Risk factors for coronary heart disease in the white community of Durban

Y. K. SEEDAT, F. G. H. MAYET, E. GOUWS

Abstract **Coronary heart disease (CHD) is the leading cause of death among the white and Indian populations of Durban. This was a community-based study of the white population of Durban, which is predominantly English-speaking. There were 396 subjects (194 men, 202 women) aged 15 - 69 years. A history of CHD was present in 9,3% of the subjects. The important risk factors were hypercholesterolaemia, hypertension and smoking. The minor risk factors were obesity, hypertriglyceridaemia, hyperuricaemia, a sedentary occupation and a history of CHD in the immediate family. Electrocardiograph abnormalities denoting CHD were present in 17% of subjects. A study of the major risk factors showed that 35,1% (age and sex adjusted) had at least one major risk factor at the higher level (level A) and 33,8% (age and sex adjusted) at the lower risk levels (level B). When the combination of risk factors was taken into account, 15,2% and 28% had two major risk factors, one each at levels A and B respectively. On average the percentage of men and women with one risk factor or more increased with age. A protective high-density lipoprotein/total cholesterol ratio ≥ 20% was present in 53,5% of the respondents. Because of the severe nature of CHD, an intensive programme for the primary prevention of CHD risk factors should be instituted.**

S Afr Med J 1994; **84:** 257-262.

Coronary heart disease (CHD) is common in South Africa and is a major clinical problem.[1,2] It is the leading cause of death among economically active white South Africans, the age-standardised mortality rates (MRs) for men being higher than those in a number of other countries with a high CHD mortality rate.[1] However, there has been an observed decline in MRs among whites of both sexes, which suggests that preventable major risk factors may be coming under control.[3] There is an impression among health professionals that Afrikaans-speaking and Jewish people are especially prone to the disease, but this has been difficult to prove.[3] A three-community study of a rural Afrikaans-speaking community in the south-western Cape revealed major risk factors in the great majority of the study population aged more than 44 years.[4] No previous study has examined the risk factors which lead to CHD in an urban white population, however. This paper describes the first white urban study which examined risk factors for CHD in a white community that was largely English-speaking.

Department of Medicine, University of Natal, Durban
Y. K. SEEDAT, O.M.S.S., M.D. (N.U.IREL.), PH.D. (MED. SCI.), F.R.C.P. (LOND.), F.R.C.P. (IREL.), F.A.C.P., F.C.P. (S.A.), F.A.C.C., F.C.C.P., F.I.C.A., F.I.C.N.
F. G. H. MAYET, M.D.
Institute for Biostatistics of the South African Medical Research Council, Parowvallei, CP
E. GOUWS, B.SC. HONS

Accepted 28 Apr 1993.

Subjects and methods

A coronary risk factor survey was carried out among whites living in the metropolitan area of Durban. It was similar to our previous study of risk factors for CHD in the Indian population of Durban.[5] The sample size and selection were based on the 1985 population census figures for the urban Durban area in which the white population numbered 215 344 (total population of Durban 446 318),[6] together with information from the various municipalities of the number of dwellings in each residential area. A fixed percentage population of 0,18% per area was studied.

Household addresses were randomly selected from the latest ratepayers' and electricity consumers' records. At each address one member of the family aged between 15 and 69 years was randomly selected for participation in the survey. Exclusion criteria were: (*i*) pregnancy and lactation; (*ii*) being bed-ridden; (*iii*) mental retardation; (*iv*) carcinoma; and (*v*) anti-tuberculosis therapy. The study was undertaken in July 1987 and completed over a 2-year period.

Each household was visited on at least three occasions. On 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 appointment time made. During the second visit a risk factor questionnaire was completed by interview and a physical examination carried out. A single interviewer, a qualified nursing sister who was intensively trained to administer the questionnaire in a standardised manner, was employed. The questionnaire elicited the following: (*i*) biological, ethnic and socio-economic data; (*ii*) a present or previous history of angina pectoris or pain from possible myocardial infarction — the London School of Hygiene questionnaire for chest pain (Rose Questionnaire[7]) was used; (*iii*) medical history of hypertension, diabetes, CHD, gout and stroke — respondents were asked to produce all medication for inspection; (*iv*) history of hypertension, diabetes, CHD and stroke in parents, siblings and grandparents; (*v*) smoking habits of both present and former smokers; (*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; (*viii*) dietary history — a 24-hour dietary recall was coded in detail; and (*ix*) details of coronary-prone behaviour — the Bortner Short Rating Scale was used to assess this.[8]

The physical examination entailed blood pressure recordings, which were taken after respondents had been seated for at least 30 minutes. According to American Heart Association (AHA) guidelines,[9] readings were taken three times and the lowest recorded (phase V) by a single observer, who had been standardised against an experienced clinician. The anthropometric study entailed height measured to the nearest 0,5 cm and mass measured with the respondent in light clothing and without shoes. A resting 12-lead electrocardiograph (ECG) was coded according to the revised Minnesota Code Manual.[10]

At the third visit 20 ml blood were collected from all respondents, who had been asked to fast for 14 hours. The blood 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 measurements were made by means of kits provided by Boehringer Mannheim: serum cholesterol levels 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 measurement; for 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 used for testing glucose levels, and the Precinorm and Precipath for low and high controls respectively. A glucose tolerance test using 75 g glucose was performed and fasting and 2-hour blood samples were collected. Glucose tolerance was classified according to the criteria of the World Health Organisation's study group.[11] 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 glucose loading was ≥ 11,1 mmol/l or both. Haemoglobin levels were measured with a Spencer Wells haemoglobinometer.

All equipment was regularly calibrated and the mercury manometer and ECG recorder met with the specifications laid down by the AHA.[9] ECGs were read by two independent 'blind' observers and disputes were settled by discussion. Reference standards were included in each run of blood chemical tests. Split samples were sent to the laboratories of the National Research Institute for Nutritional Diseases of the South African Medical Research Council for comparison of readings.

Statistical methods

Descriptive statistics were calculated for the various risk factors within each age and sex grouping, as well as for each sex and for the total sample.

Crude overall prevalences were age- and sex-adjusted using the 1985 census figures for the white population of metropolitan Durban. The Human Sciences Research Council's estimates of census undercount were taken into account.[6]

The prevalence of combinations of risk factors such as hypercholesterolaemia, smoking, hypertension and diabetes was calculated. Diabetes was measured with only one cut-off point, whereas the other 3 were considered at 2 cut-off points.

The association between hypercholesterolaemia and other risk factors was tested after the study population was stratified for age (< 45 years v. ≥ 45 years). The Breslow-Day test for homogeneity of the odds ratio was used to test for homogeneity across strata. If the association in one stratum was found to differ from that in the other, the association within a stratum was tested by means of the chi-square test. For homogeneous strata, the Cochran-Mantel-Haenszel (CMH) test was used to assess the overall association. In cases where significant associations were found, odds ratios and 95% confidence intervals were calculated.

For the association between CHD and risk factors an analysis stratified for age and sex was performed. Homogeneity of association across strata was tested as described above. In all cases the Breslow-Day test indicated that the associations were homogeneous and the CMH statistic was therefore used to assess the significance of the association.

Results

There was a 93% response rate; the refusal rate of 7% was replaced in order to obtain our final sample of 396. The vast majority of the sample (91%) were English-speaking. The age and sex distribution is given in Table I.

Among the men 32% were managers or clerical workers, 13% were professionals, 20% were artisans and semi-skilled workers, 10% were salesworkers and 11% were retired. Among the women 33% were managers or clerical workers, 12% were professionals and 40% were housewives.

From the Rose Questionnaire,[7] a positive history of CHD was obtained for 11,9% of the men and 9,5% of the women in the total sample (Table II). When the analysis of a 12-lead ECG was included, a surprisingly high prevalence of changes was obtained. Resting ECG tracings were analysed in 274 subjects (137 men, 137 women). In 16% of men and 8% of women abnormalities that could be coded were detected. The overall prevalence of ECG abnormalities was 36,5%. The ECG findings of CHD according to the CORIS criteria are shown in Table III.

TABLE II.
Positive history of CHD (Rose Questionnaire)

	Men		Women	
Age (yrs)	No.	%	No.	%
15 - 24	1	0,5	0	0,0
25 - 34	1	0,5	3	1,5
35 - 44	4	2,1	6	3,0
45 - 54	1	0,5	3	1,5
55 - 69	16	8,3	7	3,5
Total (crude)	23	11,9	19	9,5
Age-adjusted		10,8		7,9

Total number in study sample = 396; number with history of CHD = 42; percentage with history of CHD = 10,6; age- and sex-adjusted = 9,3.

TABLE I.
Age and sex distribution

	No.		%		% of population*	
Age (yrs)	Men	Women	Men	Women	Men	Women
15 - 24	20	20	10,3	9,9	23,9	22,8
25 - 34	41	36	21,1	17,8	21,8	19,9
35 - 44	38	61	19,6	30,2	18,2	16,7
45 - 54	41	33	21,1	16,3	13,2	14,0
55 - 69	54	52	27,8	25,7	22,9	26,6
Total	194	202	100,0	100,0	100,0	100,0

*1985 census figures, adjusted for undercount using HSRC estimates.
No. of respondents = 396.

TABLE III.
Relevant ECG findings denoting CHD

Minnesota code	ECG abnormality	Men (%)	Women (%)
1.1	Large Q waves	5,83	5,10
1.2	Medium Q waves	8,0	2,91
3.1	LVH	–	–
4.1	Large ST depression	1,45	–
5.1	Large T waves	–	–
5.2	Medium T waves	0,72	–
7.1	LBBB	–	–
7.2	RBBB	–	–

LVH = left ventricular hypertrophy; LBBB = left bundle-branch block; RBBB = right bundle-branch block.

Descriptive statistics of selected risk factors

Table IV shows the age and sex breakdown of mean levels (± SD) of selected risk factors. The total serum cholesterol levels rose with age in both sexes. The serum HDL-cholesterol levels were higher in women and showed no age-related trend. The serum triglyceride levels and mean systolic and diastolic blood pressures also increased with age in both sexes. Serum uric acid levels were similar in both sexes. Body mass index (BMI) values (wt/ht^2) were similar in both sexes. The number of smokers (≥ 10 cigarettes per day) was similar in both sexes. The age distribution in this group showed smoking to be more common in young men in contrast to women where the incidence was almost equally distributed in all age groups.

Prevalence of major reversible risk factors

The prevalences of hypercholesterolaemia, smoking and hypertension were examined at two levels of risk: level A, which is the conventional cut-off point, and level B, a level at which the risk of CHD is higher than average (Pooling Project[12]) (Table V). A total serum cholesterol level of ≥ 6,5 mmol/l was detected in 27,8% of both

TABLE IV.
Descriptive statistics of selected risk factors (mean ± SD)

	Men					Women				
Age group (yrs)	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69
No. of patients	20	41	38	41	53	20	35	61	33	52
Serum total cholesterol (mmol/l)	4,9 ± 0,7	5,6 ± 1,1	5,9 ± 1,2	6,3 ± 1,4	6,2 ± 1,1	5,0 ± 1,1	5,4 ± 1,2	5,7 ± 1,1	6,1 ± 1,2	7,2 ± 1,4
HDL cholesterol (mmol/l)	1,1 ± 0,2	1,1 ± 0,3	1,4 ± 0,7	1,2 ± 0,4	1,3 ± 0,5	1,3 ± 0,3	1,3 ± 0,6	1,5 ± 0,5	1,6 ± 0,8	1,3 ± 0,4
HDL/TC ratio (%)	21,8 ± 5,5	20,6 ± 7,1	25,9 ± 16,4	19,6 ± 9,2	20,6 ± 9,3	27,1 ± 7,3	25,3 ± 10,4	26,3 ± 11,6	28,0 ± 17,6	19,2 ± 5,9
Triglycerides (mmol/l)	1,2 ± 0,5	1,7 ± 1,1	1,7 ± 1,4	1,9 ± 1,3	1,8 ± 0,8	1,1 ± 0,4	1,3 ± 1,1	1,3 ± 0,9	1,3 ± 0,5	2,1 ± 1,0
Systolic blood pressure (mmHg)	117,3 ± 12,3	122,6 ± 11,8	128,2 ± 13,5	130,6 ± 16,8	150,5 ± 24,6	115,1 ± 12,2	112,4 ± 12,3	122,2 ± 18,8	130,2 ± 18,8	140,8 ± 23
Diastolic blood pressure (mmHg)	72,1 ± 8,8	74,9 ± 11,5	78,3 ± 9,4	81,4 ± 9,4	84,3 ± 10,0	69,5 ± 10,0	69,8 ± 13,5	76,0 ± 11,2	79,5 ± 9,9	79,8 ± 9,9
Uric acid (mmol/l)	0,38 ± 0,23	0,33 ± 0,07	0,37 ± 0,07	0,34 ± 0,08	0,35 ± 0,08	0,26 ± 0,06	0,25 ± 0,08	0,26 ± 0,08	0,26 ± 0,10	0,34 ± 0,28
BMI	21,8 ± 2,4	24,1 ± 3,9	26,3 ± 4,7	25,7 ± 3,9	26,1 ± 3,7	21,9 ± 4,1	22,1 ± 4,2	22,9 ± 4,5	23,7 ± 4,1	25,6 ± 4,7
Cigarette smokers ≥ 10/d (%)	25,0	52,5	31,6	24,4	16,7	20,0	27,8	24,6	21,2	25,0
Fasting blood sugar (mmol/l) at 0 hours	4,3 ± 1,3	4,1 ± 0,9	4,1 ± 1,0	4,2 ± 0,9	4,8 ± 1,8	4,3 ± 0,4	4,5 ± 0,8	4,4 ± 1,7	4,2 ± 0,7	4,8 ± 1,8
2 hours	4,8 ± 0,7	5,0 ± 1,0	5,5 ± 1,6	5,3 ± 1,2	6,3 ± 3,7	5,3 ± 0,8	5,2 ± 1,1	5,0 ± 0,9	5,3 ± 1,4	6,1 ± 3,1

TABLE V.
Prevalence (%) of major reversible risk factors in the total study sample

	Men							Women								
Age group (yrs)	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	Total* sample	Age/sex‡ adjusted
Serum cholesterol																
A ≥ 6,5 mmol/l	5,3	31,7	29,7	41,5	42,3	33,7	27,8	5,6	17,7	21,8	33,3	61,2	31,8	27,7	32,7	27,8
B ≥ 5,7 mmol/l	10,5	41,5	51,4	63,4	73,1	53,7	44,8	33,3	35,3	41,8	69,7	91,8	57,7	53,0	55,7	49,0
Smoking																
A ≥ 10 cigarettes/d	25,0	52,5	31,6	24,4	16,7	29,5	29,9	20,0	27,8	24,6	21,2	25,0	24,3	23,8	26,8	26,8
B ≥ 1 cigarette/d	31,6	53,9	29,7	29,3	18,5	31,6	32,0	20,0	27,8	29,5	21,2	30,8	27,2	26,2	29,3	29,0
Blood pressure																
A ≥ 160/95 mmHg and/or treatment	0,00	4,9	13,2	12,2	57,4	22,2	18,0	0,00	5,6	9,8	21,2	40,4	17,8	16,3	20,0	17,2
B ≥ 140/90 mmHg	10,0	22,0	31,6	39,0	75,9	41,2	35,1	0,00	8,3	24,6	42,4	63,5	32,2	28,2	36,6	31,6
Diabetes mellitus																
Blood sugar at 0 h. ≥ 7,8 mmol/l and/or 2 h. ≥ 11,0 mmol/l + known diabetics	5,3	0,00	0,00	0,00	8,5	2,7	2,6	0,00	3,1	1,9	6,1	6,5	3,9	3,5	3,3	3,0

* Crude
† Age-adjusted using 1985 census figures for white population of metropolitan Durban.
‡ Age- and sex-adjusted using 1985 census figures for white population of metropolitan Durban.
The major risk factors are considered at two levels:
A = level where conventional higher cut-off points are used.
B = level where cut-off points approximate the lower levels associated with increased risk.

men and women. Smoking was regarded as a risk factor and occurred in 29,9% of men and 23,8% of women. Hypertension (systolic pressure ≥ 160 mmHg and/or diastolic pressure ≥ 95 mmHg) occurred in 17,2% (18% of men and 16,3% of women). Based on a modified glucose tolerance test and the presence of a positive history, 3% were diabetic. Thus in the total study sample 27,8% were hypercholesterolaemic, 17,2% were hypertensive and 26,8% were at risk from smoking. When these parameters were taken into account, many more of the subjects were at risk.

Combination of major reversible risk factors

As shown in Table VI, 53,6% of respondents had at least one risk factor at level A, and 69,4% at least one at level B when hypercholesterolaemia, smoking and diabetes were considered. When the combination of risk factors was taken into account, 15,2% and 28% had two major risk factors at levels A and B respectively. On average, the percentage of men and women with one risk factor or more increased with age.

TABLE VI.
Prevalence (%) of combination of major risk factors in the total study sample

	Men							Women								
Age group (yrs)	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	Total* sample	Age/sex‡ adjusted
Level A																
None	65,0	36,6	47,4	43,9	24,1	39,7	43,3	75,0	61,1	55,7	39,4	21,2	47,0	49,5	43,4	46,4
1 factor	35,0	39,0	34,2	36,6	38,9	37,1	36,6	25,0	30,6	37,7	39,4	36,5	35,2	33,7	36,1	35,1
2 factors	0,00	24,4	15,8	17,1	27,8	19,6	16,5	0,00	5,6	1,6	21,2	34,6	13,9	13,9	16,7	15,2
3 factors	0,00	0,00	2,6	2,4	9,3	3,6	3,1	0,00	0,00	4,9	0,00	7,7	3,5	3,0	3,5	3,0
4 factors	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	2,8	0,00	0,00	0,00	0,5	0,00	0,3	0,25
1 or more factors	35,0	63,4	52,6	56,1	75,9	60,3	56,2	25,0	38,9	44,3	60,6	78,9	53,0	50,6	56,6	53,6
Level B																
None	50,0	31,7	29,0	22,0	7,4	24,2	28,4	55,0	52,8	37,7	9,1	3,9	28,7	31,7	26,5	30,1
1 factor	40,0	26,8	34,2	39,0	27,8	32,5	32,5	40,0	27,8	36,1	45,5	30,8	35,2	35,2	33,8	33,8
2 factors	10,0	34,2	31,6	26,8	51,9	34,5	30,9	5,0	16,7	21,3	42,4	42,3	27,7	25,2	31,1	28,0
3 factors	0,00	7,3	5,3	12,2	13,0	8,8	7,2	0,00	0,00	4,9	3,0	21,2	7,4	6,9	8,1	7,1
4 factors	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	2,8	0,00	0,00	1,9	1,0	1,0	0,5	0,5
1 or more factors	50,0	68,3	71,1	78,1	92,6	75,8	70,6	45,0	47,2	62,3	90,9	96,2	71,3	68,3	73,5	69,4

* Crude
† Age-adjusted using 1985 census figures for white population of metropolitan Durban.
‡ Age- and sex-adjusted using 1985 census figures for white population of metropolitan Durban.
The major risk factors are considered at two levels:
A = level where conventional higher cut-off points are used.
B = level where cut-off points approximate the lower levels associated with increased risk.

TABLE VII.
Prevalence (%) of minor risk factors

	Men							Women								
Age group (yrs)	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	15 - 24	25 - 34	35 - 44	45 - 54	55 - 69	Total*	Age† adjusted	Total* sample	Age/sex‡ adjusted
No. of patients	20	41	38	41	54	194		20	36	61	33	52	202		396	
Reversible risk factors																
BMI																
Obesity (≥ 30)	0,00	7,3	18,4	14,6	9,4	10,9	8,8	5,0	5,6	6,6	6,1	13,5	7,9	7,4	9,4	8,1
Overweight (≥ 25 in men; ≥ 24 in women)	5,0	31,7	57,9	51,2	58,5	45,6	37,6	20,0	22,2	27,9	33,3	50,0	32,7	31,2	39,0	34,3
Type A behaviour (Bortner ≥ 55)	20,0	46,3	39,5	31,7	22,2	32,5	30,9	15,0	22,2	31,2	39,4	25,0	27,7	25,7	30,1	28,3
Sedentary workday activity	45,0	14,6	18,4	12,2	13,0	17,5	21,6	36,8	11,4	19,7	24,2	21,2	21,0	23,5	19,3	22,6
Hyperuricaemia (≥ 0,42 mmol/l in men, ≥ 0,34 mmol/l in women)	27,8	9,8	21,6	14,6	19,2	17,5	18,0	0,00	14,7	9,1	3,0	30,6	13,8	12,9	15,6	15,4
Alcohol (past and current)	90,0	90,0	78,4	87,5	87,0	86,4	85,1	50,0	77,8	73,3	72,7	67,3	70,1	66,8	78,1	75,8
Oral contraceptive	–	–	–	–	–	–	–	30,0	22,2	16,4	–	–	–	–	–	–
Inactivity/leisure (< 2000 kcal/wk)	65,0	85,4	94,7	87,8	92,6	87,6	84,0	100,0	100,0	95,1	96,9	96,2	97,0	97,5	92,4	90,1
Work (< 7700 kcal/wk)	95,0	95,1	86,8	100,0	92,5	93,8	92,8	100,0	97,1	96,7	93,9	96,2	96,5	96,5	95,2	94,7
Triglycerides (≥ 2 mmol/l)	10,5	26,8	21,6	34,2	38,5	29,0	24,7	5,6	8,8	9,1	9,1	46,9	18,5	17,3	23,8	21,0
Non-reversible risk factors																
History of chest pain	0,00	0,00	0,00	0,00	1,9	0,5	0,5	0,00	2,8	0,00	0,00	1,9	1,0	1,0	0,8	0,75
Family history of CHD	60,0	56,1	63,2	68,3	53,7	59,8	59,8	60,0	66,7	72,1	66,7	69,2	68,3	67,3	64,1	63,6
Protective levels of HDL:TC (20%)	57,9	53,7	56,8	29,3	38,5	45,3	46,9	77,8	75,8	72,7	66,7	38,8	63,8	60,0	54,5	53,5

* Crude
† Age-adjusted using 1985 census figures for white population of metropolitan Durban.
‡ Age- and sex-adjusted using 1985 census figures for white population of metropolitan Durba.

Prevalence of minor risk factors and protective HDL/total cholesterol (TC) ratio

The prevalence of minor risk factors is shown in Table VII. Based on BMI, obesity occurred in 8,1% while 34,3% were overweight. An interesting feature was that the BMI was almost the same in both sexes. Hyperuricaemia occurred in 15,4% and was common in both sexes. A positive history of CHD in immediate family members, viz. grandparents, parents and/or siblings, was obtained from 63,6% of respondents.

The HDL/TC ratio was regarded as protective when the HDL concentration was 20% or more of the TC level. This ratio was found to be protective in 53,5% of respondents. Elevated serum triglyceride levels were found in 21% of respondents.

Association of risk factors with a history of CHD

As shown in Table II a personal history of CHD was obtained from 42 respondents (10,6%). There were 23 men (11,9%) and 19 women (9,5%). The mean age of those with a positive history was 52,7 ± 13 years while that of subjects without a history of CHD was 42,8 ± 14 years. Table VIII shows the prevalence of selected risk factors in those with or without a history of CHD.

The association between risk factors and the presence of CHD was tested after stratification for age and sex. Fifteen respondents (35,7%) with CHD were younger and 27 (64,3%) older than 45 years.

We did not control for diabetes as was the case in the Indian study,[5] since only 12 of the respondents (3,32%) had a positive history of diabetes; none of these had CHD. Hypertension (P = 0,037) was significantly associated with CHD history. A family history of CHD was almost significant (P = 0,075). The odds of respondents with hypertension having a history of CHD are 2,24 times those of respondents with normal levels (95% CI 1,05 - 4,76). The odds of respondents with a family history of CHD having CHD themselves are 1,96 times those of respondents with normal levels (95% CI 0,93 - 4,12). This is shown in Table IX.

Association between hypercholesterolaemia and other risk factors

The data (Table X) showed that 27,8% of the respondents were hypercholesterolaemic (level A). Hypercholesterolaemia was significantly associated with high triglyceride levels (P < 0,0001). In the younger group (< 45 years) hypercholesterolaemia was significantly associated with overweight (P < 0,0001) and hypertension (P < 0,0001). These associations were not significant for respondents older than 45 years.

TABLE IX.
Testing association between selected risk factors and CHD history

Risk factor	Cochran-Mantel-Haenszel P-value	Odds ratio*	95% CI
Hypertension	0,037	2,24	(1,05;4,76)
CHD family history	0,075	1,96	(0,93;4,12)
Smoking (⩾ 10 cigarettes/d)	0,103		
Triglyceride	0,120		
Hypercholesterolaemia	0,641		
HDL cholesterol	0,431		
Hyperuricaemia	0,808		
Overweight	0,546		
Obesity	0,506		
Bortner type A personality	0,966		
Diabetes	0,172		
Educational status	0,919		
Physical activity (work, leisure)	0,757		
HDL:TC ratio	0,479		

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

TABLE X.
Association between hypercholesterolaemia and other risk factors

Risk factor	Cochran-Mantel-Haenszel P-value	Odds ratio	95% CI
Triglycerides	< 0,0001	7,09	(4,31; 11,628)
HDL/TC < 20	< 0,0001	5,99	(3,745; 9,524)
Family history of CHD	0,230		
Hyperuricaemia	< 0,0001	3,06	(1,73; 5,41)
Obesity	0,073	1,889	(0,943; 3,785)
Smoke ⩾ 10 cigarettes/d	0,053	1,641	(0,994; 2,708)
Bortner type A	0,648		
Diabetes	0,051	3,497	(0,995; 12,286)

The association of hypercholesterolaemia with the smoking of more than 10 cigarettes per day (P = 0,053) and obesity (P = 0,073) was close to significance in both instances. Family history of CHD and type A personality[8] were not significantly associated with hypercholesterolaemia.

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	22	18	40	167	170	337
Mean age (years)	55,3 ± 12,8	49,5 ± 12,9	52,7 ± 13,0	42,4 ± 14,0	43,2 ± 14,1	42,8 ± 14,0
Hypercholesterolaemia	40,9	38,9	40,0	32,9	31,2	32,1
Hypertension	47,8	26,3	38,1	18,8	17,1	18,0
Overweight	56,5	36,8	47,6	44,4	32,6	38,3
Obesity	8,7	5,3	7,1	11,2	8,3	9,7
Hyperuricaemia	9,1	22,2	15,0	18,7	12,9	15,8
Smoking ⩾ 10 cigarettes/d	13,0	15,8	14,3	31,4	24,9	28,0
Diabetes	0,00	0,00	0,00	3,1	4,4	3,7
Family history of CHD	73,9	79,0	76,2	58,2	67,4	63,0

Discussion

Despite the decline in CHD incidence in South African whites the MRs remain high. Overall, when South African whites' ranking was compared with those of other Western countries studied, it was found to have changed from 2nd place in 1970 to 4th position for men and 3rd for women in 1982.[3] Our study showed that a high number of subjects, 42 out of 396 (10,6%), had a positive history of CHD as determined by the Rose Questionnaire[7] (Table II). Moreover, relevant ECG changes denoting CHD occurred in 16% of the men and 8% of the women (Table III).

In this study the prevalence of hypertension (blood pressure ≥ 160 mmHg systolic and/or ≥ 95 mmHg diastolic) was 17,2%. This figure was similar to that in a previous study of the white community of Durban.[13] Hypercholesterolaemia was common and occurred in 27,8% of the white subjects when the cut-off level for serum cholesterol was ≥ 6,5 mmol/l and 49% when the cut-off level was ≥ 5,7 mmol/l. Moreover, a ratio of protective levels of HDL to total cholesterol of more than 20% was present in only 53,5% of the subjects (men 46,9%, women 54,5%). This contrasts with studies in the black population in which a ratio of protective levels of HDL to total cholesterol of more than 20% was observed in 81,3% of our study[14] and 96% of the blacks in the Cape Peninsula.[15] This study showed that 29,9% of the men and 26,8% of the women smoked more than 10 cigarettes a day. This contrasts with our studies of Indian[5] and black women in Durban[14] in which the prevalence was 5,3% and 3,4% respectively. A cross-sectional study of tobacco-smoking in a large rural community of 7 188 white subjects aged 15-64 years revealed that 48,1% of men and 17,9% of women were smokers.[16] The Rembrandt Tobacco Corporation estimated in 1980/1981 that 44% of white adult South African men and 36% of white adult women were smokers.[17] Smoking in our study was particularly common in young white men between the ages of 25 and 34 years (52,5%) (Table V).

This study has a minor deficiency in that haemostatic variables were not studied. However, when the study was initiated, haemostatic variables like raised fibrinogen levels[18,19] and increased plasma levels of a rapid inhibitor of t-PA,[20] and their role in CHD were not fully appreciated.

An important implication of our results is that personal factors such as hypertension, hypercholesterolaemia and smoking emerge as major risk factors for CHD in the white population of Durban. Our data, together with other data like the CORIS study,[4] justify the implementation of educational and interventional programmes in the white population of South Africa, particularly at younger ages.

We wish to thank Sr Mary Veale for her meticulous work in this study. This research project was financially supported by the South African Medical Research Council.

REFERENCES

1. Wyndham CH. Mortality from cardiovascular diseases in the various population groups in the Republic of South Africa. *S Afr Med J* 1979; **56:** 1023-1030.
2. South African Medical Research Council. *Review of South African Mortality (1984)* (Technical Report No. 1). Parowvallei, CP: MRC, 1987.
3. Balfe DL, Steinberg WJ, Kustner HGV. Comparison of the decline in the ischaemic heart disease mortality rate in the RSA with that in other countries. *S Afr Med J* 1988; **74:** 551-553.
4. Rossouw JE, Du Plessis JP, Benade AJS, *et al.* Coronary risk factor screening in three communities: the CORIS baseline study. *S Afr Med J* 1983; **64:** 430-436.
5. Seedat YK, Mayet FGH, Khan S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; **78:** 447-454.
6. *Central Statistical Services Population Census* 1985 (Report No. 02/85/01). Pretoria: Government Printer, 1986.
7. Rose GA, Blackburn H, Gillum RF, Prineas RJ. *Cardiovascular Survey Methods.* 2nd ed. Geneva: World Health Organisation, 1982.
8. Bortner RW. A short rating scale as a potential measure of pattern A behaviour. *J Chron Dis* 1969; **22:** 87-91.
9. American Heart Association. *Recommendations for Human Blood Pressure Determination by Sphygmomanometers.* Dallas: AHA, 1980.
10. Prineas RJ, Crow RS, Blackburn H. *The Minnesota Code Manual of Electrocardiographic Findings.* Boston: John Wright, 1982.
11. World Health Organisation. Diabetes mellitus. *WHO Tech Rep Ser* 1985; No. 727: 9-12.
12. Pooling Project Research Group. Relationship of blood pressure, serum cholesterol, smoking habits, relative weight and ECG abnormalities to incidence of major coronary events: final report of the Pooling Project. *J Chron Dis* 1978; **31:** 201-306.
13. Seedat YK, Seedat MA, Veale M. The prevalence of hypertension in urban whites. *S Afr Med J* 1980; **57:** 1025-1030.
14. Seedat YK, Mayet FGH, Latiff GH, Joubert G. Risk factors and coronary heart disease in Durban blacks — the missing links. *S Afr Med J* 1992; **82:** 521-526.
15. Steyn K, Jooste PL, Bourne L, *et al.* Risk factors for coronary heart disease in the black population of the Cape Peninsula: the BRISK study. *S Afr Med J* 1991; **79:** 480-485.
16. Steenkamp E, Jooste PL, Christopher KJ. The prevalence of smoking and its relationship to other coronary risk factors in a rural white community. *S Afr Med J* 1988; **73:** 402-405.
17. Yach D. Economic aspects of smoking in South Africa. *S Afr Med J* 1982; **62:** 167-170.
18. Meade TW, Brozovic M, Chakrabarti RR, *et al.* Haemostatic function and ischaemic heart disease: principal results of the Northwick Park heart study. *Lancet* 1986; **2:** 533-537.
19. Kannel WB, Wolf PA, Castelli WP, D'Agostino RB. Fibrinogen and risk of cardiovascular disease. *JAMA* 1987; **258:** 1183-1186.
20. Hamsten A, Wiman B, de Faire U, Blomback M. Increased plasma levels of a rapid inhibitor of tissue plasminogen activator in young survivors of myocardial infarction. *New Engl J Med* 1985; **313:** 1557-1563.