Controls* n *(%)*	*OR (CI 95%)*[a]
MUFA (g)			
< 39	35 (21)	42 (25.2)	1 (reference)
–46	46 (27.5)	42 (25.2)	0.27 (0.11–0.69)
47–54	41 (24.5)	41 (24.5)	0.65 (0.24–1.79)
⩾54	45 (27)	42 (25.2)	0.52 (0.20–1.38)
Linear trend			$P=0.247$
PUFA (g)			
<11	27 (16.2)	42 (25.2)	1 (reference)
11–14	36 (21.6)	42 (25.2)	0.90 (0.28–2.92)
15–17	38 (22.7)	41 (24.5)	3.59 (1.06–12.17)
⩾18	66 (39.5)	42 (25.2)	5.88 (1.38–25.02)
Linear trend			$P=0.011$
SFA (g)			
< 23	36 (21.6)	42 (25.1)	1 (reference)
23–28	39 (23.4)	42 (25.2)	0.90 (0.36–2.28)
29–33	43 (25.7)	41 (24.6)	1.22 (0.47–3.15)
⩾34	49 (29.3)	42 (25.2)	0.80 (0.29–2.19)
Linear trend			$P=0.822$
Ratio MUFA/PUFA			
< 2.8	62 (37.1)	42 (25.2)	1 (reference)
2.8–3.3	49 (29.3)	41 (24.5)	0.44 (0.16–1.18)
3.4–3.9	27 (16.2)	42 (25.2)	0.22 (0.07–0.67)
⩾4.0	29 (17.4)	42 (25.2)	0.20 (0.07–0.60)
Linear trend			$P=0.002$
Omega-3 fatty acids (g)			
< 11	50 (29.9)	42 (25.2)	1 (reference)
11–14	42 (25.2)	41 (24.5)	0.90 (0.35–2.28)
15–17	47 (28.1)	42 (25.2)	1.47 (0.59–3.64)
⩾18	28 (16.8)	42 (25.2)	1.27 (0.47–3.39)
Linear trend			$P=0.561$
Omega-6 fatty acids (g)			
< 11	35 (21.0)	42 (25.2)	1 (reference)
11–14	32 (19.2)	41 (24.5)	0.89 (0.30–2.63)
15–17	54 (32.3)	42 (25.2)	2.35 (0.84–6.55)
⩾18	46 (27.5)	42 (25.2)	3.41 (1.05–11.15)
Linear trend			$P=0.012$

Abbreviations: MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.
[a]Adjusted for total energy intake, age, calcium intake, vitamin C serum level, physical activity (METS-h/week), types of fat, underlying disease, number of stairs at home, Katz's index and ear disability, and PUFA (when relevant).

Table 4 Serum lipids and fracture risk. Multivariable analysis

Variable	*Cases* n *(%)*	*Controls* n *(%)*	*OR (CI 95%)*[a]
Total cholesterol (mg/dl)			
< 191	30 (18.5)	42 (25.3)	1 (reference)
191–212	47 (29)	41 (24.7)	2.42 (0.80–7.34)
213–240	42 (25.9)	42 (25.3)	1.92 (0.68–5.39)
⩾241	43 (26.5)	41 (24.7)	0.94 (0.34–2.63)
Missing	5	1	
Linear trend			$P=0.285$
HDL cholesterol (mg/dl)			
< 45	31 (19.4)	43 (26)	1 (reference)
45–52	49 (30.6)	39 (23.6)	1.90 (0.60–6.75)
53–61	36 (22.5)	44 (26.7)	0.31 (0.08–1.21)
⩾62	44 (27.5)	39 (23.6)	0.29 (0.08–1.09)
Missing	7	2	
Linear trend			$P=0.033$
Total cholesterol/HDL			
⩽3.42	34 (21.3)	41 (24.8)	1 (reference)
3.43–4.04	42 (26.3)	41 (24.8)	1.80 (0.60–5.39)
4.05–5.01	51 (31.9)	43 (26.1)	3.39 (1.11–10.38)
⩾5.02	33 (20.2)	40 (24.2)	3.21 (1.05–9.77)
Missing	7	2	
Linear trend			$P=0.119$

Abbreviation: HDL, high-density lipoprotein.
[a]Adjusted for age, total energy intake, MUFA intake, PUFA intake, calcium intake, vitamin C serum level, physical activity (METS-h/week), underlying disease, number of stairs at home, Katz's index and ear disability.

the role of MUFA on the risk of osteoporotic fractures. In addition, we found that higher serum HDL–cholesterol levels were associated with lower fracture risk.

The inverse association between MUFA and fracture risk that we report here could be attributed to the high consumption of MUFA-rich olive oil, which is typical of the Mediterranean food pattern. The major fat present in olive oil is the monounsaturated 18-carbon oleic acid. A wide array of studies have shown that olive oil exerts important anti-inflammatory effects (Visioli and Galli 2002; Esposito *et al*., 2004; Beauchamp *et al*., 2005; Serrano-Martínez *et al*., 2005) and antioxidant effects (Ramírez-Tortosa *et al*., 1999; Visioli and Galli, 2002; Pitsavos *et al*., 2005), which may explain why it has been reported to be negatively associated with the risk of CHD in epidemiologic studies (Fernandez-Jarne *et al*., 2002; Barzi *et al*., 2003). These anti-inflammatory and antioxidant effects have been partly explained because of the high content of polyphenols present in virgin olive oil (Carrasco-Pancorbo *et al*., 2005).

Ageing and oestrogen deficiency induce inflammatory and oxidant conditions that are involved in the development of bone loss, osteoporosis and a higher likelihood of low-energy fractures. In this context, a sufficient load of antioxidants supplied by a diet rich in olive oil may prevent bone loss through the scavenging of free radicals. The anti-inflammatory components present in olive oil may also act by averting the increased plasma concentration of proinflammatory cytokines (interleukin-6, tumour necrosis factor-α) involved in bone resorption among postmenopausal women (Zheng *et al*., 1997).

A Mediterranean-type MUFA-rich diet can also affect bone metabolism because a MUFA-rich diet might interfere with the actions of prostaglandins. Prostaglandins, especially PGE2, stimulate bone resorption by increasing the number and activity of osteoclasts (Raisz, 1999). Most of the potent stimulators of bone resorption increase prostaglandin production in bone by induction of COX-2, and the ability of polyphenols present in olive oil to exert a dose-dependent inhibition of the enzyme COX-2 has been recently shown (Beauchamp *et al*., 2005). In addition, oleic acid is an inhibitor of prostaglandin PGE2 synthesis, the major prostaglandin involved in bone metabolism (de La Puerta Vázquez *et al*., 2004). Normal or moderate levels of PGE2 support bone formation, whereas greater quantities promote bone resorption (Watkins *et al*., 2001).

Furthermore, animal studies have shown that virgin olive oil prevents inflammation-induced bone loss (Puel *et al*., 2004), thus providing a potential mechanistic support to our findings. The reduced risk of fractures associated with high HDL-cholesterol level is consistent with the protection observed for MUFA, because a high MUFA intake, typical of the Mediterranean food pattern, has been repeatedly shown to increase HDL levels (Mensink and Katan, 1992; Mensink *et al*., 2003; Esposito *et al*., 2004).

It is not surprising that in other cultural contexts, with a very different food pattern, no association between MUFA and fracture risk (or bone density) might have been found, because a major source of MUFA in Northern European countries or the US is not olive oil, but meat and meat products. This diverging pattern of MUFA sources may explain why, contrary to our findings, a longitudinal study conducted in the UK found an inverse association between MUFA intake and change in bone mineral density (Macdonald *et al*., 2004). However, that study reported a considerably stronger detrimental effect for PUFA than for MUFA, and this is not very far from our results.

We found a higher risk association with PUFA intake and specifically with omega-6 fatty acid intake. Recent investigations indicate that the type and amount of PUFA influence bone formation and osteoblastic cell functions (Watkins *et al*., 2003), with evidence of an adverse effect for omega-6 PUFA (Watkins *et al*., 2000; Macdonald *et al*., 2004). Also, the intake of omega-6 fatty acids has been previously shown to be associated with a lower bone mineral density, and a high ratio of omega-6 to omega-3 has been reported to be detrimental for bone formation (Albertazzi and Coupland, 2002). In the Rancho Bernardo Study, after assessing bone mineral density in 1532 community-dwelling men and women aged 45–90 years, an increasing ratio of total dietary omega-6 to omega-3 fatty acids was significantly and independently associated with lower bone mineral density (Weiss *et al*., 2005). The results of the investigation conducted by Macdonald *et al*., (2004) also suggested that PUFAs are harmful to bone in women, but, to our

knowledge, no previous epidemiologic study has reported a direct association between PUFA intake and a higher fracture risk.

In summary, our results are encouraging because they suggest that MUFA intake may help to prevent osteoporosis-associated fractures in the elderly. However, further studies are needed to confirm our results.

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