High Prevalence of Type 2 Diabetes in All Ethnic Groups, Including Europeans, in a British Inner City

Relative poverty, history, inactivity, or 21st century Europe?

LISA RISTE, PHD
FARIDA KHAN, MSC
KENNEDY CRUICKSHANK, MB, MD, FRCP

OBJECTIVE — To compare the prevalence of type 2 diabetes in white Europeans and individuals of African-Caribbean and Pakistani descent.

RESEARCH DESIGN AND METHODS — Random sampling of population-based registers in inner-city Manchester, Britain's third most impoverished area. A total of 1,318 people (25–79 years of age) were screened (minimum response 67%); 533 individuals without known diabetes underwent 2-h glucose tolerance testing, classified by 1999 World Health Organization criteria.

RESULTS — More than 60% of individuals reported household annual income <£10,000 ($15,000) per year. Energetic physical activity was rare and obesity was common. Age-standardized (35–79 years) prevalence (mean 95% CI) of known and newly detected diabetes was 20% (17–24%) in Europeans, 22% (18–26%) in African-Caribbeans, and 33% (25–41%) in Pakistanis. Minimum prevalence (assuming all individuals not tested were normoglycemic) was 11% (8–14%), 19% (15–23%), and 32% (24–40%), respectively. Marked changes in prevalence represent only small shifts in glucose distributions. Regression models showed that greater waist girth, lower height, and older age were independently related to plasma glucose levels, as was physical activity. Substituting BMI and waist-to-hip ratio revealed their powerful contribution.

CONCLUSIONS — A surprisingly high prevalence of diabetes, despite expected increases with new lower criteria, was found in Europeans, as previously established in Caribbeans and Pakistanis. Lower height eliminated ethnic differences in regression models. History and relative poverty, which cosegregate with obesity and physical inactivity, are likely contributors. Whatever the causes, the implications for health services are alarming, although substantial preventive opportunities through small reversals of glucose distributions are the challenge.

Diabetes Care **24:1377–1383, 2001**

Rates of type 2 diabetes have been rising around the world (1,2). The increase in prevalence has accelerated due to aging population structures in developed countries and increasing obesity globally. High prevalence occurs in the Caribbean (3,4), West Africa (5,6), and among these communities in Britain (7–12), as in individuals of Indian subcontinental origin, with heterogeneity in subgroups (13). Whether these occur as a result of genetic or environmental factors remains unclear; the case against a major genetic contribution has been made elsewhere (14,15).

Poverty has been underrecognized as a contributor to prevalence of type 2 diabetes. However, in Britain, standardardized mortality ratios from reported diabetes, known to be seriously underrecorded (16), correlate closely with deprivation scores and inner cities (17). Prevalence is higher in individuals exposed to more deprivation, individually and at the level of the electoral ward (18–21). Patients in more deprived areas were less likely to receive insulin therapy, and those who did receive insulin had poorer diabetes control (22).

Herein we compare the prevalence of type 2 diabetes among African-Caribbeans in inner-city Manchester (part of an international collaborative study examining nutrition and the emergence of cardiovascular disease in African origin populations) (6,23–27) with data from individuals of local European origin (aged 25–79 years) and a smaller sample of the local residents of Pakistani descent (aged 35–79 years). We examine the contribution of deprivation, measured by reported income in an area with poor socioeconomic indexes, education level, physical activity, and obesity.

RESEARCH DESIGN AND METHODS

— Because no formal ethnic registers exist, population-based registers for patients aged 25–79 years held by local health centers were randomly sampled. Centers were approached within the Hulme, Moss Side, and Cheetham Hill areas of Manchester, where many African-Caribbean and Pakistani-origin people lived. Seven larger centers were recruited. The Pakistani community, more easily identifiable by name on the registers, was sampled with age restricted to 35–79 years due to needing Urdu-speaking staff (coordinated by F.K.). Up to two appointment letters were sent and were signed by the patient's doctor to encourage attendance. Ethical approval was obtained from the local Ethics Committee. All participants provided written con-

From the Clinical Epidemiology Group, University of Manchester Medical School, Manchester, M13 9PT, U.K. E-mail: clinep@man.ac.uk.
Address correspondence and reprint requests to Dr. J.K. Cruickshank, University of Manchester Medical School, Manchester M13 9PT, U.K.
Received for publication 22 December 2000 and accepted in revised form 12 April 2001.
A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

sent and were advised that if they were diagnosed as having diabetes, then they would be referred to their primary care physician.

At interview, participants were asked the ethnicity of their grandparents, requiring three sharing the same ethnic group to classify. In addition, birthplaces of grandparents and parents were ascertained. When these were unknown, participants selected their ethnic group from the Census list (28). African-Caribbean was defined as Caribbean origin and African descent. People of direct West African origin were excluded, because numbers were low and cultural and dietary habits are quite different from African-Caribbeans. Other ethnic groups were not included in regression models because of low numbers.

After completion of a questionnaire of personal and family history, carefully standardized anthropometry was performed with the patient wearing light clothing (after removing shoes). Weight was measured using calibrated scales to 0.1 kg and height was measured to 0.1 cm using a stadiometer with the head in the Frankfort plane. BMI was recorded as weight (kilograms) divided by height (meters) squared. Two waist and hip circumferences were measured using a thin metal tape to 0.1 cm. If readings differed by >0.5 cm, a third measurement was recorded and the mean of all measurements was analyzed (27).

Physical activity type, both recreational and occupational, and duration over the past week was assessed by a validated questionnaire (29) and allocated into one of five categories (light housework only to sustained vigorous activity).

Participants were asked (in Punjabi or Urdu, if necessary) whether they had ever been diagnosed as having diabetes ("sugar"). Glucose tolerance tests were performed in individuals without a previous diagnosis, randomly sampling one third of Europeans, two thirds of African-Caribbeans, and all Pakistanis. After venipuncture fasting, participants drank 75 g anhydrous glucose in 300 ml water over 2–3 min, and blood samples were collected exactly 120 min later. Plasma glucose was assayed using a standard glucose oxidase analyzer; results were classified primarily by the 1999 World Health Organization criteria (30), with diabetes defined as fasting blood glucose level ≥7.0 mmol/l and/or 2-h blood glucose level ≥11.1 mmol/l, a combination of recent,

Table 1—*Basic characteristics of the male and female participants aged 35–79 years*

	European		African-Caribbean		Pakistani		Total	
	Men (n = 219)	Women (n = 252)	Men (n = 131)	Women (n = 185)	Men (n = 67)	Women (n = 65)	Men (n = 502)	Women (n = 520)
Age (years)	56.0 (54–57)	55.6 (54–57)	58.1 (57–60)	54.8 (53–56)	50.4 (48–53)	50.5 (48–53)	55.8 (55–57)	54.5 (54–55)
Percent born in U.K.	65.6 (59–72)	73.1 (68–79)	2.3 (0–5)	2.7 (0–5)	0	0	29.5 (26–34)	37.4 (33–42)
Time in U.K. (years)	34.8 (29–41)	34.5 (30–39)	34.0 (32–36)	31.9 (31–33)	24.6 (22–27)	19.3 (17–22)	30.8 (29–32)	27.7 (26–29)
Household income (%)								
<$15,000 (£10,000)	63.6 (57–70)	69.3 (64–75)	64.6 (57–72)	63.1 (56–70)	56.3 (44–68)	31.1 (20–42)	68.9 (65–73)	75.2 (71–79)
Percent single	18.0 (13–23)	12.0 (8–16)	18.0 (12–24)	20.0 (14–26)	0	1.5 (0–4.5)	23.5 (20–27)	23.9 (21–27)
Percent physically active								
3 × 20 min/week	37.8 (23–53)	29.4 (13–46)	25.4 (14–37)	33.8 (23–45)	6.8 (0–13)	5.2 (0–11)	22.0 (16–28)	22.7 (16–29)
In manual tasks	30.6	14.7 (15–28)	25.4 (19–43)	35.2 (24–46)	10.2 (2–18)	3.4 (0–8)	21.4 (15–28)	19.6 (13–26)
Age leaving school (years)	15.3 (15–15)	15.7 (15–16)	16.3 (15–18)	18.4 (16–21)	17.6 (15–20)	17.1 (11–23)	15.5 (15–16)	15.6 (15–16)
Height (cm)	170.3 (169–171)	159.2 (158–160)	171.0 (170–172)	160.8 (160–162)	168.4 (167–170)	156.0 (154–158)	170.2 (170–171)	159.4 (159–160)
Weight (kg)	79.5 (78–81)	69.0 (67–71)	78.7 (77–81)	78.0 (76–80)	78.1 (75–81)	72.2 (69–75)	78.6 (77–80)	72.3 (71–74)
BMI (kg/m^2)	27.4 (27–28)	27.2 (27–28)	26.9 (26–27)	30.2 (29–31)	27.5 (27–29)	29.6 (28–31)	27.1 (27–27)	28.4 (28–29)
Waist (cm)	96.6 (95–98)	84.6 (83–86)	92.8 (91–94)	91.3 (89–93)	99.9 (98–102)	96.5 (94–99)	95.4 (94–96)	88.4 (87–90)
Hip (cm)	103.4 (102–105)	104.8 (103–106)	101.0 (100–102)	109.2 (107–111)	104.7 (102–107)	109.3 (107–112)	102.4 (102–103)	107.0 (106–108)
Waist-to-hip ratio	0.92 (0.92–0.94)	0.81 (0.80–0.82)	0.92 (0.91–0.93)	0.84 (0.83–0.85)	0.96 (0.94–0.97)	0.88 (0.86–0.91)	0.92 (0.92–0.94)	0.83 (0.82–0.83)

Data are means (95% CI).

Table 2—*Age-standardized prevalence of known and newly detected type 2 diabetes using 1999 and 1985 World Health Organization criteria and 1997 American Diabetes Association criteria among European, African-Caribbean, and Pakistani individuals aged 35–79 years*

	European		African-Caribbean		Pakistani		Total	
	Men	Women	Men	Women	Men	Women	Men	Women
Number screened	222	259	180	188	67	65	517	546
Known diabetes	8.0 [18]	3.7 [11]	15.7 [36]	16.2 [28]	14.0 [9]	18.2 [12]	11.4 [69]	9.2 [55]
	(4.7–11.3)	(1.5–5.9)	(10.7–20.7)	(11.5–20.9)	(5.7–22.3)	(8.8–27.6)	(8.8–14.0)	(7.0–11.4)
Glucose tested (*n*)	66	83	76	95	52	52	206	243
New diabetes*	14.1 [12]	16.8 [14]	9.2 [7]	6.0 [7]	18.0 [9]	21.9 [11]	13.5 [29]	12.9 [32]
(WHO 1999)	(5.7–22.5)	(8.8–24.9)	(2.7–15.7)	(1.2–10.7)	(7.6–28.5)	(10.7–33.2)	(8.9–18.2)	(8.7–17.1)
Known and new	20.8 [30]	19.9 [25]	23.4 [43]	20.8 [35]	29.9 [18]	35.7 [23]	23.3 [98]	20.8 [87]
(WHO 1999)	(15.5–26.1)	(15.1–24.8)	(17.2–29.6)	(15.0–26.6)	(19.0–40.9)	(24.0–47.3)	(19.6–26.9)	(17.4–24.2)
Minimum prevalence†	12.4 [30]	9.2 [25]	19.6 [43]	19.0 [35]	28.0 [18]	35.3 [23]	16.9 [98]	14.9 [87]
	(8.0–16.7)	(5.7–12.8)	(13.8–25.4)	(13.4–24.7)	(17.3–38.8)	(23.6–46.9)	(13.7–20.2)	(12.0–17.9)
Known and new	18.0 [25]	11.8 [18]	23.7 [42]	20.9 [33]	26.2 [15]	36.4 [22]	20.9 [89]	18.4 [77]
(WHO 1985)	(13.3–22.7)	(8.11–15.5)	(17.9–29.5)	(15.7–26.1)	(15.7–36.7)	(24.7–48.1)	(17.6–24.2)	(15.4–21.4)
Known and new	19.2 [28]	18.8 [24]	22.9 [42]	18.3 [32]	22.2 [14]	26.7 [14]	20.7 [91]	17.2 [77]
(ADA 1997)	(14.4–24.0)	(14.3–23.3)	(17.1–28.7)	(13.3–23.3)	(12.2–32.2)	(15.9–37.5)	(17.4–24.0)	(14.3–20.1)

Data are % [*n*] (95% CI). Age standardized to the 1991 combined male and female population of England and Wales. Note that 255 individuals aged 25–34 years were not included because no comparable Pakistani group had been sampled within this age group (see text). *Prevalence in group without known diabetes attending for glucose tolerance testing; †minimum prevalence assuming that those those individuals who did not undergo glucose challenge would be normoglycernic if tested. ADA, American Diabetes Association; WHO, World Health Organization.)

lower fasting criteria (31) and older 2-h postglucose criteria (32).

We made vigorous efforts to contact nonattenders by telephone or by up to three visits at various times before classifying them as no longer residing in the area. Despite considerable staff time and effort to maintain representative population sampling, not all addresses could be visited.

Data were age-standardized to the 1991 combined male and female population for England and Wales (33) to provide ethnic specific prevalence, using 25–79 years of age and then 35–79 years of age, the age range of the Pakistani sample.

Variables with clearly skewed distributions were log-transformed. Multiple regression tested which factors were independently related to fasting and 2-h plasma glucose levels. Use of standardized coefficients from these allows direct comparison of the strength of association between variables.

RESULTS — Invitation letters were mailed to 3,539 individuals. Of these, 899 individuals (25%) had moved, 561 (16%) were no longer registered, 54 (1%) had died, and 7 were pregnant. Of the remaining 2,018 individuals, 355 (18%) refused screening, 345 (possibly residing at the listed addresses) did not attend more than two appointments, and 1,318 people attended (65%). To allow comparison of prevalence across all three ethnic groups, data in Tables 1 and 2 are restricted to people aged 35–79 years ($n = 1{,}063$).

Poverty indexes were prominent; >60% of participants reported annual incomes <$15,000 (£10,000) (Table 1). Pakistani men were significantly younger than European and African-Caribbean men, in whom mean ages were similar. Of the three ethnic groups, Pakistani men were the shortest and most obese, African-Caribbean men were tallest, and European men were intermediate for all factors. Both the waist and hip circumferences in African-Caribbean men were significantly smaller than in European and Pakistani men, the latter having significantly the largest waists, also reflected in their having the largest waist-to-hip ratios.

Pakistani women were significantly younger (mean age 50.5 years) than European and African-Caribbean women (55.6 and 54.8 years, respectively). Pakistani women were also significantly shorter (156.0 cm) than both European (159.2 cm) and African-Caribbean (160.8 cm) women. African-Caribbean women were the most obese (mean BMI 30.2 kg/m^2), although European and Pakistani women were also generally overweight (BMI >27.4 kg/m^2). Waist-to-hip ratios differed significantly across the ethnic groups: they were lowest in European women (0.81), intermediate in African-Caribbean women (0.84), and highest in Pakistani women (0.88).

Of the 1,318 individuals screened, 1,063 were aged 35–79 years. A total of 124 individuals (11.7%) had existing type 2 diabetes: 6.0% of Europeans ($n = 29$), 17.4% of African-Caribbeans ($n = 64$), and 16% of Pakistanis ($n = 21$) (Table 2). The proportions increased rapidly with age (Fig. 1). One European participant had confirmed type 1 diabetes (not included in the prevalence data and not analyzed further).

Glucose tolerance data were obtained from 449 of these 35- to 79-year-old participants (83% of the 540 people invited). These individuals were of similar mean age (54.5 years, 95% CI 53.5–55.5) to those without glucose tolerance data, but with no previous history of diabetes (55.0 years, 54.0–56.0) and were marginally more obese (28.2 kg/m^2 [27.7–28.6] vs. 27.4 kg/m^2 [26.9–27.9]) but had similar waist-to-hip ratios (0.88 [0.87–0.88] vs. 0.87 [0.86–0.88]). The percentage of individuals who had at least one first-degree relative with diabetes was 19% (15–21%) in those with glucose tolerance data and 18% (16–23%) in those without.

The prevalence of newly detected diabetes in the 35- to 79-year-old individuals (age-standardized to the general

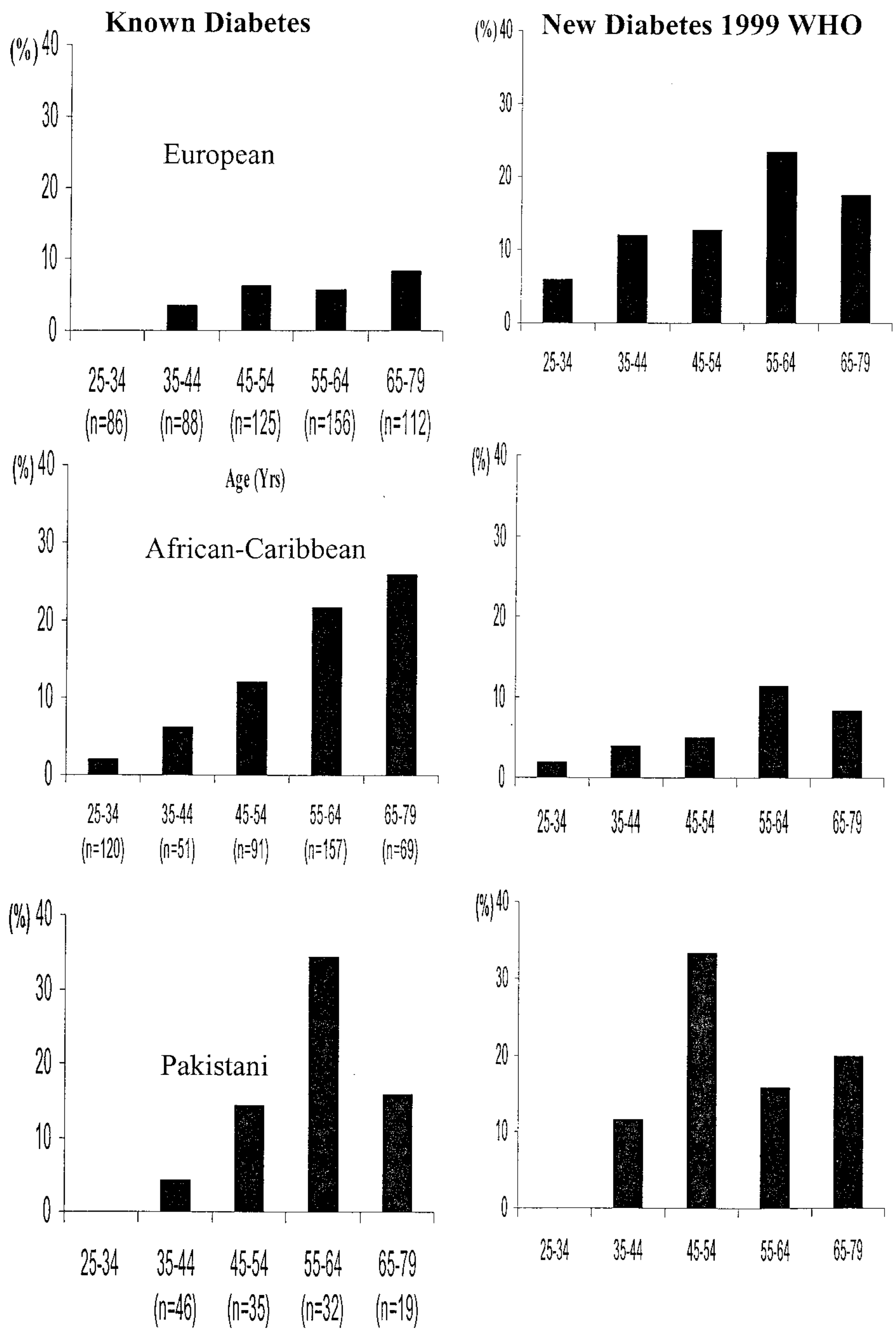

Figure 1—*Prevalence (%) of known and newly detected type 2 diabetes using 1999 World Health Organization criteria by age and ethnic group. (Numbers in parentheses represent number of individuals screened.)*

population) was high in all ethnic/sex groups, using 1999 criteria (Table 2); marked increases corresponded with age (Fig. 1). Prevalence for both older criteria illustrate which age groups are most affected by varying criteria. When 25- to 34-year-old individuals (n = 255) were included, prevalence of newly diagnosed diabetes decreased to 12.9% (9–17%) and 12.8% (9–17%) in European men and women, respectively, and decreased to 7.0% (4–10%) and 5.3% (3–8%) in African-Caribbean men and women, respectively. The distributions of fasting and 2-h blood glucose levels show how relatively small shifts lead to such high proportions of defined diabetes (Fig. 2). Sex differences were minimal.

Finally, what matters for service provision is the combined prevalence for all diabetes (known and newly detected combined). For individuals aged 35–79 years, the prevalence was 20% (17–24%) in Europeans, 22% (18–26%) in African-Caribbeans, and 33% (25–41%) in Pakistanis, based on new 1999 criteria. Based on older criteria, the same age-adjusted prevalence was 15% (12–18%), 22% (15–23%), and 31% (23–39), respectively (32), and the sex-adjusted prevalence was 19% (16–23%), 21% (17–25%), and 24% (17–32%), respectively (31). Even minimum prevalence (assuming all individuals not tested were normoglycemic) was 11% (8–14%), 19% (15–23%), and 32% (24–40%), respectively, based on the new 1999 criteria.

Forward, stepwise, multiple-regression models showed several consistent independent associations with both fasting and 2-h plasma glucose levels. In descending order of impact (shown by standardized β coefficients, P values, and impact, from unstandardized coefficients, as mmol/l glucose change), in a restricted model including physical activity (n = 231), these were higher waist circumference (0.39, $P < 0.001$, 0.88/10 cm), lower height (−0.26, $P < 0.001$, −0.90/10 cm), and age (0.13, $P = 0.04$, 0.28/10 years). Other variables were not significant ($P > 0.1$), so that ethnic differences in this model were entirely accounted for by lower height or greater waist and age. Substituting waist-to-hip ratio and BMI for their components included the latter (0.27, $P < 0.001$, 0.17 per kg/m^2) but not the former ($P = 0.2$) and added physical activity (−0.13, $P = 0.04$, −0.11/quintile). All significant variables contributed 13 and 22% to fasting and 2-h glucose variance; other variables, including income and education, added <1% in a forced inclusion model. The pattern was virtually identical in the fuller model (n = 374) with the omission of incomplete physical activity. Greater waist circumference (0.368, $P < 0.001$, 0.098/10 cm) and lower height (−0.231, $P < 0.001$, −0.092/10 cm) predicted fasting and 2-h plasma glucose levels with European ethnicity (0.132, $P = 0.007$, 0.403) in the fasting and age (0.121, $P = 0.027$, 0.03/10 years) in the 2-h models. In log glucose models, which slightly improved the variance (17 and 25%), income became an additional if borderline contributor (−0.11, $P = 0.08$, −0.02/$7,000).

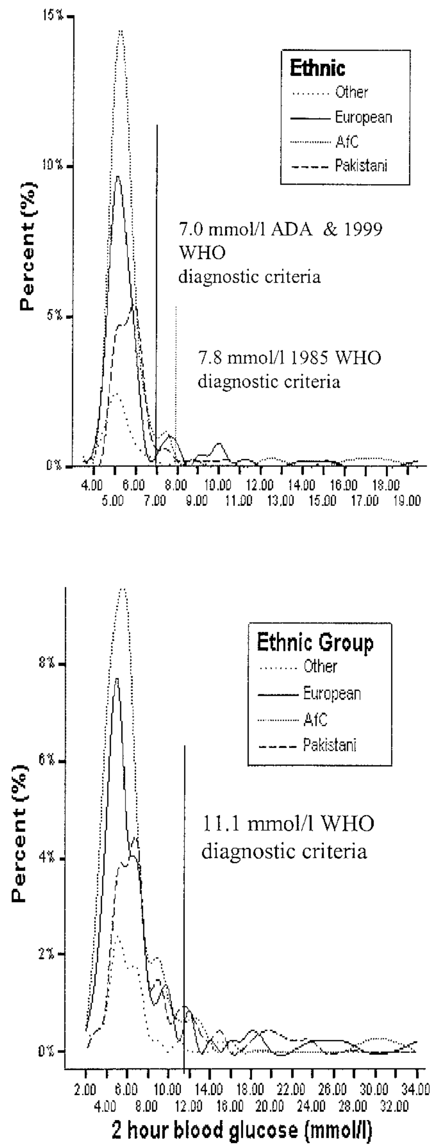

Figure 2—*Fasting and 2-h blood glucose distributions by ethnic group.*

CONCLUSIONS — As in previous English inner-city surveys, ≥30% of individuals could not be located at their listed addresses (8–11,13,34–36). Population mobility was high, partly due to the ongoing major regeneration program. This minimum response to the initial screening of ≥65% is probably a considerable underestimate because it was possible to verify addresses for only two thirds of all nonattenders; one third of the addresses were no longer accurate. We estimated a minimum response of 67% among the European and African-Caribbean groups (indistinguishable by name) and 70% among Pakistanis. Despite fieldworkers visiting addresses up to three times, the status of many individuals could not be ascertained. Therefore, we give a minimum response for the glucose challenge but believe it to be actually closer to 75–80%.

The data illustrate the burden of type 2 diabetes across all ethnic groups in this area, to which health services must respond. To avoid inflation of these prevalences by denominator uncertainty, we also provided "minimum prevalence," which assumes that all individuals not tested were normoglycemic. These still show mean prevalences (1999 criteria) in men and women (combined) of 11% (8–14%) in Europeans, 19% (15–23%) in African-Caribbeans, and 32% (24–40%) in Pakistanis (35–79 years of age), more than twice that found in the Anglo-Saxon Ely community, in which figures were uncorrected for the 74% response (37). Even the lower 95% CIs suggest the scale of the epidemic.

However, the data also illustrate the difficulties of using fasting criteria alone for diagnosing diabetes, as reported elsewhere (38–40). A study of individuals with glycosuria undergoing glucose tolerance testing found that the impaired fasting group were phenotypically different, with fewer men, less apparent insulin resistance, and more β-cell dysfunction than the 2-h impaired group (41). Postprandial rather than fasting glucose level is also more closely associated with cardiovascular disease and death across several ethnic groups (42).

Multiple regression models showed that the most modifiable variable in predicting fasting and 2-h blood glucose was body mass, although of individual components, waist circumference and height were the most prominent.

Height is of particular importance because Pakistanis, in whom prevalence was greatest, were markedly shorter (by 2–5 cm) than other ethnic groups, and in the multiple-regression models, height virtually accounted for ethnic differences in 2-h glucose, as noted previously (8). Intergenerational increases in attained height have occurred in certain ethnic groups (43).

The basis of the diabetes epidemic in individuals of West African descent has been discussed previously (6,15) and was closely associated with increasing BMI across sites, presumably reflecting inevitable declines in physical activity with urbanization. Measurement errors caused by using only reported activity probably reduced its impact in our analysis; when physical activity has been directly measured in populations, its association with lower glucose and higher blood pressure is clear (44,45). There is overwhelming evidence that these two closely related factors, probably through habits developed during adulthood in migrating groups and perhaps intergenerationally in all groups (15,46), are the major promoters of diabetes (47,48). Therefore, public health efforts across inner cites must find solutions, which probably goes beyond health promotion trials aimed just at minor behavioral change. Such trials and other major population-directed interventions seem to not have had much impact yet (49,50). In 1998, Manchester was ranked the third most impoverished area in the U.K. (51). Although the scale of deprivation is nothing compared with current poverty in third-world cities (where undernutrition is rife but coexists with dominant obesity found here), it is salutary that 160 years ago, in a famous commentary by the owner of a cotton factory, living conditions were much worse in this very area, southwest of the city center (52). How long that historical legacy takes to eliminate is unclear, remembering that slavery was only six generations ago, and among the Pakistani women, some 30% are illiterate in their own language (15,53,54). Such is the history of Manchester and many other cities, in which newer migrants have also been relatively impoverished. Poorer socioeconomic status cosegregates with obesity, even in diabetes (21), as do physical inactivity, smoking, and low birth weight, which are all known risk factors for type 2 diabetes. These pervasive influences remain common, but whatever the origins of the high prevalence of diabetes, the implications for services are alarming.

Acknowledgments— This work was supported by grants from the North-West Regional Health Authority local scheme, the National Research & Development Cardiovascular Program (WE16), and the European Commission (TS3*CT92 01242).

We thank the communities surveyed and all the trained field staff, particularly Caron Thomas, Judith Savage, Christine Amica, Caroline Greenwood, the late Stephen Griffiths, and also the local general practitioners and their staff. Without their persistent efforts this work could not have been completed.

References

1. King H, Rewers A: Global estimates for prevalence of diabetes mellitus and impaired glucose intolerance in adults. *World Health Organ Diabetes Rep Group* 16:157–177, 1993
2. Cruickshank JK: Epidemiology and geography of non-insulin dependent diabetes. In *Textbook of Diabetes*. Pickup J, Williams G, Eds. Blackwell, Oxford, U.K., 1997, p. 17–29
3. Miller GJ, Maude GH, Beckles GLA: Incidence of hypertension and non-insulin dependent diabetes mellitus and associated risk factors in a rapidly developing Caribbean community: the St James survey, Trinidad. *J Epidemiol Community Health* 50:497–504, 1996
4. Wilks R, Rotimi C, Bennett F, McFarlane-Anderson N, Kaufman JS, Anderson SG, Cooper RS, Cruickshank JK, Forrester T: Diabetes in the Caribbean: results of a population survey from Spanish Town, Jamaica. *Diabet Med* 16:875–883, 1999
5. Dodu SR: Diabetes in the tropics. *BMJ* 2:747–750, 1967
6. M'Banya JC, Cruickshank JK, Forrester T, Balkau B, Ngogang JY, Riste L, Forhan A, McFarlane-Anderson N, Bennett F, Wilks R: Standardized comparison of glucose intolerance in West African-origin populations of rural and urban Cameroon, Jamaica and Caribbean migrants to Britain. *Diabetes Care* 22:434–440, 1999
7. 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* 60: 390–396, 1988
8. Cruickshank JK, Cooper J, Burnett M, MacDuff J, Drubra U: Ethnic differences in fasting plasma C-peptide and insulin in relation to glucose tolerance and blood pressure. *Lancet* 338:842–847, 1991
9. McKeigue P, Shah B, Marmot MG: Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 337:382–386, 1991
10. Cappuccio FP, Cook DG, Atkinson RW, Strazullo P: Prevalence, detection and management of cardiovascular risk factors in different ethnic groups in South London. *Heart* 78:555–563, 1997
11. Chaturvedi N, McKeigue PM, Marmot MG: Resting and ambulatory blood pressure differences in Afro-Caribbeans and Europeans. *Hypertension* 22:90–96, 1993
12. Cruickshank JK, Beevers DG, Osbourne VL, Haynes RA, Corlett JC, Selby S: Heart attack, stroke, diabetes and hypertension in West Indians, Asians and whites in Birmingham, England. *BMJ* 281:1108, 1980
13. Bhopal R, Unwin N, White M, Yallop J, Walker L, Alberti KGGM, Harland J, Patel S, Ahmad N, Turner C, Watson B, Kaur, Kulkarni A, Laker M, Tavridou A: Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi, and European origin populations: cross sectional study. *BMJ* 319:215–220, 1999
14. Hopper J: Is type II (non-insulin-dependent) diabetes mellitus not so "genetic" after all? *Diabetologia* 42:125–127, 1998
15. Cruickshank JK, Mbanya JC, Wilks R, Balkau B, McFarlane-Anderson N, Forrester T: Sick genes, sick individuals or sick populations with chronic disease? An international example from the emergence of diabetes and high blood pressure in African-origin populations. *Int J Epidemiol* 30:111–117, 2001
16. Fuller JH, Elford J, Goldblatt P, Adelstein AM: Diabetes mortality: new light on an underestimated public health problem. *Diabetologia* 24:336–341, 1983
17. Gardner MJ, Winter PD, Barker DJP: *Atlas of Mortality from Selected Diseases in England and Wales, 1968–78*. Chichester, Wiley, 1984
18. Meadows P: Variation of diabetes mellitus prevalence in general practice and its relationship to deprivation. *Diabet Med* 12: 696–700, 1995
19. Ismail AA, Beeching NJ, Gill GV, Bellis MA: Capture-recapture-adjusted prevalence rates of type 2 diabetes are related to social deprivation. *QJM* 92:707–710, 1999
20. Connolly V, Unwin N, Sherriff P, Bilous R, Kelly W: Diabetes prevalence and socioeconomic status: a population based study showing increased prevalence of type 2 diabetes mellitus in deprived areas. *J Epidemiol Community Health* 54:173–177, 2000
21. Evans JMM, Newton RW, Ruta DA, MacDonald TM, Morris AD: Socio-economic status, obesity and prevalence of type 1 and type 2 diabetes mellitus. *Diabet Med* 17:478–480, 2000
22. Kelly WF, Mahmood R, Turner S, Elliott K: Geographical mapping of diabetes patients from deprived inner city areas shows less insulin therapy and more hyperglycaemia. *Diabet Med* 11:344–348, 1994
23. Cruickshank JK, Mbanya JC, Wilks R, Balkau B, Forrester T, Anderson SG, Mennen L, Forhan A, Riste L, McFarlane-Anderson N: Hypertension in 4 African-origin populations: current 'Rule of Halves', quality of blood pressure control and attributable risk (fraction) for cardiovascular disease. *J Hypertens* 19:41–46, 2001
24. Sharma S, Cade J, Jackson M, Mbanya JC, Chungong S, Forrester T, Bennett F, Wilks R, Balkau B, Cruickshank JK: Development of food frequency questionnaires in 3 population samples of African origin from Cameroon, Jamaica and Caribbean migrants to the UK. *Eur J Clin Nutr* 50:479–486, 1996
25. Sharma S, Cade J, Riste L, Cruickshank JK: Nutrient intakes among a UK African-Caribbean population sample compared with national population data. *Public Health Nutr* 2:469–476, 1999
26. Landman J, Cruickshank JK: A review of ethnicity, health and nutrition-related diseases in relation to migration in the United Kingdom. *Public Health Nutr*. 4:647–657, 2001
27. Riste LK: *Risk Factors Associated with Glucose Intolerance and High Blood Pressure in African-Caribbeans and Whites: Opportunities for Prevention*. PhD Thesis. Manchester, U.K., Manchester University, 1997
28. Office of Population Censuses and Surveys: *1991 Census Ethnic Group and Country of Birth*. Vol. 1. London, HMSO, 1993
29. Washburn RA, Janney CA, Fenster JR: The validity of objective physical activity monitoring in older individuals. *Res Q Exerc Sport* 61:114–117, 1990 *
30. World Health Organization Department of Noncommunicable Disease Surveillance: *Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications Part 1: Diagnosis and Classification of Diabetes Mellitus.* Geneva, World Health Org., 1999
31. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 20:1183–1197, 1997
32. World Health Organization: *Diabetes Mellitus: Report of a Study Group*. Geneva: World Health Org., 1985 (Tech. Rep. Ser., no 727)
33. Office of Population Censuses and Surveys: *1991 Census Report for Great Britain (Part 1)*. Vol. 1. London, HMSO 1993
34. Riste LK: *A Comparison of the Prevalence of Rheumatoid Arthritis in Caribbeans and Caucasians Living in Inner City Manchester.* MSc Thesis. Manchester, U.K., University of Manchester, 1992
35. Silman AJ: Age-sex registers as a screening tool for general practice: size of the wrong address problem (Letter). Br Med J (Clin Res Ed) 289:415–416, 1984
36. Fraser RC: Age-sex registers as a screening tool for general practice: size of the wrong address problem (Letter). *Br Med J (Clin Res Ed)* 289:923–924, 1984
37. Williams DR, Wareham NJ, Brown DC, Byrne CD, Clark PMS, Cox BD, Cox LJ, Day NE, Hales CN, Palmer CR, Shackleton JR, Wang TWM: Undiagnosed glucose intolerance in the community: the Isle of Ely Diabetes Project. *Diabet Med* 12:30–35, 1995
38. von Eckardstein A, Schiltze H, Assman G: Risk for diabetes mellitus in middle-aged Caucasian male participants of the PROCAM Study: implications for the definition of impaired fasting glucose by the

American Diabetes Association. *J Clin Endocrinol Metab* 85:3101–3108, 2000
39. DECODE Study Group: Glucose tolerance and mortality: comparison of WHO and American Diabetes Association diagnostic criteria. *Lancet* 354:617–621, 1999
40. Unwin N, Alberti KGMM, Bhopal R, Harland J, Watson W, White M: Comparison of the current WHO and new ADA criteria for the diagnosis of diabetes mellitus in three ethnic groups in the UK. *Diabetes Care* 15:554–555, 1998
41. Davies MJ, Raymond NT, Day JL, Hales CN, Burden AC: Impaired glucose tolerance and fasting hyperglycaemia have different characteristics. *Diabet Med* 17:433–440, 2000
42. Shaw J, Hodge AM, de Courten M, Chitson P, Zimmet PZ: Isolated post-challenge hyperglycaemia confirmed as a risk factor for mortality. *Diabetologia* 42:1050–1054, 1999
43. Kelly AM, Shaw NJ, Thomas AMC, Pynsent PB, Baker DJ: Growth of Pakistani children in relation to the 1990 growth standards. *Arch Dis Child* 77:401–405, 1997
44. Wareham NJ, Wong MY, Hennings S, Mitchell J, Rennie K, Cruickshank K, Day NE: Quantifying the association between habitual energy expenditure and blood pressure. *Int J Epidemiol* 29:655–660, 2000
45. Wareham NJ, Wong MY, Day NE: Glucose intolerance and physical inactivity: the relative importance of low habitual energy expenditure and cardiorespiratory fitness. *Am J Epidemiol* 152:132–139, 2000
46. Bhatnagar D, Anand IS, Durrington PN, Patel DJ, Wander GS, Mackness MI, Creed F, Tomenson B, Chandrashekhar Y, Winterbotham M: Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India [see comments]. *Lancet* 345:405–409, 1995
47. Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG: Prospective study of risk factors for the development of non-insulin-dependent diabetes in middle-aged British men. *BMJ* 310:560–564, 1995
48. Colditz GA, Willett WC, Rotnitzky A, Manson JE: Weight gain as a risk factor for clinical diabetes in men. *Ann Intern Med* 122:481–486, 1995
49. Imperial Cancer Research Fund OXCHECK Study Group: Effectiveness of health checks conducted by nurses in primary care: final results of the OXCHECK study. *BMJ* 310:1099–1104, 1995
50. Tudor-Smith C, Nutbeam D, Moore L, Catford J: Effects of the Heartbeat Wales programme over five years on behavioural risks for cardiovascular disease: quasi-experimental comparison of results from Wales and a matched reference area. *BMJ* 316:818–822, 1998
51. Manchester City Council Web Page. Available from http: //www.manchester.gov.uk. Accessed November 2000
52. Engels F: *Conditions of the Working Class in England*. London, Metheun, 1988
53. Cruickshank JK: Migration, cardiovascular and general health across the Caribbean diaspora. *West Ind Med J* 49 (Suppl. 2):76–81, 2000
54. Barker DJP: *Mothers, Babies and Health in Later Life*. 2nd ed. Edinburgh, Churchill Livingstone, 1998