

Coronary risk factor screening in three rural communities

The CORIS baseline study

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Summary

A three-community study of rural Afrikaans-speaking Whites in the south-western Cape revealed that the major reversible risk factors hypercholesterolaemia, hypertension and smoking, as well as 'minor' factors such as inactivity, obesity, hyperuricaemia, coronary-prone behaviour and the irreversible risk factors of chest pain, ischaemic changes on the ECG and a family history of ischaemic heart disease (IHD), 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. The interaction of high levels of lifestyle-induced risk factors with constitutional predisposition could adequately explain any excess risk of IHD in the Afrikaans-speaking community. The almost universal risk factor prevalence in this study has major implications for any preventive strategy.

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The high incidence of ischaemic heart disease (IHD) among White South Africans has been extensively documented by Wyndham,¹ using death certification data. There is an impression among health professionals that the Afrikaans-speaking and Jewish segments of the White population are especially prone to the disease, but this has been difficult to confirm because of the lack of language and cultural information on death certificates. In districts where more than 80% of the White population are Afrikaners, rates of deaths from IHD in males under the age of

50 years and in females under 55 years are higher than those for the rest of the country (see the article by J. P. G. Pretorius on p. 427 of this issue). The reasons for this excess of IHD are unknown. *A priori* they must lie in an environmental or genetic predisposition to the disease. Seftel's work on the Witwatersrand has indicated strongly that Afrikaners from that locality have a high prevalence of familial hypercholesterolaemia (FH).² A suggestion, based on heterozygote frequency in this population, that as many as 1 in every 10 young Afrikaners with myocardial infarcts may have FH has been borne out by preliminary data from Johannesburg (J. Slack and C. H. Wyndham — personal communication). If confirmed elsewhere in the RSA, these data would indicate that FH makes an important contribution to IHD. However, it would not explain the still high incidence of deaths from IHD in the residue of Whites (Afrikaners and others) without FH, and it is likely to be less important in older subjects.

A high prevalence of the classic IHD risk factors would offer an alternative explanation. Risk factors such as hypercholesterolaemia, hypertension, smoking, obesity, hyperuricaemia and coronary-prone behaviour patterns are largely environmentally induced and are therefore potentially reversible. They would be expected to interact with any constitutional predisposition to aggravate the risk of IHD.

This report concerns the first attempt to quantify IHD risk factors in an Afrikaans-speaking community. The Coronary Risk Factor Study (CORIS) was launched jointly by the South African Medical Research Council, the Department of Health and Welfare and the Human Sciences Research Council in 1978. Its aims were to establish the prevalence and intensity of risk factors in an Afrikaner community and to evaluate the effectiveness of intervention towards lowering risk factors. A baseline three-community study was to be followed by two levels of intervention intensity in two of the communities, with the third acting as control. The Stanford Study served as model.³ The duration of the study was left open, but the first large-scale evaluation of effectiveness was planned to follow 3 years of intervention and is currently under way.

The study population

During the first half of 1979 a community coronary risk factor survey was carried out in the magisterial districts of Swellendam (White population 5 860 in the 1980 census), Riversdale (5 540) and Robertson (5 320). All three towns are situated in the south-western Cape Province and are similar in cultural and socio-economic structure. Mortality rates from IHD in economically active White males averaged 214/100 000 (85/100 000 for females) in the three towns in 1970. The national figure for White South African males was 240/100 000 (72/100 000 for females) in that year (J. P. G. Pretorius — personal communication). By means of an intensive postal campaign, 3 357 White males and 3 831 White females between the ages of 15 and 64 years were

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recruited. They represented 82% of the known target population between these age limits as ascertained from ratepayers' and electricity consumers' records and a postal census, and 68% of the 1980 census population aged 15 - 64 years. Sixty-three per cent of the population lived in town, the remainder living on farms or smallholdings. One-third of the males earned a living in the agricultural sector (Table I). More than 90% of the White inhabitants were Afrikaans-speaking. The age and sex distribution are shown in Table II, together with the age distribution of the White population of the RSA and the study area population aged between 15 and 64 years as estimated during the 1980 general census. Both the CORIS sample and the study area population have an almost rectangular age distribution, which differs considerably from national figures. Ascertainment, as judged from 1980 census figures, was uniformly lower among males (63%) than among females (73%). Comparisons of educational qualifications and occupations between the CORIS sample and the 1980 census of the study area show close agreement.

TABLE I. OCCUPATIONAL CATEGORIES OF POPULATION

| | Males (%) | Females (%) |
|-------------------------------|--------------|----------------|
| Professional and technical | 8,6 | 6,6 |
| Management and administration | 9,7 | 2,2 |
| Clerical | 6,2 | 12,4 |
| Sales | 3,2 | 2,4 |
| Agriculture | 32,3 | 0,3 |
| Transport and communication | 3,9 | 0,5 |
| Services | 5,4 | 1,3 |
| Tradesmen | 7,2 | 0,7 |
| Operators and labourers | 1,3 | 0 |
| Retired | 9,3 | 0,8 |
| Housewives | 0 | 63,3 |
| Scholars and students | 12,7 | 9,5 |

Methods

Respondents completed a risk factor questionnaire and a London School of Hygiene questionnaire for chest pain⁴ by interview in a study centre set up in each community. The interviewers were dietitians who had been intensively trained in the standardized administration of the questionnaire. The questionnaire covered socio-economic items, smoking habits, a family history of IHD, personal medical history and activity patterns, as well as items on risk factor knowledge, attitudes and actions. All respondents completed a self-administered Bortner Short Rating Scale⁵ for coronary-prone (type A) behaviour. A 12% random subsample also completed a 24-hour dietary recall by interview.

A resting 12-lead ECG was recorded in the recumbent position on a Hewlett-Packard Model No 1516 Tape Terminal for later playback and coding according to the Minnesota criteria.⁶ The coding was done in two stages and at each stage two independent blind observers were used, with an arbitrator for disagreements.

Height was measured to the nearest 0,5 cm with a rigid rod anthropometer and mass was measured on a beam balance in light clothing and without shoes. Skinfolts at four sites (biceps, triceps, subscapular and supra-iliac) were measured with Harpenden skinfold calipers.

Blood pressures were measured after subjects had been seated for 5 minutes. A standard 12,5 x 23-cm cuff connected to a mercury manometer was used. The American Heart Association guidelines for measuring blood pressure⁷ were followed. The diastolic pressure was taken as the point of muffling of the Korotkoff sounds (phase IV). Readings were taken three times and the lowest reading was recorded. Only three observers were used, whose readings had been standardized against those of an experienced clinician (J.E.R.). Standardization was checked at weekly intervals and interobserver variation never exceeded 5%. Subsequent analysis of the data failed to show evidence of interobserver variation or of end-digit preference.

Non-fasting blood samples were taken with minimal stasis into a plain evacuated glass container and allowed to clot at room temperature. Serum was separated within 2 hours and analysed manually on the same day for serum cholesterol and high-density lipoprotein (HDL) cholesterol by means of the Boehringer CHOD-PAP enzymatic method and dextran sulphate-magnesium chloride precipitation. Reference standards (Ortho Diagnostics and Boehringer Precilip) were included in each run. Inter- and intra-run variation was less than 3%. Split samples assayed by the Lipid Research Clinics (LRCs) in the USA (by courtesy of Dr B. Rifkind) showed that our laboratory under-reads by approximately 10% compared with the automated colorimetric method used by the LRCs, which is in turn standardized to the Centers for Disease Control, Atlanta. The remaining serum was frozen at -20° for later determination of uric acid by the Boehringer Urica-quant method.

Interpretation of data

The prevalence of the major reversible risk factors (hypercholesterolaemia, hypertension and smoking) is considered at two levels of severity. The conventional cut-off point for hypercholesterolaemia is 6,5 mmol/l (250 mg/dl), but prospective studies⁸ have repeatedly shown that the risk of coronary heart disease (CHD) increases after 5,7 mmol/l (220 mg/dl). Both these levels are considered in this presentation. Similarly the World Health Organization⁹ definition of hypertension ($\geq 160/95$ mmHg) is commonly used, but the risk of CHD starts rising after 120/80

TABLE II. AGE AND SEX DISTRIBUTION OF WHITES IN THE RSA, IN THE STUDY AREAS, AND IN THE CORIS POPULATION SAMPLE

| Age (yrs) | % of total | | | No. of respondents | |
|--------------|------------|-----------------|-----------------|--------------------|---------|
| | RSA* | Study areas* | CORIS sample | Males | Females |
| 15 - 24 | 27,4 | 22,8 | 18,0 | 635 | 658 |
| 25 - 34 | 25,1 | 16,2 | 18,7 | 634 | 713 |
| 35 - 44 | 20,7 | 20,2 | 20,2 | 641 | 813 |
| 45 - 54 | 14,9 | 20,0 | 21,6 | 705 | 845 |
| 55 - 64 | 11,9 | 20,8 | 21,5 | 742 | 802 |
| Total | 100,0 | 100,0 | 100,0 | 3 357 | 3 831 |

*From the 5% subsample of the 1980 general census.

mmHg and is higher than average after 140/90 mmHg.⁸ The WHO defines blood pressures above 140/90 mmHg but below 160/95 as 'borderline hypertension'. Both WHO cut-off points are considered here. It should be noted that the WHO cut-off points (and most prospective studies) make use of phase V diastolic pressures, whereas this study used phase IV, which in our hands gave a reading averaging 6 mm higher than phase V. Smoking more than 10 cigarettes per day is accepted as a CHD risk factor, but the status of light smokers, pipe smokers and past smokers is less clear. The Pooling Project⁸ found pipe or cigar smokers and past smokers to be at slightly higher risk than those who smoked less than 10 cigarettes per day, but the latter category was known to include a sizeable proportion of individuals who smoked occasionally, or who smoked one or two cigarettes per day only. For the present purposes smokers are therefore considered at the level of 10 or more cigarettes per day, as well as at the level of any regular cigarette or pipe smoking, excluding occasional (less than once per day) smokers.

The body mass index (BMI) (weight/height²) was used in preference to relative weight, since the former is independent of external standards. The cut-off points for 'obesity' and for 'overweight' were those advocated by Bray.¹⁰ The Bortner Short Rating Scale⁵ used for the assessment of coronary-prone (type A) behaviour was modified to yield 12 seven-point bipolar scales for characteristics such as competitiveness, impatience and striving. Those who fell in the upper two-fifths of the possible total score of 12 - 84, i.e. 55 points or more, were arbitrarily classified as exhibiting type A behaviour.¹¹

Inactivity at work was categorized according to the data of Paffenberger and Hale¹² on longshoremen, which suggests that an energy expenditure of approximately 6 300 kJ (1 500 kcal) per day above basal is associated with a lower risk of CHD. For the purpose of this study, a weekly work expenditure of 32 300 kJ (7 700 kcal) was taken as the closest 'threshold' value (T. D. Noakes *et al.* — unpublished data). For leisure-time activity a

weekly 'threshold' of 8 400 kJ (2 000 kcal) was used, based on Paffenberger *et al.*'s findings in college alumni.¹³

For hyperuricaemia the manufacturer of the enzymatic kit (Boehringer) gives a normal upper value of 416 μ mol/l in males and 339 μ mol/l in females, and these were accepted for the present purposes.

Electrocardiographic findings suggestive of IHD were identified by the strength of their association with a history of chest pain and with age.¹⁴ In males suggestive findings included large and medium Q waves (Minnesota codes 1.1, 1.2), large and medium ST-segment depressions (4.1, 4.2), large and medium T-wave inversions (5.1, 5.2) and left and right bundle-branch blocks (7.1, 7.2). In females, Q waves, T waves and large ST depressions were considered similarly suggestive of IHD, but medium ST depressions and bundle-branch blocks were not. Left ventricular hypertrophy (3.1) was considered positive for females but not for males.

Since the CORIS population distribution differs from that of South African Whites as a whole, crude and age-adjusted prevalence figures are given.

Results

Selected risk factors stratified by age decile and sex are described in Table III. Serum total cholesterol levels rose with age in both males and females, but after reaching a mean peak (\pm SD) of 6.39 \pm 1.27 mmol/l in males aged 45 - 54 years levels declined slightly by the ages of 55 - 64 years. In females the peak was higher (7.16 \pm 1.30 mmol/l) and was reached in the highest age decile of 55 - 64 years. It is noteworthy that, except in the young, mean serum cholesterol exceeded the level of 5.7 mmol/l which is associated with an increased risk of IHD. With the exception of the age range 25 - 44 years, females had higher serum total cholesterol levels than males, largely because of their higher HDL cholesterol

TABLE III. DESCRIPTIVE STATISTICS OF SELECTED RISK FACTORS*

| | Age groups (yrs) | | | | | | | | | |
|----------------------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| | Males | | | | | Females | | | | |
| | 15-24 (635) | 25-34 (634) | 35-44 (641) | 45-54 (705) | 55-64 (742) | 15-24 (658) | 25-34 (713) | 35-44 (813) | 45-54 (845) | 55-64 (802) |
| Serum total cholesterol (mmol/l) | 4.56 \pm 0.99 | 5.72 \pm 1.31 | 6.16 \pm 1.34 | 6.39 \pm 1.27 | 6.34 \pm 1.16 | 4.96 \pm 0.97 | 5.52 \pm 1.18 | 5.87 \pm 1.25 | 6.62 \pm 1.46 | 7.16 \pm 1.30 |
| HDL cholesterol (mmol/l) | 1.14 \pm 0.24 | 1.13 \pm 0.27 | 1.12 \pm 0.29 | 1.14 \pm 0.31 | 1.15 \pm 0.32 | 1.35 \pm 0.27 | 1.41 \pm 0.30 | 1.41 \pm 0.37 | 1.46 \pm 0.40 | 1.42 \pm 0.39 |
| Systolic blood pressure (mmHg) | 124.7 \pm 12.4 | 130.4 \pm 12.2 | 133.5 \pm 16.3 | 140.2 \pm 20.2 | 149.0 \pm 23.5 | 120.1 \pm 11.1 | 123.5 \pm 12.6 | 131.4 \pm 17.5 | 144.0 \pm 22.4 | 154.7 \pm 25.3 |
| Diastolic blood pressure (mmHg) | 75.3 \pm 9.2 | 83.5 \pm 9.5 | 87.7 \pm 11.2 | 90.0 \pm 11.7 | 91.3 \pm 12.0 | 76.1 \pm 9.1 | 80.1 \pm 8.9 | 85.4 \pm 10.2 | 90.5 \pm 11.7 | 92.2 \pm 12.2 |
| Smokers (%)† | 34.3 | 53.1 | 57.5 | 51.1 | 42.6 | 11.1 | 22.5 | 23.4 | 18.7 | 11.1 |
| Cigarette smokers (%)† | 32.9 | 49.3 | 50.2 | 43.0 | 32.6 | 11.1 | 22.5 | 23.4 | 18.7 | 11.1 |
| Past cigarette smokers (%) | 6.5 | 17.5 | 25.2 | 30.6 | 38.1 | 2.4 | 6.9 | 7.9 | 9.1 | 10.1 |
| Cigarettes/d | 11.1 \pm 7.8 | 18.1 \pm 9.6 | 20.0 \pm 11.7 | 20.6 \pm 11.7 | 18.9 \pm 11.4 | 10.9 \pm 7.5 | 13.6 \pm 8.2 | 15.4 \pm 9.1 | 15.7 \pm 9.3 | 15.8 \pm 10.4 |
| BMI (wt/ht ²) | 22.9 \pm 3.3 | 25.9 \pm 3.7 | 26.4 \pm 3.6 | 27.2 \pm 4.1 | 27.0 \pm 4.0 | 22.8 \pm 3.6 | 23.2 \pm 4.6 | 25.7 \pm 5.1 | 27.4 \pm 5.6 | 28.1 \pm 5.1 |
| Type A behaviour (Bortner) | 52.0 \pm 7.8 | 54.2 \pm 9.3 | 54.0 \pm 9.5 | 52.1 \pm 10.3 | 50.2 \pm 10.2 | 51.5 \pm 8.5 | 53.0 \pm 9.2 | 52.6 \pm 10.0 | 51.3 \pm 9.9 | 49.1 \pm 10.2 |
| Serum uric acid (μ mol/l) | 364 \pm 134 | 367 \pm 83 | 362 \pm 90 | 359 \pm 96 | 374 \pm 115 | 271 \pm 72 | 258 \pm 87 | 265 \pm 79 | 288 \pm 87 | 313 \pm 95 |

*Values expressed as mean \pm SD, where appropriate.

†Includes pipe smokers but excludes occasional smokers (<1/d).

levels. Unlike total cholesterol, HDL cholesterol levels showed little trend with age in the range under study.

Mean systolic and diastolic blood pressures rose with age, females eventually having higher pressures than males. Mean blood pressures in both sexes exceeded the level of 'borderline' hypertension associated with an increased risk of CHD after the age of 44 years.

Cigarette smoking was more than twice as common among males than among females at all ages, and the mean daily cigarette consumption was also higher. In both sexes the prevalence of cigarette smoking was highest in the age groups 35 - 44 years (50,2% of males v. 23,4% of females) and declined thereafter. In this age group 75% of men and 30% of women were either current or past cigarette smokers. Among men over 34 years old past cigarette smokers had an inverse relationship to present cigarette smokers, possibly indicating a trend for those past middle age to give up the habit. Pipe-only smokers made a substantial contribution to the total percentage of smokers in men, accounting for up to 10% at the age of 55 - 64 years. Regular cigar-only smokers were infrequent and were not included in the above figures.

Mean BMIs were similar in men and women, rising from approximately 23 at 15 - 24 years to 28 - 29 at 55 - 64 years. In the higher age groups the mean BMI exceeded the 'overweight' cut-off points in both sexes. Mean Bortner scores showed little age trend. Serum uric acid levels did not show any marked age

trend apart from a slight rise in the highest female decile. Uric acid levels were higher in males than females.

The prevalence of the major reversible risk factors (hypercholesterolaemia, hypertension and smoking) is given in Table IV. Even using level A conventional cut-off points (total cholesterol $\geq 6,5$ mmol/l, blood pressure $\geq 160/95$ mmHg, smoking ≥ 10 cigarettes per day) the major risk factors were commonly present, with hypercholesterolaemia in 30,1% of males and 34,4% of females, hypertension in 22,1% of males and 22,3% of females, and cigarette smoking in 33,4% of males and 12,9% of females. At level B, using cut-off points which are based on prospective epidemiological data (total cholesterol $\geq 5,7$ mmol/l, blood pressure $\geq 140/90$ mmHg, any regular smoking), each of the major risk factors was very common, with the exception of smoking in women. According to these criteria over 50% of all subjects had hypercholesterolaemia, over 40% had hypertension and almost half of the males were smokers. Because of the strong age trends already referred to above, the prevalences of major risk factors were especially high in subjects over 44 years old; almost 70% of men and over 80% of women in the two highest age deciles combined had hypercholesterolaemia ($\geq 5,7$ mmol/l), and over 60% of men and 70% of women had hypertension ($\geq 140/90$ mmHg).

Since risk factors exert their influence independently of each other, it is interesting to note that even at the more liberal level A cut-off points 59,4% of all men and 50,5% of all women had one

TABLE IV. PREVALENCES (%) OF THE MAJOR REVERSIBLE RISK FACTORS HYPERCHOLESTEROLAEMIA, HYPERTENSION AND SMOKING*

| | Age groups (yrs) | | | | | | | | | | | |
|-------------------------------------------|------------------|----------------|----------------|----------------|----------------|------------------|----------------|----------------|----------------|----------------|----------------|------------------|
| | Males | | | | | | Females | | | | | |
| | 15-24 (635) | 25-34 (634) | 35-44 (641) | 45-54 (705) | 55-64 (742) | 15-64† (3357) | 15-24 (658) | 25-34 (713) | 35-44 (813) | 45-54 (845) | 55-64 (802) | 15-64† (3831) |
| Serum cholesterol | | | | | | | | | | | | |
| A $\geq 6,5$ mmol/l | 4,7 | 22,2 | 35,8 | 42,7 | 41,8 | 30,1 (25,6) | 5,3 | 15,6 | 26,6 | 47,5 | 68,6 | 34,4 (26,1) |
| B $\geq 5,7$ mmol/l | 10,2 | 44,1 | 62,6 | 67,8 | 70,1 | 51,9 (45,3) | 18,8 | 36,9 | 50,4 | 74,9 | 89,4 | 56,0 (46,7) |
| Blood pressure | | | | | | | | | | | | |
| A $\geq 160/95$ mmHg | 1,9 | 11,7 | 22,4 | 30,2 | 40,6 | 22,1 (17,4) | 2,0 | 5,9 | 17,4 | 34,0 | 46,3 | 22,3 (16,2) |
| B $\geq 160/95$ mmHg and/or on treatment | 1,9 | 12,1 | 22,8 | 32,1 | 43,4 | 23,3 (18,2) | 2,1 | 6,5 | 18,7 | 38,8 | 53,7 | 25,3 (18,3) |
| B $\geq 140/90$ mmHg | 10,7 | 32,6 | 44,7 | 57,6 | 67,0 | 43,6 (37,0) | 7,4 | 17,1 | 36,3 | 62,4 | 78,4 | 42,4 (32,5) |
| Smoking | | | | | | | | | | | | |
| A ≥ 10 cigarettes/d | 18,1 | 42,4 | 43,4 | 37,1 | 26,8 | 33,4 (33,3) | 6,2 | 16,3 | 18,2 | 14,4 | 8,4 | 12,9 (12,7) |
| B† | 34,3 | 53,1 | 57,5 | 51,1 | 42,6 | 47,6 (47,3) | 11,1 | 22,5 | 23,4 | 18,7 | 11,1 | 17,5 (17,7) |
| Combinations of major risk factors | | | | | | | | | | | | |
| A | | | | | | | | | | | | |
| One factor only | 22,4 | 41,9 | 40,5 | 41,1 | 42,3 | 37,9 (36,2) | 12,6 | 26,0 | 35,0 | 38,8 | 45,6 | 32,5 (28,5) |
| Two factors | 0,9 | 13,9 | 22,1 | 22,7 | 26,5 | 17,7 (14,9) | 0,5 | 5,5 | 11,6 | 25,6 | 36,2 | 16,8 (12,1) |
| Three factors | 0,3 | 2,2 | 5,6 | 7,8 | 4,6 | 4,2 (3,5) | 0,0 | 0,3 | 1,4 | 2,1 | 1,7 | 1,2 (0,9) |
| Total with one or more factors | 23,6 | 58,0 | 68,2 | 71,6 | 73,4 | 59,4 (54,6) | 13,1 | 31,8 | 48,0 | 66,5 | 83,5 | 50,5 (41,4) |
| B | | | | | | | | | | | | |
| One factor only | 37,2 | 39,5 | 33,2 | 29,2 | 27,0 | 32,9 (34,6) | 29,0 | 36,8 | 40,6 | 32,9 | 23,6 | 32,6 (33,3) |
| Two factors | 7,4 | 29,8 | 39,4 | 40,9 | 49,1 | 34,0 (29,6) | 4,0 | 15,9 | 26,0 | 48,4 | 67,2 | 33,9 (25,7) |
| Three factors | 1,1 | 10,2 | 17,6 | 21,8 | 18,2 | 14,1 (11,9) | 0,2 | 2,7 | 5,9 | 8,8 | 7,0 | 5,2 (4,1) |
| Total with one or more factors | 45,7 | 79,5 | 90,2 | 91,9 | 94,3 | 81,0 (76,1) | 33,2 | 55,4 | 72,5 | 90,1 | 97,8 | 71,1 (63,1) |

The major risk factors are considered at two levels. At level A the cut-off points are set at conventional (higher) levels, while level B cut-off points approximate the (lower) levels found to be associated with excess risk in the Pooling Project.⁸

†For all age groups combined, crude and age-adjusted (in brackets) prevalences are given.

‡Includes pipe smokers, but excludes occasional smokers (<1/d).

or more of the major risk factors. Over 70% of subjects over 44 years old had one or more of the major factors. At the more stringent level B 81,0% of men and 71,1% of women had major risk factors, including over 90% of those more than 44 years old. Although relatively few subjects had all three risk factors, an appreciable proportion had two (17,7% at level A and 34% at level B).

Certain of the 'minor' risk factors (inactivity, type A behaviour pattern, obesity, hyperuricaemia and oral contraceptive use) are presented in Table V, together with certain non-reversible risk factors (history of chest pain, electrocardiographic evidence of ischaemia and a family history of IHD). The 'minor' risk factors (so called because of a lesser predictive ability for IHD or less independent power, or because further research is needed to establish them firmly) were also very common. Less than 2% of all subjects at any age could expect some protection from IHD because of a high level of physical activity at work, and less than 25% of men and less than 10% of women had 'protective' levels of recreational physical activity. Only males aged 15 - 24 years appeared to make some attempt towards a high level of leisure-time physical activity, and even in this group only 36% reached 'protective' levels.

Type A behaviour (as defined) was present in about 40% of all subjects, and in both sexes the peak prevalence was at age 25 - 34. Type A behaviour was less prevalent in younger subjects (15 - 24 years) and in those over 54 years, and was less prevalent in women than in men. Obesity (BMI ≥ 30) increased progressively with age, and was especially common in women over 54 years old (31,7%). Less than 5% of the young (< 25 years) were obese, but 57% of all subjects were overweight. Hyperuricaemia was found in approximately 25% of males and 20% of females. Oral contraceptives were used by 8,2% of women overall, with a maximum of 21,8% in those aged 25 - 34 years.

Of the non-reversible risk factors, a history of chest pain (possible angina or infarct) was present in approximately 8% of subjects overall. An ECG suggestive of IHD was present in 13,2% of males and 7,2% of females. A history of fatal or non-fatal IHD in first-degree relatives was obtained from approximately 40% of all subjects, and 11% had a history of IHD in first-degree relatives aged under 50 years.

Consideration of combinations of the most potent reversible and non-reversible risk factors provides the information depicted in Fig. 1. Even with the three major reversible risk factor cut-off points set at level A, one or more of these three factors, a positive family history or personal evidence of IHD was present in 73,5% (68,2% after age adjustment) of males and in 67,1% (59,8% after age adjustment) of females. The majority (32%) had only a single factor, but over 20% had any two and 12% had any three.

Discussion

The overall picture that emerged from this survey was disturbing. The major reversible factors were extremely common, with the possible exception of cigarette smoking in women. Taken together and using conservative criteria, the majority of the study population had one or more major risk factors. Using more realistic (lower) level B criteria, less than 20% of men and 30% of women did not exhibit either hypercholesterolaemia, hypertension or smoking. If some of the more important non-reversible risk factors such as evidence of existing IHD or a family history of IHD are added, the picture is indeed grim (Fig. 1). It is difficult to escape the conclusion that the study communities are at extremely high risk of IHD, few individuals being completely risk-free. Individuals with more than one risk factor make up a significant proportion of the study communities, and would be at even higher risk because of risk factor synergism.¹⁵

TABLE V. PREVALENCES (%) OF MINOR REVERSIBLE RISK FACTORS AND OF NON-REVERSIBLE RISK FACTORS

| Reversible risk factors | Age groups (yrs) | | | | | | | | | | | |
|-----------------------------------------------------------------|------------------|----------------|----------------|----------------|----------------|------------------|----------------|----------------|----------------|----------------|----------------|------------------|
| | Males | | | | | | Females | | | | | |
| | 15-24 (635) | 25-34 (634) | 35-44 (641) | 45-54 (705) | 55-64 (742) | 15-64* (3357) | 15-24 (658) | 25-34 (713) | 35-44 (813) | 45-54 (845) | 55-64 (802) | 15-64* (3831) |
| Inactivity | | | | | | | | | | | | |
| Work < 32 300 kJ/wk | 99.5 | 97.5 | 98.8 | 97.9 | 99.1 | 98.5 (98.6) | 100.0 | 99.9 | 99.9 | 99.8 | 99.9 | 99.9 (99.9) |
| Leisure < 8 400 kJ/wk | 64.1 | 78.0 | 80.2 | 83.0 | 78.3 | 76.9 (75.4) | 87.7 | 91.1 | 92.0 | 90.9 | 93.8 | 91.2 (90.7) |
| Type A behaviour | | | | | | | | | | | | |
| (Bortner ≥ 55) | 37.7 | 50.9 | 45.8 | 40.8 | 34.6 | 41.8 (42.9) | 33.8 | 44.4 | 43.2 | 36.9 | 30.1 | 37.6 (38.4) |
| Obesity (BMI ≥ 30) | 3.6 | 13.2 | 14.3 | 20.9 | 19.8 | 14.7 (12.7) | 4.6 | 10.5 | 15.6 | 23.8 | 31.7 | 18.0 (14.5) |
| Overweight (BMI ≥ 25 in males, ≥ 24 in females) | 20.6 | 55.3 | 63.7 | 70.9 | 68.8 | 56.6 (51.5) | 27.7 | 40.4 | 55.6 | 72.0 | 79.7 | 56.8 (49.5) |
| Hyperuricaemia (≥ 416 μmol/l in males, ≥ 339 μmol/l in females) | 20.6 | 23.8 | 24.2 | 25.2 | 28.9 | 24.7 (23.8) | 13.5 | 8.2 | 13.7 | 23.0 | 34.4 | 19.1 (16.1) |
| Oral contraceptive use | — | — | — | — | — | — | 9.1 | 21.8 | 10.0 | 1.8 | 0.1 | 8.2 (10.3) |
| Non-reversible risk factors | | | | | | | | | | | | |
| History of chest pain | 2.0 | 3.9 | 6.8 | 11.2 | 16.6 | 8.5 (6.6) | 2.1 | 2.8 | 5.5 | 9.9 | 16.6 | 7.7 (5.9) |
| ECG suggestive of IHD | 6.3 | 7.7 | 9.6 | 16.7 | 23.7 | 13.2 (11.0) | 3.3 | 3.9 | 5.2 | 8.6 | 13.6 | 7.2 (5.9) |
| Family history of IHD | 15.4 | 25.7 | 35.5 | 48.1 | 52.2 | 36.2 (29.8) | 13.8 | 30.8 | 43.7 | 52.5 | 61.1 | 41.8 (35.1) |
| Family history of IHD before age 50 | 9.4 | 8.8 | 9.5 | 15.3 | 9.3 | 10.5 (10.1) | 6.4 | 9.4 | 13.9 | 14.6 | 11.1 | 11.3 (10.5) |

*For all age groups combined, crude and age-adjusted (in brackets) prevalences are given.

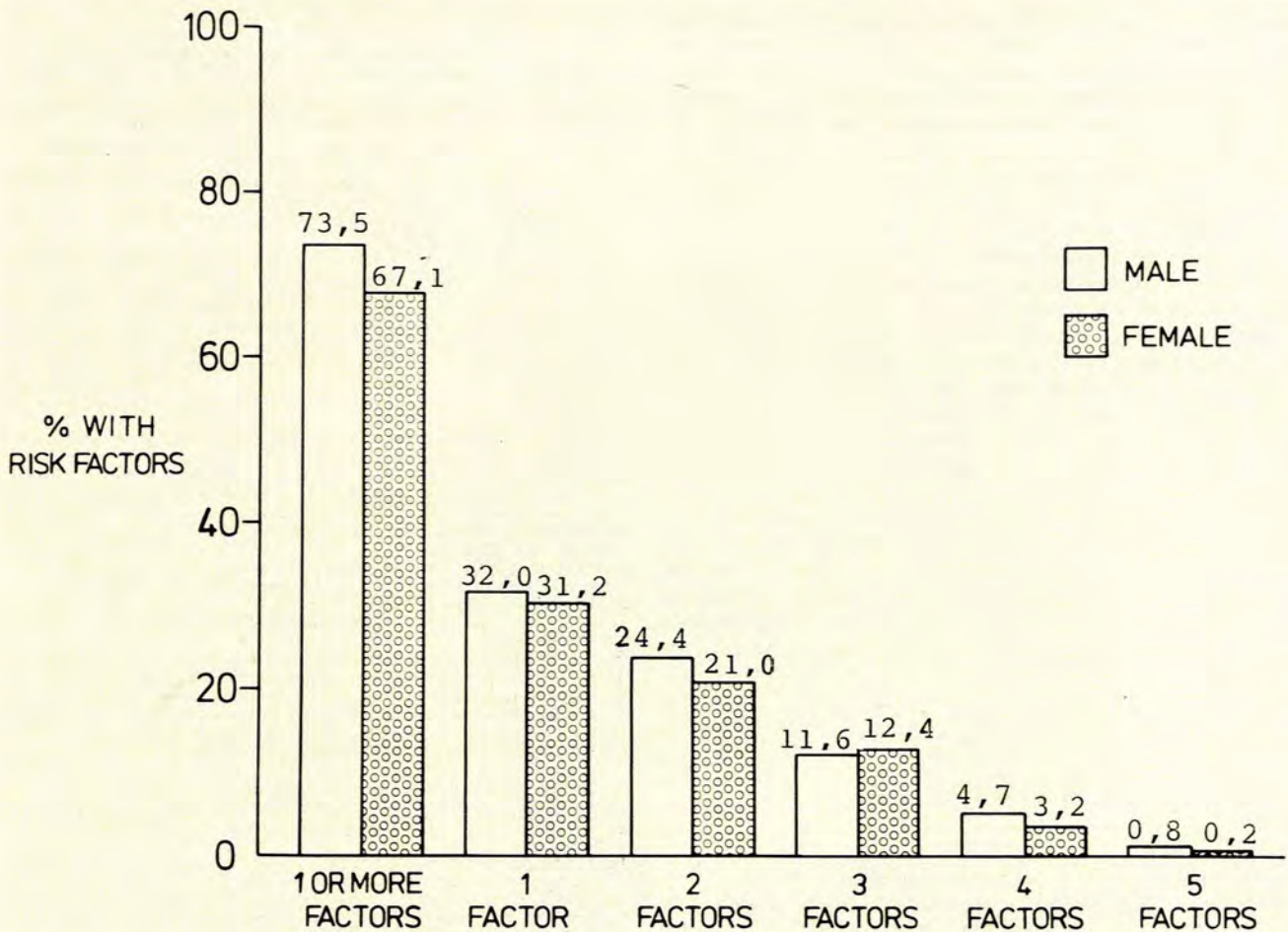


Fig. 1. Combination of the three major reversible risk factors, a family history of IHD and a history of chest pain and/or an ECG suggestive of IHD in males and females aged 15 - 64 years. (Note that the major risk factors considered are hypercholesterolaemia ($\geq 6,5$ mmol/l), hypertension ($\geq 160/95$ mmHg) and cigarette smoking (≥ 10 /d). Subjects with at least one first-degree relative with fatal or non-fatal IHD were considered to have a positive family history. See text for further details.)

This assessment of major risk prevalence is unlikely to be an overestimate in men, because the cut-off points used are based on experience gained from prospective studies in men aged between 40 and 64 years.⁸ Because of the strong age trend for total cholesterol, blood pressure and smoking, fewer men in the younger age groups were classified as having a risk factor. However, cut-off points for risk factors in the young, although not known, are likely to be lower than those applied to a middle-aged population.

Information on the power of risk factors in women is lacking, since the major prospective studies have been performed exclusively on men. However, it is almost certainly inappropriate to apply male total cholesterol cut-off points to women in view of the much higher HDL levels in the latter. A much lower rate of IHD in women in spite of similar risk factor levels (with the exception of smoking and use of the contraceptive pill) may also indicate that their cut-off points should be different, or of course it may be that other sex-related factors, not measured here, play a dominant role.

The major risk factor levels documented in this study appear to be higher than those currently pertaining in the USA, with the possible exception of smoking in women. For example, compared with Lipid Research Clinics Program data (1973 - 1976)¹⁶ the uncorrected mean serum cholesterol values of CORIS males were on average 0,8 mmol/l (30 mg/dl) higher. Corrected for interlaboratory differences, the gap widened to 1,30 mmol/l (50 mg/dl). Even larger discrepancies were found for females. Mean

systolic blood pressures in CORIS males and females were 5 - 13 mmHg higher than those of their rural age peers in the US National Health Survey (1971 - 1974).¹⁷ The age-adjusted hypertension rate found in this study (18%) is similar to that found by Seedat¹⁸ in Durban Whites (17%). In 1978, 37% of adult male Americans and 30% of females smoked cigarettes.¹⁹ In the CORIS population there were slightly more male (42%) and considerably fewer female (17%) cigarette smokers. Since there is good reason to believe that risk factor levels are currently declining in Whites in the USA,²⁰ these comparisons may be even more unfavourable than indicated above.

The high prevalence of major risk factors is compounded by similarly high prevalences of 'minor' factors such as obesity, hyperuricaemia and coronary-prone behaviour. The possible adverse effects of these are not counterbalanced by any potentially favourable factors such as physical activity. In fact, after the age of 24 levels of physical activity are remarkably low (Noakes *et al.* — unpublished data). It should therefore not be surprising that history-taking and electrocardiography revealed an excessively high prevalence of existing IHD in this population.¹⁴

It is not possible to be certain of the degree to which the results of this study are representative of the Afrikaans-speaking community. Certainly the study communities had no obviously unusual features, and they were not chosen because of a particularly high rate of CHD — in fact, their CHD rate is slightly below the national average for Whites. Nevertheless, it would be important to confirm these results in other Afrikaans-

speaking communities, both rural and urban, using standardized methodology and age adjustment. The opportunity to do so is fast disappearing in the wake of increasing public awareness of the South African IHD dilemma, which will almost certainly alter the lifestyle habits that largely determine risk factor levels. If the present findings are indeed found to be applicable to other White South Africans, they would go some way toward explaining our high IHD rate.

The debate as to whether measures aimed at prevention of CHD should be directed towards individuals at risk or towards the entire community carries on unabated (see the article by J. E. Rossouw on p. 437 of this issue). A strong body of opinion favours preventive measures only in individuals who have a major risk factor, obesity, or a family history of IHD.^{21,22} The proponents of this view argue that such an approach targets intervention on those most likely to benefit and avoids unnecessary treatment of the healthy segment of the population. While theoretically sound, this approach ignores certain important practical issues. Firstly the logistic difficulties and costs identifying those individuals at risk are enormous. Secondly, in an IHD-ridden community such as the one reported here the above definition of who is at risk is likely to include the vast majority of individuals making up that community. This implies that the community itself is at risk, and measures aimed at prevention should therefore be directed at the community rather than at the individual.

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