Incidence of hypertension and non-insulin dependent diabetes mellitus and associated risk factors in a rapidly developing Caribbean community: the St James survey, Trinidad

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Abstract

Objective – To compare the incidence rates of hypertension and non-insulin dependent diabetes mellitus in relation to ethnicity and other characteristics in a rapidly developing community.

Design – Prospective surveillance of a total community for five years.

Subjects – Cohort of 2491 men and women aged 35 to 69 years (79% response), of African, Indian and 'other' (mainly Afro-European) descent.

Results – During surveillance, secular increases occurred in fasting blood glucose concentrations in both sexes and in body mass index (BMI) in men, with apparent secular reductions in systolic blood pressure in both sexes. Incidence rates of hypertension did not differ significantly with ethnicity, ranging between 33 and 41 per 1000 person-years in men and between 27 and 32 per 1000 person-years in women. In men, the incidence of diabetes (per 1000 person-years) in Indians (24) was significantly higher than in Africans (13) and others (11). In women, the diabetic incidence was similar to that for men in Indians (23) and Africans (14), but in others was twice that in men (21). In both sexes, weight gain was an important risk factor for hypertension, whereas risk of diabetes increased with BMI at baseline. The increased risk of diabetes in Indians among men was independent of baseline BMI and blood glucose.

Conclusion – Apart from the increased risk of diabetes in Indians, ethnicity had no significant influence on incidence rates of hypertension and diabetes in Trinidad. Secular increases in blood glucose in both sexes and in BMI in men probably contributed to the concurrent increase in mortality from coronary heart disease in this community.

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Hypertension[1-4] and non-insulin dependent diabetes mellitus[5-6] (hereafter 'diabetes') are common causes of morbidity and mortality in the Caribbean. A prospective study of adults aged 35 to 69 years on the island of Trinidad (the St James survey) showed that, as elsewhere, both conditions are major risk factors for coronary heart disease[7] and for total mortality[8] in this community. Ethnic- and sex specific incidence rates for hypertension and diabetes have not so far been published for any Caribbean community, and information is lacking on risk factors for these conditions. These issues have therefore been examined in the St James survey, which was undertaken at a time (1977 to 1985) when Trinidad was experiencing a period of unprecedented affluence and a rapidly increasing mortality from coronary heart disease. The prevalence rates of hypertension and diabetes at baseline in this study have been reported previously.[7]

Methods

Details of the population and recruitment procedures have been published previously.[7 8] In brief, all men and women aged 35 to 69 years living in a defined area of Port-of-Spain, the capital of Trinidad, were identified by household census. All 1386 men resident in the area on the day of appointment and without mental handicap were recruited for study, but enrolment of women was limited to 1105 belonging to a large subsector of the survey area.

BASELINE EXAMINATION
Ethnic composition was judged from grandparental origins. Those judged to be at least 75% African or Indian ancestry were classed accordingly and the remainder (mainly Afro-European) were grouped together as 'other'. Adults of European descent were too few to be assessed as a separate group for disease-specific incidence rates. Details of the previous medical history were taken by an interviewer, who inspected all current medication. Smoking status was assessed by recording whether the respondent had ever smoked, the number of cigarettes smoked per day, and the age at which the respondent last smoked. A fasting venous blood sample (2ml) was taken by vacutainer (Becton-Dickinson) into 10 mg of potassium oxalate and 10 mg of sodium fluoride for whole blood glucose concentration determination (Nelson-Somogyi method). Throughout the study, quality control sera (Wellcome Diagnostics) and a standard (5.6 mmol/l glucose in saturated benzoic acid) were included in each run. The 12 batches of quality control material used had different glucose concentrations, and the performance of the

laboratory was therefore assessed by expressing each day's result as the ratio of the measured concentration to that specified by the manufacturer. A 50 g oral glucose tolerance test (GTT) was performed whenever the fasting glucose concentration was between 6.2 and 7.7 mmol/l. Diabetes was defined at baseline by World Health Organization (WHO) criteria,[9] with allowance for a 50 g load and analysis of whole venous blood:
(1) A fasting concentration of 7.8 mmol/l or more, or,
(2) In the GTT, a two hour concentration of 9.0 mmol/l or more, plus a similar result between 30 and 90 minutes or a fasting value of at least 7.0 mmol/l, or,
(3) Current antidiabetic therapy.

In women, all blood pressures were recorded by the one observer (GLAB) throughout the study. In men, all measurements were made by one observer at baseline (GJM) and by GLAB at follow up. Two blood pressures were recorded on each occasion with the subject seated. Measurements were taken at the left arm with a random-zero sphygmomanometer (Gelman-Hawksley), using a large cuff (bladder 33 x 15.5.cm) when the mid-arm circumference exceeded 33cm. Systolic and diastolic V pressures were recorded to the nearest 2 mmHg below. Hypertension was defined as an average systolic pressure ⩾ 160 mmHg and/or an average diastolic pressure ⩾ 95 mmHg,[10] or the current use of antihypertensive therapy.

Weight (kg), measured with the subject wearing underwear, was recorded to the nearest 0.1 kg below on a balance scale (Avery) which was calibrated at three monthly intervals. Height (m) was measured on a stadiometer (Holtain Instruments, UK) in order to calculate body mass index (BMI) as kg/m².

FOLLOW UP
Field workers visited each participant at least once each year to record details of current medication and recent illness. Respondents were encouraged to contact survey staff about medical problems and were referred for care when necessary. Emigrants were sent a questionnaire annually and examined on return visits. All survivors were recalled for further examination after five years of follow up, or sooner when they had entered at less than five years before the close of the study. Weight, blood pressure, and fasting blood glucose were measured in the same manner as at baseline, but because of a change in WHO criteria during the course of the study, a 75g oral GTT was used when the fasting blood glucose concentration was 6.2 to 7.7 mmol/l and the critical concentration for diagnostic purposes was increased from 9 to 10 mmol/1.[9] All medications were inspected for the final time.

STATISTICAL METHODS
Individual length of follow up was defined as the interval between the determinations of blood pressure and fasting blood glucose concentration at recruitment and follow up. The ethnic- and sex specific incidence rates of hypertension were expressed per 1000 person-years of observation in subjects who were not hypertensive at baseline. This method will have underestimated the true incidence because it assumed that all cases had their onset at the end of surveillance, when in fact the true time of onset was unknown. However, this error will not have affected the comparison between groups. Poisson regression was employed to derive sex specific incidence rate ratios of hypertension according to quarters of the distributions of systolic pressure, diastolic pressure, and BMI at baseline; diabetic status at baseline; and change in weight during follow up, firstly, after adjustment for age and ethnic group alone and then with additional adjustment for the other factors.

The incidence rates for diabetes were then analysed in the same manner in subjects free of the disorder at entry, with rate ratios calculated according to thirds of the distribution of fasting blood glucose, hypertensive status (No, Yes-untreated, Yes-treated) and smoking category at recruitment. Five smoking categories were used for men (non-smoker, ex-smoker, current smoker either < 10 per day, 10 to 19 per day, or 20 or more per day), but only three in women (non-smoker, ex-smoker, current smoker) because only 12% of women had ever smoked. Ethnic specific rate ratios for diabetes were calculated after adjustment for age and the other characteristics listed above.

Analysis of variance was used to assess the statistical significance of secular changes in blood pressure and blood glucose concentrations between the beginning and end of surveillance, both in the total survey population and after excluding individuals on antihypertensive therapy either at recruitment or at follow up examination. The analysis was then repeated after exclusion of baseline data on those who died during follow up.

Results
RESPONSE RATES
Table 1 gives the eligible population defined by census, the numbers of respondents at baseline and with complete data at follow up examination, and the number of deaths in the interim. Overall, 96% of those available for recruitment had their blood pressure and/or blood glucose concentration recorded at baseline, and 79% had this recorded at follow up. The length of time between baseline and follow up averaged 5.47 years in men and 5.03 years in women, during which time there were 265 deaths. Follow up examination was completed in 1325 subjects who were normotensive when first seen and in 1712 subjects who were not diabetic at baseline.

QUALITY CONTROL
Figure 1 presents the cumulative distribution of the ratio of observed to specified control glucose concentrations at baseline and at follow up. The average ratio at baseline was 0.963, compared with 0.987 at follow up. The average difference of 0.024 was statistically highly significant (p < 0.0001), but amounted to only 0.24 mmol/l (4.3 mg/dl) at a concentration of 10 mmol/l (180 mg/dl). Adjustment of

Table 1   Numbers of baseline respondents, deaths during follow up, and response rates

	Men, no (%)		Women, no (%)		Total
Baseline eligible population*	1386	(100)	1105	(100)	2491
Blood pressure and/or blood glucose:					
At baseline	1332	(96)	1049	(95)	2381
At follow up	1051	(76)	921	(83)	1972
Deaths during follow up	178	(13)	87	(8)	265
Baseline and follow up blood pressure and blood glucose concentration recorded (%):					
All subjects	1016	(100)	918	(100)	1934
Normotensive at baseline	703	(69)	622	(68)	1325
Non-diabetic at baseline	916	(90)	796	(87)	1712

* Resident in survey area on appointment date for baseline examination and without mental handicap.

Table 2   Mean (SD) body mass index according to diabetic status, hypertensive status, and treatment of hypertension at baseline

	Hypertensive status		
	Normotensive	Hypertensive-no treatment	Hypertensive-treatment
Men			
Non-diabetic	23.3 (3.8)	24.6 (4.4)	25.7 (4.2)
Diabetic	24.9 (4.7)	25.0 (3.3)	26.2 (3.0)
Women			
Non-diabetic	26.3 (5.7)	28.6 (5.5)	28.2 (5.3)
Diabetic	29.1 (5.6)	31.9 (6.4)	30.4 (5.7)

In both sexes, body mass index was significantly higher in hypertensive subjects than non-hypertensives ($p < 0.001$) and in diabetics than non-diabetics ($p = 0.01$). Increased body mass index in treated hypertensives was significant in men ($p < 0.001$) but not in women.

results for this small difference did not alter the conclusions of the statistical analysis and therefore this small drift has been ignored.

ASSOCIATIONS AT BASELINE BETWEEN HYPERTENSION, DIABETES, AND BMI

Baseline hypertension and diabetes were strongly associated in both sexes. Thus in men, 8% of normotensive subjects and 14% of those with hypertension were diabetic, or alternatively, 29% of non-diabetic men and 45% of diabetic men were hypertensive (after adjustment for age and ethnic group, $\chi^2_1 = 6.2$; $p=0.01$). In women the respective figures were 9% and 23%, or alternatively 29% and 56%, (adjusted $\chi^2_1 = 29.6$, $p < 0.0001$).

Table 2 shows that at baseline, in both sexes, those who were neither hypertensive nor diabetic had the lowest BMI. Hypertensive subjects were more obese than normotensive subjects ($p < 0.001$), and diabetics more obese than non-diabetics ($p=0.01$). There was no

evidence for interaction between hypertension and diabetes with respect to body mass. A trend was found for treated hypertensives to have a higher BMI than untreated hypertensives among men ($p < 0.001$), but not in women.

PROSPECTIVE AND SECULAR CHANGES IN BMI, BLOOD PRESSURE, AND BLOOD GLUCOSE

Figure 2 shows that prospectively, men of all ages and women up to 49 years at entry gained weight during follow up; older women either showed no change or a reduction in weight during this period. In men, the mean (SD) gain in weight of 1.52 (4.6) kg was sufficient to produce a statistically significant ($p < 0.0001$) secular increase in BMI in all age groups during the study. For example, at baseline the mean BMI for men aged 45 to 49 years was 24.2 kg/m²; five years later the mean BMI for men aged 45 to 49 years had increased by 1 kg/m² to 25.2 kg/m². In women, however, weight gain during follow up produced no significant secular change in BMI. This sex difference in secular changes was unaffected by exclusion of individuals on antihypertensive therapy.

Figure 3 shows that only in men and women aged 35 to 49 years at baseline did blood pressures tend to rise during follow up. Changes in systolic blood pressure were such that in both sexes a secular reduction in mean values was seen in all age groups ($p < 0.0001$), except men aged 45 to 49 years. In men there was significant interaction ($p=0.02$) between age and the decline in systolic pressure, the secular decline being greater in older age bands. For example, at ages 40 to 45 years the mean decline was 3.8 mmHg, while at ages 65 to 69 years it was 7.4 mmHg. A similar trend in women was not statistically significant. Age specific diastolic pressures did not change significantly in men between rounds, but fell slightly but significantly in women in age bands from 50 years upwards ($p=0.02$).

When the analysis was repeated after exclusion of those on antihypertensive therapy either at baseline or follow up examination, there was
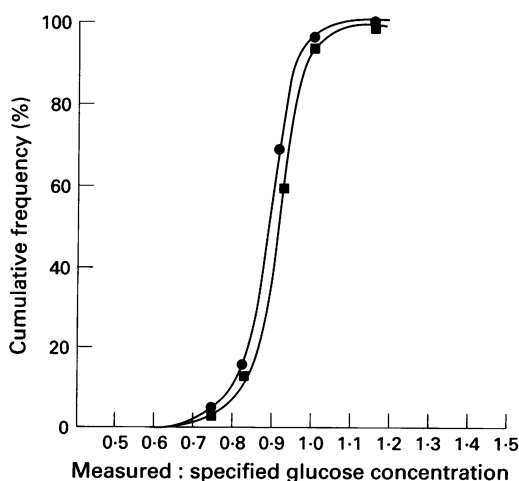

Figure 1   Cumulative frequency of the ratio of measured to specified concentration in quality control at baseline (closed circles) and at follow up (closed squares) examination.
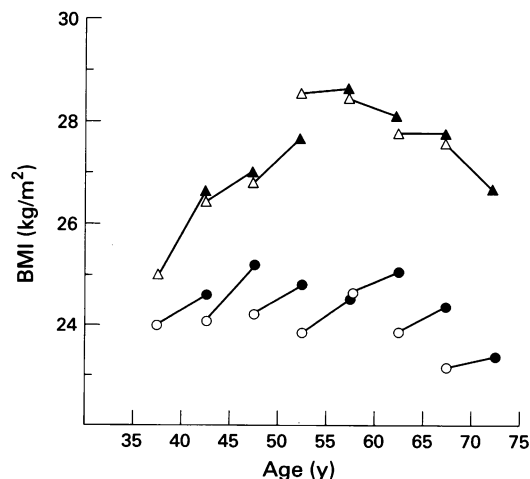

Figure 2   Mean body mass index in relation to age group, sex, and time. (Men at baseline, open circles; men at follow up, closed circles; women at baseline, open triangles; women at follow up closed triangles.)

Table 3   Relations between changes (Δ) in body weight (W), blood pressure (BP), and fasting blood glucose concentration (FBG) during follow up

	Correlation coefficient			
	Δ W	Δ Systolic BP	Δ Diastolic BP	Δ FBG
Δ W		0.15*	0.22*	− 0.04	Men (n=986)
Δ Systolic BP	0.13*		0.66*	− 0.00
Δ Diastolic BP	0.15*	0.65*		− 0.04
Δ FBG	− 0.06	− 0.08	− 0.03	
	Women (n=679)			

Statistical significance of the correlation coefficient: *p < 0.001.

no systematic difference in age specific systolic pressure between rounds in men ($F_{1, 1523}$ = 1.7, p=0.19), but a secular decline persisted in women ($F_{1, 1267}$ = 17.1, p < 0.001). Age specific diastolic pressures in those never on antihypertensive therapy showed inconsistent secular changes in men, there being an increase up to age 54 years, little change between 55 and 64 years, and a secular decline at older ages, whereas in women no difference was observed between rounds ($F_{1, 1217}$ = 0.004, p=0.95).

Figure 4 is a similar presentation of fasting concentrations of blood glucose, showing that in both sexes there was a clear secular increase in concentrations in all age bands during the study (p < 0.0001). This pattern was unchanged by exclusion of all subjects on antihypertensive treatment either at baseline or follow up examination.

The findings of these analyses of prospective and secular changes were essentially unchanged by exclusion from the baseline data of the results for those who died during follow up (for example, the baseline mean BMI for men aged 45 to 49 years was altered by only + 0.1 kg/m^2).

ASSOCIATIONS BETWEEN CHANGES DURING FOLLOW UP

Table 3 presents the correlation coefficients between changes in body weight, blood pressures, and blood glucose concentration across the study. Change in body weight was significantly and positively correlated with changes in systolic blood pressure and diastolic blood pressure in both sexes. By contrast, a change in fasting blood glucose concentration was not associated with either a change in body weight or changes in blood pressures. The explanation for this apparently anomalous relation between blood glucose and body weight became apparent in the further analysis.

INCIDENCE OF HYPERTENSION

Table 4 shows that on average 41 African men, 41 other men, 33 Indian men, and about 30 women in each ethnic group developed hypertension per 1000 person-years of observation. The lower incidence in Indians among men was not statistically significant.

After age and ethnic group adjustment, the incidence of hypertension showed a trend
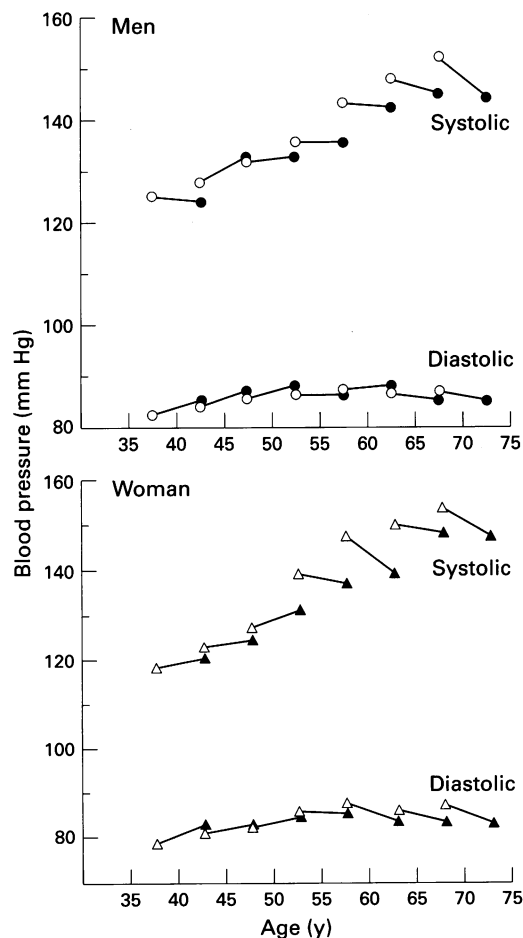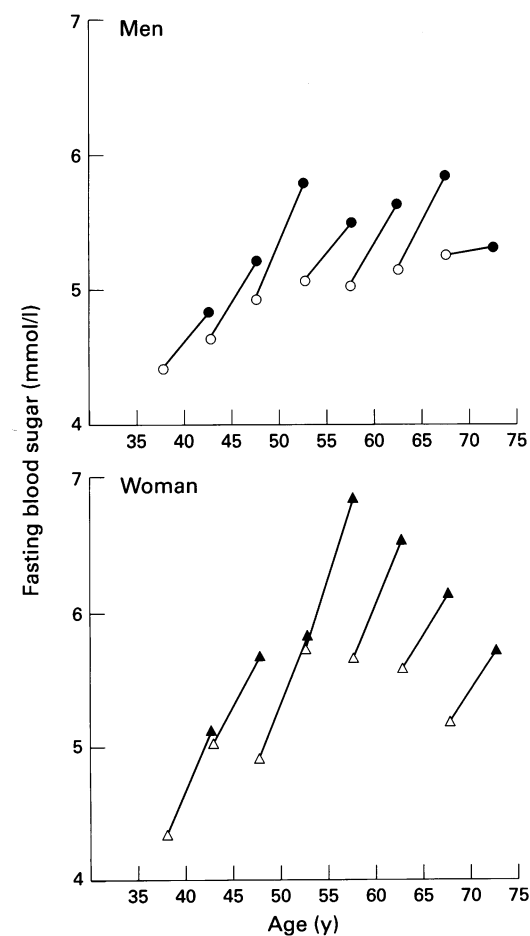

Figure 3   Mean systolic and diastolic blood pressures in relation to age group at baseline (men, open circles; women, open triangles) and at re-examination after about five years (men, closed circles; women, closed triangles).

Figure 4   Mean fasting blood glucose concentration in relation to age group at baseline (men, open circles; women open triangles) and at re-examination (men, closed circles; women, closed triangles).

Table 4   Numbers of subjects who developed diabetes and hypertension during follow up, and incidence rates (per 1000 person-years) in relation to sex and ethnic group

	Hypertension*			Diabetes†		
	No	Person-years	Rate	No	Person-years	Rate
Men:						
Indian	40	1251	32.9	34	1399	23.6
African	52	1285	41.0	24	2016	12.5
Other	53	1309	40.6	19	1764	11.0
Women						
Indian	31	1044	32.3	27	1181	22.7
African	30	1061	27.9	21	1474	14.4
Other	28	1034	27.2	28	1367	20.5

In the comparison of ethnic groups: * p > 0.1 in both sexes; † rates are statistically significantly different in men (p = 0.018) but not in women (p > 0.1).

*Table 5   Multivariate adjusted rate ratio (95% confidence interval) * for predictors of hypertension (quartiles) in men and women*

	Men		Women	
Systolic blood pressure (mmHg)				
	< 116	1.0	< 114	1.0
	116 –	3.1 (1.4 to 6.8)	114 –	1.5 (0.5 to 4.4)
	125 –	2.9 (1.3 to 6.6)	125 –	1.3 (0.4 to 4.0)
	135 +	5.0 (2.2 to 11.6)	137 +	2.0 (0.7 to 6.3)
χ^2 test for trend		p < 0.0001		p = 0.17
Diastolic blood pressure (mmHg)				
	< 74	1.0	< 73	1.0
	74 –	0.9 (0.5 to 1.8)	73 –	0.6 (0.1 to 2.4)
	80 –	1.0 (0.5 to 2.0)	78 –	2.4 (0.8 to 7.2)
	86 +	1.7 (0.9 to 3.3)	84 +	5.0 (1.7 to 14.7)
χ^2 test for trend		p = 0.029		p < 0.001
BMI				
	< 20.7	1.0	< 22.7	1.0
	20.7 –	1.0 (0.6 to 1.6)	22.7 –	1.5 (0.6 to 3.7)
	23.2 –	1.1 (0.7 to 1.9)	25.9 –	2.0 (0.8 to 4.9)
	26.0 +	1.3 (0.8 to 2.2)	29.3 +	2.5 (1.0 to 5.9)
χ^2 test for trend		p = 0.18		p = 0.019
Weight				
	< – 0.6	1.0	< 1.4	1.0
	– 0.6 –	1.4 (0.9 to 2.4)	– 1.4 –	0.9 (0.4 to 1.9)
	1.9 –	1.4 (0.8 to 2.3)	1.5 –	1.3 (0.6 to 3.0)
	4.4 +	1.8 (1.1 to 3.3)	4.3 +	2.1 (1.0 to 4.4)
χ^2 test for trend		p = 0.02		p = 0.02
Non-diabetic		1.0		1.0
Diabetic		1.3 (0.7 to 2.7)		0.7 (0.3 to 1.5)
χ^2 test		p = 0.43		p = 0.37

* Adjusted for age, ethnic group and other variables included in the table.
BMI = body mass index, kg/m²; weight = change in weight during follow up, kg.

to increase with increasing systolic blood pressure (p < 0.001), diastolic blood pressure (p < 0.001), and BMI at baseline (p < 0.01) in both sexes. Normotensive men and women entering with a systolic pressure above the upper quartile of the distribution had an incidence of hypertension about seven times that of those with pressures below the lowest quartile. The respective figures for diastolic pressure were four for men and seven for women. Among male normotensives at baseline, those entering with a BMI above the highest quartile had an incidence about twice that of those with a BMI below the lowest quartile. Normotensive women entering with a BMI above the highest quartile had an incidence approximately four times that of those with a BMI below the lowest quartile. There was a trend to increasing risk of hypertension with increasing weight gain during follow up in both sexes, but this was not statistically significant. In neither sex was there any evidence that diabetics at baseline had a higher incidence of hypertension than non-diabetics.

Table 5 shows that after adjustment for age, ethnic group, and the other characteristics included in the table, both systolic and diastolic blood pressure at recruitment continued to make independent contributions to the risk of hypertension in men, though the association was stronger for systolic pressure. In women, diastolic pressure made an independent contribution to the risk of hypertension, whereas the association of systolic pressure with risk was no longer statistically significant. The association between BMI at baseline and the incidence of hypertension was attenuated in both sexes and no longer of statistical significance in men. Weight gain during follow up was positively and significantly related to risk of hypertension in both sexes after allowance for BMI and blood pressure at entry.

INCIDENCE OF DIABETES
Table 4 shows that 24 Indian men, 13 African men and 11 other men developed diabetes per 1000 person-years, an ethnic difference that was statistically significant. In women, although ethnic differences were not statistically significant, the incidence rates in Indians (23) and Africans (14) were very similar to those for men, although in others the rate (21) was almost twice that in men.

After age and ethnic group had been allowed for, the incidence of diabetes showed a trend to increase with increasing BMI at baseline (p < 0.001 in both sexes), to decrease with increasing weight gain during follow up (p < 0.05 in both sexes), and to increase with increased fasting blood glucose at baseline (p < 0.001 in both sexes). Men in the highest quarter of the distribution of BMI at baseline had an incidence of diabetes almost five times that of those in the lowest quarter, while in women the respective figure was almost four. The incidence of diabetes in those who lost most weight during follow up (that is, more than 0.9 kg in men or 2 kg in women), was more than twice that in those who lost lesser amounts or gained weight during this period. Non-diabetic men and women with a fasting blood glucose concentration at recruitment between 4.7 and 7.79 mmol/l had respective incidences of diabetes 28 and 17 times that of those with a concentration below 4.2 mmol/l. Untreated hypertension at baseline was not accompanied by any significant increase in the incidence of diabetes in either sex, but in male hypertensives on treatment the incidence was three times that of normotensives (p < 0.001). Hypertensive therapy had no effect on the incidence of diabetes in women. At recruitment 165 (53%) of 311 hypertensive men and 218 (67%) of 325 hypertensive women, whose status was known at follow up, were taking treatment for this disorder. Among these treated hypertensives, thiazides and frusemide had been prescribed to 69% and 6% of men respectively, but to 15% and 42% of women respectively. The incidence of diabetes was unrelated to smoking status at baseline either in men or women.

Table 6 shows that the associations of the incidence of diabetes with BMI and fasting blood glucose concentration persisted in both sexes after allowance for age, ethnic group, and

*Table 6   Multivariate adjusted rate ratio   (95% confidence interval) * for predictors of diabetes   (quartiles) in men and women*

	Men		Women	
BMI				
< 21.3	1.0		< 22.9	1.0
21.3 –	1.2	(0.5 to 3.1)	22.9 –	0.7 (0.2 to 2.2)
23.9 –	2.4	(1.0 to 5.5)	26.1 –	2.0 (0.8 to 4.9)
26.5 +	2.6	(1.2 to 5.9)	29.9 +	2.3 (1.0 to 5.5)
χ^2 test for trend	p = 0.004			p = 0.07
Weight				
< – 0.9	1.0		< – 2.0	1.0
– 0.93 –	0.6	(0.3 to 1.1)	– 2.0 –	0.7 (0.3 to 1.4)
1.70 –	0.6	(0.3 to 1.2)	1.10 –	0.5 (0.2 to 1.1)
4.2 +	0.7	(0.4 to 1.3)	4.00 +	0.6 (0.3 to 1.4)
χ^2 test for trend	p = 0.21			p > 0.1
Fasting blood glucose				
(mmol/l) < 4.2	1.0		< 4.2	1.0
4.2 –	9.4	(2.2 to 40.5)	4.2 –	3.6 (0.8 to 16.6)
4.7 –	21.3	(5.1 to 88.3)	4.7 –	12.8 (3.1 to 53.6)
χ^2 test for trend	p < 0.001			p < 0.001
Normotensive	1.0			1.0
Hypertensive - no treatment	1.1	(0.5 to 2.3)		1.2 (0.6 to 2.5)
Hypertensive - treatment	1.7	(1.0 to 2.9)		1.0 (0.5 to 2.0)
χ^2 test for trend	p = 0.09			p > 0.1
Non-smoker	1.0			1.0
Ex-smoker	1.9	(1.1 to 3.6)		0.7 (0.1 to 5.2)
Current smoker				0.8 (0.3 to 2.2)
0-9/d	1.0	(0.4 to 2.6)		
10-19/d	1.3	(0.6 to 2.9)		
20 + /d	1.2	(0.6 to 2.2)		
χ^2 test for trend	p > 0.1			p > 0.1

* Adjusted for age, ethnic group and other variables included in the table.
BMI = body mass index, kg/m^2; weight = change in weight during follow up, kg.

the other characteristics included in the table, although that for BMI in women was of marginal statistical significance. The associations of onset of diabetes with weight change during follow up in both sexes and with antihypertensive therapy in men were no longer statistically significant.

The rate ratio of diabetes in Indian men was 1.9 (95% confidence interval (CI) 1.1,3.4) relative to that of African men after adjustment for age and all other characteristics included in table 6.

Discussion

Hypertension has previously been reported to be very common among men and women who entered this study, but differences in prevalence rates between ethnic groups were small and not statistically significant.[7] The ethnic comparison of incidence rates of hypertension in this community accorded with that of prevalence rates. Age standardised incidence rates for men of African descent were closely similar to those of other (that is, mainly Afro-European) ethnic descent, and in African women similar to the rates for non-African women. The age standardised incidence in Indian men was about 8 per 1000 person-years lower than in non-Indian men, a difference that was not statistically significant.

The similar prevalence rates of hypertension in Africans and Indians in Trinidad accorded with an earlier prevalence survey in the Caribbean territory of Guyana, in which the higher systolic and diastolic blood pressures in adults of African than Indian descent were explained entirely by ethnic differences in lean body habitus.[4] In the UK also, Afro-Caribbean and Indian immigrants seem to have similar blood

pressures,[11-13] differences when found[14] apparently being largely accounted for by differences in BMI.[15]

Estimates of incidence and prevalence rates of hypertension will depend upon the definition of the disorder used and the composition of the community examined by age and social class,[16] making comparisons between studies of limited value. When hypertension was defined as a diastolic pressure of 95 mmHg or more, an incidence of 4% per year was found among black adults aged 35 to 59 years in Evans County, Georgia,[17] though higher incidence rates have been reported in other studies of black communities in the USA.[18] [19] In the UK, Afro-Caribbean people (mainly from Jamaica) have higher prevalence rates of hypertension than adults of European descent,[13] [14] [20] though similar rates to those reported in people of predominantly African origin in Trinidad and the USA.[21] When black-white differences in blood pressures have been observed in the UK[20] and USA,[22] they have generally persisted when allowance was made for ethnic differences in BMI, although in one British study this ethnic difference disappeared in women after allowance for BMI.[13] In Trinidad, African men tended to have slightly, but not statistically significantly, higher blood pressures than men of European origin after adjustment for arm circumference as an index of body size.[8]

Fourteen per cent of hypertensive men were diabetic at entry, as compared with 8% of normotensive men. The respective rates in women were 23% and 9%, contrasts that remained after adjustment for age and ethnic group. This cross sectional association between hypertension and diabetes mellitus has been recognised elsewhere.[23-25] In the Chicago Heart Association detection project the associations between blood pressures and blood glucose after an oral glucose load were similar in blacks and whites after allowance for age, heart rate, and relative weight.[26] However, the cross sectional association between hypertension and diabetes in Trinidad did not extend to the prospective findings, there being no association between change in blood pressure and change in blood glucose. The incidence of hypertension was not affected by the presence of diabetes at baseline in normotensives, and the incidence of diabetes was uninfluenced by the presence of untreated hypertension.

The baseline and prospective findings in Trinidad suggested that hypertension and diabetes may frequently have a common underlying cause when they occur together, but that neither directly raises the risk of the other. One mechanism proposed for this cross sectional association is an augmented adrenergic activity with effects on insulin resistance and blood pressure.[27] Another possible factor predisposing to both conditions is abdominal or central obesity, the importance of which for the level of blood pressure,[28] [29] blood glucose concentration,[11] and the presence of diabetes[30] is well documented (measures of fat distribution were not in this routine study).

The well recognised increase in BMI in hypertensive[31] and diabetic adults[32] was present

at baseline in Trinidad. During follow up there was a significant secular increase in BMI in men but not in women. This sex difference could not have been due to errors of measurement, because a single set of scales was employed throughout the study, with frequent calibration and systematic recording to the nearest 0.1 kg. below. Presumably there had been an increase in positive energy balance in men (possibly owing to a decline in physical activity) during a period of rapid economic advance and affluence, without any similar change in women (most of whom were house-wives and mothers). Trinidad has large reserves of oil and natural gas and its own oil refining industry. Largely for this reason, the population enjoyed a period of exceptional prosperity during the 1970s and early 1980s when world prices for oil were high. Taking 1970 as the base year, by 1980 the gross domestic product had risen 2.7 times, though gradually declining thereafter. This increased economic activity created an unprecedented period of affluence, and whereas in 1980 prices stood at 3.35 times their level in 1970, earnings had risen 4.33 times over the same period. By 1985 the ratio of earnings to prices was 1.5 times that in 1970.[33]

Not unexpectedly,[16] the incidence of hypertension was positively associated with the diastolic blood pressure at baseline in both sexes, and also independently with the systolic blood pressure at entry in men. Also, after allowance for the effect of BMI at entry, weight change was positively associated with the onset of hypertension in both sexes, as reported previously.[17] Because change in weight was correlated positively with change in blood pressure and the incidence of hypertension, it might have been expected that blood pressures would have increased with the secular increase in BMI in men. However, mean age specific systolic blood pressure and diastolic blood pressure actually declined significantly during follow up, particularly at older ages, indicating that other factors had more than offset the effect of increasing age specific BMI on age specific blood pressure in men. What these factors might have been is uncertain. The finding was not due to selective loss of subjects with high blood pressure due to death during follow up. Technical artefacts can be excluded because similar declines in age specific blood pressure were observed in women, in whom all measurements were recorded by the one (Trinidadian) investigator using the same instrument and standardised technique throughout the study. One possibility may have been that the management of high blood pressure had improved during the survey, because the secular declines in systolic blood pressure in men and diastolic pressure in women were no longer apparent when individuals prescribed antihypertensive therapy were removed from the analysis. Another possibility was that readings at recruitment tended to be elevated by anxiety induced by unfamiliarity with the survey procedure, but that this effect had subsided by the time of repeat examination.

Diabetes is very common in Trinidad,

particularly in people of Indian descent.[7] At entry into this study, 20% of Indian men and 23% of Indian women had diabetes, compared with 8% of African men and 17% of African women, 4% of European men and 11% of European women (age adjusted rates).[7] Similar ethnic contrasts have been reported from the UK.[11 14] The ethnic specific incidence rates found in Trinidad accorded with those in prevalence rates, in that the highest incidence rates were observed in Indian men and women, confirming their susceptibility to diabetes.

As expected, the incidence of diabetes was highest in those who had entered with the largest BMI.[34 35] However, the incidence of diabetes was significantly increased in subjects who lost rather than gained weight during follow up, as has been observed in other studies.[36] The association probably represented an effect of the disease per se, possibly with an additional component of therapeutic weight loss, and probably explained the absence of any significant relation between changes in weight and blood glucose level during follow up. This lack of association, similar to the weak association between these two variables in the Framingham study,[37] did not imply of course that weight gain is of no consequence for the risk of diabetes.

In men, the incidence of diabetes mellitus was more than doubled by treatment of hypertension, mostly with thiazide diuretics and/or beta blockers. This side effect of these forms of antihypertensive therapy is well recognised,[38-40] and the loss of statistical significance for the relation between incidence of diabetes and antihypertensive therapy in men after allowance for blood glucose levels at baseline probably reflected the inducement of hyperglycaemia by these preparations. The pathogenic significance of this effect is debated. While the actions of these drugs on insulin and glucose metabolism may be detrimental in their own right, their antihypertensive effects appear to more than offset such risks because thiazide diuretic and beta blocker therapy reduce cardiovascular mortality in patients with increased blood pressure.[41] The lack of effect of antihypertensive therapy on blood glucose levels in women appeared to be because frusemide was the main drug prescribed.

A number of previous studies have found an association between cigarette smoking and the risk of diabetes,[42 43] but this finding was by no means universal.[44 46] No relation between smoking and the development of diabetes was shown in Trinidadian men, even after adjustment for BMI at entry, weight change, and fasting blood glucose level at baseline. This analysis lacked statistical power in women, only 7% of whom were current smokers.

The considerable increase in age specific BMI in men, the effect of weight gain on the incidence of hypertension, and the increase in age specific fasting blood glucose concentrations in both sexes during the course of the study suggested that the marked rise in affluence experienced by this community after 1973 had detrimental effects on cardiovascular risk. The long term consequences of these

changes for cardiovascular morbidity and mortality in Trinidad are unknown, but they probably account for at least part of the recent increase in mortality from coronary heart disease. Between 1981 and 1987, the mortality from coronary heart disease (per 100 000) increased markedly in Trinidadian men from 417 to 506 but then declined to 467 in 1988-89, whereas in Trinidadian women the rate increased steadily from 263 in 1981 to 319 in 1989.[47] If BMI and blood glucose levels have fallen back with the more recent economic decline and decrease in the purchasing power of wages then the experience of the 1970s and 1980s may be of less consequence in the long term. On the other hand, if raised levels of BMI and blood glucose have been sustained then Trinidad's cardiovascular death rate might deteriorate even further in future.

1 Kean BH. Blood pressure studies on West Indians and Panamanians living on the isthmus of Panama. *Arch Intern Med* 1941;68:466-75.
2 Saunders GM, Bancroft H. Blood pressure studies on negro and white men and women living in the Virgin Islands of the United States. *Am Heart J* 1942;23:410-23.
3 Johnson BC, Remington RD. A sampling study of blood pressure levels in white and negro residents of Nassau, Bahamas. *J Chron Dis* 1961;13:39-51.
4 Florey C du V, Ashcroft MT, Miller GJ. Blood pressure levels of Guyanese adults of African and Indian origin. *Am J Epidemiol* 1971;94:419-24.
5 Poon-King T. Henry MV, Rampersad F. Prevalence and natural history of diabetes in Trinidad. *Lancet* 1968; i:155-60
6 Florey C du V. Blood sugar and serum insulin levels in Jamaica, West Indies. *Adv Metabolic Disorders* 1978;9:65-91.
7 Miller GJ, Beckles GLA, Maude GH, et al. Ethnicity and other characteristics predictive of coronary heart disease in a developing community: principal results of the St James survey, Trinidad. *Int J Epidemiol* 1989;18:808-17.
8 Miller GJ, Kirkwood BR, Beckles GLA, Alexis SD, Carson DC, Byam NTA. Adult male all-cause, cardiovascular and cerebrovascular mortality in relation to ethnic group, systolic blood pressure and blood glucose concentration in Trinidad, West Indies. *Int J Epidemiol* 1988;17:62-9.
9 World Health Organization Expert Committee on Diabetes Mellitus. *Second report*. WHO Technical Report Series No 646. Geneva: WHO, 1980.
10 World Health Organization. *Hypertension and coronary heart disease. Classification and criteria for epidemiological studies*. WHO Technical Report Series No 168. Geneva: WHO, 1959.
11 McKeigue PM, Shah B. Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991;337:382-6.
12 Miller GJ, Kotecha S. Wilkinson WH, et al. Dietary and other characteristics relevant for coronary heart disease in men of Indian, West Indian and European descent in London. *Atherosclerosis* 1988;70:63-72.
13 Cruickshank JK, Jackson SHD, Beevers DG, Bannan LT, Beevers M, Stewart VL. Similarity of blood pressure in Blacks, whites and Asians in England: The Birmingham factory study. *J Hypertension* 1985;3:365-71.
14 Cruickshank JK, Cooper J. Burnett M, MacDuffJ, Drubra U. Ethnic differences in fasting Cpeptide and insulin in relation to glucose tolerance and blood pressure. *Lancet* 1991; 338:842-7.
15 McKeigue PM, Marmot MG, Syndercombe Court YD, Cottier DE, Rahman S. Riemersma RA. Diabetes, hyperinsulinaemia, and coronary risk factors in Bangladeshis in East London. *Br Heart J* 1988;60:390-6.
16 Keil JE, Tyroler HA, Sandifer SH, Boyle E. Hypertension: effects of social class and racial admixture. The results of a cohort study in the black population of Charleston, South Carolina. *Am J Public Health* 1977;67:634-9.
17 Tyroler HA, Heyden S. Hames CG. Weight and hypertension: Evans County studies of blacks and whites. In: O Paul, ed. *Epidemiology and control of hypertension*. New York: Stratton Intercontinental Medical Book Corp, 1975; 177-204.

18 Dischinger PC, Apostolides AY, Entwisle G. Hebel JR. Hypertension incidence in an innercity black population. *J Chron Dis* 1981 ;34:405-13.
19 Apostolides AY, Cutter G. Daugherty SA, et al. Three-year incidence of hypertension in thirteen US communities. *Prev Med* 1982;11:487-99.
20 Chaturvedi N. McKeigue PM, Marmot MG. Resting and ambulatory blood pressure differences in Afro-Caribbeans and Europeans. *Hypertension* 1993;22:90-6.
21 Hypertension Detection and Follow-up Program Cooperative Group. Race, education and prevalence of hypertension. *Am J Epidemiol* 1977;106:351-61.
22 McDonough JR, Garrison GE, Hames CG. Blood pressures and hypertensive disease among negroes and whites: a study in Evans County, Georgia. *Ann Intern Med* 1964;61:208-28.
23 Garcia M, McNamara P. Gordon T. Kannel WB. Cardiovascular complications in diabetics. *Diabetes* 1974;23:105-1.
24 Jarrett RJ, Keen H. McCartney M, et al. Glucose tolerance and blood pressure in two population samples: their relation to diabetes mellitus and hypertension. *Int J Epidemiol* 1978;7:15-24.
25 Barrett-Connor E, Criqui MH, Klauber MR, Holdbrook M. Diabetes and hypertension in a community of older adults. *Am J Epidemiol* 1981;113:276-84.
26 Stamler J. Rhomberg P. Schoenberger JA, et al. Multivariate analysis of the relationship of seven variables to blood pressure: findings of the Chicago Heart Association detection project in industry, 1967-1972. *J Chron Dis* 1975;28:527-48.
27 Saad MF, Lillioja S. Nyomba BL, et al. Racial differences in the relation between blood pressure and insulin resistance. *N Engl J Med* 1991;324:733-9.
28 Peiris AN, Sothmann MS, Hoffmann RG et al. Adiposity, fat distribution, and cardiovascular risk. *Ann Intern Med* 1989;110:867-72.
29 Blair D, Habicht J-P, Sims EAH, Sylwester D, Abraham S. Evidence for an increased risk of hypertension with centrally located body fat and the effect of age and sex on this risk. *Am J Epidemiol* 1984;119:526-40.
30 Feldman R. Sender AJ, Siegelaub AB. Differences in diabetic and non-diabetic fat distribution patterns by skinfold measurements. *Diabetes* 1969;18:478-86.
31 Chiang B N. Periman L V, Epstein F H. Overweight and hypertension: a review. *Circulation* 1960;39:403-21.
32 West K M. *Epidemiology of diabetes and its vascular lesions.* New York: Elsevier North Holland, 1978.
33 Republic of Trinidad and Tobago. *Annual statistical digest 1978- 1993*. Office of the Prime Minister, Central Statistical Office, Port-of-Spain, Trinidad and Tobago.
34 Medalie JH, Papier C, Herman JB, et al. Diabetes mellitus among 10,000 adult men. 1. Five year incidence and associated variables. *Israel J Med Sci* 1974;10:681-97.
35 Cassano PA, Rosner B. Vokonas PS, Weiss ST. Obesity and body fat distribution in relation to the incidence of non-insulin-dependent diabetes mellitus. A prospective cohort study of men in the normative aging study. *Am J Epidemiol* 1992;136: 1474-86.
36 Noppa H. Body weight change in relation to incidence of ischemic heart disease and change in risk factors for ischemic heart disease. *Am J Epidemiol* 1980;111:693-704.
37 Ashley FW, Kannel WB. Relation of weight change to changes in atherogenic traits: The Framingham study. *J Chron Dis* 1974;27:103-14.
38 Pollare T. Lithell H. Selinus I, Berne C. Sensitivity to insulin during treatment with atenolol and metoprolol: a randomised, double blind study of effects on carbohydrate and lipoprotein metabolism in hypertensive patients. *BMJ* 1989;298: 1152-57.
39 Skarfors ET, Selinus KI, Lithell HO. Risk factors for developing non-insulin dependent diabetes: a 10 year follow up of men in Uppsala. *BMJ* 1991;303:755-60.
40 Medical Research Council Working Party on Mild to Moderate Hypertension. Adverse reactions to bendrofluazide and propranolol for the treatment of mild hypertension. *Lancet* 1981;ii:539-43.
41 Collins R. Peto R. MacMahon S. et al. Blood pressure, stroke, and coronary heart disease. Part 2, short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *Lancet* 1990; 335:827-38.
42 Feskens EJM, Kromhout D. Cardiovascular risk factors and the 25-year incidence of diabetes mellitus in middle-aged men. The Zutphen study. *Am J Epidemiol* 1989; 130:1101-8.
43 Rimm EB, Chan J. Stampfer MJ, Colditz GA, Willett WC. Prospective study of cigarette smoking, alcohol use, and risk of diabetes in men. *BMJ* 1995;310:555-9.
44 Medalie JH, Papier CM, Goldbourt U. Herman JD. Major factors in the development of diabetes mellitus in 10,000 men. *Arch Intern Med* 1975;135:811 17.
45 Butler WJ, Ostrander LD, Carman WJ, Lamphiear DE. Diabetes mellitus in Tecumseh, Michigan. Prevalence, incidence, and associated conditions. *Am J Epidemiol* 1982;116:971-80.
46 Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG. Prospective study of risk factors for development of non-insulin dependent diabetes in middle aged British men. *BMJ* 1995;310:560-4.
47 World Health Organisation. *World health statistics annual.* Geneva: WHO, 1987-1992.