Associations Between Diabetes and Clinical Markers of Benign Prostatic Hyperplasia Among Community-Dwelling Black and White Men

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OBJECTIVE — The aim of this study was to examine associations between diabetes and clinical markers of benign prostatic hyperplasia (BPH) in community-dwelling white and black men aged 40–79 years.

RESEARCH DESIGN AND METHODS — Data from the Olmsted County Study of Urinary Symptoms and Health Status and the Flint Men's Health Study were combined for a total study sample of 2,484 men. Severity of lower urinary tract symptoms (LUTS), peak urinary flow rates, prostate volume, and serum prostate-specific antigen (PSA) levels were examined by self-reported physician-diagnosed diabetes.

RESULTS — Overall, 170 men (6.8%) reported a history of diabetes. Increased irritative LUTS and specifically nocturia were positively associated with diabetes. These patterns were consistent across race and persisted after adjustment for age, BMI, and various indicators of socioeconomic status. Furthermore, the relationship between irritative LUTS and diabetes was greater in black men. No significant associations were observed between diabetes and prostate volume, PSA level, and peak urinary flow rate.

CONCLUSIONS — Our multiethnic community-based study demonstrates positive associations between diabetes and irritative LUTS and nocturia. Moreover, the association between irritative LUTS and diabetes is increased in black men. There was no strong evidence for an association between diabetes and BPH across measures more specific to BPH (i.e., prostate volume, PSA, and peak urinary flow rate). Taken together, our findings suggest that the presence of diabetes may be less related to prostate growth and more related to the dynamic components of lower urinary tract function. Further evaluations of the association between diabetes and BPH and related racial variations are warranted.

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Abbreviations: AUASI, American Urological Association Symptom Index; BPH, benign prostatic hyperplasia; FMHS, Flint Men's Health Study; LUTS, lower urinary tract symptoms; OCS, Olmsted County Study of Urinary Symptoms and Health Status; PSA, prostate specific antigen; SES, socioeconomic status.

Benign prostatic hyperplasia (BPH) and type 2 diabetes are both highly prevalent conditions that bring millions of older American men to medical attention each year. Typically, clinicians treat BPH and type 2 diabetes as separate entities, although some have suggested that diabetes may be a risk factor for the development and progression of BPH (1–3). Clinical uncertainty stems from the fact that both diabetic neuropathy and BPH can lead to dysfunctional bladder storage and emptying, which makes it difficult to determine the extent to which diabetes and/or BPH contributes to voiding dysfunction in these patients. To more clearly understand the association between diabetes and BPH it is important to examine the full spectrum of clinical markers of BPH including lower urinary tract symptoms (LUTS), urinary flow, prostatic volume, and serum prostate-specific antigen (PSA) concentrations.

Recently, the National Health and Nutrition Examination Survey reported the prevalence of diabetes to be greater in blacks than in whites. Specifically, the prevalence in black men aged 40–59 years is 9.6%, increasing to 29.2% in men aged ≥65 years compared with 5.5 and 14.3%, respectively, in white men (4). Moreover, in a previous study, we observed a significantly greater report of moderate/severe LUTS in black men compared with that in white men, suggesting a potential racial difference in BPH (5). If diabetes and BPH are indeed related, these racial disparities, along with the dramatic increase in the incidence of type 2 diabetes in the past decade, have striking implications for the black male population and underscore the need to understand this relationship more completely.

The Olmsted County Study of Urinary Symptoms and Health Status (OCS) has served as a point of reference for prevalence and incidence estimates of measures of BPH in community-dwelling men (6). However, the lack of black men in the OCS has limited the ability to evaluate racial differences. To fill this void, the

Flint Men's Health Study (FMHS) was developed as a comparable community-based study of black men designed to mirror the OCS protocol (7). In this study we examine the associations between diabetes and clinical markers of BPH and determine whether race modifies these relationships. We did this by combining these two large comparable ongoing epidemiological studies of community-dwelling men aged 40–79 years.

RESEARCH DESIGN AND METHODS

— Details on subject selection for both the OCS and FMHS have been published previously (8,9). Briefly, the OCS and FMHS are population-based, prospective cohort studies established to evaluate the natural history of BPH in white and black male residents of Olmsted County, Minnesota, and Genesee County, Michigan, respectively. In the OCS, 2,115 of 3,874 eligible white men aged 40–79 years in 1990 without a history of prostate cancer or surgery or other conditions known to interfere with voiding, including diabetic neuropathy leading to lower limb amputation, completed the self-administered American Urological Association Symptom Index (AUASI) (10). A detailed urologic examination that included uroflowmetry, transrectal ultrasound, and serum PSA measurement was performed in a 25% random subsample (475 of 537, 88%) (11). With use of the same criteria and protocol described above, 730 of 943 eligible black men completed an interview-administered questionnaire in 1996 in the FMHS (9). Of these, 369 men underwent the comprehensive urologic examination that included, as in the OCS, uroflowmetry, transrectal ultrasound, a serum PSA measurement, and self-administered AUASI and were deemed to be free of prostate cancer (7,12). The selective participation in the clinical examination and potential resulting selection bias have been addressed previously in both studies (12,13). For the OCS, the clinic sample was comparable to the community cohort in virtually all respects, whereas in the FMHS, although there was greater participation in the clinic phase among men who reported greater LUTS, it did not bias the estimated age-specific reference ranges for PSA concentrations.

Measurements

The primary end points included the following clinical markers of BPH: LUTS, prostate volume, peak urinary flow rate, and serum PSA concentrations. Specifically, severity of LUTS was measured by the AUASI (10) on self-administered questionnaires in both the OCS (n = 2,115) and the FMHS (n = 369). Prostate volume (milliliters) determined by transrectal ultrasound, maximum urinary flow rate (milliliters per second) measured by uroflowmetry, and serum PSA (nanograms per milliliter) concentrations were collected during the clinical examination portions of the two studies (OCS, n = 475; FMHS, n = 369). Although no single surrogate measure provides a definitive nonhistological diagnosis of BPH, previous studies have demonstrated that these measures have adequate construct and predictive validity for BPH (14). As fasting plasma glucose concentrations were not available, information on diabetes was gathered by questionnaire in both the OCS (n = 2,115) and FMHS (n = 369) by querying participants as to whether they ever had diabetes diagnosed by a physician and at what age it was diagnosed. Information on age at diagnosis was used to determine those with type 1 versus type 2 diabetes. Only four men were identified as possibly having type 1 diabetes (age at diagnosis <30 years) (15,16) in the current report. Finally, information regarding marital status, education, income levels, and BMI (based on self-reported weight and height) (2) were gathered by questionnaire in both the OCS (n = 2,115) and FMHS (n = 369).

Statistical analysis

Distributions of the clinical markers of BPH were evaluated overall and by diabetes status after data elements from the OCS and FMHS were combined. Differences in median AUASI scores, individual symptoms, prostate volume, peak urinary flow rate, and total PSA levels by diabetes status were tested univariately on a continuous scale (Kruskal-Wallis) and using standard dichotomous cut points (10) (χ^2 test). Multivariable associations were also explored using logistic regression models, with adjustment for race, age, BMI, and various indicators of socioeconomic status (SES) including marital status, education, and income. An interaction term defined by the cross-product of race and diabetes status was included in all models to examine the potential for racial differences in the associations between diabetes and markers of BPH. Racial differences in these associations were also examined using the Breslow-Day test for homogeneity of odds ratios (ORs), which yielded the same results as the multivariable approach described above. All analyses were performed using SAS (SAS Institute, Cary, NC).

RESULTS

— Among the 2,484 total participants (2,115 white and 369 black men), 170 (6.8%) had a self-reported history of diabetes (Table 1). The mean ± SD ages were 56.0 ± 10.5 years overall and 62.4 ± 10.4 and 55.5 ± 10.4 years in those with and without diabetes, respectively ($P < 0.0001$). Overall, 68.3% of men were overweight/obese (BMI ≥25 kg/m^2), with 82.9 and 67.2% being overweight/obese among diabetic and nondiabetic men, respectively ($P < 0.0001$). Black men had an increased odds of having diabetes (age-adjusted OR 3.96 [95% CI 2.82–5.56]).

In bivariate analyses, men with diabetes had significantly greater median AUASI scores (7 vs. 5, $P < 0.0001$) (Table 2). Median prostate volume was also higher among these men (30.24 vs. 26.01 ml, $P = 0.008$), whereas peak urinary flow rates were significantly lower (15.2 vs. 17.4 ml/s, $P = 0.02$). No significant differences were found in median PSA levels; however, the frequency of men with PSA levels ≥2.5 ng/ml was significantly greater among those with diabetes ($P = 0.04$). The frequency of irritative symptoms was also notably higher among diabetic men (57% vs. 39%). No significant differences were observed for obstructive symptoms.

In multivariable analyses, men with diabetes were more likely to report moderate/severe LUTS (OR 1.28 [95% CI 0.88–1.85]), have an enlarged prostate (1.44 [0.76–2.72]), and have increased PSA concentrations (1.47 [0.68–3.19]), although these results did not reach statistical significance (Table 2). Finally, diabetes was associated with a significant increased risk of irritative symptoms (1.73 [1.19–2.52]). Consistent with bivariate analysis, no association was observed for obstructive symptoms.

Diabetes was not significantly associated with any individual obstructive/voiding symptoms after adjustment for age, BMI, race, marital status, education, and income (Table 3). Among irritative symptoms, diabetes was significantly associated with increased risk for nocturia (OR 2.04 [95% CI 1.38–3.02]) after adjustment for age, BMI, race, marital status, education, and income.

Figure 1 shows a comparison of ORs for the associations between clinical

Table 1—*Overall distribution of SES, health characteristics, and measures of BPH in the combined population*

	Overall*	White*	Black*	*P* value†
n	2,484	2,115	369	
Age				0.01
40–49 years	895 (36.03)	787 (37.21)	108 (29.27)	
50–59 years	728 (29.31)	612 (28.94)	116 (31.44)	
60–69 years	537 (21.62)	444 (20.99)	93 (25.20)	
70–79 years	324 (13.04)	272 (12.86)	52 (14.09)	
BMI				0.003
Normal (<25 kg/m^2)	782 (31.48)	663 (31.35)	119 (32.25)	
Overweight (25–29 kg/m^2)	1188 (47.83)	1054 (49.83)	134 (36.31)	
Obese (≥30 kg/m^2)	509 (20.49)	393 (18.58)	116 (31.44)	
Marital status				<0.0001
Married/living together	2108 (84.86)	1890 (89.36)	218 (59.08)	
Divorced, separated, or widowed	289 (11.63)	168 (7.94)	121 (32.79)	
Single	80 (3.22)	51 (2.41)	29 (7.86)	
Education				<0.0001
High school graduate	2014 (81.08)	1865 (88.18)	149 (40.38)	
Not a high school graduate	337 (13.57)	224 (10.59)	113 (30.62)	
Income				<0.0001
<$15,000	231 (9.30)	169 (7.99)	62 (16.80)	
$15,000–$34,999	670 (26.97)	585 (27.66)	85 (23.04)	
$35,000–$54,999	644 (25.93)	593 (28.04)	51 (13.82)	
≥$55,000	745 (29.99)	672 (31.77)	73 (19.78)	
History of diabetes				<0.0001
Yes	170 (6.84)	105 (4.96)	65 (17.62)	
No	2314 (93.16)	2010 (95.04)	304 (82.38)	
BPH measures				
AUASI score	5 (2, 9)	5 (2, 9)	6 (2, 11)	0.03
Obstructive score	2 (0, 5)	2 (0, 5)	2 (0, 5)	0.68
Irritative score	3 (1, 5)	3 (1, 5)	3 (2, 6)	<0.0001
Prostate volume (ml)	26.34 (20.62, 33.82)	26.44 (21.21, 34.04)	25.88 (20.08, 33.02)	0.11
Peak urinary flow rate (ml/s)	17.30 (12.30, 23.80)	17.70 (12.60, 23.90)	15.10 (10.00, 23.00)	<0.0001
PSA (ng/ml)	0.97 (0.60, 1.70)	1.00 (0.60, 1.70)	0.95 (0.55, 1.75)	0.33

Data are *n* (%) or median (25th, 75th percentile). The following variables were measured by self-report in the interview portion of the two studies (OCS, white men, *n* = 2,115; FMHS, black men, *n* = 369): age, education, income, marital status, BMI, history of diabetes, AUASI score, obstructive score, and irritative score. Prostate volume, peak urinary flow rate and PSA measures were collected in the clinical examination portion of the two studies (OCS, white men, *n* = 475; FMHS, black men, *n* = 369). *Totals vary for each variable because of missing data. †Tests of significant differences in frequencies by race based on χ^2 *P* value. Tests of significant differences in medians by race are based on the Kruskal-Wallis *P* value.

markers of BPH and diabetes adjusted for age, BMI, marital status, education, and income by race. The association between diabetes and moderate/severe LUTS was greater among black (OR 2.37 [95% CI 1.04–5.43]) compared with white men (1.07 [0.70–1.65]), albeit these differences were not statistically significant (Fig. 1). The findings are similar for obstructive and irritative symptoms. Race did not modify associations between diabetes and prostate volume, peak urinary flow rate, or PSA levels.

CONCLUSIONS — In this study we examined associations between diabetes and clinical markers of BPH in community-dwelling black and white men. In prior reports, type 2 diabetes, which affects 90–95% of people with diabetes, has been associated with bladder dysfunction, typically resulting in impairment of the detrusor (2,17). Impaired detrusor function results in a lower maximum flow rate for any given level of bladder outlet resistance and can increase postvoid residual volume and severity of LUTS (2). BPH is also characterized by its presentation of LUTS, including a reduced peak urinary flow rate and increased postvoid residual volume. The underlying pathophysiology is different because BPH does not primarily impair detrusor function but enhances bladder outlet resistance via static and dynamic components (2).

Although the association between diabetes and BPH has been examined in several studies, findings have been inconsistent. A series of cross-sectional studies from Sweden showed that physician-diagnosed type 2 diabetes, treated hypertension, obesity, low HDL cholesterol levels, and high insulin levels were significantly associated with the presence of BPH in a consecutive series of patients with LUTS referred for surgery (1,18,19). Furthermore, the Massachusetts Male Aging Study (20), the FMHS (21), and others (22) have consistently reported diabetes or glucose levels to be significantly associated with an increased risk of LUTS.

The positive associations described between measures of diabetes and BPH, however, have not been consistently observed across studies. Specifically, Boon et al. (3) examined individuals with physician-diagnosed diabetes and LUTS compared with individuals with LUTS only and found little difference in prostate vol-

Table 2—*Distribution of and associations between clinical markers of BPH and self-reported diabetes status*

				ORs (95% CI)		
	No diabetes*	History of diabetes*	*P* value†	Unadjusted	Age adjusted	Multivariable adjusted‡
n	2,314	170				
AUASI	5 (2, 9)	7 (4, 11)	<0.0001			
Prostate volume (ml)	26.01 (20.43, 33.09)	30.24 (22.05, 39.82)	0.0078			
Q_{max} (ml/s)	17.40 (12.40, 23.90)	15.20 (11.00, 23.15)	0.0188			
Total PSA (ng/ml)	0.96 (0.60, 1.70)	1.06 (0.60, 2.12)	0.1889			
LUTS severity			0.0010			
Mild/none (≤7)	1,530 (66.12)	91 (53.53)		1.00 (reference)	1.00 (reference)	1.00 (reference)
Moderate/severe (>7)	778 (33.62)	78 (45.88)		1.69 (1.23–2.31)	1.42 (1.03–1.95)	1.28 (0.88–1.85)
Obstructive symptom score			0.9069			
≤4	1,675 (72.39)	122 (71.76)		1.00 (reference)	1.00 (reference)	1.00 (reference)
>4	632 (27.31)	47 (27.65)		1.02 (0.72–1.45)	0.87 (0.61–1.24)	0.76 (0.50–1.16)
Irritative symptom score			<0.0001			
≤3	1,404 (60.67)	72 (42.35)		1.00 (reference)	1.00 (reference)	1.00 (reference)
>3	902 (38.98)	97 (57.06)		2.10 (1.53–2.88)	1.78 (1.29–2.45)	1.73 (1.19–2.52)
Prostate volume			0.0049			
≤30 ml	479 (63.19)	41 (47.13)		1.00 (reference)	1.00 (reference)	1.00 (reference)
>30 ml	257 (33.91)	42 (48.28)		1.91 (1.21–3.01)	1.40 (0.86–2.28)	1.44 (0.76–2.72)
Q_{max}			0.0119			
≥12 ml/s	1,782 (77.01)	114 (67.06)		1.00 (reference)	1.00 (reference)	1.00 (reference)
<12 ml/s	514 (22.21)	51 (30.00)		1.55 (1.10–2.19)	1.04 (0.72–1.49)	0.86 (0.56–1.32)
Total PSA			0.0381			
<2.5 ng/ml	655 (86.41)	68 (78.16)		1.00 (reference)	1.00 (reference)	1.00 (reference)
≥2.5 ng/ml	103 (13.59)	19 (21.84)		1.78 (1.03–3.08)	1.20 (0.67–2.14)	1.47 (0.68–3.19)

Data are median (25th, 75th percentile) or *n* (%) unless indicated otherwise. The following variables were measured by self-report in the interview portion of the two studies (OCS, white men, $n = 2{,}115$; FMHS, black men, $n = 369$): age, education, income, marital status, BMI, history of diabetes, AUASI score, obstructive score, and irritative score. Prostate volume, peak urinary flow rate, and PSA measures were collected in the clinical examination portion of the two studies (OCS, white men, $n = 475$; FMHS, black men, $n = 369$). *Totals vary for each variable because of missing data. †Tests of significant differences in frequencies by diabetes status based on χ^2 *P* value. Tests of significant differences in medians by diabetes status are based on the Kruskal-Wallis *P* value. ‡Measures effect of diabetes status on various clinical markers of BPH adjusted for age, BMI, race, marital status, education, and income.

ume, peak urinary flow rate, and postvoid residual volume. This study, however, relied on a control group from a referral population that did not meet the specified exclusion criteria for BPH and, thus, the effect of diabetes on LUTS was probably underestimated. Furthermore, in contrast with their finding for BPH surgery, the Normative Aging Study investigators found a nonsignificant inverse association between diabetes and clinical BPH (23). Finally, in the OCS, Burke et al. (24) observed that diabetic men reported a larger increase in AUASI score than did nondiabetic men. However, they found no differences in change of prostate volume, suggesting, perhaps, that the presence of diabetes may be less directly associated

Table 3—*Distribution of individual symptoms by self-reported diabetes status and associated ORs and 95% CIs*

			ORs (95% CI)		
	No diabetes*	History of diabetes*	Unadjusted	Age adjusted	Multivariable adjusted†
n	2,314	170			
Obstructive/voiding					
Incomplete emptying	446 (19.27)	41 (24.12)	1.33 (0.92–1.93)	1.19 (0.82–1.72)	0.93 (0.59–1.44)
Straining	296 (12.79)	17 (10.00)	0.77 (0.46–1.29)	0.75 (0.44–1.26)	0.79 (0.44–1.42)
Weak stream	748 (32.32)	56 (32.94)	1.04 (0.75–1.45)	0.84 (0.60–1.18)	0.94 (0.63–1.39)
Intermittency	550 (23.77)	45 (26.47)	1.16 (0.81–1.65)	1.00 (0.70–1.43)	1.08 (0.72–1.63)
Irritative/storage					
Urgency	753 (32.54)	66 (38.82)	1.32 (0.96–1.82)	1.11 (0.80–1.55)	1.29 (0.89–1.89)
Frequency	814 (35.18)	69 (40.59)	1.26 (0.92–1.74)	1.22 (0.89–1.69)	1.05 (0.72–1.53)
Nocturia	485 (20.96)	84 (49.41)	3.72 (2.70–5.12)	2.90 (2.09–4.03)	2.04 (1.38–3.02)

Data are *n* (%) unless indicated otherwise. ORs are based on frequency of individual symptom >1 point vs. ≤1 point. *Totals vary for each variable because of missing data. †Adjusted for age, BMI, race, marital status, education, and income.

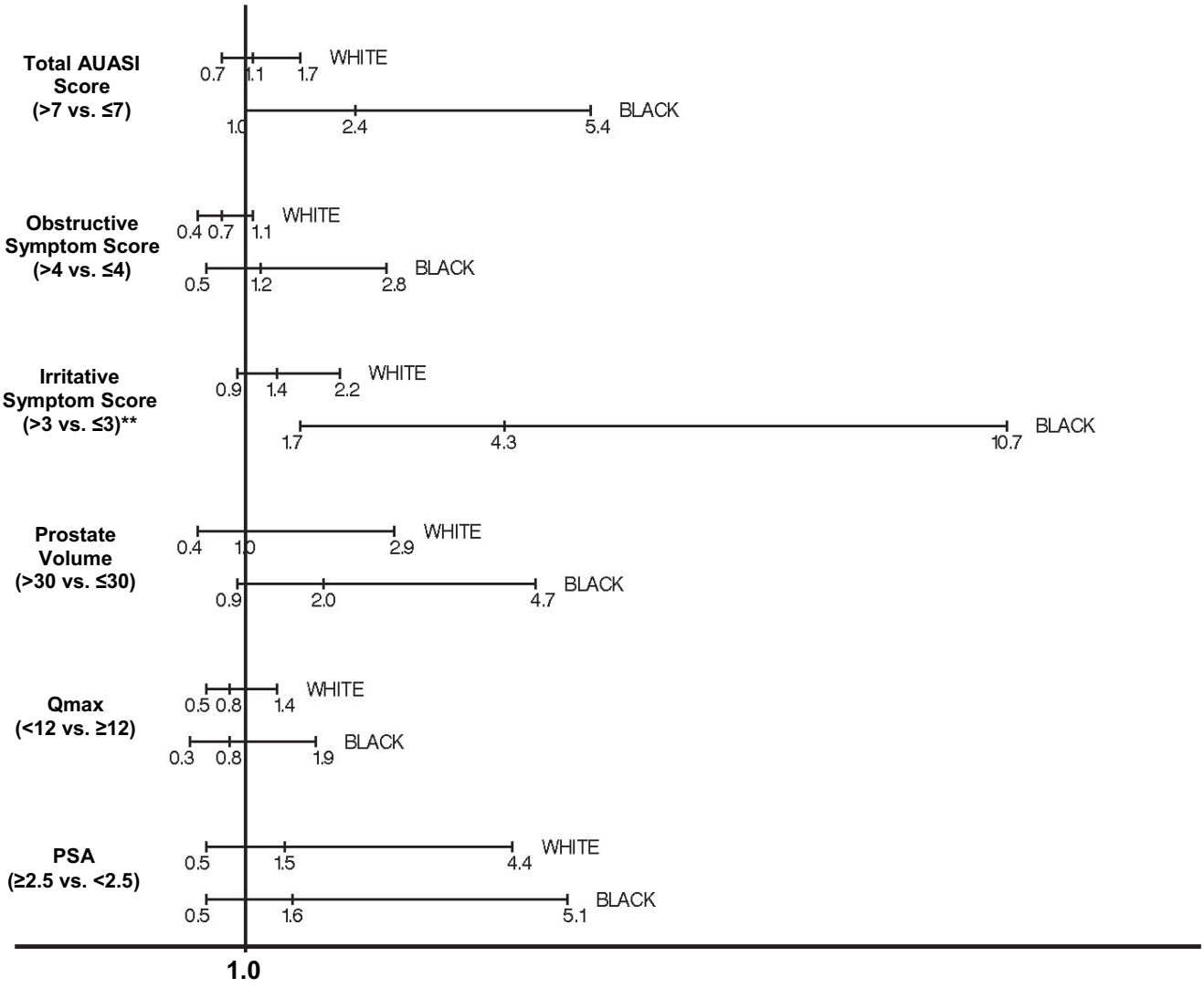

Figure 1—*Associations between BPH characteristics and diabetes status by race. ORs (95% CI) were adjusted for age, BMI, marital status, education, and income. **Interaction between diabetes and race,* $P < 0.10$.

with prostate growth and more closely associated with the dynamic components of lower urinary tract function.

Importantly, in some of the aforementioned studies markers of BPH (e.g., transurethral resection of the prostate) were used to define disease. These markers can be a poor end point for LUTS in diabetic men whose LUTS could be a result of bladder dysfunction (25). Furthermore, the failure to differentiate LUTS from BPH, along with the lack of inclusion of additional clinical markers more specific to BPH, may have contributed to the confusing evidence now seen in the literature (25). Finally, these studies were limited by their inclusion of primarily white men and lack of population-based samples (26,27), limitations that the designs of the FMHS and OCS overcome.

In this study, we observed that diabetes was significantly associated with increased irritative symptom severity. Although we also observed that diabetes was associated with increased overall severity of LUTS, increased prostate volume, increased serum PSA levels, and decreased urinary flow rates, these findings were not statistically significant and suggest that there is no strong evidence for an association between diabetes and BPH across measures. Given the lack of evidence with measures more specific to prostate disease, the association observed between diabetes and irritative LUTS is probably attributed to diabetic neuropathy and is largely driven in this study by the significant association observed specifically between diabetes and nocturia. One explanation for this finding is that poorly managed diabetes in black men would lead to increased thirst and subsequent nocturia compared with that in white men. Although nocturia may be a consequence of changes in bladder reservoir function and/or kidney function secondary to urinary tract obstruction, nocturia has been associated with diabetes in numerous reports (28–30).

There are several mechanisms by which diabetes may influence BPH, the first being changes in insulin concentrations that may, in turn, influence sex hormone concentrations (31), sympathetic

nerve activity, and/or the insulin-like growth factor axis and affect the growth of the prostate (18,32). In addition, poorly controlled diabetes can cause osmotic diuresis, which may be associated with urinary frequency and nocturia and also affect LUTS via neuropathic mechanisms, influencing both motor and sensory nerves (33). These findings are important for several reasons. Although we did not observe statistically significant associations between diabetes and more specific measures of BPH, the magnitude and direction of the associations observed across the spectrum of BPH measures suggest that diabetes may potentially influence not only the dynamic components of lower urinary tract function via the bladder but also prostatic growth. This hypothesis is demonstrated specifically in the marginal positive association observed between diabetes and prostate volume among black men. This observation could be explained, in part, by IGF-1 and IGF binding protein-3 concentrations found not only to vary by race but also to be associated with prostate growth (34,35). As both BPH and diabetes are highly prevalent conditions with significant associated health care costs and morbidity in the U.S., the potential of prostate disease as a complication of diabetes warrants further investigation in study populations with larger samples of men with diabetes.

We also observed that the association between diabetes and severity of irritative LUTS was increased among black men. The finding of racial differences in the relationship between diabetes and LUTS is consistent with previous findings from this cohort, supporting racial disparities in LUTS (5). However, it remains unclear whether this disparity is due to an underlying biological difference in the manifestation of metabolic disease, differences in perception of symptoms, or both. The increased burden of diabetes in black men in this country along with our observed finding of a stronger association between diabetes and LUTS in black men suggest that the potential impact of diabetes on bladder etiology could be of significant concern in this subset of the U.S. population.

Although this is the first study to examine the association between diabetes and clinical markers of BPH in a multiethnic population-based sample of men, there are several limitations that should be considered. First, the cross-sectional nature of the study limits our interpretation of causal relationships between diabetes and BPH within or between races. Second, this study relies on self-reported history of physician-diagnosed diabetes, which may result in the inclusion of individuals with diabetes in the control group or vice versa. It is possible that this misclassification is related to health care utilization, which may be influenced by both race and SES. However, this misclassification is not likely to be differential by markers of BPH and would most likely result in an underestimation of the association between BPH markers and diabetes. In addition, men with the most severe cases of diabetes were excluded from the OCS cohort as a result of the exclusion of individuals with end-organ damage (36). This fact coupled with the higher prevalence of diabetes in the FMHS could have contributed to the greater magnitude of the estimate of association in black men observed in this study. Finally, although the findings reveal positive associations between diabetes and various clinical markers of BPH, we cannot exclude the possibility of chance as an explanation for our findings, as the CIs for the multivariable estimates include 1. This could probably be attributed to the limited sample size available among the clinical subset. Moreover, the resulting changes with the inclusion of the SES indicators specifically with severity of LUTS are consistent with the notion that perhaps indicators of SES (i.e., education) affect perception of rather than actual occurrence of symptoms (5). However, this is the first study with comprehensive clinical data regarding measures of BPH, and estimating the magnitude of the association between BPH and diabetes is an important first step in determining whether relationships indeed exist. These potential limitations are offset by the strengths of this study, including a population-based multiethnic sample of men with a comprehensive set of clinical markers of BPH.

In summary, in this community-based study of BPH and diabetes, we have demonstrated associations between diabetes and increased severity of irritative LUTS, specifically nocturia. Moreover, the magnitude of the association between irritative LUTS and diabetes is increased in black men. Furthermore, there was no strong evidence for an association between diabetes and BPH across measures more specific to BPH (i.e., prostate volume, PSA, and peak urinary flow rate). Taken together, our findings suggest that the presence of diabetes may be less related to prostate growth and more related to the dynamic components of lower urinary tract function. Further evaluations of the association between diabetes and BPH and any racial variation in this association are warranted.

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