established, adequate biochemical investigations for homocystinuria are mandatory. The recognition of myopia in patients with thromboembolism, skeletal anomalies, or central nervous system complications should alert physicians to the possibility that "myopia plus" can have its origin in homocystinuria.

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Insulin sensitivity and regular alcohol consumption: large, prospective, cross sectional population study (Bruneck study)

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Abstract

***Objectives*—To assess the relation between regular alcohol consumption and insulin sensitivity, and to estimate the importance of insulin in the association of alcohol with multiple vascular risk factors and cardiovascular disease.**

***Design*—Prospective and cross sectional study of a large randomly selected population sample.**

***Setting*—Part of the Bruneck study 1990-5 (Bolzano province, Italy).**

***Subjects*—820 healthy non-diabetic women and men aged 40-79 years.**

***Main outcome measure*—Concentrations of fasting and post-glucose insulin, cholesterol, apolipoproteins, triglycerides, Lp(a) lipoprotein, glucose, fibrinogen, and antithrombin III; blood pressure; insulin resistance estimated by the homeostasis model assessment.**

***Results*—Fasting insulin concentrations in those who did not drink alcohol and subjects reporting low (1-50 g/day), moderate (51-99 g/day), and heavy (≥100 g/day) alcohol intake were 12.4, 10.0, 8.7, and 7.1 mU/l (P<0.001). Likewise, post-glucose insulin concentrations and estimates for insulin resistance assessed by the homeostasis model assessment decreased significantly with increasing amounts of regular alcohol consumption. These trends were independent of sex, body mass index, physical activity, cigarette smoking, medication, and diet (P<0.001). Regular alcohol intake predicted multiple changes in vascular risk factors over a five year period including increased concentrations of high density lipoprotein cholesterol and apolipoprotein A I; higher blood pressure; and decreased concentration of antithrombin III. These associations were in part attributable to the decrease in insulin concentrations observed among alcohol consumers.**

***Conclusions*—Low to moderate amounts of alcohol, when taken on a regular basis, improve insulin sensitivity. Insulin is a potential intermediate component in the association between alcohol consumption and vascular risk factors (metabolic syndrome).**

Introduction

Insulin resistance and hyperinsulinaemia are prominent predictors of risk for the development of diabetes mellitus[1] and may promote atherosclerotic diseases because of the association with multiple vascular risk factors and direct atherogenic effects.[2-6] For preventive purposes precise knowledge of environmental determinants of insulin sensitivity is mandatory. Obesity and insufficient physical activity rank among these factors. With regard to alcohol consumption, a further widespread and potentially modifiable behaviour, epidemiological evidence is sparse and restricted to low

amounts of alcohol and to women.[7 8] We investigated the relation between insulin concentrations and regular alcohol consumption (in the range 0-210 g/day) in a large population sample of men and women.

Methods

SURVEY AREA AND STUDY SUBJECTS

The town of Bruneck is located in an alpine region in the north of Italy (Bolzano province). The study population was recruited as a random sample stratified for age and sex of all inhabitants aged 40 to 79 years (n = 4793) in such a way that 125 women and 125 men of each decade were invited to participate.[9-11] The baseline examination was performed from July to November 1990 and the first follow up from July to October 1995. Participation and follow up rates were high at 93.6% and 96.5%. Subjects with cerebrovascular disease, incomplete data collection, unavailable insulin measurements, and diabetes mellitus (n = 60; World Health Organisation criteria[12]) were considered ineligible, which left 820 (1990) and 771 (1995) men and women for analysis.

CLINICAL EVALUATION AND LABORATORY METHODS

Alcohol consumption was assessed by a standardised interview based on a questionnaire.[11] Subjects were instructed to indicate their customary frequency of drinking (days a week) and the average amount of alcoholic beverages ingested on a typical occasion or during a typical day (500 ml bottles of beer and 250 ml glasses of wine equivalent to 25 g alcohol, standard drinks of spirits 8-10 g alcohol each). Additionally, self administered prospective diet records including the same alcohol items were collected over an extended period of seven days in a representative subsample (n = 404; participation rate 91%[11]). In both evaluations average alcohol consumption was quantified in terms of grams a day (g/day) and classified in four categories: abstainers (0 g/day) and light (1-50 g/day), moderate (51-99 g/day), and heavy drinkers (≥100 g/day). Advantages and validation of this categorisation, including the high reproducibility (κ coefficient 0.87), have been described previously.[11]

Subjects were coded as non-smokers or light (1-10 cigarettes), moderate (11-20), or heavy (>20) smokers on the basis of usual smoking habits. Social class was defined by the education of the subject and the occupation of the person with the highest income in the household.[9] Systolic and diastolic blood pressures were assessed as means of two independent measurements, each of which was taken after at least 10 minutes of rest. The activity score consisted of the average of the scores for work (three categories) and sports or leisure activities (0, ≤2, >2 hours a week). Blood samples were collected between 7 30 and 9 30 am after 12 hours of fasting and abstinence from smoking and two hours after a standardised oral glucose challenge.[9] Concentrations of laboratory variables were determined as follows: triglycerides (interassay coefficient of variation 4.3-5.4% for different standards) and total and high density lipoprotein cholesterol were determined enzymatically (by the cholesterol oxidase-4-phenyl-2,3-dimethyl-4-amino-5-pyrazolone and the glycerol phosphate oxidase-4-phenyl-2,3-dimethyl-4-amino-5-pyrazolone (CHOD-PAP and GPO-PAP) methods, Merck; coefficient of variation 2.2-2.4%); Lp(a) lipoprotein with an enzyme linked immunosorbent assay (ELISA) (Immuno, Vienna; 3.5-6.3%), insulin according to Hales and Randle (3.2-4.8%)[13] and by a human insulin specific radioimmunoassay (Linco Research, 3.9%); apolipoproteins by a nephelometric fixed time method (apolipoprotein A I 5.7%; apolipoprotein B 2.4%); and antithrombin III with a chromogenic assay (3.9-4.9%). Concentrations of low density lipoprotein cholesterol were calculated with the Friedewald formula and corrected for Lp(a) lipoprotein cholesterol. Insulin resistance and β cell deficit were estimated by the homoeostasis model assessment (coefficients of variation 31% and 32%).[14] Normal weight, healthy subjects aged <35 who did not drink alcohol or smoke, who had normal glucose tolerance, and who were not taking any drug treatment were assumed to have 100% β cell function and an insulin resistance of 1.

STATISTICAL ANALYSIS

Multiple regression analysis was applied to assess relations of alcohol consumption with insulin and vascular risk attributes (SPSS-X version 4.0[15] and BMDP software[16]). Levels of alcohol intake were modelled as a set of categories or as a trend. Confidence intervals and P values presented were mainly derived from the latter approach as most results showed clear (linear) trends with alcohol intake. To evaluate the consistency of these associations by sex first order interaction terms were added to the models. Variables with a skewed distribution of serum concentrations were normalised by ln (natural log) transformation. Quantitive changes in vascular risk factors expected for the variation of insulin concentration across alcohol categories were estimated from linear regression equations (of the variable on insulin and covariates) and expressed as proportions of the differences actually observed.

Results

A total of 114 women (28%) and 314 men (76%) reported regular alcohol consumption. In the population as a whole, fasting and post-glucose insulin concentrations steadily decreased with increasing daily consumption, as did ratios of insulin resistance estimated by the homeostasis model assessment (table 1). When alcohol intake was modelled as a continuous variable an increase in daily alcohol consumption of

Table 1—*Mean values of surrogates for insulin resistance and haemoglobin A_{1c}, and glucose concentrations by daily alcohol consumption (n = 820)*

Variable	Mean (SD) or median (interquartile range)*	Observed (adjusted†) mean values				P value
		Abstainers	1-50 g alcohol	51-99 g alcohol	≥ 100 g alcohol	
Fasting insulin (mU/l):	10.5 (7.0-15.7)	12.4 (10.8)	10.0 (9.7)	8.7 (9.2)	7.1 (7.9)	< 0.001
Men (mU/l) (n = 413)	8.8 (5.7-13.9)	10.9 (10.2)	10.0 (8.8)	8.6 (8.6)	6.8 (7.2)	< 0.001
Women (mU/l) (n = 407)	12.7 (8.3-16.9)	13.1 (13.5)	11.7 (12.2)	11.9 (12.1)	9.9 (9.9)	< 0.001
Post-glucose insulin (mU/l):	33.3 (19.2-57.7)	35.1 (33.7)	32.3 (30.0)	28.8 (26.3)	22.7 (23.2)	< 0.001
Insulin resistance‡ (homoeostasis model assessment)	1.13 (0.72-1.72)	1.39 (1.25)	1.13 (1.10)	0.98 (1.03)	0.86 (0.96)	< 0.001
β cell activity‡ (%) (homoeostasis model assessment)	77.5 (50.6-113.6)	91.0 (76.0)	71.3 (67.9)	56.7 (64.7)	48.4 (54.6)	0.035
Haemoglobin A_{1c} (%)	5.41 (0.43)	5.43 (5.47)	5.36 (5.40)	5.48 (5.43)	5.30 (5.26)	0.008
Fasting glucose (mmol/l)	5.39 (0.54)	5.34 (5.39)	5.36 (5.39)	5.53 (5.47)	5.53 (5.51)	0.047
Two hour glucose (mmol/l) (n = 812)	5.46 (1.97)	5.53 (5.44)	5.36 (5.40)	5.52 (5.47)	5.28 (5.39)	0.772

*Median (interquartile range) given when data were skewed.
†Adjusted for sex, age, smoking, body mass index, physical activity, and social status. P values presented were derived multiple regression models with alcohol consumption modelled as linear trend. Analyses were adjusted for smoking, sex, age, physical activity, social status, and body mass index.
‡Normal weight healthy subjects aged <35 years were assumed to have insulin resistance of 1.0 and β cell activity of 100%.

Table 2—*Mean values of potential cardiovascular risk factors and selected laboratory variables by daily alcohol consumption (n = 820)*

Variable	Mean (SD) or median (interquartile range)*	Observed (adjusted†) mean values				P value
		Abstainers (n = 391)	1-50 g alcohol (n = 244)	51-99 g alcohol (n = 115)	≥100 g alcohol (n = 70)	
High density lipoprotein cholesterol (mmol/l)	1.47 (0.37)	1.45 (1.35)	1.46 (1.43)	1.48 (1.56)	1.62 (1.68)	< 0.001
Low density lipoprotein cholesterol (mmol/l)	3.35 (0.98)	3.38 (3.33)	3.34 (3.33)	3.20 (3.22)	3.42 (3.45)	0.273
Total cholesterol (mmol/l)	5.72 (1.04)	5.72 (5.66)	5.66 (5.66)	5.77 (5.80)	5.89 (5.93)	0.123
Apolipoprotein B (g/l)	1.20 (0.32)	1.18 (1.20)	1.20 (1.21)	1.26 (1.23)	1.19 (1.18)	0.163
Apolipoprotein A I (g/l)	1.64 (0.29)	1.61 (1.55)	1.62 (1.60)	1.66 (1.71)	1.79 (1.83)	< 0.001
Triglycerides (mmol/l)	1.29 (0.90-1.78)	1.26 (1.34)	1.27 (1.30)	1.49 (1.40)	1.32 (1.28)	0.067
Lp(a) lipoprotein (mmol/l) (n = 796)	0.25 (0.11-0.58)	0.26 (0.27)	0.24 (0.25)	0.27 (0.27)	0.23 (0.22)	0.120
Systolic blood pressure (mm Hg)	143.9 (20.6)	144.4 (141.5)	141.6 (141.6)	145.0 (145.3)	148.0 (150.5)	< 0.001
Diastolic blood pressure (mm Hg)	88.5 (9.7)	88.4 (86.8)	87.7 (87.4)	89.5 (89.8)	90.6 (92.1)	< 0.001
Fibrinogen (g/l)	2.60 (0.57)	2.70 (2.73)	2.49 (2.54)	2.52 (2.47)	2.47 (2.45)	< 0.001
Antithrombin III (%)	97.1 (13.8)	99.0 (97.7)	97.2 (97.1)	94.6 (95.6)	89.7 (90.1)	< 0.001
Triiodothyronine (nmol/l)	2.44 (0.48)	2.53 (2.56)	2.41 (2.42)	2.35 (2.33)	2.27 (2.24)	< 0.001
Uric acid (μmol/l)	318 (59.5)	300.0 (330.1)	323.1 (332.8)	357.3 (333.7)	346.1 (329.8)	0.006
γ-Glutamyltransferase (U/l)	20.3 (23.4)	14.9 (17.9)	17.4 (18.6)	31.8 (30.0)	40.2 (38.4)	< 0.001

*Median (interquartile range) given when data were skewed.
†Adjusted for sex, age, smoking, body mass index (means in alcohol categories 24.9, 24.7, 25.2, 24.2 kg/m^2), physical activity (4.4, 4.3, 4.3, 4.1), and social status. P values presented were derived from multiple regression models with alcohol consumption modelled as linear trend. Analyses were adjusted for smoking, sex, age, physical activity, social status, and body mass index.

Table 3—*Changes in cardiovascular risk factors and laboratory variables between 1990 and 1995 by daily alcohol consumption (n = 771)*

Variable	Mean (SD)	Observed (adjusted*) mean values				P value
		Abstainers	1-50 g alcohol	51-99 g alcohol	≥100 g alcohol	
High density lipoprotein cholesterol (mmol/l)	0.06 (0.34)	0.03 (−0.04)	0.06 (0.03)	0.05 (0.08)	0.14 (0.21)	0.003
Total cholesterol (mmol/l)	0.18 (0.85)	0.16 (0.03)	0.21 (0.15)	0.10 (0.17)	0.18 (0.30)	0.056
Apolipoprotein B (g/l)	−0.06 (0.32)	−0.04 (−0.06)	−0.08 (−0.10)	−0.02 (−0.01)	−0.05 (−0.02)	0.456
Apolipoprotein A I (g/l)	0.02 (0.27)	−0.01 (−0.06)	0.05 (0.03)	0.01 (0.03)	0.06 (0.11)	0.012
Triglycerides (%)†	7.0% (−21% - +29%)	4.5%(1.4%)	7.1%(5.3%)	14.4% (15.6%)	8.7% (12.3%)	0.240
Systolic blood pressure (mm Hg)	3.79 (18.9)	3.57 (2.11)	2.83 (2.49)	5.08 (5.74)	8.37 (9.53)	0.013
Fibrinogen (g/l)	0.29 (0.65)	0.29 (0.33)	0.28 (0.31)	0.25 (0.24)	0.47 (0.41)	0.071
Antithrombin III (%)	1.92 (12.6)	2.81 (4.15)	1.87 (2.21)	0.33 (−0.2)	0.08 (−1.0)	0.016
Haemoglobin A_{1c} (%)	0.02 (0.34)	0.04 (0.05)	0.01 (0.01)	−0.01 (−0.02)	−0.04 (−0.04)	0.471
Fasting glucose (mmol/l)	0.12 (0.65)	0.07 (0.06)	0.14 (0.14)	0.11 (0.12)	0.31 (0.31)	0.133
Two hour glucose (mmol/l) (n = 760)	0.61 (1.01)	0.59 (0.26)	0.50 (0.38)	0.71 (0.79)	0.99 (1.17)	0.218
γ-Glutamyltransferase (U/l)	15.4 (29.7)	10.0 (7.4)	14.7 (14.0)	20.2 (21.5)	37.1 (39.2)	< 0.001

*Adjusted for sex, age, smoking, body mass index, physical activity, and social status. P values presented were derived from multiple regression models with alcohol consumption modelled as linear trend. Analyses were adjusted for smoking, sex, age, physical activity, social status, and body mass index.
†Relative changes (1995/1990) (interquartile range) given when data were skewed: ln (x_{95})−ln (x_{90}) = ln (x_{95}/x_{90}).

50 g predicted changes in insulin resistance and fasting and post-glucose insulin of −10% (95% confidence interval −5% to −14%), −14% (−4% to −22%), and −10% (−3% to −15%), respectively. These findings were consistent by sex and independent of age, body mass index, smoking, social status, and physical activity.

Of the variability of serum insulin concentration in the entire population sample, 3.9% was attributable to alcohol consumption as compared with 9.4% for obesity and 4.1% for differences in physical activity. According to the high incidence of regular alcohol consumption, the attributable fraction for men amounted to 5.8% (25% of totally explained variability of insulin).

The prevalence of impaired glucose tolerance was low and consistent in the four categories of alcohol consumption (10.2, 8.8, 10.1, and 7.9%). Exclusion of these subjects did not affect changes in insulin concentration with increasing alcohol consumption (insulin 11.7 mU/l (0 g/day) to 7.1 mU/l (≥100 g/day); P<0.001). Separate analyses that excluded or recategorised former drinkers[11] yielded similar results (insulin 12.1 mU/l (0 g/day) to 7.1 mU/l (≥100 g/day); P<0.001), as did models that adjusted for dietary differences, drugs known to interfere with insulin and glucose metabolism (corticosteroids, thyroid hormones, diuretics, oral contraceptives, β blockers, etc[17]), and markers of liver function including γ-glutamyltransferase, the transaminases, prothrombin time, and albumin concentrations.

Finally, all analyses were supplemented and consistently confirmed by calculations on the basis of alcohol quantities derived from the prospective diet records (data not presented).

Regular alcohol consumption may produce considerable changes in lipoprotein metabolism and blood clotting. In our population sample concentrations of high density lipoprotein cholesterol and apolipoprotein A I linearly increased across categories of alcohol consumption, whereas antithrombin III, fibrinogen, and triiodothyronine concentrations exhibited opposite trends (table 2). The pattern of related risk factors did not differ between sexes except for a preferential enhancement of concentration of high density lipoprotein cholesterol in the men (P<0.05 for effect modification). In a second stage we prospectively assessed changes of risk factors over five years, dependent on the average amount of daily alcohol consumption in this period. Except for the association between alcohol intake and fibrinogen concentration the trends observed correspond with the cross sectional data (table 3). Varying concentrations of insulin and insulin resistance (homoeostasis model assessment) among drinkers might explain 9-35% of changes in concentrations of high density lipoprotein cholesterol, apolipoprotein A I, and antithrombin III associated with alcohol.

Discussion

REGULAR ALCOHOL CONSUMPTION AND INSULINAEMIA

In both sexes we found a strong tendency of serum insulin concentrations to decrease with increasing

chronic alcohol consumption. This trend emerged independent of obesity, physical activity, smoking habits, social status, and drug use. The few previous reports on the relation between regular alcohol consumption and insulin concentrations were restricted to low amounts of average alcohol intake in women. In the Kaiser permanent women twins study median daily alcohol consumption amounted to 4 g a day.[7] An increment of 12 g a day (highest category of drinking) was associated with 8% lower post-glucose insulin concentrations. In a study of women in Bristol, those consuming up to 30 g a day had lower fasting insulin concentrations than did non-drinkers.[8] Consistent epidemiological evidence of lower insulin concentrations among drinkers[7 8 18 19] was further substantiated by results from an experimental animal model. In obese rats prone to atherosclerosis insulin concentration and β cell hyperplasia were reduced after long term alcohol intake.[20]

Fasting and post-glucose insulin are well established surrogate measures for insulin resistance in non-diabetic subjects,[21 22] with low concentrations of insulin as observed in alcohol consumers indicating higher insulin sensitivity. Theoretically, impaired secretion or enhanced hepatic degradation of insulin could interfere with this assumption: The latter mechanism, however, may not apply to alcohol drinking given the experimental observation of an unchanged or even reduced liver uptake of insulin after acute or chronic intake of alcohol.[23 24] Likewise, inhibition of β cell secretion was observed only after administration of high doses of ethanol,[25-27] whereas low to moderate intake of alcohol tended to enhance insulin release ("priming effect") in an experimental setting.[25 28] The homoeostasis model assessment permits differentiation between insulin resistance and β cell deficit in epidemiological surveys with the limitations of high variability in measurement and uncertain estimates of β cell function at normal glucose concentrations.[14] When we used this method we observed a significant decrease in insulin resistance across all alcohol categories. Besides, β cell deficiency as indicated by the coincidence of fasting hyperglycaemia (>6.4 mmol/l) with clearly reduced β cell activity and insulin concentrations was demonstrated in 13% of heavy drinkers.

Direct experimental evaluations of insulin sensitivity in alcohol consumers have dealt mainly with immediate effects of acute alcohol loading[25 29 30] rather than consequences of long term alcohol consumption.[31]

ASSOCIATION OF ALCOHOL CONSUMPTION WITH MULTIPLE VASCULAR RISK FACTORS

Our population study confirmed previous reports in that it assessed an increase in concentrations of high density lipoprotein cholesterol and apolipoprotein A I and higher blood pressure among alcohol consumers, extended the metabolic complex associated with drinking by a decline in antithrombin III concentration, and provided first prospective confirmation of these findings. Several mechanisms have been proposed that link regular alcohol intake and changes in vascular risk attributes. Enhanced insulin sensitivity as postulated for alcohol consumers in our study may rank among these pathways because of the broad overlap of the alcohol associated metabolic complex with components of the "syndrome X."[32 33] This hypothesis could partly explain alcohol associated variations in concentrations of high density lipoprotein cholesterol, apolipoprotein A I, and antithrombin III. With regard to systolic blood pressure, however, changes across alcohol categories (+3.6 mm Hg) were contrary to those expected (-1.1 mm Hg), probably because of an alcohol induced activation of the autonomous nervous system or enhancement of renal tubular sodium reabsorption.[25]

Key messages

- Current analysis yielded evidence of an increased insulin sensitivity among subjects who reported regular low to moderate alcohol consumption
- Regular alcohol consumption predicted multiple changes of vascular risk factors over a five year period
- This alcohol associated metabolic syndrome is in part attributable to the decline in insulin concentrations

LIMITATIONS AND SELECTED METHODOLOGICAL PROBLEMS IN QUANTITIVE ALCOHOL RESEARCH

The insulin assay used in the current study cross reacts with pro-insulin and pro-insulin split products. In a subsample of the Bruneck cohort (n = 200), however, insulin concentration was additionally measured with a specific assay (cross reactivity <0.2%). Both assessments were strongly correlated ($r = 0.85$; mean difference −8.4%; −0.8 to −15.8%) and showed an analogous inverse association with regular alcohol intake ("specific" insulin 11.0 mU/l (0 g/day) to 6.6 mU/l (≥100 g/day); P<0.001). Likewise, Haffner and coworkers reported that both types of tests perform equally well for the purpose of analysing the associations between vascular risk factors and insulin.[34]

The quality of alcohol research primarily relies on the accuracy of assessing alcohol consumption. In our survey, the non-differential response error which is believed to be the major source of incorrect ascertainment of alcohol consumption[35] was dealt with by the application of diet records in a representative subsample. Prospective assessment of daily food records over an extended period is the best possible method to minimise error in recall and overcome fluctuation in drinking behaviour and inadequate self estimation of alcohol intake.[11 36] Analysis of these data further substantiated results derived from point estimates of alcohol intake. The second important type of error, the deliberate denial of alcohol use or selective non-responding by heavy drinkers, may also be regarded as low because of a participation rate over 93% and the fairly high boundaries of socially accepted drinking in the survey area.

Alcohol is an important constituent of the European and American diet. Our survey identified regular alcohol intake as a further determinant of insulin sensitivity in a general healthy population, even though its explanatory contribution to insulin variability was only half that of obesity. Light to moderate drinking emerged as safe regarding its effects on insulin. Conclusions on severe alcohol consumption require particular caution given the associated social and health risks, potential impairment of β cell function, and occurrence of diabetes due to advanced pancreatitis induced by alcohol.

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Fall in total cholesterol concentration over five years in association with changes in fatty acid composition of cooking oil in Mauritius: cross sectional survey

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Abstract

***Objective*—To determine the extent to which reducing the saturated fatty acid composition of a ubiquitously used cooking oil influenced changes in cholesterol concentration in the population during a five year intervention programme in Mauritius.**

***Design*—Cross sectional surveys in 1987 and 1992 determined mean total cholesterol concentrations in the population. A random sample of respondents in the 1992 survey completed a nutrition questionnaire that included questions on diet in the previous 24 hours.**

***Setting*—Mauritius.**

***Intervention*—In 1987 the government of Mauritius changed the composition of the commonly used cooking oil from being mostly palm oil (high in saturated fatty acids) to being wholly soya bean oil (high in unsaturated fatty acids).**

***Subjects*—5080 and 5162 subjects in 1987 and 1992 cross sectional surveys. 2059 subjects aged 30-64 years were randomly selected from the respondents of the 1992 survey to take part in the nutrition survey**

***Main outcome measures*—Fatty acid composition of phospholipids in pooled serum samples from men and women from the two surveys; measured and predicted change in serum cholesterol concentration.**

***Results*—From 1987 to 1992 total cholesterol concentrations fell significantly by 0.79 mmol/l (P<0.001) in men and 0.82 mmol/l (P<0.001) in women. The estimated intake of saturated fatty acids decreased by 3.5% of energy intake in men and by 3.6% in women, and the intake of polyunsaturated fatty acids increased by 5.5% and 5.6% of energy intake, respectively. These changes were reflected in changes in the fatty acid composition of serum phospholipids, and according to Keys' formula these changes explained much of the decrease in serum cholesterol concentrations (predicted decrease of 0.38 mmol/l in men and by 0.40 mmol/l in women).**

***Conclusion*—Dietary changes that entailed a reduction in the saturated fat content of a ubiquitous cooking oil explained most of the observed decrease in serum cholesterol concentration over five years in the population of Mauritius.**

Introduction

In 1987 the government of Mauritius launched a non-communicable diseases intervention programme to prevent and control diseases caused by unhealthy lifestyles because such diseases were occurring at increasing rates in Mauritians.[1,2] A national survey in 1987 had confirmed high levels of cardiovascular risk factors and a high prevalence of diabetes in the population.[3-5] As well as promoting healthy lifestyles,[2] the government also limited the content of palm oil in the cooking oil used almost universally in Mauritius ("ration oil"). Palm oil is high in saturated fatty acids and was then the main component of ration oil. The