European Journal of Clinical Nutrition (1998) 52, 419–424

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Lecithin has no effect on serum lipoprotein, plasma fibrinogen and macro molecular protein complex levels in hyperlipidaemic men in a double-blind controlled study

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Objective: To examine the effects of lecithin on serum lipoprotein, plasma fibrinogen and macro molecular protein complex (MPC) levels.
Subjects and study design: Twenty free living hyperlipidaemic men participated in this double-blind study which controlled for possible indirect effects. The subjects were randomly assigned to one of three treatments: frozen yoghurt or frozen yoghurt with 20 g soya bean lecithin or frozen yoghurt with 17 g sunflower oil. Sunflower oil was used to control for the increased energy and linoleic acid intake from lecithin. Yoghurt served as the 'vehicle' for the lecithin and sunflower oil and yoghurt alone was given to one group to control for possible effects due to the yoghurt 'vehicle', as well as other environmental influences. Variables were measured with standard methods twice at baseline and after 2 and 4 weeks of treatment.
Results: Plasma linoleic acid levels increased significantly with lecithin and sunflower oil treatments indicating that compliance to the treatments were obtained. Lecithin treatment did not have significant effects on serum total cholesterol, triglyceride, high density lipoprotein cholesterol, low density lipoprotein cholesterol, apolipoprotein A, apolipoprotein B or lipoprotein (a) levels. Plasma fibrinogen and MPC levels were also not affected by lecithin therapy. Sunflower oil treatment resulted in significant increased body weight, serum TC and decreased MPC levels.
Conclusion: Lecithin treatment had no independent effects on serum lipoprotein, plasma fibrinogen or MPC levels in hyperlipidaemic men.
Sponsorship: Drybean Producers Organisation (South Africa); Chempure cc (South Africa); SACCA, Pty. Ltd. (South Africa).
Descriptors: lecithin; phospholipids; lipoproteins; lipids; fibrinogen; macro molecular protein complex

Introduction

Lecithin (phosphatidylcholine) is choline containing phospholipids which also includes glycerol esterified with fatty acids (Gurr, 1993). Lecithin has emulsifying properties and is an essential component of biomembranes and lipoproteins. Liver, egg yolk and soya beans are especially rich sources of lecithin (Mahan & Arlin, 1992). Most of the commercial lecithin brands claim hypocholesterolaemic effects and are therefore prescribed by health practitioners for treatment of increased lipoprotein levels. The assumption that lecithin lowers lipoprotein levels is based on evidence from studies done from the 1940s to the early 1980s (Davies & Murdoch, 1959; Morrison, 1958; Saba *et al*, 1978; Steiner & Domanski 1944; Tompkins & Parkin, 1980) and most of these studies were poorly designed and showed controversial results. Some of the major design errors were the following: Firstly, small, heterogenic study groups were used, for example a study group of eight consisting of men and women with different types of dyslipidaemia including familial hypercholesterolaemia, hypercholesterolaemia, combined hyperlipidaemia and normal lipidaemia (Childs *et al*, 1981; Davies & Murdoch, 1959; Greten *et al*, 1980; Saba *et al*, 1978; Tompkins & Parkin, 1980). Secondly, most studies lacked an appropriate control group (Cobb *et al*, 1980; Davies & Murdoch, 1959; Saba *et al*, 1978; Ter Welle *et al*, 1974; Tompkins & Parkin, 1980). Knuiman *et al* (1989) highlighted four out of twenty four studies that attempted to control for the fatty acid component of lecithin namely, the studies done by Greten *et al* (1980), Childs *et al* (1981), Prack *et al* (1983) and Kesaniemi & Grundy (1986). These studies could, however, not demonstrate any independent effects of lecithin on serum cholesterol. Thirdly, in the studies where control groups were included with cross-over designs, no or too short washout periods existed between control (soya bean oil or safflower oil) and lecithin treatments (Greten *et al*, 1980; Kesaniemi & Grundy, 1986). Fourthly, control and lecithin treatment periods were not randomized with no reference group either. For example, all the subjects will follow the control diet first and then switch to the experimental diet or *vice versa* (Childs *et al*, 1981; Greten *et al*, 1980; Kesaniemi & Grundy, 1986; Ter Welle *et al*, 1974).

Plasma fibrinogen is an independent risk factor for coronary heart disease and is positively associated with total cholesterol, low density lipoprotein cholesterol, triglycerides and negatively associated with high density lipoprotein cholesterol (Ernst & Resch, 1993). A macro

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Received 16 December 1997; revised 29 January 1998; accepted 9 March 1998

molecular protein complex (MPC) which form complexes with the fibrin network in blood clots, increasing clot mass and decreasing clot fibrinolysis was described. It is hypothesized that high MPC levels increase thrombotic risk (Lipinski *et al*, 1995). Veldman (1996) showed that MPC levels can be manipulated through dietary intervention in a study where pectin, a soluble dietary fibre component, decreased MPC levels significantly. No information on the effects of lecithin on plasma fibrinogen or MPC levels could be found.

In this study the effect of a new, pure (96% phospholipids) soya bean lecithin product on serum lipoproteins, plasma fibrinogen and MPC levels were investigated by using a double-blind study design in which possible indirect effects were controlled for.

Methods

Subjects

The study was approved by the Ethics Committee of the Potchefstroom University for Christian Higher Education and an informed consent form was signed by each participant. Twenty-one hyperlipidaemic men with a mean ($\pm$ standard deviation) age of 48.25 ± 9.75 y who regularly attended the Lipid Clinic at the Potchefstroom University, participated in this study.

The study was done under free living conditions. The exclusion criteria were: smoking, baseline serum total cholesterol (TC) levels < 5.2 mmol/L, baseline serum triglyceride (TG) levels > 5 mmol/L, familial hypercholesterolaemia, baseline fasting blood glucose > 6.7 mmol/L, body mass index (BMI) > 30 kg/m^2, and use of lipid lowering drugs.

Study design

At entering the Lipid Clinic all subjects were prescribed a high-fibre, low-fat diet. Compliance to the diet varied. However, all but two subjects were stabilized on their individual adapted diets. Subjects were paired off into three groups of seven subjects each according to their baseline serum TC levels, age and body mass index (BMI). The groups were then randomly assigned to one of three treatments, namely: (1) 175 g/d of frozen yoghurt; (2) 175 g/d of frozen yoghurt with 20 g soya bean lecithin; and (3) 175 g/d of frozen yoghurt with 17 g sunflower oil. The compositions of the three treatments are summarized in Table 1. By mixing the sunflower oil and soya bean lecithin into the yoghurt, blindness was ensured. Skim milk yoghurt was chosen as the 'vehicle' for the lecithin and sunflower oil because it is compatible with the low-fat diet. Yoghurt alone was given to one group to control for possible effects due to the yoghurt 'vehicle', as well as other environmental influences. Sunflower oil was included in the yoghurt of another group to control for the increased energy and linoleic acid intake from lecithin, because an intake of 20 g of SternpurPM lecithin provides 622 kJ (148 kcal) and contains 59% linoleic acid (Stern typisch Lecithin & Soja, Hamburg, Germany). After two baseline fasting blood samples (one week apart) and anthropometric measurements (height and weight) were obtained, the subjects started with the treatment. The means of the two baseline values were calculated. Fasting blood samples and anthropometric measurements were then taken after two and four weeks of treatment, BMI (kg/m^2) was calculated.

The subjects were asked to maintain their usual diet, alcohol consumption and physical activity. One of the subjects in the lecithin group was excluded from the analysis, because he did not maintain his usual lifestyle. He went on vacation and gained 7 kg.

Blood samples

The subjects were required to fast overnight (12 h). Venous blood samples were collected using a 21-gauge scalp vein infusion set. All samples were drawn with minimal stasis and between 07:00 and 10:00 to avoid effects of diurnal variation. EDTA blood was used for the analysis of haematocrit and haemoglobin. Clotted, citrated and EDTA blood was centrifuged for 15 min at 2000 *g* to yield serum for lipoprotein, plasma for haemostatic and plasma for fatty acid assays, respectively. Serum and plasma were divided into aliquots and frozen at -72°C within 30 min for later analysis.

Experimental methods

To monitor compliance, the fatty acid composition of total plasma lipids was determined with chloroform/methanol extractions as described by Smuts *et al* (1992) (coefficient of variance (CV) = 4%). Total cholesterol (TC) concentrations were determined enzymatically with the Technicon Omnipak-method ((SM-4-0139/86), Bayer, Puteaux Cedex). Low density lipoprotein cholesterol (LDL) was determined with the CHOD-Iodide method (Merck, Darmstadt, Germany). LDL was precipitated by heparin at its iso-electric point (pH 5.12). After centrifugation, the high density lipoprotein cholesterol (HDL) and the very low density lipoprotein cholesterol (VLDL) remained in the supernatant and were determined with enzymatic methods. The LDL concentration was then calculated according to

Table 1 Fatty acid composition (g) of 175 g of frozen yoghurt, without or with lecithin or sunflower oil

Nutrient	*Frozen yoghurt*[a]	*Frozen yoghurt*[a] *with 20 g Sternpur PM lecithin*[b]	*Frozen yoghurt with 17 g sunflower oil*[a]
Energy (kJ)	948	1461	1485
Fatty acids (g):			
Palmitic acid (C16:0)	0.84	4.74	1.78
Stearic acid (C18:0)	0.30	1.06	1.12
Oleic acid (C18:1, n-9)	0.70	2.62	3.69
Linoleic acid (C18:2, n-6)	0.04	11.84	10.92
α-Linolenic acid (C18:3, n-3)	0.04	1.43	0.21
Phospholipids (%)	—	96	—

[a] Composition as given by South African Food Composition Tables (Gouws & Langenhoven, 1986; Langenhoven *et al*, 1991).
[b] Composition as given by Stern typisch Lecithin & Soja, Hamburg, Germany.

the following formula: LDL = TC − cholesterol in supernatant. Apolipoprotein A (apoA) and apolipoprotein B (apoB) were determined with an immuno-chemical method (Behring Institute, Marburg, West Germany), antiserum for human apoA (code no. OUED), and human apoB (code no. OSAN). Lipoprotein (a) (Lp(a)) was determined with a radio-immunological method (Pharmacia Apo(a) RIA). Plasma fibrinogen was measured with the method of Ratnoff & Menzies (1951) (CV = 3.31%) that measures the concentration of clottable protein. The macromolecular protein complex (MPC) was measured by precipitating thrombin unclottable proteins with protamine sulfate (Sigma, St. Louis, USA, Cat. no. p-4020) (Lipinski *et al*, 1995) and then measuring the protein concentration with the method of Rotnoff & Menzies (1951) (CV = 3.6%). Haematocrit was determined with the capillary tube method and haemoglobin with a calorimetric method (Boehringer Mannheim, Cat.no. 124729).

Statistical analysis

Because a normal distribution of data could not be assumed, significant differences within groups were determined with the Wilcoxon matched pairs test and between groups with the Mann–Whitney U-test. The computer software package Statistica® was used for these analyses. 95% Confidence intervals (CI) were calculated (Conover, 1971). All data are presented as median (95% CI). A *P*-value of less than or equal to 0.05 was regarded as being statistically significant.

Results

The median (95% CI) levels of the variables during yoghurt, lecithin and sunflower oil treatments are summarized in Tables 2–5.

Table 2 indicates that plasma linoleic acid (C18:2) levels increased significantly with both lecithin and sunflower oil treatments and were also significantly higher after two weeks of treatment, compared to the yoghurt group. Plasma palmitic (C16:0) and palmitoleic acid (C16:1) levels increased significantly in the yoghurt group. After four weeks of treatment the median palmitic acid level was significantly higher compared to the lecithin and sunflower oil groups. Plasma stearic acid (C18:0) and oleic acid (C18:1) levels did not change with any of the treatments while plasma gamma-linolenic acid (C18:3, n-6) levels decreased significantly with sunflower oil treatment and alpha-linolenic acid (C18:3, n-3) levels increased with lecithin treatment.

Weight and BMI was not affected in the yoghurt and lecithin groups, but increased significantly with sunflower oil treatment. Haemoglobin levels tended to decrease in the lecithin and sunflower oil groups and the decrease was significant in the yoghurt group. These small decreases were, probably, not of any clinical significance, because reference values for haemoglobin in men are 8.68–11.16 mmol/L (Lee & Nieman, 1993). Haematocrit levels did not change with any of the treatments (Table 3).

As shown in Table 4, lecithin therapy had no effect on serum TC, TG, HDL, LDL, LDL/HDL or apoA levels. Serum apoB and Lp(a) levels increased significantly after two weeks of lecithin treatment, but these levels returned to baseline levels after four weeks of treatment. Serum TC levels increased significantly in the group who received sunflower oil. HDL-C levels decreased and LDL/HDL ratio increased significantly in the yoghurt group, but these changes were probably not of any clinical significance.

Plasma fibrinogen and MPC levels were not effected by yoghurt or lecithin treatments, but MPC-levels were significantly reduced by sunflower oil treatment (Table 5).

Discussion

The significant increases in plasma linoleic acid levels with both lecithin and sunflower oil treatments indicate good compliance to the treatments. Because the lecithin product also contains 7% w/w of alpha-linolenic acid (Stern Lecithin & Soja GmbH & Co Kg, Hamburg, Germany) the significant increase in plasma alpha-linolenic acid levels may also be indicative of good compliance to the

Table 2 Dietary compliance: median [95% confidence interval (CI)] changes in plasma fatty acids (% w/w) during treatment

		Yoghurt		*Yoghurt with Lecithin*		*Yoghurt with sunflower oil*	
		Median	*95% CI*	*Median*	*95% CI*	*Median*	*95% CI*
C16:0	B	21.06*	16.87–24.37	21.47	17.67–28.73	21.47	20.05–25.52
	M	22.70	20.10–24.64	22.33	17.65–24.72	21.20	18.33–23.1
	E	22.98$^{*\#\bullet}$	21.17–29.13	20.90$^{\#}$	17.71–25.15	21.34$^{\bullet}$	17.71–26.05
C16:1	B	1.54*	0.86–2.24	1.72	1.20–2.22	1.41	0.92–2.26
	M	1.66	0.74–2.12	1.52	0.80–2.42	1.73	0.91–2.63
	E	1.95*	0.91–4.32	1.71	0.98–3.04	1.50	1.05–2.23
C18:0	B	7.64	7.21–8.90	7.68	6.79–8.89	7.55	6.87–8.90
	M	7.56	6.87–8.65	7.32	6.82–7.99	7.16	6.86–8.25
	E	7.64	6.91–7.96	7.52	7.24–8.00	7.32	6.57–8.55
C18:1	B	23.72	20.44–25.74	22.35	19.63–24.20	22.99	16.28–27.35
	M	22.86	19.34–27.34	21.68	18.21–25.12	19.95	19.08–24.75
	E	22.77	21.73–26.79	20.59	18.82–25.13	21.68	15.37–24.97
C18:2	B	32.13	29.48–33.09	32.87$^{*\clubsuit}$	25.46–38.71	31.44$^{\heartsuit}$	27.83–43.33
	M	31.59$^{\#\bullet}$	28.20–33.92	34.59$^{\#\clubsuit}$	31.37–40.09	35.55$^{\bullet}$	31.58–41.64
	E	30.19	24.91–33.28	35.70*	29.23–39.53	36.27$^{\heartsuit}$	30.44–45.96
C18:3 (n-6)	B	0.50	0.31–0.75	0.49	0.33–0.65	0.53*	0.29–0.69
	M	0.49	0.42–0.88	0.50	0.41–0.67	0.49	0.30–0.97
	E	0.63	0.44–0.99	0.58	0.40–0.71	0.49*	0.32–1.07
C18:3 (n-3)	B	0.32	0.22–0.43	0.30$^{* \#}$	0.23–0.56	0.29	0.20–0.75
	M	0.43	0.22–0.63	0.54*	0.30–0.71	0.35	0.24–0.47
	E	0.35	0.23–0.63	0.52$^{\#}$	0.25–0.80	0.26	0.21–0.60

B: Baseline; M: Middle; E: End.
Medians (95% CI) with the same symbol differed significantly ($P \leq 0.05$).

Table 3 Median [95% confidence interval (CI)] age and changes in anthropometric, blood pressure, haematocrit and haemoglobin levels during treatment

		Yoghurt		*Yoghurt with Lecithin*		*Yoghurt with sunflower oil*	
		Median	*95% CI*	*Median*	*95% CI*	*Median*	*95% CI*
Age (y)		44.00	30–58	47.50	34–56	58.00	31–63
Weight (kg)	B	78.7	72.2–108.0	84.0	79.6–86.8	86.6*	67.6–100.1
	M	79.2	71.6–108.0	84.1	79.6–87.4	88.2*	68.2–100.0
	E	78.8	71.6–107.5	83.9	79.6–88.6	88.8	68.4–100.5
BMI (kg/m^2)	B	26.20	22.91–31.22	26.39	25.36–28.15	27.64*#	22.85–33.01
	M	25.98	23.17–31.22	26.39	25.54–28.28	28.15*	23.05–33.28
	E	25.98	23.36–31.07	26.19	25.63–28.21	28.26#	23.12–32.81
Haematocrit (%)	B	47.00	43.50–49.00	44.75	40.00–45.50	45.00	41.00–46.50
	M	48.00	44.00–52.00	45.00	40.00–47.00	47.00	36.00–50.00
	E	48.00	43.00–50.00	44.00	43.00–47.00	47.00	40.00–48.00
Haemoglobin (mmol/L)	B	11.36*	10.54–12.45	11.63	10.54–12.18	11.63	8.91–12.18
	M	11.91	10.00–13.00	11.49	7.00–12.18	11.63	7.82–12.18
	E	10.82*	10.27–11.91	10.68#	10.27–11.36	10.00#	9.18–11.91

B: Baseline; M: Middle; E: End.
Medians (95% CI) with the same symbol differed significantly ($P \leq 0.05$).

Table 4 Median [95% confidence interval (CI)] changes in serum lipoprotein levels during treatment

		Yoghurt		*Yoghurt with Lecithin*		*Yoghurt with sunflower oil*	
		Median	*95% CI*	*Median*	*95% CI*	*Median*	*95% CI*
TC (mmol/L)	B	6.40	5.32–7.62	6.34	6.12–8.03	6.76*	5.90–6.80
	M	6.52	5.73–7.78	6.82	6.22–8.41	6.78	6.03–7.50
	E	6.53	5.99–7.98	6.58	5.35–8.36	6.90*	6.08–7.12
TG (mmol/L)	B	1.93	1.43–3.15	2.13	0.96–6.05	1.99	1.06–4.56
	M	1.67	1.38–1.86	1.56	1.26–1.68	1.61	1.44–1.95
	E	1.99	1.66–3.82	1.72	1.16–2.48	1.59	0.99–2.27
HDL (mmol/L)	B	1.13*	0.95–1.36	1.12	0.95–1.49	1.05	1.00–1.48
	M	1.05	0.86–1.27	1.15	0.83–1.72	1.12	1.94–1.30
	E	1.07*	0.81–1.21	1.19	0.75–1.68	1.05	0.91–1.53
LDL (mmol/L)	B	4.99	3.99–6.07	4.91	4.22–6.53	5.25	4.44–5.49
	M	5.02	4.43–6.29	5.03	4.56–6.85	5.15	4.40–5.97
	E	4.96	4.44–6.56	4.98	3.72–6.28	5.19	4.68–5.79
LDL/HDL	B	4.63*	3.36–5.29	4.47	2.88–6.87	5.00	3.39–5.34
	M	4.77	4.47–5.99	4.54	2.80–8.25	4.30	3.52–6.35
	E	5.26*	4.15–6.13	4.38	2.21–8.37	5.07	3.40–6.36
ApoA (mmol/L)	B	1.48	1.15–1.74	1.37	1.30–1.60	1.35	1.22–1.70
	M	1.38	1.16–1.55	1.42	1.25–1.85	1.36	1.18–1.76
	E	1.40	1.18–1.51	1.47	1.27–1.65	1.34	1.10–1.70
ApoB (mmol/L)	B	1.32	1.09–1.53	1.32*	1.17–1.72	1.36	1.18–1.52
	M	1.35	1.14–1.59	1.40*#	1.23–1.86	1.40	1.17–1.51
	E	1.32	1.25–1.65	1.31#	1.10–1.70	1.40	1.12–1.51
Lp(a) (U/L)	B	200.5	17.0–944.2	202.3*	17.5–794.0	157.0	58.0–671.0
	M	194.5	13.9–431.3	241.3*	16.4–861.8	151.2	54.6–635.5
	E	153.0	14.7–453.6	213.4	9.64–324.2	173.0	63.4–670.3

B: Baseline; M: Middle; E: End.
Medians (95% CI) with the same symbol differed significantly ($P \leq 0.05$).

Table 5 Median [95% confidence interval (CI)] changes in plasma fibrinogen and macromolecular protein complex (MPC) during treatment

		Yoghurt		*Yoghurt with Lecithin*		*Yoghurt with sunflower oil*	
		Median	*95% CI*	*Median*	*95% CI*	*Median*	*95% CI*
Fibrinogen	B	2.88	1.14–3.47	3.16	1.83–4.00	3.11	1.49–4.07
	M	2.59	1.09–3.69	3.13	1.72–4.84	3.13	1.75–4.75
	E	2.40	2.16–5.44	3.68	1.69–4.38	2.56	1.66–4.00
MPC (g/L)	B	5.03	3.05–6.39	5.30	2.26–16.75	5.30*	3.27–11.16
	M	4.88	2.65–5.76	6.14	2.04–12.68	4.63	2.74–8.84
	E	4.57	2.99–8.05	5.58	1.89–7.74	3.81*	2.84–10.43

B: Baseline; M: Middle; E: End; MPC: Macro molecular protein complex.
Medians (95% CI) with the same symbol differed significantly ($P \leq 0.05$).

lecithin treatment. Plasma palmitic and palmitoleic acid levels increased significantly in the control group. These more saturated fatty acids (SFA) might have been synthesized in the body from an increased intake of carbohydrate (Lands, 1995), because it has been indicated that the SFA composition in the plasma is not a good indicator of dietary intake of SFA (Simon *et al*, 1995). The significant decrease in gamma-linolenic acid levels with sunflower oil treatment is probably of no clinical significance since the changes in the yoghurt and lecithin groups were even greater although not significant.

The salient observations of this study on free living hyperlipidaemic men were that lecithin had no effect on the measured risk factors and markers for coronary heart disease but that the sunflower oil resulted in increase in body weight, serum TC and reduced plasma MPC levels. The increased serum TC levels may probably be explained by the increase in body weight in these subjects (Newman *et al*, 1990).

Results from clinical trials of lecithin have reported total cholesterol and LDL lowering effects (Davies & Murdoch, 1959; Morrison, 1958; Saba *et al*, 1978; Steiner & Domanski, 1944; Tompkins & Parkin, 1980) while other studies showed no significant effects (Childs *et al*, 1981; Cobb *et al*, 1980; Greten *et al*, 1980; Kesaniemi & Grundy, 1986; Ter Welle *et al*, 1974). A decreasing effect of lecithin on TG-levels (Cobb *et al*, 1980; Saba *et al*, 1978; Tompkins & Parkin, 1980) and in some studies no effect (Childs *et al*, 1981; Greten *et al*, 1980; Kesaniemi & Grundy, 1986; Ter Welle *et al*, 1974) have been reported. Some studies reported no effect of lecithin on HDL levels (Greten *et al*, 1980; Kesaniemi & Grundy, 1986; Prack *et al*, 1983), while Childs *et al* (1981) reported an increasing effect on HDL, independent of its polyunsaturated fatty acid moiety. As discussed in the introduction most of these studies were poorly designed and are not comparable to the present study because of the different study designs, different amounts of lecithin used, ranging from 0.7–54 g/d (Knuiman *et al*, 1989), and different concentrations of phospholipids in lecithin products, ranging from 29% (Childs *et al*, 1981) to 96% in the present study.

It has been demonstrated in humans that intake of lecithin (Kesaniemi & Grundy, 1986) and infusion of lecithin intraduodenally (Beil & Grundy, 1980), decreases the absorption of cholesterol in the small intestine. Kesaniemi & Grundy (1986) suggested that if lecithin is given in multiple doses throughout the day, the inhibition of cholesterol absorption might be higher and that the amount of dietary cholesterol may also have an effect on the results. This may, in part, explain why lecithin did not lower TC levels in this particular study, because these subjects followed a low cholesterol diet. Other studies in which subjects followed a low cholesterol baseline diet could also not demonstrate TC lowering with lecithin (Greten *et al*, 1980; Kesaniemi & Grundy, 1986).

In this study lecithin treatment had no effect on plasma fibrinogen or MPC levels, but sunflower oil treatment decreased MPC levels significantly that may probably decrease thrombotic risk (Lipinski *et al*, 1995). This possible beneficial effect of sunflower oil on MPC needs to be further investigated.

Conclusion

Lecithin treatment had no independent effects on serum lipoproteins, plasma fibrinogen or MPC levels in free living hyperlipidaemic men in a double-blind study which controlled for possible indirect effects.

Acknowledgements—The authors wish to thank Sister Chrissie Lessing for the outstanding role she played in the handling and organizing of the subjects, the drawing of the blood and the anthropometic measurements. We also want to thank Dr. Michael Sly from Chempure cc (SA) who donated the soya bean lecithin and Christo van der Walt from SACCA, Pty. Ltd (SA) who donated the yoghurt. A special thanks to the patients from the Lipid Clinic of the Potchefstroom University who took part in the study and the Drybean Producers Organisation (SA) who supports the Lipid Clinic financially and without whom this study would not have been possible.

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