Selected risk factors for coronary heart disease in male scholars from the major South African population groups

H. C. SEFTEL, M. S. ASVAT, B. I. JOFFE, F. J. RAAL, V. R. PANZ, W. J. H. VERMAAK, M. E. LOOCK, M. C. RAJPUT, M. A. K. OMAR, M. S. JEENAH, K. STEYN, P. J. BECKER for the Seven Schools' Study Group

Abstract **A number of risk factors for coronary heart disease (CHD) in 7 groups of South African male scholars aged between 15 and 20 years were surveyed. Selection of the groups was based on socio-economic status and comprised urban and rural blacks, Indians of higher and lower socio-economic status, coloureds of higher and lower socio-economic status, and middle-class whites. Both Indian groups, both coloured groups and the whites had a much greater prevalence and severity of CHD risk factors than the two black groups. This held for total cholesterol, low-density lipoprotein cholesterol (LDLC), high-density lipoprotein cholesterol (HDLC), the HDLC/LDLC ratio, apolipoprotein B, apolipoprotein A-I, insulin, fibrinogen and mass. One exception was lipoprotein a, levels of which were higher in both black groups. In general the CHD risk factor profile was worse in the higher socio-economic groups, and it also tended to be worse in urban than in rural blacks. These findings stress the need to reduce CHD risk factors in our developed populations and to prevent their emergence in our developing peoples.**

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Department of Medicine, University of the Witwatersrand, Johannesburg
H. C. SEFTEL, DIP. MED.
M. S. ASVAT, F.C.P. (S.A.)
B. I. JOFFE, D.SC.
F. J. RAAL, F.C.P. (S.A.)
V. R. PANZ, M.SC.
Department of Chemical Pathology, University of Pretoria
W. J. H. VERMAAK, M.MED. (PATH.)
Department of Medicine, University of Pretoria and Kalafong Hospital, Pretoria
M. E. LOOCK, M.PRAX. MED.
Department of Medicine, University of Natal, Durban
M. C. RAJPUT, F.C.P. (S.A.)
M. A. K. OMAR, M.D.
MRC Research Unit for Cell Biology of Atherosclerosis, Department of Biochemistry, University of Cape Town
M. S. JEENAH, PH.D.
Centre for Epidemiological Research in Southern Africa, Medical Research Council, Parowvallei, CP
K. STEYN, M.D.
Institute for Biostatistics of the Medical Research Council (Transvaal Branch), Pretoria
P. J. BECKER, PH.D.

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Coronary heart disease (CHD) is the commonest cause of death in South African whites and Indians, and an important cause of mortality in urban coloureds.[1] It is very rare among rural blacks, although the prevalence may recently have increased among urban blacks. The available evidence suggests that the ethnic variation in CHD incidence relates to

differences in exposure to both genetic and environmental risk factors.[2-4] For example, hypercholesterolaemia, both familial and diet-related, is much commoner in white communities, such as Afrikaners and Jews, than among blacks.[5]

We know little about the CHD risk factor status in young South Africans. This information is necessary because the impact of a risk factor on CHD commonly starts in youth and atherosclerosis can develop for several decades before overt disease becomes manifest.

In the present study we surveyed the prevalence of several CHD risk factors in 7 groups of male scholars drawn from black, white, coloured and Indian schools. The selection of the groups was based on socio-economic status in order to maximise both interethnic and intra-ethnic differences in the risk factors surveyed.

Subjects and methods

A total of 859 subjects was surveyed. They were mainly Standard 9 scholars aged between 15 and 20 years. Only males were recruited to avoid the confounding effects of the menstrual cycle or of hormonal contraceptives on the variables surveyed. The following 7 groups were selected:

1. One hundred and fifty-six rural black males (BR), mean age (± SD) 19,4 ± 2,2 years, raised in the small villages and farms of the KwaNdebele homeland. Their dwellings were huts or simple 1- or 2-roomed houses without electricity or piped water. The average number of residents per dwelling was 15. Most of the parents were poorly educated and unemployment rates were high. Fathers commonly sought work in towns, where they were employed mainly as manual labourers. Otherwise both men and women did agricultural work on their plots or on white-owned farms. These scholars attended the Kwamanola High School in Allemansdrift and the Mabutheni, Babutheni and Mbonong High Schools in Mbibane. All the schools were located within the homeland and were far from main centres.

2. One hundred and two urban black males (BU), mean age 18,6 ± 1,9 years, raised in the township of Atteridgeville adjacent to Pretoria. They lived in sub-economic 1- or 2-roomed houses, the average number of residents per dwelling being about 10. Most adults were poorly educated or semiliterate. Unemployment was common and those who were employed were unskilled workers in Pretoria. The scholars attended the D. H. Peta, Holy Trinity and Hofmeyer High Schools in Atteridgeville.

3. A lower socio-economic group of 158 coloured males (CL), mean age 16,9 ± 1,0 years, mostly born and reared in Eldorado Park, a suburb of Johannesburg inhabited almost exclusively by coloured persons. Most lived in modest 2-bedroomed houses, and the average number of occupants per dwelling was about 5. Most adults had primary or secondary school education. Their occupations varied but most were in lower category jobs, such as artisans, shop assistants and unskilled workers. The scholars were recruited from the Eldorado Park, Willow Crescent and Silver Oaks Secondary Schools in Eldorado Park and the Kliptown Secondary School in the adjacent suburb of Kliptown.

4. A higher socio-economic group of 115 coloured males (CH), mean age 17,7 ± 0,8 years. Most had been born in the Cape Peninsula and had grown up in the suburbs of Bellville South, Grassy Park, Zeekoevlei and Maitland. Most lived in freestanding or semi-detached formal housing with gardens and sometimes garages. Overcrowding is not a feature of these areas, the average number of persons per dwelling being about 5. The breadwinners had primary or often secondary schooling and occasionally tertiary education. Employment was in middle to higher categories and included clerical, sales, service and trades persons, production workers, teachers and foremen. The scholars attended the Kensington, Kasselsvlei and Zeekoevlei Senior Secondary Schools.

5. A lower socio-economic group of 122 Indian males (IL), mean age 17,1 ± 0,8 years. They had been born and had grown up in the Indian suburbs of Chatsworth and Phoenix, situated about 25 km south and 20 km north of central Durban respectively. They lived in 2-bedroomed sub-economic houses, the average number of residents per dwelling being 6. Most of the parents had primary or secondary school education; their occupations varied but most would be classified as semi-skilled. These scholars attended the Newhaven and Witteklip Secondary Schools in Chatsworth and the Phoenix Secondary School.

6. A higher socio-economic group of 104 Indian males (IH), mean age 17,0 ± 0,7 years, born and raised in the Indian suburb of Lenasia situated 25 km south-west of Johannesburg. For the most part they lived in modern, well-built, attractive single- or double-storeyed homes with gardens and garages. Most belonged to extended families, but there were also many nuclear families. In either event there was little overcrowding because the homes were large, having up to 6 bedrooms. Most breadwinners had primary or secondary school education and many had upper-category occupations in business, industry, the professions and technical crafts. The scholars were recruited from the Topaz, Nirvana, Azara, Lenasia and Trinity High Schools in Lenasia.

7. One hundred and two middle-class white males (W), mean age 16,8 ± 0,6 years. The majority had been born and had grown up in the municipality of Randburg. They lived in high-quality homes with an average of 4 residents per dwelling. Their parents were well educated with secondary and tertiary qualifications and were employed in higher-category business, finance, industrial or professional occupations. All the scholars attended the Randpark Ridge High School in Randburg.

Survey procedures and observations

Permission to undertake the investigation was obtained from the school principals and the relevant educational authorities. Informed consent was obtained from the parents or guardians. The ethics committees of the various participating institutions approved the study. The scholars included in this study were sampled at random from those children whose parents or guardians gave informed consent for their children to enter the study. The sample size of at least 100 from each group was decided upon after calculations based on the magnitude of deviances that can be regarded as clinically relevant when the power of the test is 0,90 and the level of significance is 0,05.

All the boys were investigated in a comfortably warm room at the particular school by a team of 3 - 4 doctors and 4 - 5 nurses. Details of age, religion, language, ethnic group and smoking habits of the boys were recorded.

Mass was measured on a calibrated electronic digital scale and height was determined using a height meter with a cross-bar. The same instruments were used at all the schools. Body mass index (BMI) was calculated from the formula mass (kg)/height $(m)^2$. Subjects were seated while 35 ml blood was collected into appropriate Vacutainers after an overnight fast. Resuscitation facilities were available to deal with syncopal episodes, which occurred in about 5% of the subjects. Serum was separated within 4 hours of collection and stored at –70°C. The survey started in August 1990 and was completed in February 1991.

The Department of Chemical Pathology, University of Pretoria, measured the serum lipid and lipoprotein values and the Carbohydrate and Lipid Metabolism Research Group, University of the Witwatersrand, the serum insulin levels. Plasma fibrinogen was measured at the Haematology Department of the South African Institute for Medical Research.

The lipids and lipoproteins analysed were total cholesterol (TC), high-density lipoprotein cholesterol (HDLC), triglycerides (TG), low-density lipoprotein cholesterol (LDLC), lipoprotein a (Lp(a)), apolipoprotein A-I (apo A-I) and apolipoprotein B (apo B). TC was measured by automated enzymatic procedures on a Technicon SMAC system (Technicon Instruments, Tarrytown, NY). To determine HDLC, apo B-containing lipoproteins were precipitated from serum using heparin and Mg^{++} (Merck reagent kit 15007); the supernatant was used to determine HDLC by means of the CHOD-Iodide method (Merck kit 14350). Triglyceride concentrations were determined by the enzymatic method of Wahlefeld using reagents from Boehringer-Mannheim, Germany. For the determination of LDLC, LDL was precipitated from serum by heparin at its isoelectric point (Merck reagent kit 14992). After centrifugation, the cholesterol content of the supernatant was determined and this equalled the sum of HDLC and very-low-density lipoprotein cholesterol (VLDLC). LDLC was then calculated as follows: LDLC = TC - (HDLC + VLDLC). The serum Lp(a) concentration was determined by immunoradiometric assay (Pharmacia Diagnostics AB, Uppsala, Sweden). This Lp(a) immunoassay system is a solid-phase two-site immunoradiometric assay using two monoclonal antibodies in excess, directed towards the epitopes of apo(a) and apo B. Apo B and apo A-I were determined by immunonephelometric assays using the Behring Laser-Nephelometer and methods (Behring reagent kits OSAN14/15 and QUED for apo B and apo A-I respectively). Serum insulin concentrations were measured by radio-immunoassay using kits supplied by Pharmacia. Fibrinogen levels were determined by an automated coagulation system (Instrumentation Laboratory, Italy).

The measurement of the biochemical variables included commercial quality control (QC) sera and the results of the unknown samples were accepted only if the QC values fell within 2 SD of the mean provided by the manufacturer. For example, the coefficient of variation for TC determined on the QC material during the study period was 2,0%. Results from an external QC programme confirmed that there had been no biases in TC determinations and that the analytical process had been properly controlled.

Statistical analysis

The 7 groups were compared with respect to TC, HDLC, LDLC, apo A-I, apo B, Lp(a), fibrinogen, insulin, TG, height, mass and BMI in a one-way analysis of variance (ANOVA). When the groups were found to be significantly different with the *F*-test of ANOVA, specific differences among the groups were determined by employing the appropriate *t*-test at the Bonferroni adjusted level of significance for pairwise comparisons.[6]

Presentation of the data

The distributional properties of the data are illustrated in a series of figures (Fig. 1). At the top of each figure the mean and SD for the relevant variable are given. The box and whisker plots show the median (but not the mean) and the 25th and 75th percentiles as well as the minimum and maximum values. Below these plots, line illustrations are given of the statistically significant differences ($P < 0,05$ - $< 0,001$) between groups identified by the pairwise comparisons. In these line illustrations the 7 groups were arranged in *ascending* order according to the mean values of the relevant variable. Beneath them are two or more lines which must be viewed sequentially and in relation to each other. Groups that do not differ significantly are underlined by one or more lines. In contrast, those groups that are not linked to each other by underlining differ significantly from each other and from the rest. For example, in Fig. 1a for TC rural and urban blacks differ significantly from each other because they are not linked by a common line. They also differ significantly from the other 5 groups and therefore they are also not linked to these by a common line. The only other significant difference is between the CL and IH groups, as they do not share a common line. The other groups share underlining as there are no significant differences between them. In Fig. 1b for LDLC urban and rural blacks differ significantly from the other 5 groups because they are not linked to these by the first line. However, the 2 black groups do not differ from each other because they share the second line.

In order to obtain a measure of the CHD risk factor status of the scholars in the different groups, cut-off points were defined for a number of variables. For TC, LDLC, HDLC and TG the age-dependent cut-off points of the Southern African Heart Foundation's action limits were used:[7] TC — between 3,95 mmol/l and < 5,2 mmol/l was considered to constitute a moderately increased risk for CHD and > 5,2 to constitute a high risk; LDLC — between 2,4 and < 3,65 mmol/l represented moderate risk and > 3,65 high risk; HDLC — < 1 mmol/l represented an increased risk for CHD without distinguishing between moderate and high risk; and TG — > 2,1 mmol/l also represented increased risk. An apo B level between 0,76 g/l and < 1,1 g/l constituted a moderate CHD risk and > 1,1 g/l a high risk,[8] and an Lp(a) level > 300 mg/l represented increased risk.[9] Fibrinogen values > 4 g/l[10] and insulin levels > 25 U/l[11] constituted increased risk. In each group we calculated the percentages of scholars who had levels of these variables which were considered to place them at increased risk for CHD.

Results

The *F*-test has a *P*-value of < 0,0001 for all the variables examined except BMI, for which it is < 0,003.

A comparison of TC levels between the groups is shown in Fig. 1a. The urban and rural black groups have the lowest levels with the others having similar high values. The urban black group has significantly higher TC levels than the rural black group. The LDLC values shown in Fig. 1b reflect the TC values. However, unlike the TC values, there is no significant difference in LDLC levels between urban and rural blacks.

The higher TC levels in urban than in rural blacks are due to their higher HDLC levels (Fig. 1c). The urban blacks and the lower socio-economic group Indians have the highest HDLC values. In contrast, the rural blacks and the whites have the lowest values. Except for the urban and rural blacks all the other groups follow the trend of high LDLC coupled with low HDLC, and hence have relatively low HDLC/LDLC ratios (Fig. 1d). The black groups have higher HDLC/LDLC ratios, either because their HDLC levels are high or because their LDLC values are low.

The apo B (Fig. 1e) and apo A-I (Fig. 1f) values are highly correlated with LDLC and HDLC values, respectively. The black rural group has the lowest apo A-I levels. Although LDLC levels are similar in the black rural and urban groups, the urban group has significantly higher apo B levels.

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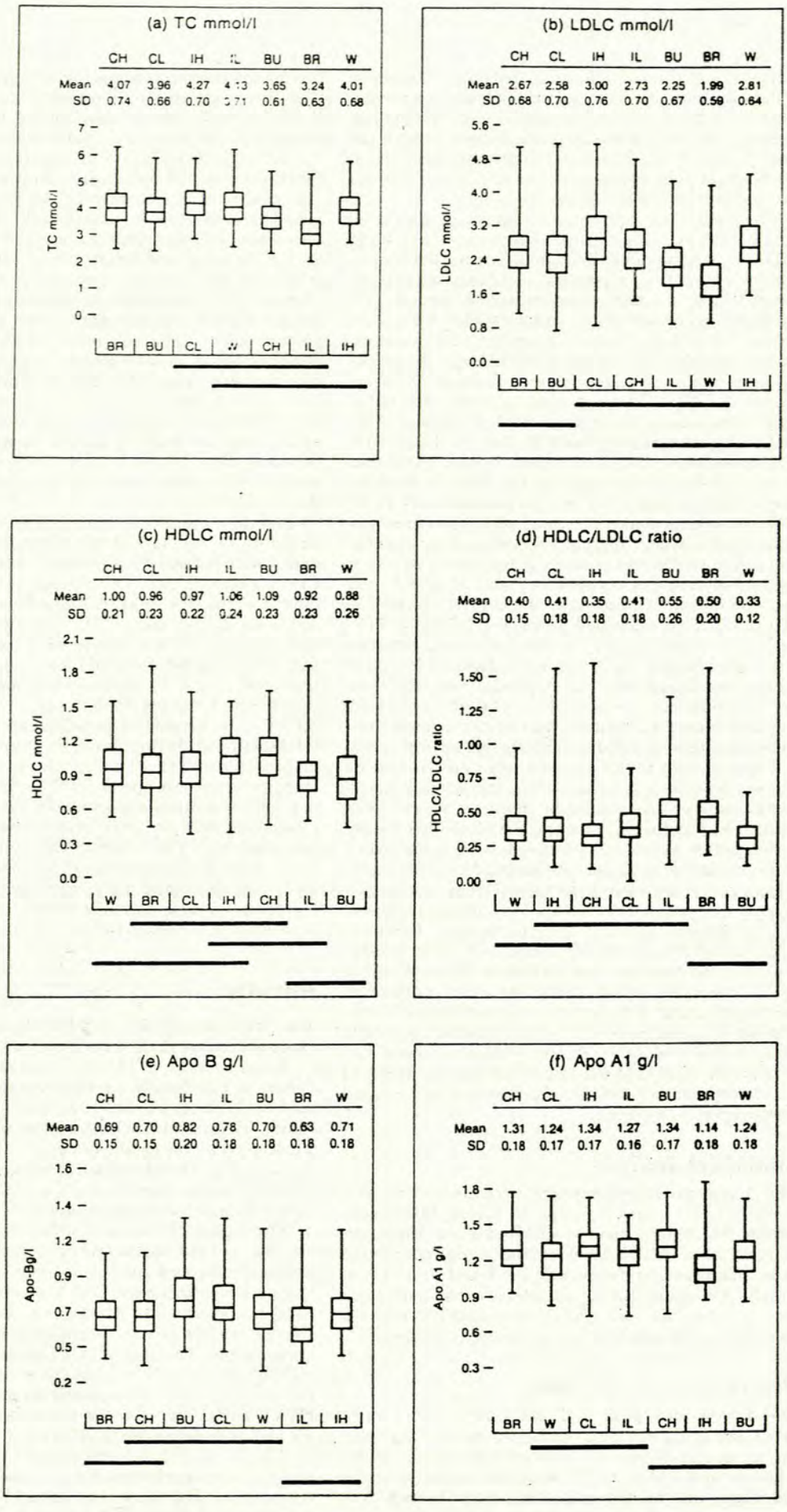

FIG. 1.
Mean, SD, distribution and statistical analysis of TC (a), LDLC (b), HDLC (c), HDLC/LDLC (d), apo B (e) and apo A-I (f) in 7 groups of male scholars. See text for explanation.

The TG values (Fig. 2a) follow a trend in which the lower socio-economic groups have lower TG values than the higher socio-economic ones in coloureds and Indians, with the difference between the two Indian groups being significant. The white group has average TG values between the lower and higher socio-economic groups. The two black groups have the highest levels. Significant positive associations ($P < 0,05$) between TG and insulin levels were found in all except the black urban and white groups.

With regard to Lp (a) (Fig. 2b), both black groups have a gaussian distribution and a high mean with the other extreme being the whites with a distribution skewed to the lower values. The Indians and coloureds have distributions that lie between the black and white groups.

Fibrinogen levels (Fig. 2c) appear to segregate according to socio-economic status, the higher socio-economic group whites, coloureds and Indians having significantly higher values than the lower socio-economic group coloureds, Indians and rural blacks. The one anomaly lies in the urban black group who, although of lower socio-economic status, have fibrinogen levels that are not significantly different from those of the higher socio-economic groups.

A comparison of insulin levels (Fig. 2d) reveals that the urban black and both Indian groups have the highest levels. The two coloured and the black rural groups have the lowest values and the whites are in between. In both Indian and both coloured groups a significant positive correlation ($P < 0,05$) exists between insulin values and levels of TC, LDLC, TG and BMI.

Fig. 2e shows that the white group had the highest mass. They were also the tallest (data not shown). The rural blacks and the lower socio-economic group Indians had the lowest mass. The differences in BMI between the 7 groups (Fig. 2f) were not so marked, although whites had significantly higher BMIs than rural blacks and the higher socio-economic group Indians and coloureds. In most groups BMI correlated positively with levels of TC, LDLC, apo B, TG and insulin and negatively with HDLC and apo A-I ($P < 0,05$).

With regard to smoking (data not shown), scholars of lower socio-economic status smoked more than those of higher socio-economic status (31,7% and 29,6% of lower socio-economic group coloureds and Indians as opposed to 21,6% and 21,2% of higher socio-economic group coloureds and Indians, respectively). Many more rural blacks smoked (39,1%) than their urban counterparts (13,7%). Only 6,9% of white participants smoked.

CHD risk factor status of groups

Table I shows the percentages of scholars in each group with levels of the variables selected which were considered to place them at increased risk for CHD. Of the subjects in both Indian groups, both coloured groups and the white group, 42 - 53% had TC levels placing them at moderate risk while 4 - 13% were at high risk. For LDLC levels the moderate- and high-risk prevalences in these 5 groups were 54 - 67% and 5 - 19% respectively, and for apo B values they were 29 - 45% and 1 - 14% respectively. The majority of the scholars in all except the lower socio-economic Indian group had low HDLC levels and about one-third of the subjects in all 5 groups had raised Lp(a) values. About one-quarter of the higher socio-economic group Indians, coloureds and whites had raised fibrinogen levels.

The CHD risk factor status of the two black groups was very different: only 31% and 9% of urban and rural blacks respectively had moderately raised TC levels and almost none had high-risk values. The situation was similar with regard to LDLC and apo B values. Only about one-third of the urban blacks had low HDLC levels, but about two-thirds of their rural fellows had low values. About one-half of both black groups had raised LP(a) values. Few rural blacks had raised fibrinogen levels.

Very few subjects in any of the 7 groups had high triglyceride or insulin values.

TABLE I.
Scholars in the 7 groups with increased risk for CHD (%)

	IH	IL	W	CH	CL	BU	BR
TC							
3,95 - < 5,2 mmol/l	53	48	51	42	43	31	9
> 5,2 mmol/l	13	8	4	10	4	1	2
LDLC							
2,4 - < 3,65 mmol/l	59	58	67	54	55	35	16
> 3,65 mmol/l	19	11	10	9	5	3	2
Apo B							
0,76 - > 1,1 g/l	45	38	23	27	29	37	20
> 1,1 g/l	14	7	2	2	1	1	3
HDLC < 1 mmol/l	56	37	67	54	62	38	70
TG > 2,1 mmol/l	1	0	0	0	1	1	0
Lp(a) > 300 mg/l	31	36	26	39	33	56	48
Fibrinogen > 4 g/l	25	12	29	22	3	16	9
Insulin > 25 IU/l	2	3	0	2	1	6	0

Discussion

This is the first survey of a variety of CHD risk factors in a large number of young South African males who were reasonably representative of the major population groups both ethnically and socio-economically. Great pains were taken to ensure that the data were valid. However, it is probable that some of the black scholars, both urban and rural, were not fasting. This may explain the high triglyceride values in both black groups and the high insulin level of the urban blacks. We are also unsure of the reported smoking data, which were based on replies to a questionnaire administered by several different observers. The cut-off points used for assessing CHD risk were to a certain extent arbitrary, being based on surveys of a number of different populations both inside and outside South Africa. For the rest we believe the findings are reliable and constitute reference standards for young males in the population groups surveyed. Also the age differences between the groups were small and unlikely to affect the validity of intergroup comparisons.

Our findings were largely what might have been expected. A recent review of CHD risk factor patterns in South Africans showed that the population groups with the higher CHD mortality were also the groups with the worst CHD risk factor profile.[12] Other investigations of young people in high CHD populations, such as the Bogalusa Heart Study in USA children, also found that CHD risk factors are commonly present in early life and are linked with the early development of atherosclerosis.[13] Our study provided considerable evidence that scholars from the South African populations at high risk for CHD also have a high prevalence and severity of CHD risk factors. This held for TC, LDLC, HDLC, the HDLC/LDLC ratio, apo B, apo A-I, insulin and fibrinogen levels, all of which were generally much more unfavourable in Indians, whites and coloureds than blacks. Upper socio-economic group Indians tended to have the worst risk factor status. The fact that a substantial proportion of South African Indians are relatively affluent may help explain why this population has the highest CHD mortality in South Africa, exceeding that of whites,[1] while our finding of a considerable CHD risk factor burden among young coloured males is in keeping with the results of a risk factor survey in adult

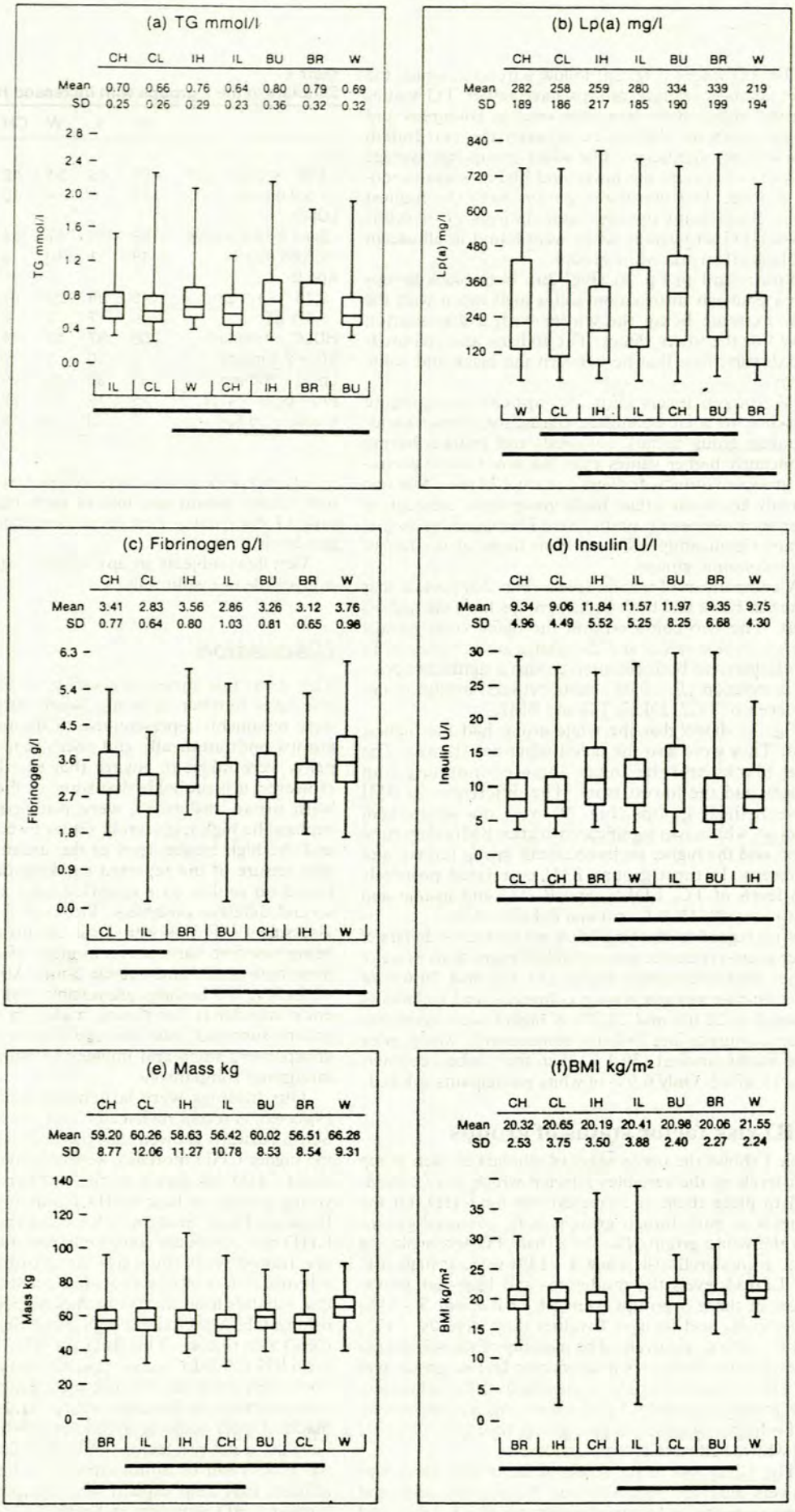

FIG. 2.
Mean, SD, distribution and statistical analysis of TG (a), Lp(a) (b), fibrinogen (c), insulin (d), mass (e) and BMI (f) in 7 groups of male scholars.

coloureds living in the Cape peninsula[3] and recent evidence that the incidence of CHD in these people now approximates that in whites (D. E. Bourne — personal communication).

Blacks, especially rural blacks, had a favourable risk profile except for their high Lp(a) levels, which have been reported in other black populations and may be genetically determined.[14] The HDLC levels of rural blacks were low but so were their LDLC values, giving them a favourable HDLC/LDLC ratio. Urban blacks had the most favourable ratio, but this was associated with high HDLC levels. The low CHD risk of the black groups could largely be due to their high level of physical activity and their diet. They were the only groups for whom dietary data were available (M. E. Loock — unpublished observations). As expected, their diet was high in carbohydrate and low in animal fat and protein. The rural diet was also low in total energy and rich in fibre, thus closely approximating the prudent diet recommended for CHD prevention.

Correlations between variables within groups were also in keeping with what has been observed generally.

Within the Indian and coloured groups, scholars of higher socio-economic status tended to have worse CHD risk profiles than their fellows of lower status. These socio-economic differentials contrast with the situation in a number of developed countries, where prevalences of CHD risk factors and CHD are distinctly lower in the upper socio-economic strata.[15] This situation emerged decades ago in adults, and more recently has also been found to hold for CHD risk in children. For example, in the USA white children of higher socio-economic status had a more favourable CHD risk factor profile than those of lower status.[16] The reasons for the worse CHD risk factor profile of upper-class Indian and coloured boys need further study, but in practical terms inadequate health education and unhealthy lifestyle are probably very important factors. Failure to correct these could mean that the incidence of CHD in Indians and coloureds will remain high among their upper socio-economic strata and even increase among their lower classes as they become more affluent.

In conclusion, this study reveals a disturbing situation. The prevalence and severity of coronary risk factors are generally high in young Indians, whites and coloureds and are becoming appreciable in urban blacks. They are greatest in the upper socio-economic strata. Urgent action is needed both to reduce these risk factors in our developed populations and to prevent their emergence in our developing peoples.

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REFERENCES

1. Wyndham CH. Trends with time of cardiovascular mortality rates in the populations of the RSA for the period 1968-1977. *S Afr Med J* 1982; **61:** 987-993.
2. Rossouw JE, Du Plessis JP, Benadé ASJ, *et al.* Coronary risk factor screening in three rural communities: the CORIS baseline study. *S Afr Med J* 1983; **64:** 430-436.
3. Steyn K, Jooste PL, Langenhoven ML, *et al.* Coronary risk factors in the coloured population of the Cape Peninsula. *S Afr Med J* 1985; **67:** 619-625.
4. Seedat YK, Mayet FGH, Kahn S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; **78:** 447-454.
5. Seftel HC, Baker SG, Jenkins T, Mendelsohn D. Prevalence of familial hypercholesterolemia in Johannesburg Jews. *Am J Med Genet* 1989; **34:** 545-547.
6. Miller RG jun, ed. *Simultaneous Statistical Inference.* 2nd ed. New York: Springer-Verlag, 1988: 67-70.
7. Rossouw JE, Steyn K, Berger GMB, *et al.* Action limits for serum total cholesterol: a statement for the medical profession by an *ad hoc* committee of the Heart Foundation of Southern Africa. *S Afr Med J* 1988; **73:** 693-700.
8. Vermaak, WJH, Kotze JP, Van der Merwe AM, *et al.* Epidemiological reference ranges for low-density lipoprotein cholesterol and apolipoprotein B for identification of increased risk of ischaemic heart disease. *S Afr Med J* 1991; **79:** 367-371.
9. Dahlen GH, Guyton JR, Attar M, Farmer JA, Judith JA. Association levels of lipoprotein Lp(a), plasma lipids and other lipoproteins with coronary artery disease documented by angiography. *Circulation* 1986; **74:** 758-765.
10. Ernst E. Plasma fibrinogen — an independent cardiovascular risk factor. *J Intern Med* 1990; **227:** 365-372.
11. Zavaroni I, Bonora E, Pagliara M, *et al.* Risk factors for coronary artery disease in healthy persons with hyperinsulinemia and normal glucose tolerance. *N Engl J Med* 1989; **320:** 702-706.
12. Steyn K, Fourie J, Bradshaw D. The impact of chronic diseases of lifestyle and their major risk factors on mortality in South Africa. *S Afr Med J* 1992; **82:** 227-231.
13. Berenson GS, Srinivasan SR, Webber LS, *et al. Cardiovascular Risk in Early Life: The Bogalusa Heart Study* (Current Concepts). Kalamazoo, Mich.: Upjohn, 1991.
14. Utermann G. The mysteries of lipoprotein(a). *Science* 1989; **246:** 904-910.
15. Rose G, Marmot MG. Social class and coronary heart disease. *Br Heart J* 1981; **45:** 13-19.
16. Walter HJ, Hofman A. Socioeconomic status, ethnic origin and risk factors for coronary heart disease in children. *Am Heart J* 1987; **113:** 812-818.