

# Prevalence of NIDDM Among Populations of the African Diaspora

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**OBJECTIVE** — Rates of non-insulin-dependent diabetes mellitus have risen sharply in recent years among blacks in the U.S. and the U.K. Increases in risk have likewise been observed in the island nations of the Caribbean and in urban West Africa. To date, however, no systematic comparison of the geographic variation of NIDDM among black populations has been undertaken.

**RESEARCH DESIGN AND METHODS** — In the course of an international collaborative study on cardiovascular disease, we used a standardized protocol to determine the rates of NIDDM and associated risk factors in populations of the African diaspora. Representative samples were drawn from sites in Nigeria, St. Lucia, Barbados, Jamaica, the United States, and the United Kingdom. A total of 4,823 individuals aged 25–74 years were recruited, all sites combined.

**RESULTS** — In sharp contrast to a prevalence of 2% in Nigeria, age-adjusted prevalences of self-reported NIDDM were 9% in the Caribbean and 11% in the U.S. and the U.K. Mean BMI ranged from 22 kg/m<sup>2</sup> among men in West Africa to 31 kg/m<sup>2</sup> in women in the U.S. Disease prevalence across sites was essentially collinear with obesity, pointing to site differences in the balance between energy intake and expenditure as the primary determinant of differential NIDDM risk among these populations.

**CONCLUSIONS** — In ethnic groups sharing a common genetic ancestry, these comparative data demonstrate the determining influence of changes in living conditions on the population risk of NIDDM.

In the last 15 years, NIDDM has been rapidly emerging as a major health threat in populations from both developed and developing countries (1,2). Particularly vulnerable have been minority ethnic populations, including African-Americans, Hispanics, and Native Americans in the U.S. and people in the U.K. who originate from the Indian subcontinent (3–7). In the western hemisphere, the rise in NIDDM prevalence and its subsequent economic burden for a whole society has perhaps been best

documented in the biethnic Caribbean nation of Trinidad and Tobago (8–11). For each ethnic group, NIDDM has been relatively uncommon among the ancestral populations (1), so that the evolution in risk appears to be driven by changes in living conditions, a hypothesis tested more formally here. Mortality from diabetes is now a burden across the Caribbean and Latin America. A detailed study of national death certificates in Jamaica, for example, revealed a 30% under-recognition of diabetes (12).

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FBG, fasting blood glucose; GTT, glucose tolerance test; ICSHIB, International Collaborative Study on Hypertension in Blacks; OR, odds ratio; PAR%, population-attributable risk percent; WHR, waist-to-hip ratio.

In parallel with an emerging understanding of the geographic distribution of NIDDM risk, intense interest has focused on the contribution of potential genetic factors, operating at the population level, for each of these groups. Thus, the unique susceptibility of Native Americans has been widely discussed under the construct of the “thrifty genotype,” and genetic effects have been sought directly among Mexican-Americans (13,14). The alternative and equally cogent hypothesis for NIDDM offering to replace, interact with, and confound such genotypic determinants is the “thrifty phenotype” (15). This theory was originally postulated with regard to low-prevalence groups in Britain, but was subsequently replicated both in Mexican-Americans (16) and in Jamaican schoolchildren (17). Empirical data to fully support either of these constructs, or establish their relative contributions, are not available as yet.

Cross-cultural studies of broadly related populations have brought a new level of sophistication to this area of research by allowing comparisons in a variety of environmental settings (18). Although migration studies or their surrogate, the multinational comparison, offer the potential to elucidate causal processes in chronic disease, they also suffer limitations. The range of social settings occupied by contemporary populations for most ethnic groups is often limited, as is the case among Native Americans. Among populations of African origin, however, the impact of modernization has been relatively limited among rural communities in much of Africa, while genetically similar groups in the U.S. and Europe experience all the consequences of an industrial lifestyle.

We sought to take advantage of the historical dispersion of populations of West Africa to examine the evolution of NIDDM risk. Along this sociocultural gradient are represented societies that include subsistence farming, developing countries, and the working class of highly industrialized urban centers.

## RESEARCH DESIGN AND METHODS

### Participant recruitment

The data for this study were obtained from

Table 1—Prevalence of diabetes by sex in populations of West African origin: the ICSHIB study, 1996

| Site                  | n     | Crude prevalence |       |       | Age-adjusted prevalence |       |       |
|-----------------------|-------|------------------|-------|-------|-------------------------|-------|-------|
|                       |       | Men              | Women | Total | Men                     | Women | Total |
| West Africa (Nigeria) | 247   | 2.8              | 2.9   | 2.8   | 2.0                     | 2.2   | 2.0   |
| Caribbean             | 2,722 | 5.1              | 9.6   | 7.7   | 4.9                     | 9.1   | 7.2   |
| Jamaica               | 820   | 5.3              | 10.9  | 8.6   | 5.3                     | 10.4  | 8.1   |
| St. Lucia             | 1089  | 2.7              | 8.9   | 6.1   | 2.8                     | 9.0   | 6.2   |
| Barbados              | 813   | 8.3              | 9.3   | 8.9   | 7.8                     | 8.4   | 8.2   |
| U.K. (Manchester)     | 336   | 17.3             | 11.8  | 14.4  | 11.0                    | 10.6  | 10.8  |
| U.S. (Maywood)        | 1,518 | 8.1              | 12.7  | 10.5  | 8.5                     | 12.3  | 10.6  |

the International Collaborative Study on Hypertension in Blacks (ICSHIB). As described in detail elsewhere (19–21), a random sample of stable residential communities were recruited. With the exception of Manchester, U.K., where participants were identified through random sampling of population registries of family health services, a door-to-door sampling procedure based on the probability proportional to size method was implemented. A geographic unit, usually a town or district, was identified with a population of about 25,000. A list of subunits, typically city blocks or villages, was then enumerated. A random sample of these subunits were selected in proportion to their size, and a fixed number of individuals were recruited from each. Field staff visited households on multiple occasions and attempted to enroll all adults between the ages of 25 and 74. The sampling strategy called for an equal number of participants in each sex for the age-groups 24–34, 35–44, 45–54, and 55–74 years. In Africa the last age category was open-ended, since many individuals did not know their exact age. Of those contacted, participation in the overall survey ranged from 65% in the U.S. to nearly 100% in rural Africa. Direct assessment of the potential for participation bias was made only in the U.S., since census data at the local level was not available elsewhere. In the U.S. survey, participants did not vary significantly in terms of modifying factors, such as age, sex, or socioeconomic status, from the general population. In each site a standardized set of questionnaires and physical measurements were made in a local clinic. The distribution of participants by site is presented in Table 1.

### Survey measurements

**Anthropometric and blood pressure measurements.** All anthropometric measurements were taken on participants with-

out shoes and in light clothing. Weight was measured on an electronic scale to the nearest 0.1 kg, and height was measured with a stadiometer attached to the wall with a headboard to the nearest 0.1 cm. Waist and hip circumferences were measured using a flexible measuring tape to the nearest 0.1 cm. BMI was calculated as current measured weight in kilograms divided by height in meters squared. Waist-to-hip ratio (WHR) was calculated as waist divided by hip circumference. Because of the narrow range for WHR, the observed values were standardized to z-scores with a mean of zero and standard deviation (SD) of one as follows:  $Z = [Y - \bar{Y}]/SD$ ; where  $Y$  is the observed value,  $\bar{Y}$  is the site-specific mean value, and  $SD$  is the associated standard deviation. Unless otherwise stated, the computed z-scores were used in all analyses evaluating the relationship between diabetes and WHR.

**Diagnostic criteria for diabetes.** For these analyses, the prevalence of diabetes was ascertained from subjects' self-report of a physician diagnosis of diabetes. Specifically, subjects were classified as diabetic if they answered yes to the following question: "Have you been told by a doctor or other health professional that you had diabetes or sugar diabetes?" History of self-reported diabetes was not asked in Nigeria given the unreliability of this information in a setting of very low level of medical screening. Prevalence estimates were, however, obtained from fasting blood glucose (FBG) from a randomly selected subset ( $n = 247$ ) of the Nigerian participants after an overnight (10- to 12-h) fast. Diabetes was defined as FBG  $\geq 6.7$  mmol/l according to the World Health Organization criteria for epidemiological studies using whole blood (22).

**Validation.** The use of self-reported diabetes prevalence may yield an underascertainment of cases due to previously

undiagnosed disease in the community. The magnitude of this bias has been observed to run as high as 20–50%, even among relatively industrialized populations (1). The use of the criterion of FBG  $\geq 6.7$  mmol/l in Nigeria is also subject to potential bias in identifying NIDDM cases. Validation was therefore necessary to demonstrate the accuracy of the prevalence estimates from the current study.

Mbanya et al. (23) conducted glucose tolerance tests (GTT) among a comparably aged urban population in Cameroon and observed adjusted NIDDM prevalence of 1.2 and 2.8% for men and women, respectively. A subset of the Jamaican ( $n = 397$ ) and U.K. participants ( $n = 336$ ) from the present study also received 75-g GTTs, yielding adjusted prevalences of 5.7 and 9.0% for men and women in Jamaica and 12.8 and 9.9% for men and women in the U.K. The GTT estimates of NIDDM prevalence were generally similar to those obtained from self-report of fasting whole blood. The largest absolute discrepancy, a prevalence estimate of 12.8% for men in the U.K. via GTT versus 11.0% by self-report, amounts to a 14% under-ascertainment (95% CI, 9–19%).

**Data analysis.** Statistical analyses were performed using programs available in SAS (24). The Student's  $t$  test was used to assess differences in continuous variables, and the  $\chi^2$  test was used for categorical variables. Both linear and logistic regression analyses were used to model the relationship between diabetes status and known risk factors. Population-attributable risk percent (PAR%) was estimated as follows:

$$\text{PAR\%} = \frac{(\text{Prev}_E)(\text{OR} - 1)}{(\text{Prev}_E)(\text{OR} - 1) + 1}$$

where  $\text{Prev}_E$  is the exposed proportion ( $\text{BMI} \geq 25 \text{ kg/m}^2$ ) and OR is the odds ratio comparing people with  $\text{BMI} < 25 \text{ kg/m}^2$  and people with  $\text{BMI} \geq 25 \text{ kg/m}^2$ , adjusting for age and sex in the logistic model (25).

**RESULTS**— The crude and age-adjusted prevalences of diabetes by site and sex are presented in Table 1. Two percent of adults were found to have diabetes in Nigeria. Within the Caribbean, rates varied from 3% among men in St. Lucia to 11% among women in Jamaica. Findings in the U.S. and the U.K. were similar, yielding an average of  $\sim 11\%$ . With the notable exception of the U.K., there was a clear sex-specific

Table 2—Sex-specific mean level for six anthropometric variables among people of West African origin: the ICSHIB study, 1996

|                   | Height (cm) | Weight (kg) | BMI (kg/m <sup>2</sup> ) | Waist (cm)  | Hip (cm)     | WHR         |
|-------------------|-------------|-------------|--------------------------|-------------|--------------|-------------|
| <b>Men</b>        |             |             |                          |             |              |             |
| Nigeria           | 168.3 ± 7.3 | 61.5 ± 11.0 | 21.7 ± 3.6               | 77.3 ± 8.4  | 88.3 ± 8.2   | 0.88 ± 0.06 |
| Jamaica           | 172.1 ± 6.7 | 69.4 ± 12.7 | 23.4 ± 4.0               | 79.9 ± 11.3 | 95.1 ± 7.8   | 0.84 ± 0.07 |
| St. Lucia         | 173.5 ± 7.5 | 73.0 ± 11.4 | 24.3 ± 3.7               | 82.7 ± 9.5  | 95.3 ± 7.4   | 0.87 ± 0.06 |
| Barbados          | 171.9 ± 7.4 | 76.4 ± 13.2 | 25.9 ± 4.3               | 86.2 ± 11.3 | 97.8 ± 7.7   | 0.88 ± 0.07 |
| U.K. (Manchester) | 172.5 ± 7.4 | 79.3 ± 12.5 | 26.6 ± 3.6               | 91.4 ± 10.5 | 101.3 ± 7.4  | 0.90 ± 0.07 |
| U.S. (Maywood)    | 176.5 ± 7.3 | 84.5 ± 18.0 | 27.1 ± 5.5               | 92.4 ± 14.0 | 103.4 ± 10.7 | 0.89 ± 0.07 |
| <b>Women</b>      |             |             |                          |             |              |             |
| Nigeria           | 158.3 ± 6.7 | 56.6 ± 12.3 | 22.6 ± 4.7               | 73.9 ± 9.6  | 93.5 ± 10.8  | 0.79 ± 0.06 |
| Jamaica           | 160.3 ± 6.1 | 70.4 ± 17.6 | 27.4 ± 6.5               | 82.2 ± 13.0 | 103.0 ± 12.8 | 0.80 ± 0.07 |
| St. Lucia         | 162.3 ± 6.8 | 72.3 ± 17.0 | 27.3 ± 6.2               | 85.5 ± 13.4 | 103.7 ± 13.1 | 0.82 ± 0.07 |
| Barbados          | 160.1 ± 6.4 | 75.2 ± 16.3 | 29.4 ± 6.4               | 87.1 ± 12.6 | 106.7 ± 12.8 | 0.82 ± 0.07 |
| U.K. (Manchester) | 161.5 ± 6.2 | 74.8 ± 16.0 | 28.6 ± 5.9               | 87.1 ± 14.2 | 106.9 ± 12.1 | 0.81 ± 0.08 |
| U.S. (Maywood)    | 163.4 ± 6.4 | 82.4 ± 20.9 | 30.8 ± 7.7               | 91.4 ± 15.4 | 111.8 ± 15.0 | 0.82 ± 0.08 |

variation, with women having the higher prevalence in all the sites.

Mean values and associated standard deviations of the anthropometric variables are displayed in Table 2. Stature increased by an average of 8.2 cm and 5.1 cm for men and women from Nigeria to suburban Chicago. There was little difference between Afro-Caribbeans in the U.K. and in Jamaica, from which >80% of the U.K. sample had migrated an average of 30 years

previously. However, the disproportionate increase in weight led to a striking gradient in average BMI. The leanest group consisted of men in Nigeria, where the mean BMI was 21.7 kg/m<sup>2</sup>. BMI was greater among women in every site. The largest relative difference between men and women was found in Jamaica, where the BMI of women exceeded that of men by 17%. Across geographic locations, site-specific values for BMI were highly related to preva-

lence of diabetes and explained 65% of the geographic variation in an ecological regression model controlling for age, sex, and site (Fig. 1). Because of high levels of collinearity, other anthropometric variables were not included as independent factors in the ecological analysis.

To estimate site-specific trends related to obesity, prevalences were calculated at each quartile of BMI (Table 3). In general, prevalence estimates increased with increasing

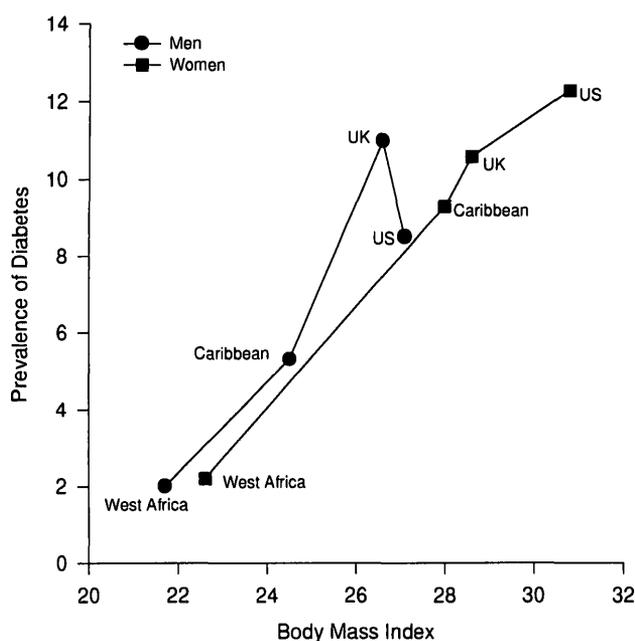


Figure 1—Prevalence of diabetes by mean BMI and sex in populations of West African origin: the ICSHIB Study, 1996. West Africa: Nigeria; Caribbean: Jamaica, St. Lucia, and Barbados; U.K.: Manchester; U.S.: Maywood, IL. Ecological regression  $R^2 = 0.65$ .

Table 3—Age- and sex-adjusted prevalence of diabetes by quartiles of BMI and site: the ICSHIB study, 1996

| Site       | Quartiles of BMI |      |      |      |      |
|------------|------------------|------|------|------|------|
|            | Q1               | Q2   | Q3   | Q4   |      |
| Nigeria    | Prev             | 1.7  | 3.1  | 1.6  | 4.9  |
|            | BMI              | 17.5 | 19.4 | 22.0 | 27.6 |
| Jamaica    | Prev             | 4.5  | 12.5 | 8.2  | 9.8  |
|            | BMI              | 19.6 | 23.3 | 26.8 | 32.8 |
| St. Lucia  | Prev             | 1.8  | 4.1  | 7.0  | 11.2 |
|            | BMI              | 20.1 | 23.9 | 27.0 | 32.8 |
| Barbados   | Prev             | 7.6  | 7.0  | 8.1  | 10.8 |
|            | BMI              | 21.7 | 25.6 | 29.0 | 35.1 |
| Manchester | Prev             | 9.4  | 8.5  | 11.1 | 15.3 |
|            | BMI              | 22.2 | 23.2 | 28.9 | 34.4 |
| Maywood    | Prev             | 6.6  | 9.9  | 9.0  | 17.5 |
|            | BMI              | 21.9 | 26.3 | 30.3 | 38.4 |

Prev, prevalence of diabetes.

Table 4—Prevalence of self-reported diabetes by age-groups, BMI, and site: the ICSHIB study, 1996

| Site       | Age-groups |       |       |      |
|------------|------------|-------|-------|------|
|            | 25-34      | 35-44 | 45-55 | 55+  |
| Nigeria    |            |       |       |      |
| Prev       | 0.0        | 0.0   | 5.7   | 2.9  |
| BMI        | 21.8       | 22.0  | 22.6  | 20.7 |
| Jamaica    |            |       |       |      |
| Prev       | 1.9        | 6.3   | 9.7   | 15.9 |
| BMI        | 25.1       | 26.1  | 26.3  | 25.1 |
| St. Lucia  |            |       |       |      |
| Prev       | 0.6        | 2.7   | 7.7   | 15.0 |
| BMI        | 25.2       | 26.0  | 26.7  | 26.2 |
| Barbados   |            |       |       |      |
| Prev       | 1.5        | 4.2   | 12.2  | 16.3 |
| BMI        | 26.9       | 27.2  | 28.9  | 28.3 |
| Manchester |            |       |       |      |
| Prev       | 2.0        | 9.0   | 13.5  | 20.5 |
| BMI        | 25.6       | 26.8  | 28.1  | 29.2 |
| Maywood    |            |       |       |      |
| Prev       | 2.2        | 5.3   | 10.4  | 25.7 |
| BMI        | 28.5       | 28.5  | 30.0  | 30.0 |

Prev, prevalence of diabetes adjusted for sex.

quartiles of BMI, although the pattern was somewhat uneven between the second and third quartiles across sites. The observed uneven trend in the middle quartiles may be due in part to small numbers of affected participants in some sites. The pattern by age was more consistent, except among the oldest group in Nigeria, where selective mortality may have been a factor (Table 4).

The impact of WHR on diabetes prevalence was also examined across sites (Table 5). Because the distribution of values for WHR is narrower, populations were divided into tertiles based on the data from all sites combined. In all groups, a consistent monotonic increase in prevalence was noted as WHR increased, with a fourfold excess in

Table 5—Prevalence of self-reported diabetes by age-groups, WHR, and site: the ICSHIB study, 1996

| Site       | Tertiles of WHR |           |       |
|------------|-----------------|-----------|-------|
|            | ≤0.81           | 0.81-0.87 | ≥0.88 |
| Nigeria    | 0.0             | 2.0       | 5.1   |
| Jamaica    | 5.7             | 9.6       | 16.2  |
| St. Lucia  | 2.4             | 5.1       | 11.3  |
| Barbados   | 4.2             | 9.0       | 14.3  |
| Manchester | 5.7             | 13.2      | 20.9  |
| Maywood    | 5.9             | 7.7       | 17.8  |

Prevalence of diabetes adjusted for sex, per 100.

Table 6—ORs, 95% CI, and PAR% comparing people with BMI <25 to those with BMI ≥25: the ICSHIB study, 1996

| Site              | Percentage BMI ≥25 | OR (95% CI)   | PAR% |
|-------------------|--------------------|---------------|------|
| Nigeria           | 18.6               | 1.8 (0.3-9.7) | 13.0 |
| Jamaica           | 48.6               | 1.5 (0.9-2.6) | 20.0 |
| St. Lucia         | 51.9               | 2.5 (1.4-4.4) | 43.8 |
| Barbados          | 65.7               | 1.6 (0.9-2.8) | 28.3 |
| U.K. (Manchester) | 67.4               | 1.6 (0.8-3.2) | 28.8 |
| U.S. (Maywood)    | 69.1               | 1.8 (1.2-2.7) | 35.6 |

OR is from the logistic regression model adjusted for age and sex.

the highest compared with the lowest tertile. Thus, at widely varying levels of overall obesity across sites as defined by BMI, WHR was independently associated with risk within each society, with an odds ratio (OR) that stabilized around 1.4 from a logistic model controlling for BMI, age, and sex.

Assessment of the impact of overweight was further examined through use of multiple logistic regression analysis (Table 6). In comparison with people with a BMI <25, a BMI of 25 or higher was associated with an OR that stabilized around 1.6; a single exception to this pattern was noted, namely in St. Lucia where the OR was 2.5. The addition of WHR to this model reduced the impact of BMI by varying degrees across site. PAR% was assessed by site, using as the reference category people with a BMI <25 kg/m<sup>2</sup>. PAR% estimates ranged from a low of 13% in Nigeria to a high of 48% in St. Lucia (Table 6). These findings suggest that if reduction in weight has a similar effect as found in other populations, encouraging individuals in these societies to maintain weight in the range of a BMI of 25 would reduce the burden of diabetes (26).

**CONCLUSIONS** — The history of the African diaspora has created a broad spectrum of modern populations that display the full range of risk for cardiovascular diseases. The prevalence of hypertension among U.S. blacks is among the highest reported, particularly in the rural South (27,28). Coronary heart disease is likewise more common among U.S. blacks than among whites, and this difference is most marked for women (29). A wide range of hypertension prevalences have now been documented for populations of West African origin, ranging from 16% in rural Nigeria to 33% in the urban U.S. (19). In the comparative study presented here, a similarly broad pattern of risk can be

observed for NIDDM. While prevalences are not likely to be more than 2% in a population sample of comparable age structure in West Africa, among blacks in the U.S. and the U.K. about 1 in 10 adults over the age of 25 will have been diagnosed with NIDDM.

Standardized surveys reviewed by King and Rewers (1) demonstrated a range of prevalences from about 1% in China to 40% among the Pima Indians and Melanesians, although U.S. blacks and East Africans were the only black populations included. The current report provides evidence of a finely graded population risk from a lower extreme among the lean and physically active Nigerians to the high levels in the largely sedentary population in the U.S. While we assessed individual risk only through anthropometric values, it should be recognized that these are at best a proxy for obesity itself and may also reflect low levels of physical activity.

In a complementary study in the same populations in Nigeria, Jamaica, and the U.S., we demonstrated that while BMI and percent body fat are reasonably well correlated within each group ( $r = 0.75$ ), large differences in body composition existed at the same absolute level of BMI (30). Thus, adipose tissue comprised ~15% of body weight for a man with a BMI of 15 kg/m<sup>2</sup> in Nigeria and 30% for a man with a comparable BMI in the U.S.. This disparity most certainly represents the training effect of exercise.

Although excess body fat may have some direct negative metabolic consequences, it is possible to think of overweight as an indicator variable for sedentarism. Despite the recognized importance of low levels of physical activity as a predisposing factor for NIDDM, relatively few opportunities exist to break the confounding between obesity and a sedentary lifestyle; this confounding is particularly difficult to overcome at the individual level in societies like the U.S. where virtually

everyone is sedentary. Ecological comparisons can sometimes provide an alternative approach to the study of highly correlated factors in homogeneous groups.

Based on studies of energy expenditure in The Gambia and our own observations in Chicago, a 60-kg individual expends 15% more calories in physical activity in Africa, or about 350 kcal/day (31). This one-to-one correspondence of inactivity and obesity may not hold for all the groups we studied, however. The lack of correspondence may in turn influence patterns of NIDDM prevalence, which was 2% in Nigerian men and 5% in Jamaican men. Jamaican men were almost as lean as Nigerians, where physical work is much more a necessity of life. A twofold male:female differential in diabetes risk was also apparent in Jamaica, where the sexual dimorphism of BMI was most striking (21). Low levels of obesity among Jamaican men might be a result of relatively low calorie intake and moderate physical activity, and it would be of interest to examine energy expenditure directly in this group. Thus, it may be possible in future studies to utilize the ecological contrasts identified here as opportunities to examine aspects of risk which are difficult to assess at the individual level.

Approximately 75% of the ancestral population of U.S. blacks came from the coastal region of West Africa, from The Gambia to Zaire (32), and roughly half of those caught up in the European slave trade were from the region corresponding to modern-day Nigeria (33). It is now well documented that African populations are of greater internal genetic diversity than are populations outside Africa (34). This heterogeneity is further increased in the western hemisphere by the admixture of European genes, currently estimated at 25% among U.S. blacks (35). Only approximate answers can be offered, therefore, on the general relatedness of these groups. More useful information will be derived from studies of specific candidate loci for the medical condition of interest. Early findings do suggest strong similarities for specific alleles in genes associated with blood pressure control between Nigerians, Jamaicans, and U.S. blacks (36,37). Although candidate genes for NIDDM have been identified (38,39), definition of genes that condition risk will require much more work. Estimating the average genetic risk of specific populations will be an even more difficult task. These considerations highlight the need for caution in declaring

"genetic predisposition" as the explanation for increased risk of NIDDM in U.S. minority groups (14,40-43).

Several limitations must be taken into account in the interpretation of these data. Analyses were based on medically diagnosed cases; clearly a substantial proportion of undiagnosed individuals were present in these population samples, and this proportion may have varied across sites. As described above, however, validation data are available from each of the regions. Although the screening procedure used in Nigeria was insensitive, it was sufficient to rule out a prevalence greater than 1-3%. Moreover, large community-based surveys in Cameroon and Tanzania have confirmed a prevalence of 1-2% based on glucose tolerance testing (1,2,23).

A more likely source of underestimation is the high mortality that may be experienced by people with diabetes in sub-Saharan Africa, which would reduce prevalence. GTTs were available in the survey samples from Jamaica and Manchester, as noted, and the prevalence data on blacks from national surveys in the U.S. are broadly consistent with our findings (7,44). In addition, the ORs associated with obesity within each sample were generally consistent, further suggesting that little differential bias in the method of ascertainment occurred. WHR played a similar role, although the differential impact of these anthropometric variables across sites was not explored given the limitations in sample size.

In conclusion, in this series of community-based surveys we have documented a wide range of NIDDM risk among populations of the African diaspora. This distribution is highly correlated with obesity, which is likely to reflect the underlying differences in levels of physical activity. Overweight was associated with a surprisingly consistent relationship to disease risk within each group, and in the ecological analysis it accounted for the majority of the between-site variability in prevalence. Direct measurement of calories expended in physical exercise would provide useful additional insight into the more fundamental determinants of NIDDM risk among these groups.

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## References

1. King H, Rewers M: Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care* 16:157-177, 1993
2. Swai AB, Lutale J, McLarty DG: Diabetes in tropical Africa: a prospective study, 1981-7. I. Characteristics of newly presenting patients in Dar es Salaam, Tanzania, 1981-7. *Br Med J* 300:1103-1106, 1990
3. Cowie CC, Harris MI, Silverman RE, Johnson EW, Rust KF: Effect of multiple risk factors on differences between blacks and whites in the prevalence of non-insulin-dependent diabetes mellitus in the United States. *Am J Epidemiol* 137:719-732, 1993
4. Raymond CA: Diabetes in Mexican-Americans: pressing problem in a growing population. *JAMA* 259:1772, 1988
5. Cruickshank JK: Diabetes: contrasts between people of black (West African), Indian and white European origin. In *Ethnic Factors in Health and Disease*. Cruickshank JK, Beevers DG, Eds. London, Wright, 1989, p. 289-304
6. 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
7. Cruickshank JK, Cooper J, Burnett M, MacDuff J, Drubra U: Ethnic differences in fasting C-peptide and insulin in relation to glucose tolerance and blood pressure. *Lancet* 338:842-847, 1991
8. Poon-King T, Henry MV, Rampersad F: Prevalence and natural history of diabetes in Trinidad. *Lancet* i:155-160, 1968
9. Beckles GLA, Miller GJ, Kirkwood BR, Alexis SD, Carson DC, Byam N: High total and cardiovascular disease mortality in adults of Indian descent in Trinidad, unexplained by coronary risk factors. *Lancet* i:1298-1301, 1986
10. Miller GJ, Kirkwood BR, Beckles GLA, Alexis SD, Carson DC, Byam N: 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* 17:62-69, 1988
11. Gulliford MC: Epidemiological transition in Trinidad and Tobago, West Indies 1953-1992. *Int J Epidemiol* 25:357-365, 1996
12. Alleyne SI, Cruickshank JK, Golding AL, Morrison EY: Mortality from diabetes mellitus in Jamaica. *Bull Pan Am Health Org* 23:306-314, 1989
13. Chakraborty R, Ferrell RE, Stern MP, Haffner SM, Hazuda HP, Rosenthal M: Relationship of prevalence of non-insulin-dependent diabetes mellitus to Amerindian admixture in the Mexican Americans of San Antonio, Texas. *Genet Epidemiol*

- 3:435-454, 1986
14. Zimmet P, Serjeantson S, Dowse G, Finch C, Collins V: Diabetes mellitus and cardiovascular disease in developing populations: hunter-gatherers in the fast lane. In *Sugars in Nutrition*. Nestlé Nutrition Workshop Series, Vol. 25. Gracy M, Kretchmer N, Rossi E, Eds. New York, Vevey/Raven, 1991, p. 19
  15. Hales CN, Barker DJP: Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 35: 595-601, 1992
  16. Stern MP: Diabetes and cardiovascular disease: the "common soil" hypothesis. *Diabetes* 44:369-374, 1995
  17. Forrester TE, Wilks RJ, Bennett FI, Simeon D, Osmond C, Allen M, Chung AP, Scott P: Foetal growth and cardiovascular risk factors in Jamaican school-children. *Br Med J* 312:156-160, 1996
  18. Stern MP, Gonzalez C, Mitchell BD, Villapando E, Hafner SM, Hazuda HP: Genetic and environmental determinants of type II diabetes in Mexico City and San Antonio. *Diabetes* 41:484-492, 1992
  19. Cooper RS, Rotimi CN, Ataman SL, McGee D, Osotimehin B, Kadiri S, Muna W, Kingue S, Fraser H, Forrester T, Bennett F, Wilks R: Prevalence of hypertension in seven populations of African origin. *Am J Public Health*. In press
  20. Ataman SL, Cooper RS, Rotimi CN, McGee D, Osotimehin B, Kadiri S, Kingue S, Muna W, Fraser H, Forrester T, Wilks R: Standardization of blood pressure measurement in an international comparative study. *J Clin Epidemiol* 49:869-877, 1996
  21. Rotimi CN, Cooper RS, Ataman SL, Osotimehin B, Kadiri S, Muna W, Kingue S, Fraser H, McGee D: Distribution of anthropometric variables and the prevalence of obesity in populations of West African origin: the International Collaborative Study on Hypertension in Blacks (ICSHIB). *Obesity Res* 3 (Suppl. 2):95S-105S, 1995
  22. World Health Organization: *WHO Expert Committee on Diabetes Mellitus. Second Report*. Geneva, World Health Organization, 1980 (Tech. Rep. Ser. no. 626)
  23. Mbanya JC, Wilks R, Bennett F, Jackson M, Forrester T, Riste L, Forhan A, Balkau B, Cruickshank JK: Standardized study of glucose tolerance and diabetes prevalence in four African (origin) populations in Cameroon, Jamaica, and migrants to Britain. *Diabetologia*. In press
  24. *SAS User's Guide: Statistics, Version 6*. Cary, NC, SAS Institute Inc, 1989
  25. Hennekens CH, Buring JE: *Epidemiology in Medicine*. Boston, Little, Brown, 1987, p. 90-93
  26. Wing RR, Koeske R, Epstein LH, Nowalk P, Gooding W, Becker D: Long-term effects of modest weight loss in type II diabetic patients. *Arch Intern Med* 147:1749-1753, 1987
  27. Schoenberger JA, Carter M, Eckenfels EJ, Frate D, Logan E, Nelson K, Pelz W, Rois-tacher R, Shimkin D: Hypertension in Holmes County, Mississippi. In *Epidemiology and Control of Hypertension*. Paul O, Ed. Miami, FL, Symposia Specialists, 1975, p. 485-502
  28. Prineas RJ, Gillum RF: U.S. epidemiology of hypertension in blacks. In *Hypertension in Blacks: Epidemiology, Pathophysiology and Treatment*. Hall WD, Saunders E, Shulman NB, Eds. Chicago, Year Book, 1985, p. 17-37
  29. Liao Y, Cooper RS: Continued adverse trends in coronary heart disease mortality among blacks, 1980-1991. *Public Health Reports* 110:572-579, 1995
  30. Luke A, Durazo-Arvizu R, Rotimi C, Pre-witt TE, Forrester T, Ogunbiyi OJ, Owoaje E, Schoeller DA, McGee D, Cooper RS: Relationship between body mass index and body fat in black population samples from Nigeria, Jamaica and the United States. *Am J Epidemiol*. In press
  31. Singh BYJ, Prentice AM, Diza E, Coward WA, Ashford J, Sawyer M, Whitehead RG: Energy expenditure of Gambian women during peak agricultural activity measured by the doubly-labeled water method. *Br J Nutr* 62:315-329, 1989
  32. Curtin PD: *The Atlantic Slave Trade: A Census*. Milwaukee, WI, University of Wisconsin Press, 1969
  33. Harris JE: *Global Dimensions of the African Diaspora*. Washington, DC, Howard University Press, 1993
  34. Hill AVS, Allsopp CEM, Kwiatkowski D, Taylor TE, Yates SNR, Anstey NM, Wirima JJ, Brewster DR, McMichael AJ, Molyneux ME: Extensive genetic diversity in the HLA class II region of Africans, with a focally predominant allele, DRB1\*1304. *Proc Natl Acad Sci USA* 89:2277-2281, 1992
  35. Chakraborty R, Kamboh MI, Ferrel RE: 'Unique' alleles in admixed populations: a strategy for determining 'hereditary' population differences of disease frequencies. *Ethn Dis* 1:245-256, 1991
  36. Rotimi C, Morrison L, Cooper R, Oyejide C, Effiong E, Ladipo M, Osotimehin B, Ward R: The role of the angiotensinogen gene in human hypertension: lack of an association of the M235T allele among African Americans. *Hypertension* 24:591-594, 1994
  37. Rotimi C, Puras A, Cooper RS, McFarlane-Anderson N, Morrison L, Ogunbiyi O, Forester T, Ward R: Polymorphisms of the genes in the renin-angiotensin system among Nigerians, Jamaicans and African Americans. *Hypertension* 27:558-563, 1996
  38. Ferrell RE, Iyengar S: Molecular studies on the genetics of non-insulin dependent diabetes mellitus. *Am J Hum Biol* 5:415-424, 1993
  39. Hanis CL, Boerwinkle E, Chakraborty R, Ellsworth DL, Concannon P, Stirling B, Morrison VA, Wapelhorst B, Spielman RS, Gogolinewens KJ, Shephard JM, Williams SR, Risch N, Hinds D, Iwasaka N, Ogata M, Omori Y, Petzold C, Rietzsch H, Schroder HE, Schulze J, Cox NJ, Menzel S, Boriraj VV, Chen X: A genome-wide search for the human non-insulin dependent (type 2) diabetes genes reveals a major susceptibility locus on chromosome 2. *Nat Genet* 13:161-166, 1996
  40. Harris MI: Epidemiologic correlates of NIDDM in Hispanics, whites and blacks in the US population. *Diabetes Care* 14 (Suppl. 3):639-648, 1991
  41. Cooper RS, Rotimi C: Hypertension in populations of West African origin: is there a genetic predisposition? *J Hypertens* 12:215-227, 1994
  42. Fujimoto WY, Leonetti DL, Kinyhoun JL, Newell-Morris L, Shuman WP, Stolov WC, Wahl PW: Prevalence of diabetes mellitus and impaired glucose tolerance among second-generation Japanese-American men. *Diabetes* 36:721-729, 1987
  43. *Diabetes in America*, 2nd Edition. Bethesda, MD, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 1995 (NIH publ. no. 95-1468)