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EFFECT OF PARTIAL REPLACEMENT OF VISIBLE FAT BY GHEE (CLARIFIED BUTTER) ON SERUM LIPID PROFILE

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Abstract : A randomised controlled trial with a parallel design was conducted on 24 healthy young volunteers who were divided into two groups. After a lead-in period of 2 wk, the experimental group (n = 11; 9 male, 2 female) had for 8 wk a lactovegetarian diet providing about 25% of the energy intake in the form of fat, of which ghee provided 10 en% and the remaining fat energy came from mustard oil and invisible fat. The control group (n = 13; 8 male, 5 female) had a similar diet except that all visible fat was in the form of mustard oil. In neither group was there any significant change in the serum lipid profile at any point in time. At 8 wk, 2 volunteers in the experimental group, and 1 volunteer in the control group had more than 20% rise in serum total cholesterol as compared to their 0 wk values. There was also an appreciable increase in HDL cholesterol at 8 wk in the experimental group, but it was not statistically significant. Consuming ghee at the level of 10 en% in a vegetarian diet generally has no effect on the serum lipid profile of young, healthy, physically active individuals, but a few individuals may respond differently.

Key words : ghee (clarified butter) fats dietary fats serum lipids cholesterol lipoproteins

INTRODUCTION

Ghee is a prestigious traditional cooking medium in India. For some time, however, medical opinion has been against its use on the basis of the generalization that being an animal fat, it is saturated; and being saturated, it would raise serum cholesterol, which in turn would increase the risk of atherosclerosis. However, saturated fats are not completely to be avoided. The step I diet of the National Cholesterol Education

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Program (NCEP) of USA provides up to 30% of the energy intake as fat, of which saturated (SFA), monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) should be about 10% each (1). Further, composition-wise, ghee has about 65% SFA, and 32% MUFA (2). The MUFA in ghee may neutralize the adverse effects, if any, of SFA as in case of palm oil (3). Studies on ghee are needed not only to evaluate whether or not ghee is harmful, but also to investigate whether it is beneficial. Because of its high MUFA content, it may raise HDL cholesterol (4), and its content of conjugated linolenic acid may confer on it anti-carcinogenic properties (5). On the other hand, it has also been proposed that the cholesterol oxides in ghee might render it atherogenic (6). Specific information on the effects of ghee on serum lipid profile is scanty. In a study on 7 subjects done at the National Institute of Nutrition (NIN), Hyderabad, when the subjects shifted from a diet providing 50 g of visible fat per day in the form of groundnut oil to one providing it in the form of 35 g ghee and 15 g groundnut oil for 8 wk, the mean levels of serum cholesterol and triglycerides were not altered, but in two subjects serum total cholesterol increased by more than 20 mg/dL (7). The study was, thus, inconclusive. More studies on the effect of ghee on serum lipid profile are clearly required. The present study is a step in that direction.

METHODS

Subjects

The study was conducted on 35 healthy, physically active adult volunteers (24 male and 11 female), all of them inmates of Sri Aurobindo Ashram, New Delhi. However, only 24 volunteers (17 male and 7 female) completed the study. None of the subjects smoked or used alcohol. All the subjects had all their meals at a common kitchen in the ashram. The meals were vegetarian, and the principal cooking medium was mustard oil.

Experimental design

The study was conducted in the form of a randomised controlled trial with a parallel design. The study started with a lead-in period of 2 wk, during which the subjects were requested to consume a relatively constant vegetarian diet, take no ghee at all, avoid baked foods (because they may contain ghee), maintain a relatively constant level of physical activity, and make no changes in the cooking medium.

After the lead-in period of 2 wk, the subjects were randomly divided into two groups, Group A (n = 11; 9 male, 2 female) and Group B (n = 13; 8 male, 5 female). Group A (experimental) consumed for 8 wk a diet in which ghee provided 10% of the energy intake while total energy from fats was 25% of the energy intake. Group B (control) consumed for 8 wk a similar diet except that all visible fat came from mustard oil as during the lead-in period and, by and large, even before that.

Ghee was provided to Group A in one dish (potato curry) such that the subject would get 5% of the day's energy intake from the visible fat in the dish at lunch, and 5% at dinner. Group B also received the potato

curry, but for them it was prepared in mustard oil. The investigator who analysed the blood samples did not know to which group a given subject belonged.

Fasting blood samples were collected at the beginning of the lead-in period (–2 wk), the end of the lead-in period (0 wk), at 4 wk and at 8 wk.

The protocol of the study was approved by the Ethics Committee of the All India Institute of Medical Sciences, and the volunteers gave their informed written consent for participation in the study.

Measurements

Total cholesterol, total triglycerides, HDL cholesterol, LDL cholesterol and lipoprotein(a) (Lp-a) were estimated using kits from Randox Laboratories Ltd., Ardmore, U.K. VLDL cholesterol and total cholesterol:HDL cholesterol ratio were calculated from the above measurements. Lp-a was determined only a 0 wk and 8 wk.

Statistical analysis

The effect of ghee on serum lipids was analysed by repeated measure analysis using SYSTAT version 7.0. In case a variable showed a significant change ($P<0.05$), two-way ANOVA was applied to compare the values at the beginning of the dietary period (0 wk) with those at the middle (4 wk) and at the end (8 wk). If ANOVA gave significant F values, Neuman Keul's multiple range test was applied to evaluate the significance.

RESULTS

The physical characteristics of the subjects at 0 wk and 8 wk are shown in Table I. There were no significant differences in characteristics between the two groups. Further, there was no significant change in body weight, body mass index, or waist-hip ratio in either group during the study. Analysis of the average daily diet on a typical day revealed that in the experimental group fat energy% was 24.2% and the PUFA:SFA ratio 0.71, and the corresponding values in the control group were 20.7% and 1.16.

TABLE I: Characteristics of the subjects.

	Group A (experimental)	*Group B (control)*
Age (years)	27.09±5.99	26.31±4.81
Weight (kg)		
At 0 wk	54.95±11.26	55.19±7.01
At 8 wk	54.23±11.71	54.42±7.57
Height (cm)	163.27±8.54	162.15±7.14
BMI (kg/m^2)		
At 0 wk	20.58±3.97	20.93±1.51
At 8 wk	20.27±4.05	20.61±1.62
Waist/hip ratio		
At 0 wk	0.83±0.06	0.84±0.06
At 8 wk	0.85±0.05	0.83±0.06

All values are mean±standard deviation.

The serum lipoprotein profiles at different points during the study in experimental group and control group have been shown in Tables II and III respectively. Lp-a values, in both the groups, have been shown in Table IV. The serum lipid profile in the experimental group at the beginning

TABLE II: Serum lipid profile in the experimental (ghee) group, Group A.

Variable	*Value (mean ± SD)*			
	-2 wk	*0 wk*	*4 wk*	*8 wk*
Total cholesterol (mg/dL)	175.18±38.50	153.55±35.47	177.82±51.59	159.55±34.27
LDL cholesterol (mg/dL)	98.29±33.47	95.02±28.85	94.79±37.12	96.33±35.17
HDL cholesterol (mg/dL)	57.91±9.35	41.64±6.87	62.36±15.08	54.51±10.89
Total cholesterol/HDL cholesterol	3.11±0.88	3.70±0.69	2.88±0.57	3.03±0.75
VLDL cholesterol (mg/dL)	19.00±6.44	16.49±6.49	20.65±12.86	13.78±4.48
Triglycerides (mg/dL)	95.00±32.20	82.45±32.45	103.27±64.27	68.91±22.39

TABLE III: Serum lipid profile in the control group, Group B.

Variable	*Value (mean ± SD)*			
	-2 wk	*0 wk*	*4 wk*	*8 wk*
Total cholesterol (mg/dL)	163.92±39.74	146.92±29.54	154.62±39.71	142.54±31.10
LDL cholesterol (mg/dL)	101.52±32.56	91.18±28.91	90.29±42.79	82.64±36.69
HDL cholesterol (mg/dL)	43.23±9.20	40.35±9.50	48.19±8.23	46.38±11.72
Total cholesterol/HDL cholesterol	3.84±0.86	3.80±0.99	3.33±1.13	3.30±1.20
VLDL cholesterol (mg/dL)	19.14±6.54	15.42±6.46	16.09±4.04	13.48±4.95
Triglycerides (mg/dL)	95.69±32.68	77.08±32.28	80.46±20.21	65.46±20.17

TABLE IV: Serum lipoprotein(a) during the study.

Experimental group (Group A)		*Control group (Group B)*	
0 wk	*8 wk*	*0 wk*	*8 wk*
38.15±19.69	42.07±20.81	25.13±15.02	32.00± 15.19

Values are expressed in mg/dL (Mean±S.D.).

(0 wk) and end (8 wk) of the dietary period has been shown in Fig. 1.

In neither group was there any significant change in the serum lipid profile at any point in time.

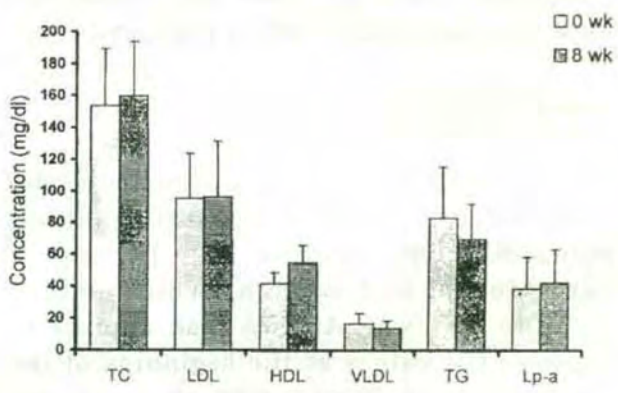

Fig. 1: Serum lipid profile in the experimental (ghee) group at the beginning (0 wk) and end (8 wk) of the dietary period. The bars represent mean and standard deviation.

DISCUSSION

In the present study replacing mustard oil by ghee such that ghee provided about 10% of the energy intake in a diet providing about 25% of the energy intake as fat did not lead to any significant change in the serum lipid profile. The absence of any change was not surprising in view of the composition of ghee. The ghee used in our study was analysed and found to have 56.7% SFA and 35.4% MUFA. Thus, like palm oil, ghee is also a saturated fat but with a substantial quantity of MUFA. Hence, like palm oil, it may also be expected to have no significant effect on serum lipid profile (3).

There was an appreciable rise in HDL cholesterol at 8 wk as compared to 0 wk in the experimental group (Table II). Although the change was not statistically significant, it may be a genuine change attributable to the high MUFA content of ghee (4).

Although the experimental group as a whole did not show any significant change in lipid profile, some individuals had appreciable changes. At 8 wk, two subjects in the experimental group had more than 20% rise in serum total cholesterol as compared to their 0 wk values. But one subject in the control group also had a similar rise. Further, one subject in each group showed a fall of more than 20% in the serum cholesterol at 8 wk as compared to the 0 wk value. No change in mean values but appreciable variation in individual response was observed also by Ghafoorunissa (7). The individual variation in response may be genetically determined (8).

An interesting observation in relation to individual variation in response was that at 4 wk, 6 subjects in the experimental group had more than 20% rise in serum cholesterol as compared to their 0 wk values. This is reflected also in the higher mean value and higher S.D. at 4 wk (Table II). But by 8 wk, only 2 of these subjects continued to have the hypercholesterolaemic response to ghee. Thus it appears the homeostatic mechanism for regulation of serum cholesterol takes about 8 wk to be effective when disturbed by a higher intake of saturated fat.

The experimental and control groups were similar in almost every respect considered relevant to the study, but in spite of randomisation, at −2 wk the HDL cholesterol level of the experimental group was considerably higher than that of the control group (Tables II and III). However, the difference became negligible by 0 wk. Thus the lead-in period of 2 wk was necessary and sufficient to make the two groups completely comparable, as has been observed also by Ghafoorunissa (7).

The present study suggests that the adverse opinion about ghee prevalent in the medical community may not be entirely valid. However, caution is required before these results are extrapolated to the entire population because the subjects of this study were all young, healthy, normolipidaemic, vegetarian, physically active, and following a yogic lifestyle in the salubrious environment of an ashram. Thus, further studies are required on a larger number of subjects, particularly on those who may be older, hyperlipidaemic and following different lifestyles.

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