

Risk factors for coronary heart disease in the Indians of Durban

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Summary

Coronary heart disease (CHD) is a major problem in migrant Indians throughout the world. In South Africa it has reached 'epidemic' proportions. A field survey was conducted among Indians in the metropolitan area of Durban to determine the prevalence and known risk factors for CHD. In a study of 778 subjects aged 15 - 69 years (408 men), 15,3% (sex and age adjusted 13,4%) had a history of CHD. The important risk factors in men were hypercholesterolaemia, hypertriglyceridaemia, diabetes, and smoking, and in women diabetes, hypercholesterolaemia, and hypertriglyceridaemia. The minor risk factors were hyperuricaemia, sedentary occupation, obesity in women and a positive family history of CHD. A study of the major risk factors leading to CHD showed that 52% (sex and age adjusted 45,5%) had at least one major risk factor at the higher (level A) and 68% (sex and age adjusted 61,9%) at the lower (level B) risk levels. Diabetes mellitus was strongly associated with a positive history of CHD. In 47,6% (sex and age adjusted 48,2%) of the total group resting ECG abnormalities were found that could be coded. Because of the severe nature of CHD in the migrant Indian, an immediate and intensive programme of primary prevention of CHD risk factors should be instituted.

S Afr Med J 1990; 78: 447-454.

In South Africa coronary heart disease (CHD) in Indians and whites is a major problem. Cardiovascular mortality rates (MRs) issued by Central Statistical Services show that, because of the high MRs for CHD and hypertensive disease in older Indians, approximately 50% of 'deaths from all causes' were due to diseases of the circulatory system in Indians over the age of 45 years. The MR for diabetes is extremely high in both male and female Indians.¹ In an MR analysis Wyndham² concluded that: 'Cardiovascular diseases are as much an "epidemic" among Asians as they are among white South Africans and judging by the high MRs for all three cardiovascular diseases, IHD, CVD and hypertensive disease, the "epidemic" is of more serious proportions in Asians than it is in whites.' Walker³ felt that from the age of 50 years onwards the South African Indian population, which enjoys much better economic circumstances than the indigent rural populations in India, had very little life-expectancy advantage; the main cause of death had changed from infectious to degenerative diseases. South African Indians were worse off in terms of life expect-

tancy than Indians in India beyond middle age. The high incidence of CHD in the metropolitan hospitals of Durban has been recorded.⁴⁻⁷ In a review of 31 000 admissions over a 10-year period to the medical wards of the R. K. Khan Hospital, a hospital for Indian patients, it was found that CHD accounted for 11% of all medical admissions and 24% of all deaths. A disturbing feature was that 22% of the patients admitted to the coronary care unit of the hospital were under the age of 45 years.⁵ In view of the documentation of this high incidence of CHD, a field survey was conducted among Indians to determine the prevalence of known risk factors for CHD in the community. The baseline findings are reported here.

Subjects and methods

Sample selection

A coronary risk-factor survey was carried out in the Indian population living in the metropolitan area of Durban, Natal. The majority of South African Indians live in Natal and most of them in Durban. The total Indian population of South Africa, according to the 1980 population census figures, is 821 320, forming 3,3% of the total population of the country, while the 665 340 Indians resident in Natal make up 28,9% of the total population of the province.⁸

Durban, like the rest of South Africa, is divided into zones set aside specifically for different ethnic groups. The Indian group areas are scattered throughout metropolitan Durban. A representative random sample of the population, residing in these various geographical areas set aside for Indians, was selected for the survey. The sample size was determined so as to make it most practical. The 1980 population census figures were used to ascertain the total Indian population of metropolitan Durban (499 520) and that for each residential area. The latter information, together with the number of dwellings in each area, were obtained from the various municipalities included in metropolitan Durban. Using the number of dwellings and the population in each group area, a fixed percentage population of 0,24% per area was selected for the study.

The sampling was by random selection. Eligible respondents had to be within the age range of 15 - 69 years and permanent residents within the geographical area of metropolitan Durban. The exclusion criteria were: pregnancy and lactation, being bed-ridden, mental retardation, carcinoma, and antituberculosis therapy.

Household addresses were randomly selected from the latest ratepayers' and electricity consumers' records. At each address one member of the family was selected randomly for participation in the survey. The study started in May 1984 and was completed over a 2-year period.

Methods

Each household was visited on at least three occasions. In the first visit a letter of introduction and instruction was presented to the household. The field-worker stressed the purpose of the survey and urged people to participate. During this visit, the respondent was chosen and a suitable appoint-

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Accepted 2 May 1990.

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ment time was made. During the second visit a risk-factor questionnaire was completed by interview and a physical examination carried out. Only 2 interviewers, who were intensively trained and standardised in administering the questionnaire, were employed. The questionnaire consisted of sections on: (i) biological, ethnic and socio-economic data; (ii) the London School of Hygiene questionnaire for chest pain (Rose Questionnaire) was used to elicit a present or previous history of angina pectoris or pain of possible myocardial infarction;⁹ (iii) medical history of hypertension, diabetes, CHD, gout and stroke was recorded — respondents were asked to produce all medication for inspection; (iv) history of hypertension, diabetes, CHD and stroke in parents, siblings and grandparents was coded; (v) smoking habits of both present and past smokers was noted; (vi) alcohol intake — a record was made of the quantity of beer, wine and spirits consumed in an average week; (vii) physical activity both at work and during leisure time was coded; (viii) dietary history — a 24-hour dietary recall was coded in detail; and (ix) the Bortner Short Rating Scale for coronary-prone behaviour was also completed.¹⁰

The physical examination consisted of: (i) blood pressure recordings, which were taken after respondents had been seated for at least 30 minutes, according to American Heart Association (AHA) guidelines;¹¹ readings were taken three times and the lowest was recorded (phase V) (a single observer, who had been standardised against an experienced clinician, recorded all blood pressures); (ii) the anthropometric study consisted of: (a) height measured to the nearest 0,5 cm; and (b) mass measured with the respondent in light clothing and without shoes; (iii) a resting 12-lead ECG was coded according to the revised Minnesota Code Manual.¹²

For the third visit all respondents were asked to fast for 14 hours and then 20 ml blood was collected. This was allowed to clot at room temperature and, after spinning, the serum was frozen and stored for later assay for cholesterol, triglyceride, high-density lipoprotein (HDL) cholesterol, uric acid and glucose levels. The haemoglobin level was also measured. The following tests, using kits provided by Boehringer Mannheim, were performed: serum cholesterol by the CHOD-PAP enzymatic method; and serum uric acid and triglyceride levels by the peridochrom GPO-PAP enzymatic colorimetric method. Controls were included in each batch — the special control provided was used for HDL-cholesterol, and for the serum cholesterol, uric acid and triglycerides the preclip low control and preclip EL high control samples were used. The peridochrom GOD-PAP enzymatic colorimetric method was also used for testing glucose levels, the precinorm and precipath low and high controls respectively. A glucose tolerance test using 75 g glucose was performed and a fasting and 2-hour blood sample collected. Glucose tolerance was classified according to the criteria given in a report of the World Health Organisation's study group.¹³ Diabetes was diagnosed if the concentration of glucose in venous plasma was $> 7,8$ mmol/l

or if the concentration of glucose in venous plasma 2 hours after the glucose loading was $\geq 11,1$ mmol/l or both. Haemoglobin levels were measured in a Spencer Wells haemoglobinometer.

All equipment was regularly calibrated and the mercury manometer and ECG recorder met with specifications as laid down by the AHA. ECGs were read by two independent 'blind' observers and disputes were settled by discussion. As regards blood chemical tests, reference standards were included in each run. Split samples were sent to the laboratories of the National Research Institute for Nutritional Diseases of the South African Medical Research Council, Parowvallei, CP, for comparison of readings.

Statistical methods

Descriptive statistics of risk factors consisting of means and standard deviations in the case of continuous variables and percentages in the case of categorical variables, were calculated for each age and sex grouping, as well as for each sex and the total sample. In addition, the number of respondents who had combinations of the risk factors hypercholesterolaemia, smoking, hypertension, and diabetes, were determined. Diabetes was measured using one cut-off point, whereas the other three variables were assessed at 2 cut-off points. Crude prevalences for each sex were age-adjusted using the 1985 population figures for the Indian population of metropolitan Durban, after taking into account the Human Sciences Research Council's (HSRC) estimates of census undercount.¹⁴ The crude overall estimates were age and sex adjusted using the same census data. In the text crude rates are given, except where indicated as adjusted (adj).

To assess the association between hypercholesterolaemia and other risk factors, a stratified analysis was carried out, stratifying by age as defined by < 45 years, and ≥ 45 years. To test for homogeneity of association across the strata, the Breslow-Day test for homogeneity of the odds ratio¹⁵ was used. If the association in the one stratum was found to differ from that in the other, the association within a stratum was tested using the chi-square test. The significance level was defined as 5%. If the association was found to be homogeneous the Cochran-Mantel-Haenszel (CMH) statistic for general association¹⁵ was used to assess the overall association.

To assess the association between history of CHD and other risk factors, a stratified analysis was carried out, stratifying by age (< 45 years/ ≥ 45 years), sex, and presence of diabetes. Homogeneity of association across the strata was tested as outlined above. In all cases the Breslow-Day test indicated that the association was homogeneous. The CMH statistic was then used to assess the association. In the cases where a significant association was found, the odds ratio was calculated, with a 95% confidence interval (CI).

TABLE I. AGE AND SEX DISTRIBUTION OF THE STUDY SAMPLE AND THE INDIAN POPULATION* OF METROPOLITAN DURBAN

Age (yrs)	No. in sample		Percentage		% in population	
	Men	Women	Men	Women	Men	Women
15 - 24	108	69	26,5	18,65	32,3	31,2
25 - 34	91	79	22,3	21,35	26,4	26,0
35 - 44	95	103	23,3	27,80	20,3	20,3
45 - 54	69	75	16,9	20,30	12,4	13,0
55 - 69	45	44	11,0	11,90	8,5	9,5
Total	408	370	100,0	100,0		

* 1985 census figures, adjusted for undercount using HSRC estimates.
Total No. of respondents = 778.

TABLE VI. PREVALENCE (%) OF COMBINATION OF MAJOR RISK FACTORS IN TOTAL STUDY SAMPLE

Level	Men					Women					Total	Age/sex adj†				
	Age groups (yrs)					Age groups (yrs)										
	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age adj.†	15 - 24	25 - 34	35 - 44			45 - 54	55 - 69	Total*	Age adj.†
Level A																
None	70,1	43,2	23,2	11,0	20,0	37,6	41,5	90,3	81,2	55,0	34,9	17,1	58,3	67,1	47,6	54,5
1 factor	25,8	45,7	41,5	34,4	30,0	35,7	35,7	9,7	16,0	30,0	33,3	46,3	26,0	21,8	31,1	28,6
2 factors	4,1	8,6	30,5	36,0	42,5	20,9	18,1	2,8	2,8	13,0	25,8	26,8	12,7	9,0	17,0	13,5
3 factors		1,2	3,7	17,2	5,0	4,7	3,7			2,0	6,1	9,8	3,0	2,1	3,8	2,9
4 factors		1,2	1,2	1,6	2,5	1,1	1,0							0,0	0,6	0,5
1 or more factors	29,9	56,8	76,8	89,0	80,0	62,4	58,5	9,7	18,8	45,0	65,1	82,9	41,7	32,9	52,4	45,5
Level B																
None	47,4	21,0	8,5	4,7	10,0	21,2	23,8	85,5	56,5	36,0	16,7	7,3	42,0	52,1	31,2	38,1
1 factor	38,1	48,2	40,2	12,5	20,0	34,3	36,3	14,5	34,8	38,0	35,4	29,3	31,7	28,6	33,0	32,5
2 factors	12,4	25,9	36,6	51,6	45,0	31,3	28,7	8,7	8,7	18,0	31,8	41,5	18,3	13,7	25,1	21,1
3 factors	2,1	3,7	13,4	18,8	22,5	10,2	8,8			8,0	13,6	22,0	7,7	5,4	9,0	7,0
4 factors		1,2	1,2	12,5	2,5	3,0	2,4				1,5		0,3	0,2	1,7	1,3
1 or more factors	52,6	79,0	91,5	95,3	90,0	78,8	76,2	14,5	43,5	64,0	83,3	92,7	58,0	47,9	68,8	61,9

* Crude.
† Age adjusted using 1985 Census figures for Indian population of Metropolitan Durban.
‡ Age and sex adjusted using 1985 Census figures for Indian population of Metropolitan Durban.

in this group. When considering worktime activity, 28,9% of men and 18% of women were involved in sedentary activity (i.e. did no significant walking or hard physical labour during work).

Association between hypercholesterolaemia and other risk factors

The data showed that 22,3% of men and 20,0% of women were hypercholesterolaemic (level A) but this difference was not significant (chi-square; *P* value = 0,110). There was, however, a highly significant association between age and high serum cholesterol levels (chi-square; *P* value = 0,001). When stratifying for age it was found that in the under 45-year-old age group, 14,8% of subjects were hypercholesterolaemic out of a total of 540. In the age group ≥ 45 years 32,6% of the respondents had high levels of cholesterol out of 230 subjects. With age stratified as above, hypercholesterolaemia was significantly associated with high levels of triglycerides (CMH; *P* = 0,001), and a non-protective ratio of HDL to TC (CMH; *P* = 0,001). The association of hypercholesterolaemia with family history of CHD (CMH; *P* = 0,053) and hyperuricaemia (CMH; *P* = 0,067) was close to significance. No significant association of hypercholesterolaemia with obesity (CMH; *P* = 0,548) or hard physical activity (CMH; *P* = 0,295) was found. In the young age group there was a significant association with smoking 10 or more cigarettes per day (chi-square; *P* = 0,812). Similarly there was a highly significant association with hypertension in the young age group (chi-square; *P* = 0,001) but not in the older group (chi-square; *P* = 0,499). No significant overall association with diabetes was found (CMH; *P* = 0,499) but in the younger age group there was a tendency for a higher proportion of hypercholesterolaemia to exist in the diabetics (23,7%) than in the non-diabetic group (13,9%).

Association of risk factors with history of CHD

As shown in Tables II and III, a personal history of CHD, as based on the Rose Questionnaire, was obtained from 119 respondents (15,3%). There were more women (70) than men (49) in this group. The mean age of those with a positive history was 44,2 ± 13,5 years while that of subjects without a history of CHD was 36,5 ± 13,3 years. Table VIII shows the prevalence of selected risk factors in those with and without a personal history of CHD.

The association between these risk factors and the presence of CHD history was tested after stratifying for age (< 45 years; ≥ 45 years) and sex. A highly significant association was found between diabetes and CHD history (CMH; *P* < 0,001). The odds ratio indicated that the odds of diabetics having a history of CHD, are 2,02 those of respondents without diabetes. The 95% confidence interval of the odds ratio was 1,39 : 2,94. Similarly, a highly significant association was found between serum triglyceride level and CHD history (CMH; *P* < 0,001). The odds of respondents with high levels of serum triglyceride having a history of CHD are 2,15 those of subjects with normal levels. The 95% confidence interval of the odds ratio was 1,54 - 2,99. It was, however, observed that 30% of the subjects with hypertriglyceridaemia were diabetics.

It was therefore decided to assess the association between a history of CHD and other risk factors by controlling for age and sex as well as diabetes, as shown in Table IX. It was found that high levels of triglyceride (CMH; *P* = 0,001), hypercholesterolaemia (CMH; *P* = 0,023) and low level of education (< std 5) (CMH; *P* = 0,042) were significantly associated with CHD. The odds ratio of respondents with high levels of serum triglycerides having a history of CHD are 1,89 those of respondents with normal levels (95% CI 1,35 - 2,67).

TABLE VII. PREVALENCE (%) OF MINOR RISK FACTORS

Risk factors	Men					Total*	Age adj.†	Women					Total	Age adj.†	Total sample	Age adj.‡
	Age group (yrs)							Age groups (yrs)								
	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69			15 - 24	25 - 34	35 - 44	45 - 54	55 - 69				
No. of patients	108	91	95	69	45	408		69	79	103	75	44	370		778	
Reversible factors																
BMI obesity (≥ 30)	2,8	1,1	4,2	4,3	4,4	3,2	3,0	7,2	8,9	23,3	40,0	34,1	21,6	17,5	12,4	10,5
Overweight (≥ 25 in men; ≥ 24 in women)	9,3	17,6	34,7	24,6	28,9	21,8	20,3	23,2	46,8	67,0	80,0	77,3	58,4	50,4	40,1	35,8
Type A behaviour (Bortner ≥ 55)	14,8	24,2	28,4	17,4	8,9	19,9	19,8	33,3	10,1	10,7	10,7	2,3	13,8	17,0	17,0	18,3
Sedentary workday activity	46,7	32,2	18,7	14,1	18,8	28,9	30,6	41,2	10,1	12,6	16,2	15,0	18,4	21,7	23,8	26,1
Hyperuricaemia ($\geq 0,42$ mmol/l in men, $\geq 0,34$ mmol/l in women)	8,4	12,2	11,8	8,8	11,1	10,4	10,4	4,3	12,8	12,6	23,3	31,8	15,5	13,1	12,9	11,8
Alcohol (past and current)	34,3	64,4	70,2	76,8	77,2	61,5	58,7	4,4	5,1	2,9	8,0	2,3	4,6	4,5	34,4	30,9
Oral contraceptive inactivity	—	—	—	—	—	—	—	17,4	22,8	5,3	—	—	9,7	—	—	—
Leisure (< 2000 kcal/wk)	88,9	95,6	91,6	92,7	—	92,9	92,7	94,2	97,5	96,1	100,0	100,0	97,3	96,7	95,0	94,7
Work (< 7700 kcal/wk)	98,1	98,9	95,6	96,9	96,9	97,4	—	100,0	100,0	100,0	100,0	100,0	100,0	—	98,7	—
Non-reversible factors																
History of chest pain	7,4	4,4	13,7	15,9	28,9	12,0	10,9	5,8	12,7	23,3	28,0	25,0	18,9	15,7	15,3	13,4
Family history of CHD	43,5	40,7	38,9	40,6	26,7	39,5	40,0	52,5	44,3	35,9	44,0	43,2	43,2	45,1	41,3	42,6
Protective levels of HDL:TC ($\geq 20\%$)	78,5	48,9	45,2	45,6	48,9	55,3	57,1	88,4	71,8	57,3	45,2	43,2	62,1	68,2	58,6	62,7

* Crude.

† Age adjusted using 1985 Census figures for Indian population of Metropolitan Durban.

‡ Age and sex adjusted using 1985 Census figures for Indian population of Metropolitan Durban.

TABLE VIII. PREVALENCE (%) OF RISK FACTORS IN SUBJECTS WITH HISTORY OF CHD

	History of CHD			No history of CHD		
	Men	Women	Total	Men	Women	Total
No. of subjects	49	70	119	359	300	659
Mean age (yrs)	44,3 ± 15,9	44,1 ± 11,7		35,5 ± 13,5	37,5 ± 13,0	
Hypercholesterolaemia	33,3	29,4	31,0	20,9	15,1	18,2
Hypertension	40,8	25,7	31,9	14,2	18,7	16,2
Overweight	20,4	67,1	47,9	22,0	56,3	37,6
Obesity	4,1	27,1	17,7	3,1	20,3	10,9
Hyperuricaemia	12,5	16,2	14,7	10,1	15,4	12,5
Smoking > 10 cigs/d	44,9	11,4	25,2	41,5	4,3	24,6
Diabetes	21,6	21,5	21,6	11,1	9,9	10,6
Family history of CHD	42,9	50,0	47,1	39,0	41,7	40,2

TABLE IX. TESTING ASSOCIATION BETWEEN SELECTED RISK FACTORS AND CHD HISTORY, USING THE COCHRAN-MANTEL-HAENSZEL STATISTIC

Risk factor	CMH P value	Odds ratio*	95% CI
Diabetes	0,001	2,02	1,39 - 2,94
Triglyceride	0,001	1,89	1,35 - 2,67
Hypercholesterolaemia	0,023	1,52	1,06 - 2,19
Educational status	0,042	1,52	1,12 - 2,27
CHD family history	0,059	1,39	0,98 - 1,97
Hypertension	0,079	1,41	0,96 - 2,08
HDL cholesterol	0,208		
Smoking (> 10 cigs/d)	0,249		
Obesity	0,540		
Physical activity (work, leisure)	0,613		
Hyperuricaemia	0,735		
Overweight	0,756		
Bortner type A personality	0,804		

* Controlled for age/sex/diabetes for all factors except diabetes where only age/sex were controlled for.

The odds of hypercholesterolaemic subjects are 1,52 those of normocholesterolaemic patients (95% CI 1,06 - 2,19). The odds of people with a lower education level having history of CHD are 1,52 those of respondents with a high level (95% CI 1,12 - 2,27). In the case of family history of CHD (CMH; $P = 0,059$) and hypertension (CMH; $P = 0,079$), which were close to significant, the odds ratios were 1,39 (95% CI 0,98 - 1,97) and 1,41 (95% CI 0,96 - 2,08) respectively.

Discussion

The majority of Indian South Africans are the descendants of indentured labourers brought to Natal between 1860 and 1911 to develop the country's sugar belt. The Indian population of South Africa is composed of both Dravidian and Aryan stock, each having distinct cultural and anthropological characteristics.¹⁸⁻²⁰ There are more than 11 million people dispersed throughout the world who can trace their ancestry to the Indian sub-continent.²¹ Despite their great cultural and geographical diversity, migrant Indians have a high mortality from CHD in comparison with other ethnic groups locally. Early indications came from countries with long-established

Indian populations — Singapore,²² Fiji,²³ South Africa,⁴ Uganda,²⁴ and Trinidad.²⁵ Recently, similar reports have come from the UK.²⁶⁻²⁸ In their study of diet and CHD rates among the Indian community, McKeigue *et al.*²⁹ could not explain the incidence of CHD by the risk factors they measured. They concluded that the identification of factors responsible for the high mortality and morbidity due to CHD experienced by people of Indian origin around the world is of the utmost importance for the understanding and prevention of this disease.²⁹

In our study on the Rose Questionnaire,⁹ a positive personal history was obtained in 15,3% (adj. 13,4%) of the respondents. Resting ECG tracings showed that 47,6% (adj. 48,2%) of the subjects had abnormalities that could be coded. A study of the prevalence of CHD in an urban population in northern India³⁰ found that 62% of the men and 88% of the women had clinically silent CHD. In our study the important risk factors were hypercholesterolaemia, hypertriglyceridaemia, diabetes, and smoking in the men; and diabetes, hypercholesterolaemia and hypertriglyceridaemia in the women (Tables V and VI). The minor risk factors were hyperuricaemia, sedentary occupation, obesity in women, and a positive family history of CHD (Table VII). In spite of a high prevalence of CHD, the serum HDL : TC ratio was found to be favourable in 58,6% (adj. 62,7%) of respondents.

Diabetes mellitus was present in 15,8% (adj. 12,4%) of the total sample (Table V). A high prevalence of diabetes has been reported in migrant Indians in South Africa,^{31,32} Fiji,³³ Trinidad,³⁴ Singapore,³⁵ and Southall Borough of London.³⁶ The prevalence in Indians in India was believed to be low.³⁷ Recent reports from India have shown that the prevalence of diabetes is high and is comparable with the high prevalence seen in migrant Indian populations.^{38,39} However, as stated in an editorial in *The Lancet*,⁴⁰ it is strange that neither of the British studies^{28,41} mentioned non-insulin-dependent diabetes mellitus as a major cause of CHD in the Indian population of the UK. Miller *et al.*⁴² found that fasting blood glucose results were unrelated to CHD in the Indian population of Trinidad. Beckles *et al.*⁴³ noted that ethnic differences in risk factors were not explained by systolic blood pressure, fasting blood sugar concentration, serum HDL or LDL concentration, or smoking habits. Differences in risk of cardiovascular death between Indian and white men seemed to be accounted for by the high prevalence of diabetes in men (19%) but other ethnic contrasts in mortality rates were unrelated to diabetes mellitus.

A three-community study (the CORIS study) of rural Afrikaans-speaking whites in the south-western Cape (a population group that is prone to CHD)¹⁶ revealed that the major risk factors — hypercholesterolaemia, hypertension and smoking — were very common. The minor risk factors, such as inactivity, obesity, hyperuricaemia, coronary prone behaviour,

ischaemic changes on ECG and a family history of CHD, were exceedingly common. Singly or in combination the major risk factors were present in the great majority of the study population after the age of 44 years. Our study showed that the risk factors leading to CHD in Indians were similar to the Afrikaans-speaking whites, except that diabetes was common in the Indian population. No mention is made by the authors of the CORIS study of the prevalence of diabetes in rural Afrikaans-speaking whites⁴³ or in the study of coronary risk factors of the 'mixed' population of the Cape Peninsula.⁴⁴

In conclusion, CHD is a major cause of morbidity and mortality in the Indian population of South Africa. A study of the major risk factors leading to CHD showed that 52% (adj. 45,5%) had at least one major risk factor at level A and 68% (adj. 61,9%) at level B (Table VI). Diabetes mellitus was strongly associated with a positive history of CHD. Because of the severe nature of the 'epidemic' of CHD in the Indian population, an immediate and intensive programme of primary prevention of CHD risk factors should be instituted.

This study was supported by the South African Medical Research Council, the Heart Foundation of Southern Africa and the South African Sugar Association. We wish to thank Professor J. E. Rossouw formerly Director of the National Research Institute of Nutritional Diseases of the South African Medical Research Council for his help and encouragement, Mrs Linda van Rooyen for the biochemical assays and Mrs R. Billy for typing the manuscript.

Drs S. Khan and S. R. Somers were Medical Research Council post-intern scholars.

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