

Fatty Acid Nutriture in Hospitalized Elderly Women

Anne Schmuck, PhD, Annick Villet, PhD, Nicole Payen, PhD, Josette Alary, PhD, Alain Franco, MD, and Anne-Marie Roussel, PhD, FACN

GREPO (A.S., A.V., J.A., A-M.R.), Université Joseph Fourier, La Tronche; Laboratoire Interuniversitaire de Gérologie de Grenoble (A.S., A.F.), and Laboratoire de Biochimie A (N.P.), CHU de Grenoble, FRANCE

Key words: hospitalized elderly, fatty acids, dietary intake, EFA deficiency

Objective: The aim of this study was to measure the fatty acid (FA) dietary intakes and the FA composition of plasma total lipids in a selected group of hospitalized elderly patients.

Methods: Twenty-three women aged 76 to 99 years were recruited. FA were analyzed in 5-day duplicate portions and in plasma by gas liquid chromatography.

Results: The hospitalized elderly women ingested an average of 5.22 megajoules (MJ) and 45.9 g of lipids per day. Polyunsaturated fatty acids (PUFA) represented 11.0% and saturated fatty acids (SFA) 53.6% of the lipid intake. Minimal recommendations for linoleic acid intake were reached in average, but 32% of the patients ingested less than 3 g of linoleic acid/d. Eighty-six percent received less than 0.5% of energy from α -linolenic acid and 64% had low intakes in very long-chain n-3 FA. In parallel, these patients presented several biochemical signs of essential fatty acids (EFA) insufficiency (decrease in linoleic acid, increase in monounsaturated fatty acids (MUFA), in n-7 FA and in indexes of δ -6 and δ -9 desaturase activities).

Conclusions: Hospitalized elderly patients have low PUFA intakes and show biochemical indices of EFA insufficiency. These patients might benefit from a nutritional supplementation providing both EFA and antioxidant micronutrients to limit the risk of skin troubles, immune system impairment and vascular disease often observed in institutionalized elderly subjects.

INTRODUCTION

Because of the major role played by essential fatty acids (EFA) and their derivatives in lipid metabolism, platelet functions, immune system, inflammatory response, and epidermal functions [1], it is important to assure optimal EFA intakes. An adequate balance between n-3 and n-6 families is also required [2]. Elderly people represent a population at risk of nutritional imbalance due to low energy intakes and to altered food choices [3]. A possible alteration of FA metabolism upon aging has been reported. The activity of δ -6 desaturase seems particularly concerned, as shown in animals [4,5]. These modifications could worsen the consequences of low EFA dietary intakes in older subjects. However, only few studies have been dedicated to the determination of FA intakes in the elderly in relation with biochemical indicators of EFA status. Given the possible consequences of EFA deficiency, it is important to

know the FA nutriture of populations at risk among which the institutionalized elderly. The present work was therefore undertaken to determine the actual FA dietary intakes and plasma concentrations in a group of hospitalized elderly patients.

SUBJECTS AND METHODS

Subjects

Twenty-three elderly women (mean age 86 years, range 76 to 99) hospitalized in the department of Gerontology of the Grenoble University Hospital were recruited. This population was composed of patients in rehabilitation following a femoral neck fracture and patients in long-term care (>2 months). Only those in stable medical condition without hepatic, renal, gastrointestinal, or malignant disease were selected.

Abbreviations: EFA=essential fatty acids, FA=fatty acids, FAME=fatty acid methyl esters, HDL=high-density lipoproteins, LDL=low-density lipoproteins, MJ=megajoules, MUFA=monounsaturated fatty acids, PUFA=polyunsaturated fatty acids, P/S=ratio of polyunsaturated to saturated fatty acids, RDA=recommended dietary allowances, RDI=recommended dietary intake, SFA=saturated fatty acids, TBARS=thiobarbituric acid reactive substances.

Address reprint requests to: Anne Marie Roussel, Pharm D, PhD, FACN, GREPO, UFR de Pharmacie, Domaine de la Merci, 38700 La Tronche, FRANCE.

None of the selected patients were receiving tube feeding but several of the long-stay patients needed assistance with or were dependent on others for feeding. Patients were receiving set meals composed of either regular diets or pureed foods, according to their dental or mental status. Menus were composed by dietitians to be well-balanced.

Informed consent was obtained from the subjects or from a close family member for mentally-impaired patients. The study protocol was approved by the ethics committee of the hospital.

Diet Collection and Food Analyses

The dietary study was done in the hospitalized elderly patients during 5 consecutive days using the duplicate portion technique, as described previously [6,7]. Food analyses were performed for each patient on a 5-day food composite. Dietary total lipids were extracted and measured according to the AFNOR recommendations [8]. An aliquot of the dietary fat was frozen at -20°C for FA analysis. After saponification with a solution of 0.5 mol/L of sodium hydroxide in methanol, FA were transesterified with 14% (wt/vol) boron trifluoride in methanol (Sigma, France) at 95°C for 30 minutes [9,10]. Fatty acid methyl esters (FAME) were then extracted with hexane containing 0.005% (wt/vol) butylated hydroxytoluene and analyzed by GLC using a Varian Star 3400 CX (Varian, 91941 Les Ulis-Cedex, France) fitted with a flame ionization detector and a capillary column HP INNOVAX (60m \times 0.32 mm \times 0.15 μm) (Hewlett-Packard, 91947 Les Ulis-cedex, France). The make up and carrying gas was N_2 at a flow rate of 1 ml/minute (head column pressure: 15 psi). The running conditions were: initial temperature 100°C during 5 minutes, followed by an increase ($10^{\circ}\text{C}/\text{minutes}$) to 200°C for 20 minutes, followed by another increase ($3^{\circ}\text{C}/\text{minute}$) to 230°C for 5 minutes. The injection and detection temperature were 230°C and 250°C respectively. FAME were identified by comparison with the retention times of FAME standards (Sigma, Saint Quentin Fallavier, France). Results were expressed as percentage of total FA (chain length between 14 and 22 carbon atoms). The daily FA intakes (g/day) were also calculated.

Blood Collection and Analysis

Fasting blood was collected between 6:00 and 7:30 a.m. on the last day of the diet collection using evacuated tubes (Becton Dickinson, Meylan, France). Ethylene diamine tetra-acetic acid-containing tubes were used for FA determination. Samples were kept on ice and centrifuged within 30 minutes. Plasma was stored at -80°C for FA analysis.

Fatty acids of plasma lipids were analyzed as follows. After addition of the internal standard, heptadecanoic acid (Sigma, France), total plasma lipids were extracted with a single phase system (hexane/isopropanol 3:2, by vol) [11,12]. FA were transesterified and extracted as described above. They were expressed in relative amount (%) and in absolute concentration (mmol/L).

Lipid peroxidation was monitored by fluorometric determination of thiobarbituric acid reactive substances (TBARS) in plasma as described by Richard et al [13].

Statistics

Statistical calculations were performed with the PCSM statistical software (Deltasoftware, Meylan, France). Normality of data distribution was tested using the Kolmogorov-Smirnov test. Results were compared using the Student's *t*-test. Data were analyzed for statistical significance with a probability level of 0.05.

RESULTS

The hospitalized elderly women ingested an average of 5.22 MJ/day. Total lipids (45.9 ± 14.0 g/day) and SFA represented respectively 32.8% and 17.6% of the energy intake (Table 1). Linoleic acid and α -linolenic acid represented 3.0% and 0.35% of the total energy intake, respectively. The composition of dietary fat is shown in Table 2. Dietary lipids contained 53.6% of SFA, 35.4% of MUFA, and 11.0% of PUFA. The ratio of PUFA to SFA (P/S) in the diets was 0.21. The two major FA were oleic acid and palmitic acid (29.9% and 29.0% of the FA intake, respectively). The minimal recommendation for linoleic acid (1% of energy intake) [14,15] was reached in all patients. Thirty-two percent of the subjects had linoleic acid intakes below the minimal range recommended by the National Research Council (3 g/day) [14]. Eighty-six percent received less than 0.5% of energy from α -linolenic acid. Only one patient reached the French recommendations for α -linolenic acid (0.8 g/day) [15]. The intake of very long-chain n-3 FA (C20:5, C22:5, and C22:6) corresponded to less than 0.1% of energy intake in 64% of the patients.

The FA composition of total plasma lipids is shown in Table 3. A high relative content in 16:0, 18:1n-9, 16:1n-7, and 18:1n-7 was observed along with a low relative content in 18:2 n-6 compared to laboratory reference values. The ratio of 16:1n-7 to 18:2n-6, used as an indicator of linoleic acid deficiency [16], was increased in the hospitalized elderly subjects. Indexes of

Table 1. FA Dietary Intakes (% of Total Energy Intake) in Relation to Recommendations

	Dietary intake ^a	Recommendations
Lipids ^b	32.82 ± 4.37	30–35 [15,22].
C18:2n-6 ^b	3.03 ± 0.76	1–2 (minimum) [14]. 5–6 (optimum) [15].
C18:3n-3 ^b	0.35 ± 0.16	0.5–1 [15].
Very long-chain n-3 ^b	0.11 ± 0.06	0.1–0.2 [15,28].
SFA ^b	17.60 ± 2.79	<10 [14,22].

^a Each value represents the mean \pm SD of n determinations, where n=23 and represents the number of the elderly patients.

^b Expressed as percentage of total energy intake.
SFA=saturated fatty acids.

Table 2. Composition of Fatty Acid Dietary Intake

	Composition of dietary lipids ^b	Dietary intake ^c
C10:0	1.06 ± 0.42	0.46 ± 0.20
C12:0	2.84 ± 0.85	1.30 ± 0.56
C14:0	8.36 ± 1.06	3.80 ± 1.12
C15:0	0.87 ± 0.11	0.40 ± 0.12
C16:0	28.95 ± 2.28	13.14 ± 3.65
C16:1n-7	1.65 ± 0.17	0.76 ± 0.24
C17:0	0.56 ± 0.11	0.26 ± 0.10
C18:0	10.14 ± 1.11	4.70 ± 1.61
C18:1n-9	29.87 ± 2.95	13.80 ± 4.72
C18:1n-7 cis	2.23 ± 0.87	1.04 ± 0.59
C18:1n-7 trans	0.51 ± 0.10	0.24 ± 0.08
C18:2n-6	9.21 ± 1.74	4.32 ± 1.82
C18:3n-6	0.10 ± 0.03	0.04 ± 0.02
C18:3n-3	1.08 ± 0.49	0.50 ± 0.30
C20:4n-6	0.19 ± 0.06	0.08 ± 0.03
C20:5n-3	0.10 ± 0.07	0.04 ± 0.03
C22:5n-3	0.09 ± 0.04	0.04 ± 0.03
C22:6n-3	0.15 ± 0.11	0.07 ± 0.06
Others ^d	2.05 ± 0.38	0.95 ± 0.38
SFA	53.57 ± 4.31	24.44 ± 7.14
MUFA	35.41 ± 2.94	16.34 ± 5.49
PUFA	11.02 ± 2.01	5.14 ± 2.11
18:2n-6/18:3n-3	9.91 ± 4.03	
P/S	0.21 ± 0.05	

^a Each value represents the mean±SD of n determinations, where n=23 and represents the number of the hospitalized elderly patients.

^b Expressed as percentage of total fatty acids.

^c Expressed as 1 g day.

^d Fatty acids representing individually less than 0.35% of the dietary lipids (16:1n-9, 17:1n-3, 20:0, 20:1n-9, 20:1n-7, 20:2n-6, 20:3n-6, 22:0, 22:1n-9, 22:4n-6 and 24:0).

MUFA=monounsaturated fatty acids, PUFA=polyunsaturated fatty acids, SFA=saturated fatty acids, P/S=ratio of polyunsaturated fatty acids.

δ-6 and δ-9 desaturase activity [16] were also increased in the hospitalized elderly (Table 3).

The total cholesterol and HDL-cholesterol concentrations were low (5.38±1.11 and 1.18±0.30 mmol/L, respectively) whereas LDL-cholesterol and triglyceride concentrations (3.54±0.98 and 1.43±0.48 mmol/L, respectively) were close to usual laboratory values.

Lipid peroxidation was estimated by the measurement of TBARs in plasma. The TBARs plasma concentration was in the range of normal values obtained in free-living subjects aged 65 to 79 recruited for another protocol. However the ratio of TBARs to PUFA appeared significantly increased in plasma of hospitalized elderly patients compared to the healthy elderly subjects (0.59±0.11 vs 0.49±0.08 μmol/mmol, respectively, p<0.01).

DISCUSSION

Our study provides detailed analytical data about FA ingested by hospitalized elderly patients. There are very little data

Table 3. Fatty Acid Composition of Total Plasma Lipids

	Hospitalized patients ^b	Reference values ^c	p value ^a
14:0	1.33 ± 0.30	1.01 ± 0.44	0.005
16:0	21.47 ± 1.23	19.91 ± 1.55	<0.0001
16:1n-7	3.89 ± 1.17	2.04 ± 0.78	<0.0001
18:0	6.07 ± 0.55	6.63 ± 0.91	0.012
18:1n-9	23.23 ± 2.07	16.64 ± 2.17	<0.0001
18:1n-7	1.98 ± 0.43	1.55 ± 0.29	<0.0001
18:2n-6	26.15 ± 4.01	35.55 ± 4.42	<0.0001
18:3n-6	0.58 ± 0.26	0.42 ± 0.15	0.015
18:3n-3	0.48 ± 0.17	0.49 ± 0.11	0.80
20:2n-6+20:3n-9	0.43 ± 0.14	0.33 ± 0.06	0.006
20:3n-6	1.77 ± 0.32	1.55 ± 0.26	0.012
20:4n-6	8.40 ± 1.77	8.63 ± 1.35	0.62
20:5n-3	0.81 ± 0.30	1.29 ± 0.92	0.016
22:5n-3	0.58 ± 0.19	0.64 ± 0.18	0.30
22:6n-3	2.56 ± 0.52	3.01 ± 0.78	0.023
SFA	28.87 ± 1.60	27.56 ± 2.90	0.01
MUFA	29.10 ± 2.88	20.23 ± 3.24	<0.0001
PUFA	41.99 ± 3.33	52.21 ± 3.70	<0.0001
n-3	4.41 ± 0.79	5.33 ± 1.40	0.008
n-6	37.60 ± 3.53	46.88 ± 3.54	<0.0001
n-7	5.87 ± 1.56	3.59 ± 0.99	<0.0001
P/S	1.46 ± 0.18	1.91 ± 0.25	<0.0001
n-3/n-6	0.12 ± 0.03	0.11 ± 0.03	0.57
16:1n-7/18:2n-6	0.16 ± 0.06	0.06 ± 0.03	<0.0001
20:3n-6/18:2n-6	0.07 ± 0.02	0.04 ± 0.01	<0.0001
18:1n-9/18:0	3.85 ± 0.46	2.56 ± 0.55	<0.0001
16:1n-7/16:0	0.18 ± 0.06	0.10 ± 0.04	<0.0001

^a Expressed as percentage of total fatty acids.

^b Each value represents the mean±SD of n determinations, where n=23 and represents the number of the hospitalized patients.

^c References values obtained from 25 normolipidemic women aged <55 years (mean 38).

MUFA=monounsaturated fatty acids, PUFA=polyunsaturated fatty acids, SFA=saturated fatty acids, P/S=ratio of polyunsaturated to saturated fatty acids.

about the FA dietary intakes of institutionalized elderly subjects, probably because of obvious difficulties in estimating the dietary intakes of very aged and often disabled people. Data concerning FA intakes in free-living elderly people are more numerous. However, usually only data about total saturated, monounsaturated, PUFA, and sometimes linoleic acid are provided [17–19]. The preciseness of these data can be questioned because food composition tables were used in a majority of cases. Furthermore, Dabadie et al [20] showed that the composition of fat remaining on the dishes at the end of the meal was different from that of the fat ingested. Conversely, in the duplicate diet technique, the closest copy of ingested portions is reproduced and chemically analyzed.

The average fat intake measured in our population is lower than most data obtained in institutionalized [21] and in free-living elderly subjects [17–19] using food composition tables. Our data are strikingly close to those obtained by the chemical analysis of hospital meals provided to healthy French middle-aged subjects [20]. The partition of FA in dietary fat is different from current recommendations [14,15,22]. For an optimal prevention of cardiovascular disease, SFA should represent less

than 10% of the energy intake [14]. The value obtained here is 17.6%. In addition, palmitic acid and myristic acid, known to have the highest hypercholesterolemic effect [23] represented 70% of the SFA on average. This could be harmful in terms of cholesterolemia and therefore of cardiovascular morbidity. However, Krumholz et al [24] recently showed that hypercholesterolemia or low HDL-cholesterol may not be important risk factors for cardiovascular diseases in subjects older than 70 years. Actually, our hospitalized subjects presented rather low cholesterol concentrations in plasma. From recent studies, it has appeared that low cholesterolemia corresponds to an important risk of malnutrition in elderly subjects [25]. Below 4.13 mmol/L, the risk of mortality increases [26]. Twenty-two percent of our patients were below this range. The low total cholesterol values found in the hospitalized patients were mainly related to low HDL-cholesterol concentrations. This could be related to living conditions and insufficient physical activity. Moreover, Wilson et al [27] showed that total and LDL-cholesterol levels increase until age of 55 to 60 years in men and 70 to 75 years in women, and then start to decline. The cholesterol concentrations have probably already declined significantly in our patients. Therefore, the consumption of a high-SFA diet may not be as harmful in this population of 86 years-old patients as it would be in younger adults, provided adequate amounts of EFA are ingested.

The linoleic acid intake of the patients was below the optimal recommendation for French elderly people (5 to 8 g/day) [15]. One-third of the patients failed to ingest the minimum of 3 g/day recommended by the National Research Council [14]. According to Bjerve [28] the minimal recommendation for α -linolenic acid is 0.29 g/day in the near absence of long-chain n-3 FA. Twenty-two percent of our patients failed to ingest this amount, whereas 91% percent of the intakes were below the French recommendation for elderly subjects (0.8 g/day) [15]. A degradation of PUFA in foods before consumption could account for these low intakes, particularly in the case of arachidonic acid and n-3 FA. These FA are highly unsaturated and sensitive to oxidation [29]. Current recommendations do not provide values concerning the needs of elderly subjects for very long-chain PUFA. However, it has been suggested that 20:4n-6 and 20:5n-3, the precursors of eicosanoids, could also become essential for older subjects because of an impairment of desaturase activity with aging [4,5]. Further studies should be undertaken to define the specific needs of older subjects for these long chain PUFA. Relative to energy, the 18:3n-3 intake measured here is very close to the one calculated by Ascittimoura et al [21] (0.34% vs. 0.35%).

Consistent with these marginal EFA intakes, the hospitalized patients presented several biochemical signs suggesting an EFA malnutrition. A proportion of 18:2n-6 below 28% and a proportion of 16:1n-7 above 2.6% of total FA indicate EFA deficiency according to Siguel et al [30]. These criteria were met in 70% and 87% of our hospitalized patients, respectively. Only one subject presented none of these signs. As previously

observed in EFA-deficient subjects [16], these patients presented a low P/S ratio and a low proportion of n-6 FA in blood lipids. An increase in the proportion of n-7 FA was also observed. The high indexes of δ -6 and δ -9 desaturase activities were also in favor of an EFA insufficiency in our patients [16]. It is noteworthy that the proportion of subjects presenting biochemical signs of EFA insufficiency was much higher than the proportion of subjects with 18:2n-6 intakes below minimal recommendations. This is in agreement with the lack of significant correlation between the intake of linoleic acid and its concentration in plasma (data not shown). Although this should be interpreted with care due to the small sample size, it suggests an impairment of linoleic acid absorption and metabolism in these patients.

Besides insufficient dietary intakes, other factors could contribute to EFA insufficiency in these patients. Metabolic disturbances related to aging could be one, the average age of the subjects being 86 years. A rise in the proportion of oleic acid, a decrease in n-6 FA, total PUFA and P/S ratio were already described in plasma and cell lipids of elderly subjects compared to younger controls [19,20,31,32]. In the present study, we also observed these differences between hospitalized elderly subjects and laboratory reference values obtained from younger subjects (25 normolipidemic women aged <55 years). All these studies suggest an alteration of the plasma FA profile upon aging and hospitalization. A study with a group of free living older subjects is being carried out to respectively determine, first the effects of age and second those of hospitalization.

PUFA are known to be one of the main targets of free-radical attack. Therefore, oxidative stress could impair EFA insufficiency through lipid peroxidation. Lipid peroxidation is generally reported to increase with age [33,34]. Schäfer and Thorling [35] showed the importance of expressing lipid peroxidation products relative to the available fatty acids. In the present case, the ratio of TBARS to PUFA was significantly higher in the hospitalized elderly compared to the value obtained in free-living elderly women. This suggests an increased relative lipid peroxidation in the hospitalized elderly patients. This is consistent with the high indexes of δ -6 and δ -9 desaturase activity expressed respectively as show in Table 3, by the ratio 16:1n-7/18:2n-6 and 20:3n-6/18:2n-6 in hospitalized elderly women. Indeed, Cabré et al [36] suggested that an increased oxidative stress could lead to enhanced desaturation and elongation of EFA to maintain the long-chain PUFA status of cell membranes.

In conclusion, the present study showed that hospitalized elderly patients present marginal EFA intakes and biochemical signs of EFA deficiency which could be partly related to an increased lipid peroxidation in these subjects. It is not possible to define if the observed decreased PUFA in hospitalized women is related to hospitalization or aging. Further studies are needed to compare these results with those obtained in an age-matched control group. However, dietary interventions

could be considered in institutionalized elderly patients. They could limit the risk of skin troubles, immune system impairment, and vascular disease often observed in institutionalized elderly subjects, which may be partly related to EFA deficiency.

ACKNOWLEDGMENTS

This work was supported in part by the Rhône-Alpes Region and the Aguetant laboratory (Lyon, France).

We are grateful to L. Sauze, C. Tavel-Besson, and Y. Grateloux for technical assistance, and to the staff of the Geriatric department (Pavillon E. Chatin) for helping with the diet collection. We thank C. Garcia de La Rosa and the dietitians' department of the Grenoble Hospital (J. Vaccari).

The reading of this manuscript by Dr. R.A. Anderson, PhD, is greatly acknowledged.

REFERENCES

1. Sardesai VM: Nutritional role of polyunsaturated fatty acids. *J Nutr Biochem* 3:154-166, 1992.
2. Lands WEM: Biochemistry and physiology of n-3 fatty acids. *FASEB J* 6:2530-536, 1992.
3. Munro HN, Danford DE: Nutrition, aging, and the elderly. New York: Plenum Press, 1989.
4. Bordoni A, Biagi PL, Turchetto E, Hrelia S: Aging influence on delta-6-desaturase activity and fatty acid composition of rat liver microsomes. *Biochem Int* 17:1001-1009, 1988.
5. Hrelia S, Bordoni A, Celadon M, Turchetto E, Biagi PL, Rossi CA: Age-related changes in linoleate and α -linolenate desaturation by rat liver microsomes. *Biochem Biophys Res Commun* 163:348-355, 1989.
6. Schmuck A, Ravel A, Coudray C, Alary J, Franco A, Roussel AM: Antioxidant vitamins in hospitalized elderly patients: analysed dietary intakes and biochemical status. *Eur J Clin Nutr* 50:473-478, 1996.
7. Schmuck A, Roussel AM, Arnaud J, Ducros V, Favier A, Franco A: Analyzed dietary intakes, plasma concentrations of zinc, copper, and selenium, and related enzyme activities in hospitalized elderly women. *J Am Coll Nutr* 15:462-468, 1996.
8. AFNOR: Détermination de l'extrait à l'oxyde diéthylique NF V 18-104 Méthode CEE 2^{ème} directive. Collection des Normes Françaises, AFNOR ed., 1981.
9. Slover HT, Lanza E: Quantitative analysis of food fatty acids by capillary gas chromatography. *J Am Oil Chem* 56:933-943, 1979.
10. Rockerbie RA, Dodson RD, Frohlich J: Gas-chromatographic analysis of patterns of fatty acids of cholesteryl esters and phosphatidylcholine. *Clin Chem* 25:1411-1414, 1979.
11. Hara A, Radin NS: Lipid extraction of tissue with a low toxicity solvent. *Anal Biochem* 90:420-426, 1978.
12. Kolarovic L, Fournier NC: A comparison of extraction methods for the isolation of phospholipids from biological sources. *Anal Biochem* 156:244-250, 1986.
13. Richard MJ, Portal B, Méo J, Coudray C, Hadjian A, Favier A: Malondialdehyde kit evaluated for determining plasma and lipoprotein fractions that react with thiobarbituric acid. *Clin Chem* 38:704-709, 1992.
14. National Research Council: "Recommended Dietary Allowances," 10th ed. Washington DC: National Academy Press, 1989.
15. Lemarchal P, Bourre JM, Darcet P, Durand G, Klère J, Legrand P, Mendy F, Renaud S, Zwobada F: Apports nutritionnels conseillés en acides gras essentiels. In Dupin H, Abraham J, Giachetti I (eds): "Apports Nutritionnels Conseillés pour la Population Française", 2^{ème} ed. Tec & Doc Lavoisier, Paris: pp 74-81, 1992.
16. Lepage G, Levy E, Ronco N, Smith L, Galéano N, Roy CC: Direct transesterification of plasma fatty acids for the diagnosis of essential fatty acid deficiency in cystic fibrosis. *J Lipid Res* 30:1483-1490, 1989.
17. Euronut SENECA Investigators: Intake of energy and nutrients. *Eur J Clin Nutr* 45 (Suppl. 3):105-119, 1991.
18. Löwik MRH, Westenbrink S, Hulshof KFAM, Kistenmaker C, Hermus RJJ: Nutrition and aging: dietary intake of "apparently healthy" elderly (Dutch Nutrition Surveillance System). *J Am Coll Nutr* 8:347-356, 1989.
19. Olivieri O, Stanzial AM, Girelli D, Trevisan MT, Guarini P, Terzi M, Caffi S, Fontana F, Casaril M, Ferrari S, Corrocher R: Selenium status, fatty acids, vitamins A and E, and aging: the nove study. *Am J Clin Nutr* 60:510-517, 1994.
20. Dabadie H, Castera A, Lacomère RP, Bernard M, Mordret F, Chazan JB, Paccalin J: Consommation lipidique en France dans une collectivité. *Cah Nutr Diét* 26:197-202, 1991.
21. Ascitti-Moura L, Guillaud JC, Fuchs F, Richard D, Klepping J: Fatty acid composition of serum lipids and its relation to diet in an elderly institutionalized population. *Am J Clin Nutr* 48:980-987, 1988.
22. Anonymous, WHO and FAO Joint Consultation: Fats and oils in human nutrition. *Nutr Rev* 53:202-205, 1995.
23. Grundy SM, Denke MA: Dietary influences on serum lipids and lipoproteins. *J Lipid Res* 31:1149-1172, 1990.
24. Krumholz HM, Seeman TE, Merrill SS, Mendes de Leon CF, Vaccarino V, Silverman DI, Tsukahara R, Ostfeld AM, Berkman LF: Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years. *JAMA* 272:1335-1340, 1994.
25. Schlienger JL: The cholesterol controversy. *Presse Med* 24:471-473, 1995, (in French).
26. Forette B, Tortrat D, Wolmark Y: Cholesterol and mortality in elderly women. *Lancet* i:868-870, 1989.
27. Wilson PWF, Anderson KM, Harris T, Kannel WB, Castelli WP: Determinants of change in total cholesterol and HDL-C with age: the Framingham study. *J Gerontol* 49:M252-M257, 1994.
28. Bjerve KS: Omega 3 fatty acid deficiency in man: implications for the requirement of alpha-linolenic acid and long-chain omega 3 fatty acids. In Simopoulos AP, Kifer RR, Martin RE, Barlow SM (eds): "Health Effects of Omega 3 Polyunsaturated Fatty Acids in Seafoods." Vol. 66. Basel: Karger, pp 133-142, 1991.
29. Duthie GG: Lipid peroxidation. *Eur J Clin Nutr* 47:759-764, 1993.
30. Siguel EN, Lerman RH: Altered fatty acid metabolism in patients with angiographically documented coronary artery disease. *Metabolism* 43:982-993, 1994.

31. Bjerve KS, Fougner KJ, Midthjell K, Bonna K: n-3 fatty acids in old age. *J Int Med* 225 (Suppl. 1):191–196, 1989.
32. Véricel E, Croset M, Perrot L, Renaud S, Lagarde M: Platelets and aging. II—plasma lipoproteins and fatty acid profiles. *Thromb Res* 49:451–462, 1988.
33. Rodriguez-Martinez MA, Ruiz-Torres A: Homeostasis between lipid peroxidation and antioxidant enzyme activities in healthy human aging. *Mech Ageing Dev* 66:213–222, 1992.
34. Zarling EJ, Mobarhan S, Bowen P, Kamath S: Pulmonary pentane excretion increases with age in healthy subjects. *Mech Ageing Dev* 67:141–147, 1993.
35. Schäfer L, Thorling EB: Lipid peroxidation and antioxidant supplementation in old age. *Scand J Clin Lab Invest* 50:69–75, 1990.
36. Cabré E, Periago JL, Mingorance MD, Fernandez-Banares F, Abad A, Esteve M, Gil A, Lachica M, Gonzales-Huix F, Gassull MA: Factors related to the plasma fatty acid profile in healthy subjects, with special reference to antioxidant micronutrient status: a multivariate analysis. *Am J Clin Nutr* 55:831–837, 1991.
37. Horrobin DF: Nutritional and medical importance of gamma-linolenic acid. *Prog Lipid Res* 31:163–194, 1992.

Received August 1997; revision accepted April 1998.