Black-White Differences in Risk of Developing Retinopathy Among Individuals With Type 2 Diabetes

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OBJECTIVE — To assess racial differences in risk of developing retinopathy among individuals with type 2 diabetes, after taking into account differences in the distribution of risk factors for retinopathy.

RESEARCH DESIGN AND METHODS — The participants were 105 individuals with type 2 diabetes, aged 40–69 years, who had no evidence of retinopathy at the time of a diabetic eye disease screening project. After an average of 4 years of follow-up, the subjects were reevaluated using nonmydriatic fundus photography.

RESULTS — Retinopathy occurred more often among black than white participants (50 vs. 19%). This difference could not be explained by differences in risk factors for retinopathy or potential confounders (odds ratio [95% CI] 2.96 [1.00–8.78] after adjustment for level of glycosylated hemoglobin, systolic blood pressure, type of diabetes treatment, and sex).

CONCLUSIONS — These results are consistent with the concept that racial differences in risk of developing retinopathy exist among individuals with type 2 diabetes and that these differences may be caused by differential (genetic) susceptibility to the adverse effects of increased levels of blood glucose and/or blood pressure. Discovery of the etiology of this differential susceptibility would allow us to identify and target secondary prevention efforts to individuals with type 2 diabetes who are at increased risk of retinopathy.

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Population data suggest that U.S. black individuals are at higher risk of blindness due to diabetic retinopathy than are U.S. white individuals, and that not all of this difference is due to the increased risk of diabetes in the black population (1). This difference, if present, may be due to increased prevalence of risk factors for diabetic retinopathy among black individuals, a greater impact of identified risk factors for diabetic retinopathy among black individuals, or increased prevalence in as-yet-unidentified risk factors for diabetic retinopathy. Cross-sectional data suggest that African-Americans have a higher prevalence of retinopathy than non-Hispanic whites (2,3), that this higher prevalence may be explained by differences in diabetes severity and glycemic control (3), and that a specific blood pressure may confer a greater risk of retinopathy among black individuals than it does among white individuals (4).

To our knowledge, no longitudinal data are available to address this issue. Identification of racial differences in risk of diabetic complications, and the sources of such differences, will permit clinicians to focus secondary prevention efforts on individuals at particularly high risk of complications. We report here the results of a small study designed to assess the differences in risk of developing retinopathy between black and white individuals with type 2 diabetes.

RESEARCH DESIGN AND METHODS

Study population

The study population was drawn from a group of 2,357 adults with diabetes who participated in a screening program for diabetic visual impairment (DVIP) from 1989 to 1991. DVIP targeted areas of the state of Maryland with high diabetes-related morbidity and mortality and was conducted at sites convenient to potential participants (such as local medical clinics and senior centers). Individuals with type 2 diabetes, without evidence of retinopathy, between 40 and 69 years of age and living in Baltimore, Maryland, at the time of the original screening were targeted for recontact. The diagnosis of type 2 diabetes was based on onset characteristics reported by the participant (age at onset, use of insulin, absence of ketoacidosis). Retinopathy status was assessed using nonmydriatic fundus photography, as described below. We attempted to balance the study population with respect to age, follow-up time, sex, and race by focusing recruitment efforts on equal numbers of individuals in each decade of life and number of years of follow-up, stratified on race and sex.

Initial contact for the 268 potential participants in this study was by mail, with a follow-up telephone call by the study coordinator (M. Sheldon-Rubio), a diabetes educator, to explain the study, answer questions, and schedule an appointment for interested individuals. Although we attempted to obtain current addresses through searches of local telephone books published since their DVIP participation, 81 individuals could not be contacted by letter or telephone; for 14 additional individuals, we felt certain that the telephone number was correct but we were never able to talk

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Abbreviations: DVIP, screening program for diabetic visual impairment; OR, odds ratio.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

to the DVIP participant. In five cases, family members informed us that the DVIP participant was deceased. Of the 168 potential participants we were able to contact, 58 did not participate: 15 had health or family problems; 8 had scheduling problems; 4 were temporarily out of town; 1 had moved out of the area; 2 stated they had transportation problems (even though we offered additional help); 2 denied having diabetes; 18 refused for undisclosed reasons; and 8 agreed to participate but did not keep scheduled appointments. There were 110 individuals (65% of those successfully contacted) who participated in this study.

Informed consent

The clinics were held at the Outpatient General Clinical Research Center at the Johns Hopkins University School of Medicine. The protocol was reviewed and approved by the institutional review board, the Joint Committee on Clinical Investigation, at the Johns Hopkins University School of Medicine. Written informed consent was obtained from each participant immediately after arrival at the clinic.

Retinopathy assessment

For both the DVIP screening and this follow-up study, retinal photographs were obtained with a nonmydriatic camera (Canon CR3-45NM fundus camera, a nonstereoscopic 45-degree fixed-angle camera; Canon, Tokyo). One photograph was taken for each eye, centering between the disc and the macula. The 35-mm film for the slides was processed routinely. An ophthalmologist (S.H.S.) examined the photographs to determine if retinopathy was present, and if so, to assess its severity (5). He did not examine the participants, so his rating of the degree of retinopathy was not affected by knowledge of other clinical characteristics of the participant, eliminating one potential source of bias. The photographs for 12 participants (6 black [10.7%] and 6 white [12.2%]) could not be evaluated because of poor quality or other eye conditions. To assess repeatability of retinopathy assessment, photographs from 15 individuals were sent to S.H.S. a second time as part of the regular study batches. Agreement for severity ratings of evaluable eyes was 85% (weighted kappa statistic 0.76) (6), comparable to published intrarater agreement for nonmydriatic cameras (7).

Laboratory measurements

To assess degree of diabetic control, fasting plasma glucose was measured by the glucose oxidase method (8), and glycosylated hemoglobin levels were measured by agarose gel electrophoresis (9) using standard clinical laboratory methods at the Johns Hopkins Hospital clinical laboratory.

Physical measurements

Resting blood pressure was measured by a research nurse using a standard sphygmomanometer with the participant seated. If a participant's blood pressure was above normal limits, a second reading was taken after the person had had additional time to rest. Height and weight were measured without shoes or outdoor clothing. Hip and waist circumferences were measured in a standard fashion (10).

Questionnaire

At the clinic, participants were asked to complete a questionnaire about their medical history and personal habits. It included risk factors for diabetic complications and information about diabetes onset and treatment.

Statistical methods

The analysis sample includes 105 individuals instead of 110: 1 woman's age was incorrectly recorded in the DVIP data, and she was much older than the specified age range; and 4 individuals were excluded because further checking of the DVIP data indicated that they either had had retinopathy or uninterpretable photographs at the first screening. Although both eyes were rated, each person was included only once in these analyses; the rating of the eye with the higher grade of retinopathy was used as each person's outcome. To assess possible differences between participants and nonparticipants, characteristics from the DVIP screening were compared (such as age, duration of diabetes, blood pressure) using nonparametric statistics (nonparametric analysis of variance, χ^2 statistic) (11). We also used these methods for preliminary analyses of information from the study population (i.e., comparisons of black versus white participants, those with retinopathy versus those without, interrelationships among measurements).

To assess racial differences in risk of developing retinopathy, accounting for differences in potential confounders, we used multiple logistic regression with a manual forward stepwise approach (12). Since the goal of the multivariate analyses was to adjust for potential confounders rather than predict retinopathy risk, decisions about which variables to include in the final model were based on change in the odds ratio (OR) for race rather than on statistical tests (12).

RESULTS

Comparison of participants and nonparticipants

Participants and nonparticipants in the follow-up study were similar in age, race, blood pressure, blood pressure medication status, duration of diabetes, and type of diabetes treatment (Table 1). Women were significantly less likely to participate than were men ($P = 0.03$).

Table 1—*Comparison of DVIP screening characteristics for participants and nonparticipants in the follow-up study*

Characteristic at first screening	Participants	Nonparticipants
n	110	158
Age (years)	58 (50–63)	58 (50–63)
Systolic blood pressure (mmHg)	136 (126–150)	136 (126–148)
Diastolic blood pressure (mmHg)	83 (80–88)	82 (78–88)
Women	54.5 (60)	67.7 (107)*
Black race	51.8 (57)	49.4 (78)
Duration of diabetes (years)		
<5	59.1 (65)	54.4 (86)
5–9	20.0 (22)	26.6 (42)
≥10	20.9 (23)	19.0 (30)
Use of blood pressure medication	45.5 (35)	55.1 (54)
Diabetes treatment		
Insulin	10.0 (11)	19.0 (30)
Oral agents	61.8 (68)	57.0 (90)
Neither insulin nor oral agents	28.2 (31)	24.1 (38)

Data are medians (interquartile ranges) or % (*n*). *$P = 0.03$ for participants vs. nonparticipants.

Table 2—*Distributions of age, follow-up time, duration of diabetes, and age at onset of diabetes stratified by race and sex*

Characteristic	Race and sex			
	White women	Black women	White men	Black men
n	22	34	27	22
Age at first screening*	55 (51–60)	55 (48–62)	60 (54–63)	60.5 (48–65)
Age at follow-up†	59.5 (56–73)	58.5 (53–66)	65 (59–69)	65 (53–69)
Length of follow-up	4.2 (3.8–4.4)	4.1 (3.5–5.1)	4.2 (3.7–5.5)	3.7 (3.3–4.2)
Duration of diabetes	6.5 (6–11)	9.5 (6–20)	10 (7–17)	8.5 (5–12)
Age at diagnosis of diabetes‡	51 (44–53)	46 (42–51)	53 (46–58)	55 (44–60)

Data are median years (interquartile ranges). *P = 0.02, †P = 0.03, and ‡P = 0.05 for women vs. men.

Race and sex differences in risk factors for retinopathy and potential intermediary variables

Black participants did not differ from white participants in age at first screening, age at or length of follow-up, age at diagnosis, or duration of diabetes. Women were about 6 years younger than men at first screening and at follow-up (P = 0.02 and P = 0.03, respectively), and were diagnosed at a younger age than men (P = 0.05) (Table 2).

Significant race and sex differences were found in longer-term diabetes control, as measured by glycosylated hemoglobin: black women had the highest levels of glycosylated hemoglobin, followed by black men, white women, and white men (Table 3). Fasting plasma glucose levels were significantly higher in black than in white participants; women did not differ significantly from men.

No significant race or sex differences were observed in systolic blood pressure at follow-up, systolic or diastolic blood pressure from the first screening, change in systolic blood from the first screening to follow-up, or obesity measures (BMI, waist circumference, waist-to-hip ratio) (Table 3). Diastolic blood pressure at follow-up was significantly higher among black than white participants.

Comparison of individuals with and without retinopathy

Participants with and without retinopathy at follow-up did not differ with respect to age at follow-up or length of follow-up (Table 4). Those with retinopathy reported a longer duration of diabetes and a younger age at diagnosis than those without retinopathy.

Participants with retinopathy showed poorer diabetic control, with higher glycosylated hemoglobin and fasting glucose levels than those without retinopathy (Table 5). There were no significant differences in blood pressure or obesity measures by retinopathy status.

Racial differences in risk of developing retinopathy

Black individuals with type 2 diabetes were significantly more likely to have developed retinopathy within the follow-up period than were white individuals: 25 (50%) black and 8 (19%) white participants had retinopathy at follow-up (P = 0.002). After adjustment for sex, glycosylated hemoglobin level, systolic blood pressure at follow-up, and type of treatment (insulin, oral agent, other), black participants were still at higher risk of developing retinopathy than white participants (OR 2.96; 95% CI 1.00–8.78) (Table 6). The association of insulin treatment with retinopathy is an interesting observation that might reflect the severity of diabetes.

CONCLUSIONS — The results of this small prospective study indicate that black individuals with type 2 diabetes are more likely to develop retinopathy than white individuals with type 2 diabetes, even after taking into account potential risk factors for

Table 3—*Distributions of measurements of diabetic control, blood pressure (at follow-up and at first screening), and obesity stratified by race and sex*

Characteristic	Race and sex			
	White women	Black women	White men	Black men
n	22	34	27	22
Glycosylated hemoglobin (% total Hb)*†	11.4 (9.6 to 13.2)	13.1 (11.5 to 15.8)	10.2 (8.3 to 12.2)	12.3 (9.7 to 14.2)
Fasting glucose (mmol/l)‡	10.0 (7.4 to 14.5)	12.8 (9.7 to 17.5)	10.2 (7.2 to 11.7)	12.1 (8.7 to 14.3)
Systolic blood pressure (mmHg)				
Follow-up	132 (122 to 150)	140 (128 to 160)	140 (130 to 150)	142 (124 to 154)
First screening	135 (118 to 150)	134 (124 to 150)	134 (130 to 148)	140 (128 to 150)
Change (follow-up − first)	1 (−16 to 12)	6 (−12 to 22)	2 (−10 to 10)	8 (−10 to 14)
Diastolic blood pressure (mmHg)				
Follow-up§	79 (70 to 90)	84 (80 to 90)	80 (74 to 90)	85 (80 to 90)
First screening	82 (78 to 86)	84 (80 to 88)	84 (78 to 90)	85 (80 to 90)
Change (follow-up − first)	−2 (−6 to 0)	2 (−10 to 6)	−2 (−10 to 2)	1 (−8 to 6)
Obesity				
BMI (kg/m^2)	31.7 (26.8 to 35.7)	31.4 (27.3 to 36.3)	28.4 (25.1 to 30.5)	29.6 (26.5 to 36.3)
Waist-to-hip ratio	0.98 (0.92 to 1.04)	0.93 (0.89 to 1.00)	0.97 (0.92 to 1.02)	0.98 (0.94 to 1.02)
Waist (cm)	110.5 (99.1 to 119.4)	106.7 (94.0 to 119.4)	101.6 (94.0 to 111.8)	104.1 (94.0 to 119.4)

Data are medians (interquartile ranges). *P = 0.0004, ‡P = 0.002, and §P = 0.004 for blacks vs. whites; †P = 0.01 for women vs. men.

Table 4—*Distributions of age, follow-up time, duration of diabetes, and age at onset of diabetes stratified by retinopathy status*

Characteristic	Retinopathy	No retinopathy
n	33	60
Age at first screening*	53 (48–60)	59 (52.5–63)
Age at follow-up	57 (52–65)	63 (57–67.5)
Length of follow-up	4.3 (3.5–5.1)	4.0 (3.5–4.6)
Duration of diabetes†	10 (7–15)	8 (6–10.5)
Age at diagnosis of diabetes‡	45 (42–51)	52.5 (46–58)

Data are medians (interquartile ranges) and are given in years. For 12 individuals, the photographs were missing or were of inadequate quality for reading. *P = 0.05, †P = 0.03, and ‡P = 0.005 for no retinopathy vs. any retinopathy.

retinopathy. A recently published prospective study of proliferative diabetic retinopathy among individuals with type 1 diabetes also observed a higher risk in African-Americans than in white participants (13); however, these differences could be accounted for by differences in baseline glycemic control, baseline retinopathy grade, and length of follow-up. To our knowledge, there are no published prospective studies of racial differences in risk of developing diabetic retinopathy among people with type 2 diabetes.

One difficult decision was whether to include certain variables as potential confounders in the multivariate analyses, because some of these variables may actually be co-outcomes of the process that causes retinopathy. For example, is the increase in systolic blood pressure over time an indicator of increasing risk of retinopathy because it is a risk factor for retinopathy, or because one process is causing both the increase in blood pressure and changes in the retinal blood vessels? Including this variable in multivariate analyses would be appropriate if it is a risk factor, but would result in overadjustment and might obscure a racial difference if it is a co-outcome of the pathogenic process leading to retinopathy.

Because the DVIP study targeted individuals who were at increased risk of having undiagnosed diabetic retinopathy, our study population may not be representative of the general population of individuals with diabetes. However, the validity of the comparison between black and white participants is supported by the observation that these two groups were quite similar on many important characteristics; we were able to adjust statistically for any observed differences in risk factors for retinopathy.

One limitation of our data is that the retinal photographs were obtained with a nonmydriatic camera (Canon CR3–45NM fundus camera), which is a nonstereoscopic 45-degree fixed-angle camera. With this method, the entire retina was not seen, so changes in the more peripheral parts of the retina may not have been detected; Klein et al. (7) estimated that 8–15% of diabetic retinopathy may be missed by a nonmydriatic camera. Another limitation is that the photographs are not of sufficient quality to detect the more subtle changes of diabetic retinopathy, such as intraretinal microvascular abnormalities. Several studies have addressed these issues; three examples follow. Comparing the rating of retinopathy (into four diagnostic categories) using a nonmydriatic camera and a standard fundus camera, Klein et al. (7) found exact agreement in 82% of the photographs from a predominantly white population; 13% of the photographs taken using the nonmydriatic camera could not be graded. For the nonmydriatic camera, interrater agreement was 85% and intrarater agreement was 81%. In a more racially and ethnically mixed group (52% non-Hispanic white, 38% Mexican-American, 10% African-American), Pugh et al. (14) found 72% exact agreement (weighted kappa statistic 0.62) between the nonmydriatic camera and stereoscopic photographs of seven standard fields; 14% of the photographs taken using the nonmydriatic camera could not be graded. In a British study including both Indians and Europeans, agreement between photographic assessment of retinopathy (using a nonmydriatic camera) and ophthalmoscopy by an ophthalmologist was 72% and did not differ between racial groups (15). Most studies have judged nonmydriatic fundus photography to be adequate for screening for diabetic retinopathy. There is no evidence of racial or ethnic differences in the validity of this type of screening—something that, if present, could have biased our study. We therefore believe that the observed racial differences are not affected by the limitations of our methodology.

To confirm our findings of racial differences in risk of retinopathy among individuals with type 2 diabetes, future work should include larger longitudinal studies in ethnically diverse populations with regular assessment of retinopathy status and risk factors for retinopathy; these studies

Table 5—*Distributions of measurements of diabetic control, blood pressure (at follow-up and at first screening), and obesity stratified by retinopathy status*

Characteristic	Retinopathy	No retinopathy
n	33	60
Glycosylated hemoglobin (% total Hb)*	13.0 (10.7 to 15.3)	10.9 (9.4 to 13.1)
Fasting glucose (mmol/l)†	12.8 (8.8 to 16.9)	10.3 (7.5 to 14.2)
Systolic blood pressure (mmHg)		
Follow-up	140 (128 to 160)	140 (124 to 150)
First screening	130 (120 to 144)	139 (128 to 150)
Change (follow-up − first)	8 (−4 to 24)	2 (−14 to 9)
Diastolic blood pressure (mmHg)		
Follow-up	86 (70 to 90)	80 (79 to 90)
First screening	82 (80 to 86)	84 (80 to 88)
Change (follow-up − first)	0 (−10 to 6)	−1 (−9 to 4)
Obesity		
BMI (kg/m^2)	29.4 (26.9 to 36.1)	28.6 (26.2 to 35.3)
Waist-to-hip ratio	0.96 (0.90 to 1.00)	0.97 (0.92 to 1.03)
Waist (cm)	101.6 (96.5 to 119.4)	106.7 (94.0 to 115.6)

Data are medians (interquartile ranges). For 12 individuals, the photographs were missing or were of inadequate quality for reading. *P = 0.005, †P = 0.01 for retinopathy vs. no retinopathy.

Table 6—*Results of multiple logistic regression analysis showing the effect of race and sex on risk of developing retinopathy after controlling for the effects of diabetic control and systolic blood pressure at follow-up*

Variable	OR (95% CI)
Black race	2.96 (1.00–8.78)
Woman	1.91 (0.66–5.52)
Glycosylated hemoglobin (% total Hb)	1.10 (0.92–1.32)
Systolic blood pressure (mmHg)	1.01 (0.99–1.04)
Type of treatment	
Insulin	12.36 (1.06–143.62)
Oral agent	3.06 (0.31–29.77)

should be designed to have sufficient power to examine the etiology of the observed racial differences in risk. Because a high proportion of individuals with type 2 diabetes eventually develop some degree of retinopathy, racial differences in rate of progression of retinopathy also need to be considered in longitudinal studies. To prevent vision loss in individuals with type 2 diabetes, rate of progression of retinopathy may be as important a factor as risk of developing retinopathy.

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