Incident Type 2 Diabetes Mellitus in African American and White Adults
The Atherosclerosis Risk in Communities Study

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Diabetes mellitus imposes a major burden on the public health of the United States, leading annually to more than 300,000 deaths and about $100 billion in total costs. Approximately 90% of diabetic Americans are classified as having type 2 diabetes. Data from studies of nationally representative samples indicate that, compared with their white counterparts, African American men are 20% to 50% more likely and African American women more than 100% more likely to have or to develop diabetes. One possible explanation for this excess risk is racial differences in the prevalence of established risk factors for type 2 diabetes, such as adiposity, physical inactivity, low socioeconomic status, and family history of diabetes. The identification of potentially modifiable risk factors as contributors to excess diabetes risk in African Americans would suggest possible targets for prevention strategies. Unfortunately, previous investigations in this area have been limited by cross-sectional study designs, samples atypical of the general population, and lack of data on diabetes-related health behaviors and traits, such as fasting blood glucose levels.

We conducted a prospective study of a community-based, biracial cohort of middle-aged adults with the following objectives: to compare the risk of incident diabetes in African Americans vs whites, to determine the extent to which excess diabetes risk in African Americans was explained by racial differences in established diabetes risk factors, and to identify explanatory factors for racial disparities.

Setting and Participants A total of 2646 African American and 9461 white adults aged 45 to 64 years without diabetes at baseline, sampled from 4 US communities.

Main Outcome Measures Incident type 2 diabetes, ascertained by self-report of physician diagnosis, use of diabetes medications, or fasting glucose level of at least 7.0 mmol/L (126 mg/dL), compared among white and African American subjects and by presence of potentially modifiable risk factors.

Results Diabetes incidence per 1000 person-years was about 2.4-fold greater in African American women (25.1 [95% confidence interval, 22.4-28.1] vs 10.4 [95% CI, 9.4-11.4]) and about 1.5-fold greater in men (23.4 [95% CI, 19.9-27.2] vs 15.9 [95% CI, 14.6-17.2]) than in their white counterparts (P < .001). Results from proportional hazards regression models indicated that racial differences in potentially modifiable risk factors, particularly adiposity, accounted for 47.8% of the excess risk in African American women but accounted for little excess risk in African American men. Compared with their white counterparts, African American men and women had higher blood pressures before diabetes onset (diastolic blood pressure difference = 5.6 mm Hg in women and 8.4 mm Hg in men; P = .005).

Conclusions Our data indicate that compared with their white counterparts, middle-aged African Americans are at greater risk of developing type 2 diabetes and have higher blood pressure prior to development of diabetes. In women, almost 50% of this excess risk might be related to potentially modifiable factors.

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tors, and to compare diabetes-related traits (eg, blood pressure and plasma lipid concentrations) in African Americans and whites 3 to 9 years before the onset of diabetes. We hypothesized that an adverse profile of potentially modifiable risk factors in African Americans would lead to a substantial racial disparity in incident diabetes risk.

METHODS

Setting

The Atherosclerosis Risk in Communities (ARIC) Study is an ongoing, longitudinal cohort study of atherosclerotic cardiovascular disease in 15792 adults aged 45 to 64 years at baseline. The cohort was selected by probability sampling from 4 US communities: Forsyth County, North Carolina; Jackson, Miss; the northwest suburbs of Minneapolis, Minn; and Washington County, Maryland. By design, the Jackson, Miss, site exclusively recruited African Americans, thereby accounting for 90% of African Americans in the study. Most of the remaining African Americans came from the Forsyth County cohort. The sampling procedures and methods used in ARIC have been described in detail elsewhere.13 Baseline visits were conducted from 1986 through 1989. Participants were followed up subsequently by annual telephone interviews and clinic visits every 3 years, for a total of 9 years of follow-up.

Participants

To construct the cohort for the present analysis, we excluded individuals who met any of the following conditions: diabetes mellitus present at baseline (n=1870); race other than white or African American (n=45); fasted for 8 hours or less before baseline visit (n=337); had missing baseline data on exposures or diabetes status (n=393). We also excluded individuals in the upper or lower 1% of dietary energy intake who were presumed to represent outliers (n=237). After these exclusions, we were left with a cohort of 12910 of whom 803 (6.2%) failed to return to the clinic, leaving 12107 participants with data on incident diabetes.

Assessment of Baseline Characteristics

Interview and Questionnaires. Information on age, sex, race, educational attainment, and family history of diabetes was based on self-report. A positive family history of diabetes was defined by participant report of diabetes in either biological parent. Parents whose diabetes status could not be recalled were classified as nondiabetic. Physical activity was assessed using a modified version of the questionnaire developed by Baecke et al.19 Activity was classified as either sports-related (eg, jogging) or non–sports-related leisure activity (eg, gardening) and measured on a 5-point scale, with 1 indicating the lowest level of activity and 5 the highest. Cigarette use was classified as never, former, or current. Alcohol consumption was assessed by the question: “During a typical week, how many glasses of wine/cans of beer/mixed drinks do you consume?” After pooling across beverage type, individuals were classified by the number of alcoholic drinks consumed per day. Dietary energy intake was assessed using an interviewer-administered, modified version of the 61-item food frequency questionnaire developed by Willett et al.15

Laboratory Evaluation. Participants were asked to fast for at least 12 hours before morning blood collection. After applying a tourniquet, blood was drawn from the antecubital vein while participants were seated. Blood specimens were collected into vacuum tubes containing serum-separator gel (glucose, insulin, creatinine and uric acid chemicals) and EDTA (lipids). Tubes were centrifuged at 3000g for 10 minutes at 4°C. After separation, aliquots were quickly frozen at −70°C until analysis was performed (within a few weeks). Serum glucose was assessed by a modified hexokinase/glucose-6-phosphate dehydrogenase procedure. Standard radioimmunoassay was used to determine serum insulin level. Total cholesterol16 and triglycerides17 were measured by enzymatic methods, high-density lipoprotein cholesterol was measured after dextran-magnesium precipitation,18 and low-density lipoprotein cholesterol was calculated using the Friedewald equation.19

Ascertainment of Diabetes Mellitus

Individuals were classified as having diabetes mellitus if any of the following criteria, adapted from 1997 American Diabetes Association criteria,20 were met: fasting serum glucose levels of at least 7.0 mmol/L (126 mg/dL), nonfasting glucose levels of at least 11.1 mmol/L (200 mg/dL), current use of medications prescribed to treat diabetes (eg, insulin or sulfonylureas), or a positive response to the question “Has a doctor ever told you that you had diabetes (sugar in the blood)?” In this study, individuals with diabetes at baseline were excluded. Individuals without diabetes at baseline who subsequently met any of these criteria at visits 2, 3, or 4 were considered to have incident diabetes. All incident cases of diabetes were classified as type 2, because the age of onset in this middle-aged cohort was between 45 and 73 years.

Analysis

Initial analyses focused on potential explanations for the excess risk of diabetes in African Americans compared with whites. The statistical significance of baseline differences between African Americans and whites regarding established and suspected risk factors21,22 for type 2 diabetes was assessed using t tests and χ² tests. Diabetes incidence rates were determined using a person-years approach, and a test of proportions was used to assess difference in incident rates of type 2 diabetes between African Americans and whites. The relative risks (RRs) of incident diabetes in African Americans vs whites were determined using proportional hazards models. The base model adjusted for 2 established nonmodifiable risk factors: age and family history. Subsequent models were created by introducing groups of potentially modifiable risk factors in sequence.

The extent to which groups of covariates appeared to explain the excess risk of diabetes in African Ameri-
cans was characterized by calculating the percent reduction in RR (PR) associated with adjustment according to the formula, \( \text{PR} = \frac{(r_a - r_b)}{r_a - 1} \), where \( r_a \) is the RR of diabetes in African Americans vs whites in the base model, adjusted for age and family history; \( r_b \) is the RR after additional adjustment for a group of covariates; and \( r_a - 1 \) is the excess risk of diabetes in African Americans vs whites.23 To assess the robustness of these results, we conducted 2 subsidiary analyses. First, diabetes was reclassified at baseline and during follow-up using a fasting glucose level of at least 7.8 mmol/L (140 mg/dL), which corresponds more closely to 1985 World Health Organization criteria. Second, analyses were confined to the 3172 participants who resided in Forsyth County, North Carolina, thereby minimizing the possibility of geographic confounding that arises from the concentration of African American participants in Jackson, Miss.

Subsequent analyses focused on biological traits (eg, blood pressure) that accompany diabetes but which are not known to be causal.24 In these analyses, men and women were stratified by diabetes status during follow-up, i.e., no diabetes throughout follow-up vs diabetes at visits 2, 3, or 4. Tests were used to determine the statistical significance of differences in baseline traits between African Americans and whites. Analysis of variance was used to test for interaction on an additive scale between race and diabetes status with regard to each trait.

**RESULTS**

**Risk Factors for Diabetes at Baseline**

Table 1 summarizes the presence of established and suspected risk factors for diabetes at baseline. Apart from age, the profile of established risk factors for diabetes was clearly worse in African American women than in their white counterparts. In particular, African American women had fewer years of formal education, were more likely to report a family history of diabetes, had greater measures of adiposity (including body mass index [BMI] and ratio of hip-to-waist circumference), and reported less physical activity during leisure time. A similar racial disparity of established diabetes risk factors prevailed in men, with the notable exception of adiposity, which was similar in African American and white men.

Other health behaviors hypothesized to influence the risk of diabetes showed less consistent racial patterns. Compared with their white counterparts, African American women were less likely to have ever smoked cigarettes, whereas African American men were equally likely to have ever smoked and more likely to be smoking currently. Typical consumption of alcoholic beverages was lower in African American men and women than in their white counterparts. In particular, African American women had fewer years of formal education, were more likely to report a family history of diabetes, had greater measures of adiposity (including body mass index [BMI] and ratio of hip-to-waist circumference), and reported less physical activity during leisure time. A similar racial disparity of established diabetes risk factors prevailed in men, with the notable exception of adiposity, which was similar in African American and white men.

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Table 1. Established and Suspected Diabetes Risk Factors in 12,107 Adults Without Diabetes at Baseline Aged 45 to 64 Years, by Sex and Race*  

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Women</th>
<th></th>
<th>Men</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>African American (n = 1670)</td>
<td>White (n = 5093)</td>
<td>African American (n = 976)</td>
<td>White (n = 4368)</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>52.8 (5.6)</td>
<td>53.8 (5.6)</td>
<td>53.5 (5.9)</td>
<td>54.6 (5.7)</td>
</tr>
<tr>
<td>Education, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 years or less</td>
<td>591 (35.4)</td>
<td>731 (14.4)</td>
<td>379 (38.8)</td>
<td>705 (16.1)</td>
</tr>
<tr>
<td>High school graduate</td>
<td>513 (30.7)</td>
<td>2625 (51.5)</td>
<td>262 (26.9)</td>
<td>1709 (39.2)</td>
</tr>
<tr>
<td>Attended college</td>
<td>566 (33.9)</td>
<td>1737 (34.1)</td>
<td>335 (34.3)</td>
<td>1964 (44.7)</td>
</tr>
<tr>
<td>Family history of diabetes, No. (%)</td>
<td>433 (25.9)</td>
<td>1169 (22.9)</td>
<td>230 (23.6)</td>
<td>916 (21.0)</td>
</tr>
<tr>
<td>Body mass index, mean (SD), kg/m²</td>
<td>30.2 (6.5)</td>
<td>26.2 (6.1)</td>
<td>27.5 (4.6)</td>
<td>27.2 (3.8)</td>
</tr>
<tr>
<td>Waist-to-hip ratio, mean (SD)</td>
<td>0.894 (0.081)</td>
<td>0.885 (0.078)</td>
<td>0.935 (0.054)</td>
<td>0.965 (0.51)</td>
</tr>
<tr>
<td>Physical activity index†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sports-related</td>
<td>2.10 (0.65)</td>
<td>2.44 (0.77)</td>
<td>2.28 (0.75)</td>
<td>2.70 (0.82)</td>
</tr>
<tr>
<td>Leisure, non–sports-related</td>
<td>2.11 (0.58)</td>
<td>2.52 (0.54)</td>
<td>2.08 (0.58)</td>
<td>2.43 (0.52)</td>
</tr>
<tr>
<td>Cigarette use, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>976 (58.4)</td>
<td>2599 (51.0)</td>
<td>288 (29.5)</td>
<td>1271 (29.1)</td>
</tr>
<tr>
<td>Former</td>
<td>277 (16.6)</td>
<td>1292 (25.4)</td>
<td>342 (35.0)</td>
<td>2094 (47.9)</td>
</tr>
<tr>
<td>Current</td>
<td>417 (25.0)</td>
<td>1202 (23.6)</td>
<td>346 (35.5)</td>
<td>1003 (23.0)</td>
</tr>
<tr>
<td>Alcohol consumption, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>971 (58.1)</td>
<td>1221 (24.0)</td>
<td>217 (22.2)</td>
<td>448 (10.3)</td>
</tr>
<tr>
<td>Former</td>
<td>312 (18.7)</td>
<td>659 (12.9)</td>
<td>249 (25.5)</td>
<td>823 (18.8)</td>
</tr>
<tr>
<td>Current</td>
<td>387 (23.2)</td>
<td>3213 (63.1)</td>
<td>510 (52.3)</td>
<td>3097 (70.9)</td>
</tr>
<tr>
<td>Dietary energy, mean (SD), kJ</td>
<td>6406 (2494)</td>
<td>6251 (2222)</td>
<td>7226 (2640)</td>
<td>7381 (2628)</td>
</tr>
</tbody>
</table>

*Family history was classified as "positive" if either biological parent was known to have diabetes. All sex-specific differences between African Americans and whites were statistically significant (P < .05) except family history and energy intake in men (P = .09 and P = .10, respectively).
†See “Methods” section for descriptions. Index ranges from 1 (least active) to 5 (most active).

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Incident Type 2 Diabetes Mellitus

During 9 years of follow-up, there were 459 incident cases of diabetes in African Americans and 966 incident cases in whites, corresponding to substantially higher sex-specific incidence rates in African Americans than in whites (TABLE 2). In both absolute and relative terms, the excess risk of diabetes in African Americans vs whites was greater in women (absolute risk difference, 14.7 per 1000 person-years; risk ratio = 2.41) than in men (absolute risk difference, 7.5 per 1000 person-years; risk ratio = 1.47).

Multivariate Analyses

To determine the extent to which the observed excess risk of diabetes in African Americans might be explained by racial differences in established and suspected diabetes risk factors at baseline, we constructed a series of sex-specific proportional hazards models. With adjustment for age and family history (established, unmodifiable risk factors), African American women were more than 2 1/2 times more likely to develop diabetes than their white counterparts (TABLE 3). These RRs were attenuated slightly by additional adjustment for education (a marker of socioeconomic status) and for diabetes-related health behaviors, but more so by additional adjustment for BMI and ratio of waist-to-hip circumferences. Thus, racial differences in potentially modifiable risk factors for diabetes, particularly adiposity, accounted for 47.8% of the excess risk of diabetes in African American women.

The pattern of adjusted risk was markedly different in men. After adjustment for age and family history in the base model, the RR of incident diabetes in African American vs white men was 1.58 (95% confidence interval, 1.32-1.99). Little or none of this excess risk appeared to be explained by racial differences in potentially modifiable risk factors: in the final multivariate model, the RR in African American vs white men was virtually unchanged from the base-line model (relative risk, 1.62; 95% confidence interval, 1.32-1.99).

These results appeared robust in 2 subsidiary analyses. First, after reclassification of diabetes using a cutoff of 7.8 mmol/L (140 mg/dL), the RR of diabetes in African American vs white women was 2.74 in the base model and 1.86 in the fully adjusted model, indicating 50% excess risk explained; in contrast, racial differences in established or suspected diabetes risk factors did not appear to explain any of the excess risk in African American men (data not shown). Second, an identical pattern of risk was obtained when the analysis was limited to residents of Forsyth County, North Carolina (data not shown).

Diabetes-Related Traits at Baseline

We compared diabetes-related biological traits in African Americans vs whites who went on to develop diabetes during the course of follow-up. Since these traits were assessed at baseline, they characterize a prediabetic state, 3 to 9 years before the onset of disease. To provide a context for these racial comparisons in individuals who did go on to develop diabetes, we examined the same traits in individuals who did not go on to develop diabetes throughout follow-up.

Compared with their white counterparts, African American women who subsequently developed diabetes had higher systolic and diastolic blood pressure, were more likely to have hypertension, and had higher levels of insulin and high-density lipoprotein cholesterol but lower concentrations of plasma triglycerides (P for all <.005; TABLE 4). Other diabetes-related traits—including fasting levels of glucose, creatinine, uric acid, and low-

Table 2. Incident Type 2 Diabetes Mellitus in 12 107 Adults Without Diabetes at Baseline, by Sex and Race*

<table>
<thead>
<tr>
<th>No. of persons at risk</th>
<th>African American</th>
<th>White</th>
<th>Person-years of follow-up</th>
<th>African American</th>
<th>White</th>
<th>Incidence cases of diabetes</th>
<th>African American</th>
<th>White</th>
<th>Incidence per 1000 person-years (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>25.1 (22.4-28.1)</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10.4 (9.4-11.4)</td>
</tr>
</tbody>
</table>

*P<.001 for difference in incidence of type 2 diabetes in African Americans vs whites, by sex. CI indicates confidence interval.
density lipoprotein cholesterol—were similar at baseline in African American and white women who would later go on to develop diabetes. Racial differences in most “diabetes-related” traits were actually greater in women who did not develop diabetes throughout follow-up. This was particularly true for blood pressure, hypertension, and serum concentration of uric acid.

Similar patterns of traits in the prediabetic state were observed in African American and white men (data not shown). Compared with their white counterparts, African American men who would go on to develop diabetes had substantially higher systolic and diastolic blood pressure, were more likely to have hypertension, and had slightly higher fasting levels of creatinine, uric acid, and high-density lipoprotein cholesterol, but lower levels of glucose and triglycerides (P for all <.005). As in women, these differences were also present in men who did not develop diabetes throughout follow-up.

**COMMENT**

Middle-aged African Americans, particularly women, are at substantially higher risk for incident type 2 diabetes than their white counterparts. An adverse profile of established diabetes risk factors appears to account for much of the excess risk in African American women and to a lesser extent in African American men. Much of this contrast appears to stem from sex differences in the disparity between African Americans and whites in adiposity, which was substantial in women but negligible in men. Racial differences in the prediabetic state largely reflect patterns in the general population of individuals without diabetes. Compared with their white counterparts, African Americans who subsequently developed diabetes had higher blood pressure and more hypertension, but similar (or even more favorable) lipid profiles. Strengths of the ARIC study that support these conclusions are its prospective design; its large, population-based sample; and its attention to standardization.

Nonetheless, several possible limitations of the study deserve mention. The fact that more than 90% of the African American participants were recruited from a single site (Jackson, Miss) raises the possibility that our results might be confounded by racial differences in geographic distribution. However, the similarity of results obtained in a sample of African Americans and whites from Forsyth County, North Carolina, mitigates this possibility. Treatment of family history of diabetes as a nonmodifiable (ie, genetic) risk factor is an oversimplification: shared behaviors and environmental exposures contribute to familial clustering as well.

Most important, like all racial and ethnic comparisons of disease risk, it is likely that our results are influenced by residual confounding, especially regarding socioeconomic status and health behaviors. In the United States, race is a complex marker for sociocultural and historical factors, for which commonly used variables like educational attainment or self-reported physical activity serve as rough proxies, at best. Therefore, the residual excess risk of type 2 diabetes in African Americans after multivariate adjustment should be interpreted cautiously. Likewise, estimates for percentage of excess risk explained by diabetes risk factors probably represent underestimates.

Since 1965, 8 published studies have investigated the excess risk of diabetes in African Americans vs whites. Of these, 4 studied prevalent diabetes using cross-sectional designs and 2 focused on samples atypical of the general population. Thus, the only previous population-based data regarding incident diabetes in African Americans vs whites come from the First National Health and Nutrition Examination Survey (NHANES I) Follow-up Study. In this nationally representative sample of 1500 African Americans and 9500 whites...
aged 25 to 70 years, followed up for 16 years, the RR of incident diabetes for African Americans vs whites was 2.1 among women and 1.6 among men. After adjustment for age, BMI, ratio of subscapular to triceps skin folds, education, and physical activity, African Americans remained at higher risk for diabetes but only at lower levels of adiposity. In men, the RR of diabetes in African Americans vs whites fell from 1.5 at a BMI of 25 kg/m² to 1.0 at a BMI of 30 kg/m²; in women, it fell from 1.6 to 1.3. These results are generally similar to ours.

In contrast, absolute diabetes incidence rates appeared 2- to 3-fold higher in men and women of both races in our study than in the NHANES I Epidemiologic Follow-up Study. For example, after accounting for differences in follow-up time, diabetes incidence (per 1000 person-years) in African American women aged 45 to 64 years in NHANES I was approximately 11.2 vs 25.1 in our study. A similar disparity was observed when incidence in whites was compared with previous data from Rancho Bernardo, Calif., and San Antonio, Tex. Possible explanations for this disparity include differences in age structure, in regional factors, and in diagnostic criteria. Favoring the latter explanation is the similarity of results in whites in our study to those observed in a study of residents of Rochester, Minn, with diagnostic criteria that included a fasting glucose level of at least 6.1 mmol/L (110 mg/dL).

Data from prior studies on racial differences in physiological traits prior to the onset of diabetes are limited. Like the ARIC Study, the NHANES I Epidemiologic Follow-up Study demonstrated a racial disparity in systolic and diastolic blood pressure that was greater in those who remained in a nondiabetic state throughout follow-up (blood pressure difference between African Americans vs whites was 7.6 mm Hg vs 5.2 mm Hg in women and 9.1 mm Hg vs 4.9 mm Hg in men) than in those who subsequently developed diabetes (3.6 mm Hg vs 3.3 mm Hg in women and 3.1 mm Hg vs 3.1 mm Hg in men, respectively). In contrast, there was little difference in blood pressure between African American and white men selected to participate in the Multiple Risk Factor Intervention Trial, whether or not they developed diabetes. That trial also differed from our study regarding racial differences in several metabolic traits, including levels of fasting glucose, uric acid, and high-density and low-density lipoprotein cholesterol. However, since the selection process for the Multiple Risk Factor Intervention Trial was largely based on blood pressure and plasma lipids, it is likely that these comparisons were influenced by selection bias.

Growing evidence for a syndrome of insulin resistance as a “common soil” for both diabetes and vascular disease raises the question of whether differences in such a syndrome between African Americans and whites might explain racial patterns in diabetes complications. Compared with their white counterparts, African Americans with diabetes are at much higher risk for end-stage renal disease, lower extremity amputation, and blindness—all strongly related to hyperglycemia and high blood pressure but weakly related to dyslipidemia. In contrast, the risk of ischemic heart disease and cardiovascular disease mortality appears similar in both African Americans and whites with diabetes. These macrovascular complications are strongly related to dyslipidemia, as well as to high blood pressure. Therefore, the prediabetic pattern of metabolic traits observed in our study raises the possibility that preexisting high blood pressure—but not dyslipidemia or hyperglycemia—in African Americans who go on to develop diabetes might contribute to their excess risk of diabetic microvascular complications.

Our study has several implications. It supports the notion that almost half of the excess risk of type 2 diabetes in African American women might be attenuated by prevention strategies aimed at weight reduction, dietary modification, and increased physical activity. It also suggests high blood pressure in the prediabetic state as a potential target for interventions designed to reduce the excess risk of microvascular complications in African Americans with diabetes. Finally, it should stimulate research into novel risk factors that might contribute to a more complete picture of excess diabetes risk in African Americans, including susceptibility gene variants and adverse environmental exposures in early life.

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