

Coronary heart disease risk factors in a rural and urban Orange Free State black population

W. F. Mollentze, A. J. Moore, A. F. Steyn, G. Joubert, K. Steyn, G. M. Oosthuizen, D. J. V. Weich

Objective. To determine and compare the prevalence of ischaemic heart disease (IHD) risk factors in a rural and an urban black population.

Design. A survey to determine the prevalence of hypertension, diabetes mellitus, smoking, obesity, central obesity and dyslipidaemia in black subjects 25 years and older.

Setting. The indigenous black populations of QwaQwa and Mangaung.

Participants. A random sample of 950 households was selected from each area. From each household an unrelated male and/or female subject was selected in a standardised way. From QwaQwa 853 subjects (279 men and 574 women) and from Mangaung 758 subjects (290 men and 468 women) participated in the study. The response rate was 68% and 62% respectively for QwaQwa and Mangaung.

Main outcome measures. Few urban-rural differences in the prevalence of IHD risk factors were found in this study. A low prevalence of clustering of major IHD risk factors was noted.

Results. The age- and sex-adjusted prevalences of hypertension were 29% in QwaQwa and 30,3% in Mangaung. Diabetes was present in 4,8% of the QwaQwa sample and 6% of the Mangaung sample. The prevalence of heavy smoking in the Mangaung sample was almost double that of the QwaQwa sample and mostly confined to men. High-risk hypercholesterolaemia was present in 12,5% of QwaQwa and 6% of Mangaung men in the

25 - 34-year age group. The corresponding figures for moderate-risk hypercholesterolaemia were 34% and 44,8% and both levels of risk declined with increasing age. The mean body mass index of women in both samples exceeded 25 kg/m².

Conclusion. All the elements for a potential epidemic of atherosclerotic cardiovascular disease are present in the study populations. The similarity of findings in the two samples may be indicative of the advanced stage of urbanisation and westernisation of the rural group. It is alarming that subjects in the younger age groups tended to have the highest prevalences of moderate and even high-risk hypercholesterolaemia.

S Afr Med J 1995; **85**: 90-96.

More than 75% of South Africa's population will be urbanised by the turn of the century and this process will affect mainly the black population.¹ With increasing urbanisation and the adoption of Western lifestyles and dietary habits, risk factor prevalence, morbidity and mortality from cardiovascular diseases in black subjects are expected to increase sharply in future. Hypertension is already common and severe in urban blacks in South Africa.^{2,3} The incidence rate for stroke in an urban black population was reported to be 1,01 per 1 000 per year with a peak of 10,31 per 1 000 per annum for men in the 65 - 74-year age group.⁴ In the same study hypertension was present in 69,8% of stroke patients.

IHD is still a rare condition in urban black South Africans with an estimated incidence of 10 per 100 000 for the population of Soweto.⁵ Accurate figures reflecting the actual prevalence of IHD in the black population are, however, lacking. Apart from a recent study in the Cape Peninsula⁶ no community-based data that describe the IHD risk factor profile of black South Africans are available.

A new dimension was recently added to the concept of IHD risk factors with the description of insulin resistance and the clustering of IHD risk factors such as hyperinsulinaemia, impaired glucose tolerance, increased plasma triglyceride (TG) and decreased high-density lipoprotein cholesterol (HDL) concentrations.⁷ To this was added the role of upper-body obesity in the pathogenesis of hyperinsulinaemia.⁸ The waist-to-hip circumference ratio (WHR) has already become firmly established as a simple measure of regional fat distribution and as an independent IHD risk factor.⁹

This study was undertaken because no information was available on the prevalence of IHD risk factors in urban and rural black populations of the Orange Free State, and because the metabolic consequences of central obesity have not been described in South African blacks. The methods, study populations and sampling procedures are presented in this paper. An overview of the coronary heart disease risk factor profile of these samples will also be provided.

Department of Internal Medicine and Division of Biostatistics, University of the Orange Free State, Bloemfontein

W. F. Mollentze, M.B. CH.B., M.MED. (INT.), F.C.P. (S.A.)

A. J. Moore, M.B. CH.B.

A. F. Steyn, M.B. CH.B., M.MED. (INT.)

G. Joubert, B.A., B.SC. (HONS)

G. M. Oosthuizen, PH.D.

D. J. V. Weich, M.B. CH.B., M.MED. (INT.), D.M., F.C.C.P.

Centre for Epidemiological Research in Southern Africa, Medical Research Council, Parowvallei, W. Cape

K. Steyn, M.SC., N.E.D., M.D.

Methods

Study populations and sampling procedures

QwaQwa with its population of 183 000 (1985 census) is the traditional homeland of the Sesotho-speaking people and was previously the most rural black population within the borders of the Orange Free State. QwaQwa is situated in the foothills of the Drakensberg and shares borders with Natal, Lesotho and the Orange Free State. In recent years urbanisation and industrialisation have affected this region profoundly and the capital, Phuthaditjhaba, has typical urban elements. Outside the city the majority of the population still follow a rural or partly rural lifestyle.

The sampling frame consisted of adults 25 years and older. All villages as well as Phuthaditjhaba were proportionally represented in the random sample based on the 1985 census population. Nine hundred and fifty households were randomly selected by means of maps in the case of Phuthaditjhaba and recent aerial photographs in the case of remote and rural areas. The sampling procedure was designed by Professor D. Stoker of the Human Sciences Research Council. An unrelated male and/or female respondent was selected from each household in a standardised way. A household was defined as a group of people who cook and eat together. Rural QwaQwa is divided into 12 tribal regions and permission was obtained from each tribal leader before we entered and worked in a particular area. The study commenced in June 1989 and was completed in December 1990.

The urban sample was drawn from Mangaung, the black residential area of Bloemfontein. The size of this population was reported to be 79 330 in the 1985 census. From maps of the area all the residential plots were counted and numbered and 950 housing units were randomly selected. An unrelated male and/or female respondent was also selected from each household in the same standardised way as in QwaQwa. This part of the study commenced in April 1990 and was completed in October 1991.

Measurements

The households identified were visited by two fieldworkers of whom at least one was a staff nurse. The study was explained and selected respondents were requested to attend a local clinic in the case of QwaQwa or the outpatient department at Pelonomi Hospital in the case of Mangaung; actual data collection took place at these locations. Subjects were requested to fast overnight. At the clinic or hospital the details of the study were again explained to the participants in their own language by a registered nurse and signed consent was obtained from all participants. Oral temperature was recorded and if abnormal, measurements were postponed. Basal blood samples were collected for the estimation of plasma glucose, total serum cholesterol (TC), HDLC, TG levels and a biochemistry profile. This was followed by a standard 75 g oral glucose tolerance test.¹⁰ Blood samples for plasma glucose were kept on ice until they could be spun down. Samples were stored at -20°C until the specimens could be analysed.

Subjects wearing only light indoor clothing were weighed on a Seca beam scale and their height measured on the same scale. A standardised clinical examination was performed and blood pressure was measured twice in the supine position by means of a Hawksley random zero sphygmomanometer and a standard 12,5 x 23 cm cuff. The diastolic pressure was taken as the point of disappearance of Korotkoff phase V sounds and the set of readings with the lowest diastolic reading was used for analysis. When the mid-upper arm circumference exceeded 35 cm a large-size cuff (15,5 x 32,5 cm) was used. In male subjects in the upright position the waist circumference was measured with a fibreglass tape measure to the nearest 0,5 cm at the level of the umbilicus at the end of expiration while the subject was breathing normally. In female subjects waist circumference was measured in the same way but at the narrowest point between the rib cage and the iliac crest. Hip circumference was measured at the level of the greater trochanter. Both measurements were taken in the horizontal plane. Mid-upper arm circumference was also measured to the nearest millimetre with the subject standing and the arm hanging down. The mid-upper arm position was determined as the halfway mark between the inferior acromial border and the tip of the olecranon process with the elbow flexed at 90° . All anthropometric measurements, clinical examinations and blood pressure measurements were performed by two doctors (W.F.M. and A.M.) after initial and periodic standardisation of techniques. Duplicate anthropometric measurements were recorded in every 10th respondent as a quality control measure.

A questionnaire was administered to collect data on demography, socio-economic status, physical activity, cardiorespiratory status, health perception, smoking and drinking habits as well as breast-feeding and contraception. Respondents were informed of abnormal clinical or laboratory findings and a referral letter was provided for participants in need of further medical attention.

Laboratory analysis

Plasma glucose levels were measured by the glucose oxidase peroxidase method. TC levels were measured by the cholesterol esterase oxidase peroxidase method (Boehringer Mannheim kit). HDLC was measured after precipitation of major lipoproteins by phosphotungstate-MgCl by the same method as for TC. Low-density lipoprotein cholesterol (LDLC) was calculated by means of the Friedewald formula.¹¹ Serum TG levels were determined by the lipoprotein lipase glycerokinase peroxidase method (Boehringer Mannheim kit). Assay precision for TC measurement had a coefficient of variation (CV) of 3 - 4%; HDLC a CV of 5 - 6% and TG a CV of 6 - 7%.

Statistical analysis

Frequencies and percentages were computed by age group and sex for categorical variables. Percentages were adjusted to the age/sex distribution of the target population. Means and standard deviations were calculated by age and sex for normally distributed continuous variables. Medians were calculated for continuous variables with skew distributions.

Results

In QwaQwa 853 respondents (279 men and 574 women) participated in the study with a response rate of 68%. In Mangaung 758 respondents (290 men and 468 women) participated in the study with a response rate of approximately 62%. There is no reason to believe that the socio-economic status of non-responders differed meaningfully from that of responders.

In Table I the composition of the 2 study samples is shown and compared with that of the actual population in each region. Older subjects were overrepresented, and younger subjects and men underrepresented in both samples.

The mean TC level for QwaQwa men in the 25 - 34-year age group was 4,7 mmol/l (Table II) and changed little with increasing age. The mean TC level for young Mangaung men was also 4,7 mmol/l but increased over the age deciles to 5,3 mmol/l in elderly (over 65 years) subjects. TC levels

for women in the 25 - 34-year age group in both populations were also identical at 4,4 mmol/l (Table III). A distinct increase in TC levels over the age deciles was evident in women from both populations and mean TC levels were 5,2 and 5,7 mmol/l respectively for QwaQwa and Mangaung women in the over 65-year age category. These exceeded the corresponding levels for men in both populations. An increase in LDLC was noted in middle-aged women in both populations, possibly related to peri- and postmenopausal hormonal changes.

HDLC levels were 1,2 and 1,4 mmol/l respectively for QwaQwa and Mangaung men in the 25 - 34-year age group and changed little over the increasing age deciles. This pattern was also evident in women. The HDLC/TC ratio for QwaQwa and Mangaung men ranged between 0,26 and 0,31 over the age deciles. The corresponding figures for women were 0,23 and 0,3 with a tendency to decrease over the age deciles for both populations.

Table I. Age and sex distribution of the populations (%)

	Age groups (yrs)					
	25 - 34	35 - 44	45 - 54	55 - 64	65+	25+
QwaQwa						
Sample (N = 853)						
Men (N = 279)	6,6	8,0	3,2	5,3	9,7	32,7
Women (N = 574)	16,6	12,9	11,6	12,0	14,2	67,3
Actual population						
Men	16,4	11,0	7,3	4,6	5,7	45,0
Women	18,1	12,1	7,8	8,1	8,9	55,0
Mangaung						
Samples (N = 758)						
Men (N = 290)	8,8	8,6	8,4	7,1	5,3	38,2
Women (N = 468)	13,9	16,6	13,9	9,0	8,4	61,8
Actual population						
Men	15,8	12,0	8,6	4,9	3,2	44,5
Women	19,7	15,0	9,9	5,8	5,1	55,5

Table II. Descriptive statistics (mean (SD)) of risk factors for IHD in men

	Age groups (yrs)				
	25 - 34	35 - 44	45 - 54	55 - 64	65+
QwaQwa					
TC (mmol/l)	4,7 (1,2)	4,9 (1,2)	4,6 (1,0)	4,6 (1,5)	4,7 (1,0)
HDLC (mmol/l)	1,2 (0,4)	1,3 (0,5)	1,2 (0,4)	1,3 (0,6)	1,2 (0,5)
HDLC/TC ratio	0,26 (0,09)	0,27 (0,11)	0,28 (0,11)	0,31 (0,12)	0,27 (0,11)
LDLC (mmol/l)	2,9 (1,1)	2,9 (1,0)	2,7 (1,0)	2,4 (0,8)	2,9 (1,0)
TG (mmol/l)	1,3 (0,9)	1,7 (1,4)	1,5 (1,0)	1,5 (1,0)	1,4 (0,7)
WHR	0,94 (0,07)	0,97 (0,07)	0,97 (0,07)	0,99 (0,07)	0,99 (0,11)
Diastolic BP (mmHg)	78 (14)	81 (15)	83 (13)	81 (10)	83 (14)
Systolic BP (mmHg)	129 (19)	133 (20)	135 (22)	141 (22)	149 (27)
No. of cigarettes smoked per day*	3	0	0	0	0
BMI (weight/height ²)	24,0 (4,7)	24,7 (5,2)	23,6 (5,8)	23,6 (4,6)	22,9 (4,5)
Mangaung					
TC (mmol/l)	4,7 (0,9)	4,9 (1,1)	5,1 (1,3)	5,1 (1,1)	5,3 (1,7)
HDLC (mmol/l)	1,4 (0,4)	1,3 (0,5)	1,4 (0,6)	1,4 (0,5)	1,4 (0,5)
HDLC/TC ratio	0,30 (0,09)	0,28 (0,12)	0,29 (0,12)	0,29 (0,11)	0,28 (0,13)
LDLC (mmol/l)	2,8 (0,9)	2,9 (1,1)	2,9 (1,3)	3,0 (0,9)	3,2 (1,6)
TG (mmol/l)	1,3 (0,9)	1,5 (1,3)	1,8 (1,9)	1,4 (0,7)	1,6 (1,1)
WHR	0,92 (0,05)	0,97 (0,07)	1,01 (0,08)	1,03 (0,10)	1,03 (0,08)
Diastolic BP (mmHg)	77 (13)	79 (11)	87 (14)	87 (15)	88 (13)
Systolic BP (mmHg)	128 (14)	128 (14)	142 (23)	146 (27)	160 (26)
No. of cigarettes smoked per day*	8	7	5	4	0
BMI (weight/height ²)	22,3 (3,2)	24,2 (4,5)	23,5 (5,7)	24,5 (6,3)	23,6 (5,4)

* Median.

Table III. Descriptive statistics (mean (SD)) of risk factors for IHD in women

	Age groups (yrs)				
	25 - 34	35 - 44	45 - 54	55 - 64	65+
QwaQwa					
TC (mmol/l)	4,4 (1,0)	4,6 (1,0)	4,8 (1,1)	5,4 (1,3)	5,2 (0,9)
HDLC (mmol/l)	1,2 (0,3)	1,2 (0,4)	1,2 (0,4)	1,2 (0,3)	1,2 (0,3)
HDLC/TC ratio	0,28 (0,08)	0,27 (0,09)	0,25 (0,1)	0,23 (0,08)	0,25 (0,08)
LDLC (mmol/l)	2,8 (1,0)	2,9 (0,9)	3,0 (1,1)	3,5 (1,2)	3,3 (0,9)
TG (mmol/l)	0,9 (0,4)	1,1 (0,6)	1,4 (0,7)	1,5 (0,8)	1,4 (0,6)
WHR	0,82 (0,09)	0,84 (0,1)	0,86 (0,1)	0,90 (0,09)	0,89 (0,09)
Diastolic BP (mmHg)	74 (15)	82 (14)	90 (17)	88 (15)	86 (19)
Systolic BP (mmHg)	125 (20)	137 (24)	148 (29)	150 (27)	160 (31)
No. of cigarettes smoked per day*	0	0	0	0	0
BMI (weight/height ²)	27,1 (5,5)	29,4 (6,6)	29,8 (7,8)	30,5 (7,3)	27,8 (8,0)
Mangaung					
TC (mmol/l)	4,4 (0,8)	4,9 (1,0)	5,5 (1,3)	5,3 (0,9)	5,7 (1,2)
HDLC (mmol/l)	1,3 (0,4)	1,4 (0,5)	1,3 (0,4)	1,4 (0,5)	1,4 (0,4)
HDLC/TC ratio	0,3 (0,08)	0,29 (0,1)	0,26 (0,09)	0,27 (0,1)	0,25 (0,09)
LDLC (mmol/l)	2,7 (0,7)	3,1 (1,0)	3,4 (1,3)	3,3 (0,9)	3,6 (1,1)
TG (mmol/l)	0,8 (0,4)	1,1 (0,6)	1,5 (1,5)	1,3 (0,6)	1,5 (0,9)
WHR	0,79 (0,06)	0,85 (0,09)	0,88 (0,1)	0,89 (0,09)	0,90 (0,09)
Diastolic BP (mmHg)	73 (13)	81 (13)	90 (16)	86 (14)	86 (19)
Systolic BP (mmHg)	125 (17)	135 (22)	154 (32)	157 (31)	169 (37)
No. of cigarettes smoked per day*	0	0	0	0	0
BMI (weight/height ²)	27,2 (6,2)	29,5 (7,0)	31,1 (7,2)	30,6 (9,0)	29,5 (7,7)

* Median.

Fasting TG levels peaked at 1,7 mmol/l for QwaQwa men in the 35 - 44-year age group. For Mangaung men the peak value was 1,8 mmol/l in the 45 - 54-year age group. Fasting TG levels tended to increase over the age deciles for women in both populations.

WHR for men exceeded that for women and WHR also increased over the age deciles for both populations. The mean increase in WHR from the lowest to the highest age group in Mangaung men exceeded that in QwaQwa men (0,11 v. 0,05).

For both populations and sexes mean diastolic and systolic blood pressures increased with age. The rise in both diastolic and systolic blood pressures across the age deciles was greater in Mangaung men than in QwaQwa men (11 v. 5 mmHg for diastolic and 32 v. 20 mmHg for systolic). The prevalence of hypertension according to WHO guidelines¹² ($\geq 160/95$ mmHg and/or taking antihypertensive medication) was 29% for QwaQwa (age- and sex-adjusted for the QwaQwa population) and more women (34,5%) than men (22,1%) were hypertensive. The prevalence of hypertension in the Mangaung population (age- and sex-adjusted for the Mangaung population) was 30,3%; the corresponding figures for women and men were 36,3% and 22,8% respectively.

A striking difference was noted between the median number of cigarettes smoked by men in the 2 populations with Mangaung men being heavier smokers (Table II).

The mean body mass index (BMI) for men in both populations (Table II) was within the normal range (20 - 25 kg/m²). However, the mean BMI of women in both populations in all age deciles exceeded 25 kg/m² (Table III).

The prevalence of the major risk factors for coronary heart disease is shown in Tables IV and V. In the 25 - 34-year age group 12,5% and 6,0% of QwaQwa and Mangaung men respectively had TC levels in the high-risk category.¹³ The prevalences of moderate-risk hypercholesterolaemia in

25 - 34-year-old men were an alarming 34% and 44,8% for QwaQwa and Mangaung respectively. The prevalence of moderate and severe hypercholesterolaemia decreased with increasing age for both sexes and populations.

Protective HDLC/TC ratios tended to decrease over the age deciles for both sexes and populations. The prevalence of hypertriglyceridaemia in women increased sharply from the 45 - 54-year age group onwards. The prevalences of diabetes were 4,8% and 6,0% in the QwaQwa and Mangaung populations respectively and the condition was more frequent in men over 45 and women over 35 years.

The prevalence of high-risk hypertension ($\geq 160/95$ or on treatment for hypertension) increased sharply over the age deciles for both sexes and populations to peak at 43,4% and 60,0% among elderly QwaQwa and Mangaung men respectively. Corresponding figures for elderly QwaQwa and Mangaung women were 66,0% and 78,1%.

Smoking of more than 10 cigarettes a day was mostly confined to male subjects. The prevalence declined over the age deciles from 25% to 6,2% in QwaQwa men and from 46,3% to 10,3% in Mangaung men. Mangaung men were heavier smokers than QwaQwa men.

The presence of at least one risk factor in QwaQwa men ranged from 29,2% to 45% across the age decile groups. In Mangaung men the situation was even worse and ranged from 40% to 51,9%. In women the situation was strikingly different and the prevalence increased from 13% to 65,8% in QwaQwa women and from 19,1% to 73,4% in Mangaung women across the age deciles.

Obesity (BMI > 30 kg/m²) was very common among women and the prevalences ranged from 27,5% to 49,0% in QwaQwa women and from 31,1% to 54,3% in Mangaung women. The corresponding prevalence of obesity among QwaQwa men ranged between 7,4% and 19,2% and among Mangaung men from 3,0% to 20,4%.

Table IV. Prevalence (%) of IHD risk factors in the male population of QwaQwa and Mangaung

	Age group (yrs)									
	QwaQwa					Mangaung				
	25 - 34	35 - 44	45 - 54	55 - 64	65+	25 - 34	35 - 44	45 - 54	55 - 64	65+
Hypercholesterolaemia — high-risk category*	12,5	6,0	0	2,2	0	6,0	3,1	3,1	0	7,9
Hypercholesterolaemia — moderate-risk category	34,0	31,3	14,8	6,7	14,6	44,8	46,2	32,8	27,8	18,4
Protective HDL/TC ratio \geq 20%	78,6	71,6	70,4	75,0	74,4	91,0	71,0	74,6	68,5	63,2
Hypertriglyceridaemia (TG \geq 2,3 mmol/l)	8,9	17,9	11,1	9,1	9,8	9,0	15,4	18,8	9,3	23,7
Hypertension (BP \geq 160/95 mmHg and/or on treatment)*	14,3	20,6	14,8	37,8	43,4	11,9	10,8	34,4	42,6	60,0
Mild hypertension (BP \geq 140/90 but < 160/95)	14,3	26,5	25,9	22,2	31,3	19,4	20,0	18,8	27,8	22,5
Diabetes mellitus	0	0	7,4	7,0	12,5	3,1	0	14,1	7,7	5,1
Smoking \geq .10 cigarettes/d*	25	18,2	22,2	8,9	6,2	46,3	35,4	35,9	24,1	10,3
Smoking other tobacco products or <10 cigarettes/d	41,1	39,4	48,2	57,8	42	26,9	29,2	29,7	37,0	35,9
Combination of risk factors at high level of risk:										
At least one IHD risk factor	39,3	29,2	37,0	40,0	45,0	49,3	40,0	45,3	51,9	46,0
At least two IHD risk factors	3,6	7,7	0	4,4	2,5	7,5	4,6	14,1	7,4	16,2
BMI \geq 30 (mass/height ²)	10,9	19,1	7,4	13,3	7,4	3,0	12,5	15,6	20,4	17,5

* The risk factor used to calculate the prevalence of the combination of risk factors.

Table V. Prevalence (%) of IHD risk factors in the female population of QwaQwa and Mangaung

	Age group (yrs)									
	QwaQwa					Mangaung				
	25 - 34	35 - 44	45 - 54	55 - 64	65+	25 - 34	35 - 44	45 - 54	55 - 64	65+
Hypercholesterolaemia — high-risk category*	4,3	0,9	1,0	3,9	0	8,6	2,4	8,7	0,0	4,7
Hypercholesterolaemia — moderate-risk category	34,0	32,1	30,6	27,5	24,8	29,6	43,7	32,7	30,9	43,6
Protective HDL/TC ratio \geq 20%	86,4	84,3	64,3	63,7	68,3	90,5	81,0	68,3	75,0	65,1
Hypertriglyceridaemia (TG \geq 2,3 mmol/l)	1,4	4,6	13,3	12,8	7,5	1,0	4,8	13,5	4,4	14,1
Hypertension (BP \geq 160/95 mmHg and/or on treatment)*	9,9	29,1	44,4	54,0	66,0	12,4	31,0	61,2	52,9	78,1
Mild hypertension (BP \geq 140/90 but < 160/95)	11,3	20,9	26,3	16,7	14,1	9,5	18,3	10,7	26,5	12,5
Diabetes mellitus	0,7	3,7	7,1	14,0	9,9	0	5,7	12,8	13,4	17,7
Smoking \geq 10 cigarettes/d*	0,7	1,9	0	0	0	0	4,8	1,9	2,9	0
Smoking other tobacco products or < 10 cigarettes/d	4,3	8,3	10,4	3,0	5,1	14,3	18,4	15,2	13,2	6,3
Combination of risk factors at high level of risk:										
At least one IHD risk factor	13,0	29,9	42,7	53,5	65,8	19,1	32,0	56,9	55,9	73,4
At least two IHD risk factors	0,7	0,9	1,0	2,0	0	1,0	3,2	7,8	0	4,7
BMI \geq 30 (mass/height ²)	27,5	41,8	42,4	49,0	31,4	31,1	42,9	54,3	47,1	42,2

* The risk factors used to calculate the prevalence of the combination of risk factors.

Discussion

This is the first comparison of two black populations in an urban and rural setting in South Africa that also describes the prevalence of IHD risk factors in rapidly developing and westernising black populations. Although IHD is still relatively rarely diagnosed in the local black population, this study suggests that all the elements for a potential epidemic of atherosclerotic cardiovascular disease in the decades to come are present. Furthermore, urban-rural disease differences in the prevalence of IHD risk factors in the populations studied were the exception rather than the rule, although the urban population of Mangaung had the worst risk profile of the two black populations overall.

TC levels were very comparable between QwaQwa and Mangaung subjects although there was a tendency towards higher values in Mangaung subjects. Compared with Cape Peninsula blacks,⁸ TC levels were also higher in the two Orange Free State populations studied for all corresponding age categories. Seedat *et al.*¹⁴ recently reported very similar TC and HDLC levels for Durban blacks. A striking feature of the present study was the emergence of a high-risk hypercholesterolaemic subgroup present in both populations and in both sexes. Even more disturbing was the large number of subjects in the moderate-risk hypercholesterolaemic category. Usually such raised cholesterol levels are due mainly to a diet high in saturated fat and animal products. An outstanding feature was that subjects in the younger age groups had higher prevalences of moderate-risk hypercholesterolaemia. This could possibly reflect the more powerful impact of urbanisation on the younger generations who may be adopting a Western diet to a greater extent than their elders. This is in contrast to the tendency for the prevalence of hypercholesterolaemia to rise with increasing age as was found in the white, coloured and Indian communities in South Africa.¹⁵⁻¹⁷ HDLC levels remained remarkably constant with increasing age for both populations and both sexes. A lower prevalence of protective HDLC/TC ratios was found in this study than was reported for Cape Peninsula blacks.

This study confirms that hypertension is at present clinically the single most prevalent cardiovascular disease in rural as well as urban adult black South Africans. Despite the differences in methodology, the prevalence of hypertension described in this study exceeds that previously described for blacks in this country.^{2,6,18} The magnitude of the problem was very similar for both populations and unlike previous studies³ no meaningful difference could be demonstrated between the urban and rural populations. Clearly, in the Orange Free State, the processes associated with urbanisation are now no longer occurring only in the cities but also in rural settings.

Although obesity was also an outstanding anthropometric feature of women in this study, the prevalence of obesity was less than that reported for Cape Peninsula black women.⁵ The consequences of obesity and especially the metabolic consequences in South African blacks have not been adequately studied. Obesity has been shown to contribute to hypertension² and may be responsible for comparable HDLC levels in black men and women.⁵ However, the association of obesity with cardiovascular disease is found mainly in a subgroup of obese persons, i.e.

a subgroup with central or android obesity.⁹ The mean WHR of women in both populations and for all age groups exceeded 0,8 which is regarded as the cut-off value for increased risk of IHD, stroke and death, independent of total body fat mass.⁹ Whether an increased WHR has the same significance for the local black population as for whites remains to be established. Given that both diabetes and hypertriglyceridaemia occurred frequently in both populations, it is reasonable to conclude that the study populations were metabolically well advanced in respect of 'westernisation' and all the concomitant chronic diseases of lifestyle.

In spite of the fact that at least one risk factor occurred as frequently in these samples as in the white population,¹³ IHD remains rare in the black population. It is clear that the small racial differences in HDLC levels alone do not explain the rarity of IHD in the black population. The low prevalence of a combination of risk factors in these populations as well as in the Cape Peninsula black population was striking. It is tempting to speculate that the absence of a combination of risk factors protects the black population against IHD. On the other hand the long incubation period of 30 - 40 years for IHD as well as the recent development of hypercholesterolaemia, now considered the essential risk factor for IHD, may point to a possible imminent cardiovascular disease epidemic in the black population of the Orange Free State.

In conclusion, the IHD risk factor profile of the black populations in this study was not as favourable as that reported for Cape Peninsula blacks. As has been suggested,⁶ measures must be taken urgently to prevent an epidemic of atherosclerotic vascular disease in the black population. In particular efforts should be made: (i) to combat smoking and hypertension in black male subjects; (ii) to emphasise the adverse effects of obesity including hypertension, especially in black women; and (iii) to propagate the importance of a healthy lifestyle including cholesterol-lowering measures, especially at school level since the youngest age category in this study displayed the highest risk in respect of cholesterol levels.

The authors are indebted to the following organisations and persons whose commitment, support and hard work made this study possible: Professor D. Stoker of the HSRC for formulating the sampling procedure; Professor E. Albertyn, former head of the Department of Community Health, University of the Orange Free State for valuable advice; The QwaQwa Department of Health and Welfare for providing staff and infrastructure; the QwaQwa Tribal authorities for their help and support; Mr T. D. Mofokeng and Mrs P. M. Rakhatla for the dedicated way in which they performed fieldwork and collected data under difficult circumstances; the Health Department of the Bloemfontein City Council for staff and assistance; the Medical Superintendent of Pelonomi Hospital for infrastructure; the staff of the diabetes clinic for assistance; Mr T. Nkomo, Mr M. P. Khunong and Mrs M. Motsabi for fieldwork and data collection; Mrs L. Vermaak for technical assistance; and the laboratory team of the Department of Chemical Pathology, UOFS, for their professional work and assistance, and the South African Broadcasting Corporation for propagating the study. We are indebted to the following organisations for financial support: the South African Medical Research Council's Centre for

Epidemiological Research in Southern Africa, The Heart Foundation of Southern Africa, Servier Laboratories South Africa (Pty) Ltd, the Department of Internal Medicine, UOFS, and the Central Research Fund of the UOFS.

REFERENCES

1. Brink AJ. People for research and development 1988. Proceedings of a conference held at the CSIR Conference Centre, Pretoria, 23-24 August 1988.
 2. Seftel HC, Johnson S, Muller EA. Distribution and biosocial correlations of blood pressure levels in Johannesburg blacks. *S Afr Med J* 1980; **57**: 313-320.
 3. Seedat YK, Seedat MA, Hackland DBT. Biosocial factors and hypertension in urban and rural Zulus. *S Afr Med J* 1982; **61**: 999-1002.
 4. Rosman KD. The epidemiology of stroke in an urban black population. *Stroke* 1986; **17**: 667-669.
 5. Walker ARP, Walker BF. Coronary disease in blacks in underdeveloped populations (Letter). *Am Heart J* 1985; **109**: 1410.
 6. Steyn K, Jooste PL, Bourne L, *et al.* Risk factors for coronary heart disease in the black population of the Cape Peninsula. *S Afr Med J* 1991; **79**: 480-485.
 7. Reaven GM. Role of insulin resistance in human disease. *Diabetes* 1988; **37**: 1595-1607.
 8. Kaplan NM. The deadly quartet: upper-body obesity, glucose intolerance, hypertriglyceridaemia and hypertension. *Arch Intern Med* 1989; **149**: 1514-1520.
 9. Björntorp P. Regional patterns of fat distribution. *Ann Intern Med* 1985; **103**: 994-995.
 10. WHO Expert Committee. *Diabetes Mellitus* (Technical Report Series No. 727). Geneva: World Health Organisation, 1985.
 11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; **18**: 499-502.
 12. WHO Expert Committee. *Arterial Hypertension* (Technical Report Series No. 628). Geneva: World Health Organisation, 1978.
 13. Rossouw JE, Steyn K, Berger GMB, *et al.* Action limits for serum total cholesterol: a statement for the medical profession by an *ad hoc* committee of the Heart Foundation of Southern Africa. *S Afr Med J* 1988; **73**: 693-700.
 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**: 251-256.
 15. Rossouw JE, Du Plessis JP, Benadé AJS, *et al.* Coronary risk factor screening in three rural communities. *S Afr Med J* 1983; **64**: 430-436.
 16. Steyn K, Jooste PL, Langenhoven ML, *et al.* Coronary risk factors in the coloured population of the Cape Peninsula. *S Afr Med J* 1985; **67**: 619-625.
 17. Seedat YK, Mayet FGH, Khan S, *et al.* Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; **78**: 447-454.
 18. Seedat YK, Seedat MA, Nkomo MN. The prevalence of hypertension in the urban Zulu. *S Afr Med J* 1978; **53**: 923-927.
-