Fatty Acid Pattern Outcomes of a Nutritional Program for Overweight and Hyperlipidemic Australian Men

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There is growing recognition of the value of changing tissue fatty acid patterns in their own right as coronary risk factors. To examine the effects of a conventional nutritional program on plasma triglyceride (TG), cholesterol ester, and phospholipid fatty acid patterns, a group of 20 hyperlipidemic men and a control group (n = 6) of normolipidemic men were followed for 6 months. As an index of change in energy balance in the hyperlipidemic men, body mass index decreased from 26.5 to 24.4 kg m^{-2} (an 8% decrease) at 6 months. Saturated fat intake fell from 46.7 to 25.3 g/day (a 46% decrease). Dietary polyunsaturated:saturated fat ratio (P:S) rose from 0.38 and to 0.70 (an 84% increase) at the 6-month review. Ethanol intake fell from 18 to 15 g/day (a 17% decrease). Changes in plasma fatty acid (FA) patterns were found in TG, cholesterol ester, and phospholipid fractions at the 6-week to 3-month period, and these changes were maintained at 6 months. Of the factors possibly contributory to plasma FA pattern change in these men, dietary FA intake underwent the greatest percentage shift and therefore probably makes an important contribution to the change. It was of interest that fatty acid patterns in plasma neutral lipids (triglyceride, cholesterol ester, and phospholipid) significantly predicted body mass index and serum total cholesterol, triglyceride, and high-density-lipoprotein cholesterol.

INTRODUCTION

Food intake methodology is widely used in nutrition research, but few opportunities are available for its validation by biochemical methods. In nutritional management of hyperlipidemia, a failure of serum lipids to fall might reflect a problem in dietary adherence not appreciated by dietary assessment or it might indicate a lack of biological response to an altered food intake [1].

In order to evaluate these questions, we have taken the opportunity to consider the change in dietary FA pattern and related changes in plasma FA patterns following nutrition advice in overweight hyperlipidemic men. Recognizing that nutritional advice for hyperlipidemia inevitably requires advice not only about quantity and quality of dietary fat, we have also endeavored to take into account the extent to which changes in energy balance and alcohol intake have been contributory. Another aspect of examining plasma FA pattern response to diet is that it may be important in its own right given recent evidence of its predictive power in relation to risk for coronary heart disease [2,3].

SUBJECTS AND METHODS

As part of a heart disease prevention program in the aluminum industry in Geelong, Australia, 20 men were identified for lipid-lowering therapy. They were followed together with six normolipidemic men (control group). Weight-for-height relationships were assessed by calculation of the body mass index [BMI (kg m^{-2})]. Subjects were classified as being overweight if BMI was 25.1–29.9 kg m^{-2} (inclusive) or obese if BMI was greater

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Table 1. Age and Changes in Body Weight and Serum Lipids in Hyperlipidemic and Normolipidemic Men over 6 Months[a–d]

	Hyperlipidemic (n = 20)			Normolipidemic (n = 6)		
Assessment	0 months	6 weeks/ 3 months	6 months	0 months	6 weeks/ 3 months	6 months
Age (years)	45.8 ± 1.8	–	–	28.2 ± 3.0	–	–
Weight (kg)	81.8 ± 2.0	76.1 ± 1.6***	75.5 ± 1.6***	75.9 ± 2.7	74.7 ± 3.1	76.0 ± 3.0
Mean change in weight from 0 months (kg)	–	–5.8 ± 0.8***	–6.3 ± 0.9***	–	–1.4 ± 0.7	–0.0 ± 0.5
Mean BMI (kg m^{-2})	26.5 ± 0.52	24.6 ± 0.43***	24.4 ± 0.47***	23.3 ± 1.0	22.9 ± 1.1	23.3 ± 1.1
Frequency of BMI > 25 (kg m^{-2})	75	45***	40***	17	17	17
Total cholesterol (mmol/L)	6.3 ± 0.3	5.7 ± 0.1*	6.1 ± 0.2	4.8 ± 0.4	4.6 ± 0.4	4.4 ± 0.3
Frequency of abnormality (%)	45	15***	30	0	0	0
Total triglyceride (mmol/L)	3.3 ± 0.4	2.2 ± 0.4***	2.1 ± 0.2***	1.1 ± 0.1	1.4 ± 0.2	1.3 ± 0.3
Frequency of abnormality (%)	85	35***	50***	0	17	0
HDL-cholesterol (mmol/L) (n = 13)	0.97 ± 0.08	1.11 ± 0.06	1.05 ± 0.04	1.46 ± 0.11	1.56 ± 0.16	1.46 ± 0.10

[a]The first assessment of weight or lipid status after lipid-lowering counseling was between 6 weeks and 3 months. Means and standard errors of the means are shown.

[b]Significance from baseline for variables except "frequency of abnormality" were determined by the Wilcoxon's two-tailed test and are indicated by *$p < 0.05$, **$p < 0.02$, ***$p < 0.01$, ****$p < 0.001$ (Table 4 only — for correlation coefficients). In this study we have used a nonparametric method (Wilcoxon's signed-rank test [18]) to evaluate differences between observations at baseline, 6 weeks to 3 months, and at 6 months; in this way, we have avoided the problems associated with regression to the mean which arise where a particular distribution of the data is assumed.

[c]A serum cholesterol > 6.5 mmol/L or a serum triglyceride > 2.0 mmol/L was regarded as abnormal. The significance of difference of frequency of abnormality compared with baseline has been evaluated by χ^2. Similar notation for significance is shown as above.

[d]Significance levels for HDL-cholesterol were calculated for n = 13 for hyperlipidemic men and n = 6 for normolipidemic men. Insufficient samples were obtained on seven subjects for HDL determinations.

than or equal to 30 kg m^{-2}. Counseling about body fatness was given if a subject's BMI was > 25 kg m^{-2}. Based on this classification, none of the test or control subjects was obese. Table 1 shows the subject characteristics for each group. The primary purpose of nutrition counseling for hyperlipidemic men was to reduce serum lipids to less than a total cholesterol of 6.5 mmol/L and triglycerides 2.0 mmol/L. It is of interest that the National Heart Foundation of Australia currently has a public health policy that total cholesterol should be less than 5.5 mmol/L, allowing for individual consideration to be given in the range to 6.5 mmol/L. Nutrition counseling included attention to degree of body fatness, total fat intake, dietary P:S ratio, cholesterol intake, and alcohol intake as appropriate. The form of dietary education varied according to the particular serum lipid problem and the existing eating pattern of the subject. The control group received no dietary advice. Progress was assessed and further counseling provided between 6 weeks and 3 months and again at 6 months. In this study no effort was made to counsel about physical activity patterns as an aid to control body fatness nor was the level of physical activity assessed.

The food intake methodology used was recall of usual intake over about the previous month with the quantities of foods estimated in terms of household measures and with the assistance of two-dimensional drawings. This period took into account the nature of rotating rosters for shift work. Food intake for days off work was also assessed. The average daily amounts of individual food items were estimated from these dietary histories. These items were then coded for determination of energy, nutrients, and fatty acid composition by computer using McCance and Widdowson's "The Composition of

Foods" [4].

Blood was collected at baseline, 6 weeks to 3 months, and again at 6 months for analysis of serum lipids and plasma FA patterns. Serum was dispatched to St. Vincent's Hospital, Sydney, for direct assay of lipids. Serum cholesterol and TG were determined by Autoanalyser techniques of colorimetry and fluorimetry, respectively. High-density-lipoprotein (HDL) cholesterol was determined following precipitation with sodium phosphotungstate and $MgCl_2$ [5]. Plasma was frozen at –20°C for FA pattern analysis at Deakin University.

Plasma lipids were extracted for FA pattern analysis in chloroform:methanol, 2:1. Total lipids were redissolved in n-heptane and separated by thin layer chromatography (TLC) using the solvent mixture hexane:diethylether:formic acid in the v/v ratio of 80:20:2. Cholesterol ester, TG, and phospholipid fractions were transmethylated in 5% sulfuric acid in methanol under nitrogen overnight at 45°C. They were extracted in n-hexane and stored at –20°C. They were analyzed by gas liquid chromatography (GLC) with a Varian 3700 instrument fitted with a silanized packed 2-m glass column of 2 mm inside diameter. The packing was prepared from 100/120 mesh Suppelcoport (the support) and a 10% polyethylene glycol adipate coating, the latter being the stationary phase. Analyses were carried out with a flame ionization detector.

Using a temperature programming mode, the GLC, in conjunction with a Varian CDS-111 integration module, was standardized with respect to the different response of individual FA methyl esters. Standard mixtures of FA methyl esters were mixed with n-heptane to a working concentration (approximately 25 mg in 5 ml). The standard FA methyl esters from Nu Chek Prep. Inc. (Elysian, MN) and Serdary Research Laboratories, Inc. (London, Ontario, Canada) were chromatographed, and the relative response of the individual methyl esters was determined by giving the 18:1 derivative an arbitrary response value of 1.

For major components, more than 10% of total FAs (16.0, 18.0, 18.1, 18.2), interassay coefficient of variation (CV) ranged from 0.6 to 1.5%; for components less than 10% (14.0, 16.1, 20.4), interassay CV ranged between 2.7 and 10.2%.

RESULTS

Effect of Nutritional Intervention on Total Serum Lipids

All 20 test patients were hyperlipidemic, but not all were hypercholesterolemic (i.e., some had a raised triglyceride level only). Hence the baseline mean cholesterol for test subjects of 6.3 mmol/L. In hyperlipidemic subjects, nutritional intervention led to significant reductions in total cholesterol and TG at the 6-week to 3-month assessment and this was maintained for TG at 6 months. The frequency of abnormality was significantly reduced for both serum cholesterol and TG at the 6-week to 3-month assessment and for TG at 6 months. While the frequency of abnormality for serum cholesterol at 6 months showed a decrease, this change was not significantly different from baseline (Table 1). For the control group, no significant reductions in serum cholesterol or TG, or in the frequency of abnormalities, was seen in the follow-up over 6 months (Table 1).

Change in Nutrient Intake (Table 2)

A decrease in energy intake was effected at both follow-up occasions by decreases in amounts of saturated and monounsaturated fat and no significant change in polyunsaturated fat. At the first follow-up, there was a significant reduction in the amount of alcohol ingested, but not in the percentage contribution to energy intake. Reduced cholesterol intake was sustained at 6 months. In the control, no significant differences in macronutrient intake occurred in the follow-up period.

Effect of Nutritional Intervention on Plasma FA Patterns

Improvements in the P:S (polyunsaturated-to-saturated FA) ratios of plasma TG (Table 3A), cholesterol ester (Table 3B), and phospholipid (Table 3C) were seen at each of the two follow-up assessments; decreases in percentage palmitic acid (16:0) were seen in all major lipid patterns, and an increase in linoleic acid (18:2ω6) was seen in TG and cholesterol ester and arachidonic acid (20:4ω6) in phospholipid.

In the control group, some significant changes in individual FAs were recorded in the TG and phospholipid fractions (Tables 3A and 3C). However, these did not affect the overall P:S ratios of the fractions. Similarly, the P:S ratio of cholesterol ester remained constant over the 6 months.

For the control group, no significant changes were found in their habitual diets. Yet, for control as well as treated men, phospholipid FA 18:1ω9 decreased and 20:4ω6 increased. Although there could be an interplay between the ω-9 series (18:1) and the ω-6 series, that the same phenomenon occurred in phospholipid FAs of hyperlipidemic and normolipidemic men argues against a change in dietary FA intake being responsible. Presumably, some other variable which we have not measured has led to fluctuations in these phospholipid FAs irrespective of our intervention.

Table 2. Energy, Macronutrients, Cholesterol, and Fatty Acid Composition of the Diet of Hyperlipidemic and Normolipidemic Men over 6 Months[a]

	Hyperlipidemic (n = 20)			Normolipidemic (n = 6)		
Assessment	0 months	6 weeks/ 3 months	6 months	0 months	6 weeks/ 3 months	6 months
Energy (kJ)	11,254 ± 564.4	7010 ± 485.9***	8016 ± 592.2***	13,636 ± 2192	12,466 ± 2262	9956 ± 2045
Sources of energy						
Protein (%)	16.2 ± 0.6	20.2 ± 0.8***	19.3 ± 0.9***	14.5 ± 1.0	14.5 ± 1.0	16.3 ± 1.4
Fat (%)	39.8 ± 1.0	34.2 ± 1.3***	34.1 ± 1.0***	40.2 ± 3.6	37.8 ± 3.1	38.7 ± 1.8
Carbohydrate (%)	39.2 ± 1.4	40.6 ± 1.6	40.7 ± 1.6	40.5 ± 2.0	38.8 ± 1.4	39.5 ± 1.8
Alcohol (%)	4.7 ± 1.2	4.9 ± 1.2	5.3 ± 1.5	4.8 ± 1.7	8.8 ± 2.4	5.5 ± 1.7
Cholesterol (mg)	444.2 ± 33.3	246.3 ± 16.9***	310.2 ± 25.1***	457.0 ± 75.1	390.9 ± 92.4	401.7 ± 78.5
Alcohol (g)	18.1 ± 4.4	11.9 ± 2.5**	15.1 ± 4.4	20.6 ± 6.4	37.4 ± 14.8	20.1 ± 6.1
Quality of fat intake						
Saturated fat (g)	46.7 ± 2.8	20.5 ± 1.2***	25.3 ± 1.9***	58.4 ± 11.8	50.6 ± 11.2	43.2 ± 9.7
Monounsaturated fat (g)	41.0 ± 2.3	19.5 ± 1.5***	22.0 ± 1.7***	44.9 ± 8.8	38.7 ± 6.9	36.7 ± 6.0
Polyunsaturated fat (g)	20.5 ± 2.9	16.9 ± 1.8	18.3 ± 1.8	32.1 ± 9.6	26.5 ± 6.1	19.5 ± 3.6
P:S ratio	0.45 ± 0.05	0.82 ± 0.05***	0.75 ± 0.06***	0.57 ± 0.13	0.56 ± 0.15	0.46 ± 0.11

[a]For levels of significance, see footnotes to Table 1.

Plasma FA Patterns as Predictors of Body Mass Index and Total Serum Lipids

In Table 4 we have used the P:S ratio in each of the plasma neutral lipid fractions, phospholipid, triglyceride, and cholesterol ester, to consider how plasma FA patterns determine degree of body fatness (BMI) and total serum lipids. Plasma phospholipid P:S ratio is the most consistently predictive when the means of the six study categories (both hyperlipidemic and normolipidemic at 0, 6 weeks/3 months, and 6 months) are considered together in correlation analysis.

DISCUSSION

FA Patterns in Hyperlipidemia and Normolipidemia

We chose a reference normolipidemic group to monitor a change in a nonintervention group, although that group was not matched for age or BMI. In our study, hyperlipidemic men had lower commencement P:S ratios in plasma TG ($p < 0.002$) and plasma phospholipid ($p < 0.05$) (two-tailed Mann–Whitney U Test [6]) but not in cholesterol ester; dietary P:S ratios for the two groups were similar at this time. At the conclusion of the study, the P:S ratios for all lipid fractions were not significantly different for hyperlipidemic as opposed to normolipidemic men, even though the dietary P:S ratio in hyperlipidemic men exceeded that in the normolipidemic men ($p < 0.05$). This emphasizes the likely difference in metabolic sensitivity to dietary FA type in hyperlipidemia.

Dietary Factors and Plasma FA Pattern Change

Changes in plasma FA composition were observed with a combination of weight reduction (reflecting a change in energy balance), increase in dietary P:S ratio, and, at the 6 weeks to 3 months assessment, a decrease in alcohol intake. Since the improvements in weight and dietary P:S ratio were maximized within the 6-week to 3-month period and maintained thereafter, and since changes were also achieved for plasma TG, phospholipid, and cholesterol ester P:S ratios, it is not possible from these data to argue a case for a greater contribution of one factor or another. Nevertheless, the greatest percentage change in a dietary variable was for P:S ratio, with an 84% increase, indicating its potential importance.

Durrington et al [7] found an induction of serum TG–FA change within 2 days of dietary P:S ratio change in normolipidemic subjects. In our overweight hyperlipidemic subjects, following dietary P:S ratio change, the plasma TG, phospholipid, and cholesterol ester FA pattern changes were in evidence at the 6-week to 3-month assessment. However, endogenous FA synthesis in, and FAs mobilized from, body fat stores might have diluted the impact of exogenous (dietary) FAs.

Table 3A. Mean Percentages of Major Fatty Acids in Plasma Triglyceride of Hyperlipidemic and Normolipidemic Men[a]

	Hyperlipidemic (n = 20)			Normolipidemic (n = 6)		
	0 months	6 weeks/ 3 months	6 months	0 months	6 weeks/ 3 months	6 months
14:0	1.89 ± 0.21	2.09 ± 0.26	1.47 ± 0.18	1.59 ± 0.45	2.25 ± 0.64	4.47 ± 1.22*
16:0	28.49 ± 0.94	26.64 ± 0.89***	25.42 ± 1.11***	26.13 ± 0.87	25.19 ± 1.34	25.22 ± 1.68
16:1	6.44 ± 0.40	6.52 ± 0.54	6.27 ± 0.37	5.74 ± 0.82	7.93 ± 1.72	5.52 ± 0.36
18:0	5.44 ± 0.40	5.99 ± 0.46	4.86 ± 0.58	7.39 ± 1.31	7.44 ± 1.09	5.36 ± 0.51
18:1	44.93 ± 1.03	44.19 ± 1.06	45.19 ± 1.44	38.06 ± 2.94	39.67 ± 2.30	34.99 ± 2.31
18:2ω6	12.65 ± 1.15	14.61 ± 1.00*	16.86 ± 1.23***	21.11 ± 3.23	17.42 ± 1.93	21.48 ± 1.62
P:S	0.38 ± 0.05	0.44 ± 0.04***	0.56 ± 0.05***	0.61 ± 0.11	0.51 ± 0.29	0.63 ± 0.30

[a]For levels of significance, refer to Table 1 footnotes.

Table 3B. Mean Percentages of Major Fatty Acids in Plasma Cholesterol Ester of Hyperlipidemic and Normolipidemic Men[a]

	Hyperlipidemic (n = 20)			Normolipidemic (n = 6)		
	0 months	6 weeks/ 3 months	6 months	0 months	6 weeks/ 3 months	6 months
16:0	13.54 ± 0.59	12.72 ± 0.51*	12.72 ± 0.49*	12.17 ± 0.33	11.72 ± 0.59	12.14 ± 0.43
16:1	7.69 ± 0.57	6.72 ± 0.59***	6.05 ± 0.49***	6.79 ± 0.53	6.12 ± 0.40	6.99 ± 0.42
18:0	3.36 ± 0.35	3.11 ± 0.34	2.84 ± 0.41	4.13 ± 0.12	4.28 ± 0.67	4.34 ± 0.44
18:1	24.63 ± 0.93	22.35 ± 0.83***	22.54 ± 0.78*	20.91 ± 1.49	20.81 ± 0.95	19.69 ± 0.93
18:2ω6	45.57 ± 1.52	48.45 ± 1.49	50.53 ± 1.16***	48.37 ± 2.86	48.32 ± 3.15	49.55 ± 1.27
20:4ω6	5.20 ± 0.72	6.69 ± 0.94	5.63 ± 0.61	7.63 ± 1.43	8.73 ± 2.11	7.31 ± 1.45
P:S	3.09 ± 0.17	3.59 ± 0.18***	3.83 ± 0.20***	3.46 ± 0.33	3.59 ± 0.35	3.46 ± 0.32

[a]For levels of significance, refer to Table 1 footnotes.

Table 3C. Mean Percentages of Major Fatty Acids in Plasma Phospholipid of Hyperlipidemic and Normolipidemic Men[a]

	Hyperlipidemic (n = 20)			Normolipidemic (n = 6)		
	0 months	6 weeks/ 3 months	6 months	0 months	6 weeks/ 3 months	6 months
16:0	32.07 ± 1.46	29.43 ± 1.50***	28.70 ± 1.61***	24.12 ± 1.67	25.39 ± 0.98	25.31 ± 1.71
16:1	3.12 ± 0.48	3.34 ± 0.50	3.14 ± 0.48	4.97 ± 0.61	3.80 ± 0.28	3.85 ± 0.52
18:0	15.71 ± 0.45	14.79 ± 0.58***	14.79 ± 0.35	13.12 ± 0.66	13.35 ± 0.25	14.33 ± 0.33
18:1	17.01 ± 0.78	15.62 ± 0.69***	14.77 ± 0.37***	17.67 ± 0.92	15.50 ± 0.47*	13.20 ± 0.26*
18:2ω6	23.00 ± 0.88	24.52 ± 0.96	23.81 ± 0.77	29.11 ± 2.04	26.92 ± 1.64	27.27 ± 1.07
20:4ω6	8.88 ± 0.86	11.02 ± 1.00*	13.20 ± 1.34***	11.08 ± 1.07	15.10 ± 1.53*	16.15 ± 1.72
P:S	0.70 ± 0.05	0.88 ± 0.07***	0.89 ± 0.07***	1.13 ± 0.30	1.10 ± 0.08	1.12 ± 0.30

[a]For levels of significance, refer to Table 1 footnotes.

The dietary improvement in P:S ratio is a reflection of a decrease in the saturated rather than an increase in the polyunsaturated fat ingested. The increase in the proportion of linoleic acid in TG and cholesterol ester (at 6 months) may still simply be a reflection of these proportionate changes in the quality of dietary fat. Alternative-

Table 4. Use of Fatty Acid Pattern in Plasma Neutral Lipids (Triglyceride, Cholesterol Ester, Phospholipid) to Predict Body Mass Index (BMI) or Total Serum Lipids.[a]

	BMI	Serum Lipids		HDL cholesterol
		Cholesterol	Triglyceride	
Polyunsaturated:saturated fatty acid ratios for				
Phospholipid	–0.97***	–0.95***	–0.99****	0.95***
Triglyceride	–0.79	–0.69	–0.86	0.66
Cholesterol ester	–0.55	–0.12	–0.47	0.15

[a]Correlation coefficients (r) show the relationship between means of the variables in six study categories. Study categories are those of both hyperlipidemic and normolipidemic at 0 and 6 weeks and 3 and 6 months.

[b]The significance of correlations are shown by ***$p < 0.01$, ****$p < 0.0001$.

ly, since the proportion of arachidonic acid in phospholipid has risen significantly, the decrease in saturated and monounsaturated fat intakes may have affected chain elongation and desaturation of linoleic acid.

A study by Dehmer et al [8] on the effect of ω-3 FA supplementation on restenosis following coronary angioplasty showed that such supplementation resulted in a reduction in platelet arachidonic acid and a rise in the ω-3 FA component. In our subjects, there was a low intake of ω-3 FAs (the men ate negligible fish), and polyunsaturated FAs ingested were predominantly ω-6. Therefore, we cannot exclude the possibility that the low baseline plasma TG ω-6 FAs is not contributed to by altered increased utilization of available dietary ω-6 FAs for prostaglandin synthesis.

Alcohol has been reported to influence plasma FAs in its own right [9]. In the present study, alcohol intake fell significantly by 6 weeks to 3 months, but this change did not persist.

Value of Evaluating Dietary FA Pattern Change

Although dietary change in P:S ratio appears to predict some of the change in plasma P:S ratios, in turn, these have been of limited value in predicting the maintenance of serum cholesterol change in the present study. That plasma FA pattern change (an increase in P:S ratio) is maintained when a decrease in serum cholesterol is not raises the possibility that there is a metabolic escape of either cholesterol production or removal from the influence of P:S ratio. The explanation would not appear to reside in a changed contribution from HDL-cholesterol since there were no significant changes in this lipoprotein fraction at 6-weeks to 3-months or 6 months. It is of interest that there is growing evidence that dietary and plasma FA changes may be predictive of coronary heart disease in their own right [2,3,10].

Effect of Plasma FA Patterns on BMI

Our data raise the possibility that FA pattern profiles achieved in plasma lipids, presumptively on the basis of diet, influence the degree of body fatness. Of course, alternatively, the way in which people ate in our study may have influenced both plasma FA patterns and obesity independent of each other or the presence of obesity may affect plasma FA patterns. Recent evidence from Jones and Schoeller [11] indicates that P:S ratios of dietary fat influence energy substrate utilization, which would be consistent with the first of our interpretations.

Effect of Plasma FA Patterns on Total Serum Lipids

A consideration of the overall relationship between the plasma neutral lipids, especially phospholipid FA patterns, and total serum lipids indicates that the higher the P:S ratio achieved in plasma the more favorable the serum lipoproteins, including HDL. This may be a reflection of dietary habits, in which case it would support what is known from metabolic studies about dietary fat and serum lipids, expressed in the Keys and Hegsted equations [12–14]. There is increasing evidence that certain groups in industrialized countries may be at risk of essential FA deficiency reflected in serum phospholipid and triglyceride FA patterns [15].

However, it is also possible that there are particular problems with respect to the handling and metabolism of fatty acids and that this may have accounted for differences in FA patterns between hyperlipidemic and normolipidemic men in our study. The total of the ω-6 fatty acids is different between the two groups at baseline. After intervention, the total of ω-6 fatty acids approached that in the normolipidemic men, but 20:4

remained low. This suggests a block in the synthesis of 20:4 or an increased utilization of 18:2 or 20:4. Either is a possibility. Age as well could have accounted for some of the differences.

Other Variables Which Might Influence Total Serum Lipids

Apart from decreased body fatness and altered fatty acid pattern status, we acknowledge that other variables (for example, alcohol, exercise, smoking habit, and age) may influence total lipids. By the very nature of the disorders (hyperlipidemia and overweight) which distinguish our treatment from control groups, general lifestyle, and not only diet, would be expected to be different between the groups. However, alcohol consumption was not different between the two groups at baseline. There was a significant reduction in alcohol intake in the hyperlipidemic group at the first, but not the second, observation point, but no significant change in alcohol intake at the first or second observation point in the control group. Prevalence of cigarette smoking was 35% for hyperlipidemic men and 33% for normolipidemic men; these did not change through the course of the study. We cannot comment on the possibility of a difference in exercise patterns because we did not formerly assess them; in counseling we made no effort to encourage a change in physical activity; on the other hand, we did not counsel against a reduction in energy expenditure which may arise through reduced physical activity [16], nor did we counsel against a change in pattern of physical activity which may occur with reduced energy intake [17]. We sought to decrease energy intake, decrease total fat intake, improve P:S ratio, and lower alcohol consumption. Thus, by the characteristics of our treated and control groups and the nature of our dietary intervention, it is likely that most of the serum lipid change can be attributed to the dietary intervention package.

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