The relationship between dietary factors and serum cholesterol values in the coloured population of the Cape Peninsula

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Summary

A cross-sectional study of 976 coloured subjects aged 15 -64 years identified a population consuming a typical Western diet. Nutrient intake, determined by the 24-hour dietary recall method, reflected a diet high in fat (37% of total energy intake) and animal protein and a polyunsaturated/saturated fatty acid ratio of 0,85. Only 32,2% of men and 27,5% of women consumed a prudent diet (Keys score ⩽ 28).

The influence of this Western diet on serum total cholesterol (TC) levels was seen to be marked when participants with a high risk of developing coronary heart disease (CHD) were compared with those with a TC level putting them at low risk; the former consumed significantly more saturated fat and had a higher mean Keys score.

Multiple linear regression analysis on TC levels of men identified six variables that explained 26,9% of the variation of TC. These were body mass index, age, the inverse of the polyunsaturated fat intake, saturated fat intake, polyunsaturated/saturated fat ratio and cholesterol intake. For women only three variables (age, the inverse of the polyunsaturated/saturated fat ratio, and body mass index) explained 30,2% of the variation of TC.

Promotion of the prudent diet to lower TC levels of the coloured population of the Cape Peninsula is an increasingly urgent priority.

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The coloured population of the Cape Peninsula has a high prevalence of coronary heart disease (CHD) risk factors.[1-4] If appropriate cut-off points[5] are used to identify all coloureds at risk of CHD according to serum cholesterol level, then hypercholesterolaemia is affecting more coloureds in the Cape Peninsula than any other CHD risk factor.

The association between raised serum cholesterol levels and a diet rich in saturated fatty acids and cholesterol and high in energy has been studied extensively in an effort to validate the diet-heart hypothesis, which links diet with CHD via serum cholesterol.[6,7] Antagonists of this hypothesis have frequently highlighted the failure of the epidemiological studies within populations to show significant associations between dietary constituents and serum cholesterol levels.[8-10]

The Western Electric Study,[11] which examined 1 900 middle-aged men, found a significant positive association between dietary scores (based on each participant's intake of cholesterol and saturated and polyunsaturated fatty acids) and their serum cholesterol level at the baseline survey. The Western Electric Study was the first to show this association in a cross-sectional survey. It was also suggested[11] that the failure to observe this diet-serum cholesterol association in other studies could have been due to methodological problems. Jacobs *et al.*[12] have pointed out that if variances in observed nutrient intake and serum cholesterol levels were sufficiently large in cross-sectional population studies, very low correlation coefficients would be expected, even when there is cause-and-effect relationship.

The purpose of the present study was to investigate the associations of nutrient intake, body mass index (BMI) and age with serum total cholesterol (TC). The study formed part of the CRISIC study in the coloured population of the Cape Peninsula, which has a typical urban westernised lifestyle.

Methods

Study population

Detailed descriptions of the CRISIC study protocol have been reported elsewhere.[1-4,13,14] The study sample consisted of an age- and sex-stratified random sample of 976 subjects from the coloured population of the Cape Peninsula. Exclusion criteria and the population from which the subjects were drawn are described elsewhere in this issue.[15]

Measurements

After completing a training course, a team of 12 professional school nurses visited participants in their homes. A short medical history was taken and anthropometric measurements were done.

Blood pressures were recorded with a mercury sphygmomanometer after subjects had been seated for at least 5 minutes. The American Heart Association guidelines for measuring blood pressures were applied. The diastolic pressure was taken as the point of muffling of the Korotkoff sounds (phase IV). Three intermittent readings were taken and the lowest was recorded.

A non-fasting blood sample was collected, and the serum was separated from the clotted blood within 6 hours and frozen at $-20°C$. TC and high-density lipoprotein cholesterol (HDL-C) values were measured on a Gilford auto-analyser using the Boehringer CHOD-PAP enzymatic method. HDL-C was measured after precipitation of the apoprotein-B-containing lipoproteins with dextran sulphate-magnesium chloride. Triglyceride levels were determined by the Boehringer enzymatic Peridochrom method. In each case the Gilford auto-analyser was calibrated against Precilip or Precilip EL control sera, using values supplied by Boehringer Mannheim for the specific test kit in question. The control samples were included in each batch analysed. The low-density lipoprotein cholesterol (LDL-C) value was calculated using the Friedewald equation[16] and was not calculated for subjects with triglyceride values above 4,56 mmol/l.

Participants were classified on the basis of their serum TC value as being at low risk, moderate risk or high risk of developing CHD. The cut-off points used for the classification were those formulated by the *ad hoc* committee of the Heart Foundation of Southern Africa.[5]

The food intake pattern for this population has been described.[13,14] For the purpose of this study the nutrient intakes of participants with high and low serum TC levels were compared. To determine the nutrient intake of the participants, the nurses were trained by experienced dieticians in completing a dietary questionnaire, which included a 24-hour dietary recall. The 24-hour recall method has been found to be satisfactory for the interpretation of the dietary intake of groups larger than 50 respondents.[17] Interviewers were trained how to estimate and record the amounts of food eaten and the methods of food preparation as accurately as possible. The amounts of food recorded in the 24-hour dietary recall were converted by three dieticians to weights of food eaten and then coded, using the *NRIND Food Composition Tables*.[18] This enabled analysis of food intake in terms of nutrients.

Statistical and descriptive analysis

The following nutrients were considered when investigating associations with serum cholesterol levels: total energy intake, protein (animal and plant), fat (saturated, mono- and polyunsaturated fatty acids), cholesterol and dietary fibre. The accuracy of reported alcohol intake is notoriously poor, so the association of alcohol intake with lipid levels were not studied. Because of the difference in total energy intake between men and women, nutrient intakes were expressed per 4,2 MJ (1 000 kcal). The polyunsaturated/saturated fatty acid ratio (P/S ratio) of the diet was calculated, as was the diet score of Keys *et al.*[19,20] (Keys score). The Keys score was calculated as follows:

$$1{,}26\,(2S - P) + 1{,}5\,\sqrt{1\,000\,C/E},$$

where S = % energy (% E) from saturated fatty acids; P = % E from polyunsaturated fatty acids; C = dietary cholesterol (mg/d); and E = daily energy intake (kcal).

A calculation of the Keys score of the 'prudent diet', based on the US Dietary Goals, resulted in a value of 28 (where S = 10, P = 10, C = 300 and E = 2 700). The less prudent the diet, the higher the Keys score. The study population was divided into three groups on the basis of the Keys score. The first group (685 subjects) had a high score (> 28; typical Western diet), the second group (230) had a moderate score (> 17 but $\leqslant 28$) and the third group had a low score ($\leqslant 17$). A score of 17 was chosen as cut-off point to comply to the requirements of the 24-hour dietary recall methodology to have at least 50 persons in a group when comparing groups.[17] A Keys score of 14 was calculated for a high-carbohydrate traditional African diet pattern associated with low serum TC. LDL-C values for the three groups were compared to establish the influence of their differing diets on LDL-C.

Multiple linear regression analysis on TC was carried out for men and women separately to determine which dietary and other factors contribute independently to the variation of these lipids. The non-dietary variables considered in the regression were those shown by means of univariate and sub-group analysis to be correlated with TC. These were age, systolic and diastolic blood pressure, BMI, energy expenditure during work and leisure time, and occupancy rate (a measure of socio-economic standing). HDL-C was found not to be associated with the TC level. The dietary variables considered were those shown by the diet-heart hypothesis[6,8-11] to be associated with TC and other serum lipids. They were saturated fat intake, polyunsaturated fat intake, P/S ratio, plant protein intake, animal protein intake, alcohol intake and Keys score. Mono-unsaturated fatty acids were found not to be associated with TC.

BMI (weight (kg)/height^2 (m)) was used as a measure of obesity.[21] The occupancy rate, a measure of socio-economic standing, is defined as the number of occupants in a household divided by the number of habitable rooms in the house. In addition, to investigate the relationship between hypercholesterolaemia and socio-economic standing, the occupational categories of employed participants were determined using the

TABLE I. ENERGY AND MACRONUTRIENT INTAKE IN THE CAPE PENINSULA COLOURED POPULATION

	Men		Women	
	Mean ± SD	Median*	Mean ± SD	Median*
Energy intake (kcal/d)	2 301 ± 896	2 204 (1 673 - 2 816)	1 657 ± 765	1 538 (1 124 - 2 008)
Total protein intake (g/d)	84,6 ± 37,2		60,8 ± 28,7	
Total protein intake (% of total energy)	15,0 ± 4,5		15,1 ± 4,6	
Animal protein/total protein ratio	0,67 ± 0,16	0,69 (0,57 - 0,78)	0,67 ± 0,16	0,69 (0,58 - 0,78)
Total fat intake (g/d)	96,3 ± 46,4	89,4 (63,3 - 120,8)	70,3 ± 40,0	64,1 (42,8 - 89,6)
Total fat intake (% of total energy)	36,9 ± 8,8		37,0 ± 8,3	
Saturated fat intake (g/d)	29,9 ± 15,7		22,2 ± 13,9	
Saturated fat (% of total energy)	11,5 ± 3,8		11,6 ± 3,6	
Polyunsaturated fat intake (g/d)	22,9 ± 14,2	20,1 (12,6 - 30,6)	17,1 ± 11,4	14,9 (9,0 - 22,5)
Polyunsaturated fat intake (% of total energy)	8,6 ± 3,7		9,0 ± 3,7	
Dietary P/S ratio	0,83 ± 0,45	0,77 (0,47 - 1,1)	0,85 ± 0,45	0,76 (0,53 - 1,1)
Dietary cholesterol intake (mg/d)	363 ± 287	275 (157 - 479)	264 ± 223	193 (112 - 349)
Dietary cholesterol intake (mg/1 000 kcal)	162 ± 131	123 (77 - 198)	159 ± 117	129 (83 - 200)
Keys score	35,9 ± 14,2		35,8 ± 13,3	
Carbohydrate intake (g/d)	257 ± 115		191 ± 92	
Carbohydrate (% of total energy)	45,0 ± 11,1		46,7 ± 9,5	
Added sugar (% of total energy)	15,0 ± 9,0	13,9 (9,0 - 20,0)	16,4 ± 8,8	15,9 (10,5 - 21,8)

*For skewed frequency distribution the median and interquartile ranges (in brackets) are given to describe the distribution more accurately.

Centre for Applied Social Sciences scales described by Schlemmer and Stopforth[22] for occupations in South Africa. This scale is inversely related to socio-economic status.

Results

The detailed findings of the dietary study in the coloured population of the Cape Peninsula have been published elsewhere[13,14] and are summarised in Table I to illustrate the typical Western-type diet, high in fat, cholesterol and animal protein and with a high Keys score, that formed part of this population's lifestyle. When the prudent guidelines were individually applied, very few participants met any of these cut-off points (Table II).

TABLE II. RESPONDENTS MEETING PRUDENT DIETARY GUIDELINES (%)

	Men	Women
⩽ 30% energy from total fat	18,4	18,3
⩽ 10% energy from saturated fat	36,0	32,5
⩾ 10% energy from polyunsaturated fat	33,5	35,9
⩽ 100 mg cholesterol/1 000 kcal	36,8	35,3
⩾ 10 g fibre/1 000 kcal	10,9	18,9
Keys score ⩽ 28	32,2	27,5

When nutrient intake patterns of participants whose serum TC values put them at high risk of developing CHD were compared with patterns for the low-risk group, showed significantly higher saturated fat intake was found to be associated with high serum TC levels (Table III). The Keys score was also significantly higher in the high TC group.

Fig. 1 illustrates the significantly higher LDL-C level for participants with a dietary Keys score above 28 (mean ± SD 3,44 ± 1,09 mmol/l) compared with the LDL-C level for those with a Keys score of 17 - 28 (3,24 ± 0,94 mmol/l) ($P =$ 0,0136; Student's t-test). The mean LDL-C value for the group with a low Keys score (⩽ 17) (2,99 ± 1,00 mmol/l) was again lower than that for the group with a score of 17 - 28, but did not reach statistical significance when the t-test was applied ($P = 0,0755$).

TABLE III. NUTRIENT INTAKE OF RESPONDENTS WITH HIGH AND LOW CHD RISK SERUM CHOLESTEROL LEVEL

	Mean* (± SD) of high CHD risk group	Mean* (± SD) of low CHD risk group	*P*-value (*t*-test)
No.	147	245	
Mean age (yrs)	37,9	41,9	
Serum TC (mmol/l)	7,51 ± 1,24	4,52 ± 0,64	
Energy intake (kcal/d)	1 968 ± 902	1 970 ± 900	0,98
% energy from animal protein	10,8 ± 5,0	10,0 ± 4,9	0,14
% energy from fat	37,5 ± 9,0	35,8 ± 9,0	0,08
% energy from saturated fat	12,3 ± 3,5	10,8 ± 3,5	0,0001†
% energy from polyunsaturated fat	8,6 ± 3,8	8,7 ± 3,8	0,92
P/S ratio	0,78 ± 0,47	0,87 ± 0,47	0,08
Cholesterol intake (mg/d)	310 ± 237	284 ± 236	0,60
Fibre intake (g/d)	13,0 ± 8,6	12,4 ± 8,5	0,77
Keys score	7,8 ± 13,3	33,4 ± 13,3	0,0018†

*Adjusted, using analysis of covariance, to overall mean age of 40,4 years.
†Significant ($P < 0,05$).

Univariate analyses identified a number of the dietary factors that were significantly but weakly correlated with TC and LDL-C. These variables and other risk factors were entered into a stepwise multiple regression, producing a subset of variables that contributed significantly to the regression of TC (Table IV).

Table IV shows that for men 26,9% of the variation of TC could be explained by six variables. These were, in order of their contribution, BMI, age, the inverse of the polyunsaturated fat intake, saturated fat intake, the P/S ratio and cholesterol

TABLE IV. STEPWISE MULTIPLE LINEAR REGRESSION ANALYSES ON TOTAL CHOLESTEROL IN THE COLOURED POPULATION

Variable	β-coefficient	Standard error of β	Standardised coefficient[§]	Partial correlation (r)[¶]	P-value	Adjusted R^2[‖]
Men						
BMI	**4,1551**	**0,5159**	**0,3382**	**0,3482**	**< 0,0001**	**0,1829**
Age	**0,9233**	**0,1471**	**0,2722**	**0,2782**	**< 0,0001**	**0,2511**
Polyunsaturated fat (g/d)*	**−0,9241**	**0,2862**	**−0,2692**	**−0,1473**	**0,0013**	**0,2531**
Saturated fat (g/d)*	**0,5595**	**0,2370**	**0,1820**	**0,1083**	**0,0186**	**0,2567**
P/S ratio*	**18,1551**	**7,9260**	**0,1684**	**0,1051**	**0,0224**	**0,2637**
Cholesterol (mg/d)*	**0,01557**	**0,0074**	**0,0922**	**0,0969**	**0,0354**	**0,2690**
Keys score†	**0,3640**	**0,1352**	**0,1070**	**0,1228**	**0,0074**	**0,2609**
Women						
Age	**1,5695**	**0,1388**	**0,4715**	**0,4549**	**< 0,0001**	**0,2756**
P/S ratio	**−13,2138**	**3,9630**	**−0,1258**	**−0,1489**	**0,0009**	**0,2882**
BMI	**0,9361**	**0,2910**	**0,1344**	**0,1438**	**0,0014**	**0,3015**

*These variables could be replaced by the Keys Score† in the regression for men.
§ Coefficient of standardised variables.
¶ Remaining correlation after allowing for the effect of the other variables.
‖ Propart of variation of cholesterol accounted for by the predictor variable adjusted for degrees of freedom.

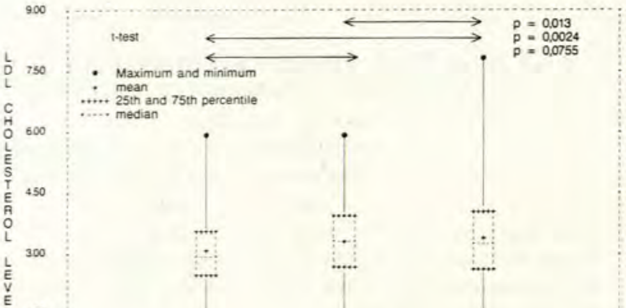

Fig. 1. Comparison of serum LDL-C levels of three groups of participants, with dietary Keys scores of ⩽ 17, > 17 - ⩽ 28 and > 28.

intake. When the last four variables were replaced by the Keys score of the diet 26,1% of the TC variation could be explained. For women 30,2% of the variation of TC could be explained by the variables age, the inverse of the P/S ratio, and BMI.

Further analysis of the data showed that the assessment of socio-economic standing pointed to hypercholesterolaemic participants having higher socio-economic standing than normocholesterolaemic participants. The former group had a significantly lower occupancy rate ($P = 0{,}0076$) and higher occupational category ($P < 0{,}001$) than the latter group.

Discussion

In 1955 the mean TC value for a group of coloured men aged 40 - 58 years was 5,27 mmol/l.[23] In 1982 the mean value for a group of men of the same age in the CRISIC study was 6,13 mmol/l. Although this difference could be explained in part by the different techniques of TC determination in the two studies, it is unlikely that there has in fact been a rise in the mean TC level for this population over the 27-year period, particularly when changes in diet are considered.

Batson,[24] using a different methodology (food purchasing habits), reported fat to represent 26% of energy intake for coloureds in 1953. This was substantially lower than the 35% for whites and higher than the 17% for blacks reported in the same study. Brock and Bronté-Stewart[25] also found that fat intake for coloureds fell between that for whites and that for blacks in 1955 (± 80, 100 and 55 g/d respectively). The 37% of energy derived from fat in the present study represents a dramatic change in fat consumption over the last 3 decades, especially when compared with current white intake of 35% of energy from fat.[26] Diets of blacks in 1971 remained lower in fat, intake of which ranged from 21% to 30% of energy, the more well-off families consuming the greatest proportion of fat.[27]

Table II shows that only 32,2% of men and 27,5% of women had a Keys score that reflected a prudent diet and that most of the coloured population consumed a diet that would raise the serum cholesterol level. The coloured population tended to consume as much fat (or more) than the white population,[26] but for the other prudent guidelines their diets compared favourably with those of whites. Their cholesterol intake was lower (313 mg v. 380 mg) and their P/S ratios higher (0,78 - 1,00 v. 0,48 - 0,59[25]). The high P/S ratios could be due to the use of plant oil (mainly sunflower) for cooking by 92% of the study sample. It should be noted that, because of within-person variation of dietary intake being larger in the 24-hour dietary recall method, the distribution will be broad-based, and a relatively large proportion will fall outside any specific cut-off point on either side of the means. The findings set out in Table II are therefore likely to be overestimations in some cases and underestimations in others.

This cross-sectional study showed that saturated fat intake and the Keys score, which combines dietary saturated fat, polyunsaturated fat and dietary cholesterol, were independently correlated with serum TC values in the coloured population of the Cape Peninsula. These correlations could be shown when comparing subjects with low and high TC values as well as in the stepwise multiple linear regression analysis on TC. The correlations were weak, but when the constraints of cross-sectional studies in proving such relationships are kept in mind,[12] they may be more meaningful in practical terms than is suggested by the statistical criteria.

As seen from Table IV, 26,9% of the variation of TC for men and 30,2% for women could be explained by variables identified by means of stepwise multiple linear regression analysis. There is an anomaly in the results for men in that the coefficient for the P/S ratio is positive, although it is expected to be negative, as was the case for women. This is explained by its strong positive correlation with grams of polyunsaturated fat intake per day, and the stronger inverse relationship of grams of polyunsaturated fat intake per day with TC. Thus, when grams of polyunsaturated fat intake per day is fitted, the residual variation becomes positively correlated with the P/S ratio.

Despite the tremendous variation in LDL-C levels for participants following diets with different Keys scores, a significantly lower LDL-C level at Keys score $\leqslant 28$ suggests that such a diet should be recommended for the coloured population of the Cape Peninsula, especially since 70% of this population (685 of our subjects) do not follow such a diet.

The results depicted in Fig. 1 generally suggest that a group of persons on a diet with a low Keys score tends to have lower LDL-C levels than a group on a diet with higher Keys scores. Fig. 1 also illustrates varying responses within the three categories of dietary Keys scores. The comparison only reaches statistically higher TC levels when the group with a Keys score over 28 is compared with the lower Keys scores groups. The use of the Keys score in a free-living population and as a measure of their usual diet can be questioned, because the Keys score was developed in a metabolic ward situation during a 3-week period. McNamara *et al.*[28] also criticises the Keys score because oleic acid was omitted from the formula. Despite these limitations it is of interest that in this cross-sectional study LDL-C values were found to be significantly higher for subjects consuming a typical Western diet (Keys score > 28) than for those on a prudent diet (Keys score $\leqslant 28$).

It has been shown elsewhere[1-4] that this urbanised population is at high risk of developing CHD. They smoke heavily, many of them suffer from hypertension, and obesity and hypercholesterolaemia are very common. There is some indication[4] that this situation has largely developed in this population over the last 3 decades, along with marked urbanisation and socio-economic improvements. The national CHD mortality rate for coloureds has not yet reached that of the other westernised populations in South Africa,[29] but the rate for the coloured population of Cape Town is higher than that for rural coloureds. Using data of the Central Statistical Services, CHD mortality rates calculated for the period 1981 - 1985 were 176,3 and 145,5 per 100 000 for white and coloured men aged 15 - 64 years in the Cape Peninsula, respectively. Over the same period the CHD mortality rate for coloured women aged 35 years in the same area exceeded that for white women (62,6 and 52,6/100 000, respectively). For the purpose of comparison these rates were age-standardised against an international reference population.[29]

An appropriate intervention programme to modify all the CHD risk factors in this population group, with the prudent diet playing a central role, is necessary if the high CHD rate that contributes so significantly to mortality in the Asian and white populations of South Africa is to be averted in the coloured population. The present results indicate the importance of dietary modification in such an intervention programme.

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