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Hyperglycemia, Hyperinsulinemia, Insulin Resistance and the Risk of BPH/LUTS Severity and Progression Over Time in Community Dwelling Black Men: The Flint Men's Health Study

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Abstract

Objective—To examine the associations between fasting serum glucose, insulin concentrations, and insulin resistance and BPH in a population-based cohort of African-American men.

Methods—Using the Flint Men's Health Study (FMHS), we examined how fasting serum glucose and insulin concentrations and calculated HOMA-IR related to burden and progression of clinical markers of BPH in African-American men aged 40–79.

Results—Among 369 men at baseline, mean age was 56.6 years and approximately 70% were overweight/obese (BMI ≥ 25 kg/m²). 148 men (34.4%) reported moderate to severe lower urinary tract symptoms (LUTS) (AUASI ≥ 8). There were no significant trends of metabolic disturbances as measured by serum glucose, insulin or HOMA-IR in men with indications of BPH compared to those without.

Conclusion—In this population-based study of African-American men aged 40–79, we did not observe any significant associations between hyperglycemia, hyperinsulinemia and insulin resistance and burden and progression of BPH after adjustment for age and BMI. This may be due in part to the single measurement and glucose and insulin which may not adequately reflect average glucose metabolism. Further studies examining measures of long-term glycemic control and BPH in racially diverse populations are warranted.

Keywords

diabetes; BPH; LUTS; diabetes; men; aging

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INTRODUCTION

Benign prostatic hyperplasia (BPH) is the most common benign neoplasm in American men. Marked by the progressive development of lower urinary tract symptoms (LUTS), its prevalence increases with age. Over 6.5 million American men meet criteria for treatment. In 2010 alone, 4.5 million visits to physicians' offices were made for BPH and over 87,000 BPH-related surgeries were performed. Exclusive of medical therapy, direct costs of BPH treatment exceed \$1.3 billion annually.¹

Despite its substantial public health impact, much of the etiology and natural history of BPH remains to be elucidated. Previous causal models have focused primarily on sex steroid hormones, essential to normal prostate growth and development, and genetic susceptibility. However, accumulating evidence indicates that metabolic disturbances including abnormal glucose homeostasis, hyperinsulinemia, and insulin resistance may increase the risk of BPH.² In addition, there is a growing body of evidence that suggests diabetes is positively associated with BPH/LUTS.^{3–8} If substantiated, associations of BPH with these abnormalities might suggest novel interventions for prevention and treatment.

The association between metabolic disturbances and BPH/LUTS may be of particular importance among African-American men. It is well established that African-American men are more commonly diagnosed with metabolic conditions such as diabetes, hypertension and obesity when compared to Caucasian men.⁹ These metabolic conditions have all been shown to be positively associated with BPH/LUTS.^{10,11}

Furthermore, the prevalence of LUTS may be greater among African-American men.¹² Therefore, it is important to further our understanding regarding the association between glycemic control and BPH/LUTS among African-American men due to their increased burden of both metabolic diseases and BPH/LUTS.

To explore this association, we used data from a population-based study of African-American men, the *Flint Men's Health Study* (FMHS) to determine whether fasting serum glucose and insulin concentrations, and insulin resistance calculated by the Homeostasis Model of Assessment – Insulin Resistance (HOMA-IR) were associated with the burden and progression of BPH as assessed by LUTS, prostate volume, urinary flow rates and prostate-specific antigen (PSA) concentrations in African-American men. With the incidence of these metabolic abnormalities reaching epidemic proportions in the U.S. and disproportionately increased in African-Americans,¹³ a potential causal relationship with BPH would have substantial implications for the health of the aging male population and underscores the need to further elucidate these processes.

MATERIALS & METHODS

Study Population

Details on subject selection for the FMHS have been previously published.¹⁴ Briefly, the FMHS is a population-based study of black men residing in Genesee County, Michigan. In the FMHS, 730 of 943 eligible black men completed an interview-administered questionnaire in 1996. Of these, 369 men underwent a comprehensive urologic examination, which included, uroflowmetry, transrectal ultrasound (TRUS), serum prostate-specific antigen (PSA) measurement and self-administered American Urological Associations Symptom Index (AUASI) and were deemed to be free of prostate cancer. Four years after baseline (2000), the 369 men who participated in the baseline clinical exam were re-contacted and invited to complete the same study protocol (except for TRUS) described

above. Of the 369 men, 186 (50%) were available and agreed to participate at follow-up. Men who reported treatment or surgery for BPH were excluded.

Measurements

Information on diabetes was gathered by questionnaire in the FMHS at baseline. Participants were asked whether they had ever been diagnosed by a physician to have diabetes mellitus and in which year they were diagnosed. Treatment for diabetes was defined by patient report of use of oral diabetes medications or insulin and categorized based on the highest level of treatment used. Fasting serum insulin and glucose were measured using blood collected during the clinical exam phase of the study at baseline. Assays were performed at the Mayo Clinic Immunochemical Core Laboratory (ICL). Glucose was measured by the hexokinase method (Diagnostic Chemicals Limited, Oxford, CT). Inter-assay variabilities are 3.1% at 84 mg/dL and 2.8% at 292 mg/dL. Insulin was measured by a double antibody radioimmunoassay as described by Hayashi et al.¹⁵ Inter-assay variability is 3.4% at 25 μ U/ml. Information regarding presence/absence of hyperglycemia was assessed via measurement of glucose concentrations as described above. Insulin resistance was assessed via measurement of 1) hyperinsulinemia using circulating insulin levels described above and 2) the Homeostasis Assessment Model - IR (HOMA-IR), a validated model that assesses degree of insulin resistance using the following formula established by Matthews, et al.: $HOMA-IR = (\text{fasting serum glucose (mg/dL)} * \text{fasting serum insulin } (\mu\text{U/mL})) / 405$.¹⁶

The primary endpoints included the following clinical markers of BPH: LUTS severity, maximum urinary flow rate, prostate volume, and serum PSA concentrations. Specifically, LUTS severity was measured by the American Urological Association Symptom Index (AUASI) score via self-administered questionnaires.¹⁷ Prostate volume (ml) determined by transrectal ultrasound, maximum urinary flow rate (ml/sec) measured by uroflowmetry and serum PSA (ng/ml) concentrations were collected during the clinical exam. Although no single surrogate measure provides a definitive non-histologic diagnosis of BPH, previous studies have demonstrated that these measures have adequate construct and predictive validity for BPH.¹⁸

Statistical Analysis

Clinical characteristics of the study population were evaluated at baseline. Distributions (quartiles) of fasting glucose and insulin concentrations and HOMA-IR values were compared by BPH status using chi-square tests. The empirical distribution of annual change (points/year) in AUASI score was calculated by dividing the difference between the baseline and follow-up AUASI score by the number of years between the measurements. Annual percent change for maximum urinary flow rate and PSA concentration was calculated by dividing the difference between baseline and follow-up measure by the product of the baseline measure and the time between the two measures multiplied by 100 (for percent). Annual change in BPH measures were then examined by baseline levels of insulin, glucose and HOMA-IR.

RESULTS

Among the 369 African-American men at baseline, 65 (17.6%) had a self-reported history of diabetes with 56 (15%) reporting control of their disease with either diet/exercise, oral medications or insulin. (Table 1) Mean age at baseline was 56.6 years (standard deviation [SD], 10.3). Overall, 68% of men were overweight/obese (Body Mass Index (BMI) ≥ 25 kg/m²). 41% (n=148) and 34% (n=122) men reported moderate/severe and reduced urinary flow rates, respectively. A greater proportion of men reported irritative symptoms (49%)

compared to obstructive symptoms (29%). Over a third of the population had an ultrasound determined enlarged prostate ($>30\text{cm}^3$). (Table 1)

In bivariate analyses, we examined the distribution of moderate/severe LUTS, enlarged prostate, decreased urinary flow and elevated PSA concentrations by quartiles of fasting glucose, insulin and HOMA-IR. Overall, there were no significant associations between metabolic disturbances as measured by serum glucose, insulin or HOMA-IR and indications of BPH. (Table 2) In multivariable analyses adjusted for age and BMI, mean serum glucose, insulin and HOMA-IR were not significantly associated with LUTS, prostate volume, peak flow rates or total PSA. (data not shown) Finally, no significant differences were observed for annual change in BPH characteristics by serum glucose, insulin and HOMA-IR (Table 3).

DISCUSSION

In this population based-study of African-American men, we examined the association between fasting glucose, insulin and calculated HOMA-IR and measures of BPH. In contrast to several studies that have reported elevated risk of BPH with markers of hyperglycemia, hyperinsulinemia and insulin resistance, we did not observe significant associations between fasting serum glucose, insulin and HOMA-IR and markers of BPH.

A number of previous studies suggest a relationship exists between measures of glucose homeostasis and BPH.² Parsons et al. found that fasting glucose was positively associated with BPH in a cross-sectional study of 422 men.¹⁹ Annual BPH growth rate was also found to be greater among men with higher fasting plasma glucose levels.²⁰ Higher serum insulin levels were found to increase the risk of BPH in a population of Chinese men.²¹ Similar to the findings from this study, Parsons and colleagues found that HOMA-IR was not associated with LUTS in the Osteoporotic Fractures in Men Study.⁷

There are several possible explanations for the observed lack of association. First, it is possible due to the day-to-day intra-individual variation, a one-time measurement of glucose and insulin is not a reliable measure of a person's glycemic control.

Glycosylated haemoglobin levels, in contrast, are an indicator of average glycemia for the previous three months and may be a better marker of long-term glucose control than a single measurement. At least one study that examined HbA1c levels and measures of BPH observed a strong positive association.²² Additionally, in this cohort, we have previously observed significant elevated odds of LUTS in men with diabetes and specifically in diabetic men not taking medications for their condition suggesting that the condition and indications for its severity do in fact demonstrate increased risk for urinary symptoms.²³ While we observed in that study that diabetes was associated with increased LUTS, the lack of associations with prostate volume and serum PSA levels, measures more specific to prostate disease, suggest the impact of diabetes on LUTS is likely attributed to glycosuria and increased urine volumes (diabetic neuropathy). Another possible explanation for the observed lack of association may be due to racial differences in the relationships between glucose homeostasis, hyperinsulinemia, insulin resistance and BPH measures. Previous studies observing strong associations were limited to primarily Caucasian men. It is possible that the sensitivity to the hypothesized BPH effects of insulin varies among ethnic or racial groups and individuals.

There are several mechanisms by which diabetes may potentially influence BPH. First, the trophic effect of increased insulin concentrations observed during the development of type 2 diabetes might induce an enlarged prostate. Because of its structural similarity to insulin-like growth factor (IGF), insulin can bind to the IGF receptor in prostate cells, possibly

activating the receptor to induce growth and proliferation. Alternatively, as insulin levels increase, IGF-1 binding protein (IGFBP-1) declines, thus increasing the bioavailability of IGF.²⁴ Several studies have observed various components of the IGF axis to be associated with the risk of BPH and LUTS.^{22,25,26} High insulin levels may also increase sympathetic nerve activity, which may contribute to an increase of prostate smooth muscle tone.²⁴ Additionally, hyperglycemia itself may play a role by increasing cystolic-free calcium in smooth muscle cells as well as in neural tissue leading to sympathetic nervous system activation. This would coincide with observations by Rohrmann et al. of increased LUTS severity in men with elevated post load glucose concentration as well as with a higher percentage of glycosylated hemoglobin compared with men with lower levels.²² Finally, insulin may influence BPH risk directly by increasing the transcription of genes involved in sex hormone metabolism and thus, influence androgens and estrogens, or indirectly through altered hormone metabolism as a result of obesity.²⁴

Although this is one of the few studies to examine the association between metabolic disturbances and progression of clinical markers of BPH in a population-based sample of African-American men, there are several limitations that should be considered. First, as previously mentioned, this study relies on a one-time measurement of glucose, insulin and HOMA-IR, which due to large intra-individual variability may have only a low ability to characterize a person's long-term glycemic level. Second, the biochemical measures and BPH markers were assessed simultaneously at baseline, therefore we cannot establish temporal sequence with clarity. However, we did not observe an impact of glucose, insulin and HOMA-IR on risk of BPH measures at follow-up. Finally, our small sample size and substantial loss to follow-up may have limited us from observing significant associations. Of the 730 men who completed the interview phase at baseline, 379 men participated in the clinical phase of the study. Owing to issues of consent, we were only able to recontact and invite the 369 men who participated in the clinical phase of the baseline study who had been found to be free of prostate cancer for participation in the follow-up study. Of those 369 men, 186 men who agreed to participate in the follow-up study. When we examined those who participated in the follow-up study and compared them with those who refused or were lost to follow-up, we did not observe any statistically significant differences in mean age, PSA or baseline AUASI scores. These data suggest that older men or men experiencing more significant urologic problems were not more likely to participate in the follow-up study, minimizing the potential for selection bias on the estimates of BPH measures. Importantly, this is one of few studies with comprehensive clinical data regarding measures of BPH and estimating the magnitude of the association between measures of metabolic disturbance and BPH progression is an important first step in determining whether relationships indeed exist. These potential limitations are offset by the strengths of this study, including a longitudinal, population-based sample of African-American men with comprehensive set of clinical markers of BPH and fasting measures of glucose and insulin.

As both BPH and diabetes are highly prevalent conditions of significant burden in the US, the potential of prostate and bladder disease as complications of poorly controlled diabetes warrants further investigation in study populations with larger samples of men with diabetes and identifies a target for primary and secondary prevention.

CONCLUSION

In this community-based study of BPH in African-American men, we did not observe any associations between baseline fasting serum glucose, insulin and HOMA-IR and increased burden or progression of LUTS, prostate volume, PSA concentrations and decreased urinary flow rates. Previous findings from this cohort, however, suggest strong associations between self-reported history of diabetes and LUTS. These findings suggest that the presence of

diabetes and subsequent poor glycemic control may be less related to prostate growth and more related to the dynamic components of lower urinary tract function. Further evaluations of the associations between diabetes, measures of long term glycemic control, and BPH progression among racially diverse populations are warranted.

Acknowledgments

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Table 1

Overall Distribution of Subject Characteristics at Baseline

Characteristic	N(%)
Age (years)	
40–49	107 (29.0)
50–59	117 (31.7)
60–69	93 (25.2)
70–79	52 (14.1)
BMI (kg/m ²)	
Normal (<25 kg/m ²)	119 (32.3)
Overweight (25–29 kg/m ²)	134 (36.3)
Obese (≥ 30 kg/m ²)	116 (31.4)
Physician diagnosed diabetes	
Yes	65 (17.6)
No	304 (82.4)
Diabetes control among diabetics	
Diet/exercise	9 (2.5)
Oral Medications	24 (6.7)
Insulin	23 (6.4)
AUA symptom score	
Mild/None (AUASI ≤ 7)	214 (59.1)
Moderate/Severe (AUASI >7)	148 (40.9)
Obstructive symptom score	
Mild/None (≤ 4)	257 (71.0)
Moderate/Severe (>4)	105 (29.0)
Irritative symptom score	
Mild/None (≤ 3)	186 (51.5)
Moderate/Severe (>3)	175 (48.5)
Prostate volume (cm ³)*	
Normal (≤ 30)	227 (65.6)
Enlarged (>30)	119 (34.4)
Maximal urinary flow rate (mL/s)*	
Normal (≥ 12)	229 (65.2)
Reduced (<12)	122 (34.8)
Serum PSA (ng/mL)*	
≤ 2.5	316 (85.6)
>2.5	53 (14.4)

TABLE 2

Distribution of Baseline Fasting Serum Glucose, Insulin and HOMA (quartiles) by BPH/LUTS Measures

	Lower Urinary Tract Symptoms				Prostate Volume			PSA Concentrations			Peak Urinary Flow Rate		
	Moderate/Severe (AUASI>7)	None/Mild (AUASI ≤ 7)	P	Enlarged (PV>30) *	Normal (PV ≤ 30)	P	Elevated (PSA>2.5) *	Normal (PSA ≤ 2.5)	P *	Decreased (Qmax<12)	Normal (Qmax ≥ 12)	P *	
Fasting Serum Glucose (mg/dL)			0.2			0.5			0.5			0.4	
Q1 (90.0)	41(46.6)	47(53.4)		29(33.3)	58(66.7)		8(8.9)	82(91.1)		26(30.2)	60(69.8)		
Q2 (90.1–101.0)	32(38.1)	52(61.9)		28(35.4)	51(64.6)		16(18.8)	69(81.2)		29(35.8)	52(64.2)		
Q3 (101.1–121.0)	26(34.2)	50(65.8)		28(37.8)	46(62.2)		15(19.7)	61(80.3)		24(33.3)	48(66.7)		
Q4 (121)	30(39.5)	46(60.5)		27(38.0)	44(62.0)		8(10.1)	71(89.9)		28(37.3)	47(62.7)		
Fasting Serum Insulin (μU/ml)			0.4			0.9			0.5			0.8	
Q1 (5.5)	37(44.1)	47(55.9)		29(34.9)	54(65.1)		13(15.1)	73(84.9)		35(42.7)	47(57.3)		
Q2 (5.6–9.9)	28(35.9)	50(64.1)		28(37.3)	47(62.7)		10(12.5)	70(87.5)		20(26.3)	56(73.7)		
Q3 (10.0–24.9)	35(43.2)	46(56.8)		28(36.8)	48(63.2)		15(18.3)	67(81.7)		23(30.3)	53(69.7)		
Q4 (25.0)	29(35.8)	52(64.2)		27(35.1)	50(64.9)		9(11.0)	73(89.0)		29(36.3)	51(63.7)		
HOMA-IR			0.7			0.8			0.3			0.9	
Q1 (1.3)	33(41.8)	46(58.2)		28(35.0)	52(65.0)		12(14.6)	70(85.4)		34(44.2)	43(55.8)		
Q2 (1.4–2.5)	34(41.0)	49(59.0)		30(39.0)	47(61.0)		15(18.1)	68(81.9)		20(25.6)	58(74.4)		
Q3 (2.6–7.4)	31(38.3)	50(61.7)		27(35.5)	49(64.5)		11(13.3)	72(86.7)		23(29.1)	56(70.9)		
Q4 (7.5)	31(38.3)	50(61.7)		27(34.6)	51(65.4)		9(11.0)	73(89.0)		30(37.5)	50(62.5)		

* Based on Mantel-Haenszel Chi-Square Test

Annual Changes in BPH/LUTS characteristics over time by diabetes status in community-dwelling white and black men at baseline

Table 3

	AUASI score (points)	Maximum urinary flow rate (%)	Total PSA (%)
Fasting Serum Glucose (mg/dL)			
Q1 (90.0)	-0.1(6.1)	-3.2(10.2)	0.4(0.7)
Q2 (90.1–101.0)	-0.6(5.4)	-1.1(10.4)	0.3(1.0)
Q3 (101.1–121.0)	-0.3(3.8)	-2.2(7.5)	0.7(1.2)
Q4 (121)	0.9(7.5)	-3.1(10.3)	0.1(0.9)
Fasting Serum Insulin (μU/ml)			
Q1 (5.5)	-1.3 (6.1)	-2.3(10.5)	0.6(1.2)
Q2 (5.6–9.9)	0.4 (5.1)	-2.2(10.4)	0.3(0.8)
Q3 (10.0–24.9)	-0.7(6.7)	-4.1(8.7)	0.4(0.9)
Q4 (25.0)	1.4(5.7)	-1.2(10.2)	0.1(0.7)
HOMA-IR			
Q1 (1.3)	-0.5(6.3)	-2.3(10.3)	0.6(1.1)
Q2 (1.4–2.5)	-1.1(4.0)	-2.2(10.0)	-0.4(0.9)
Q3 (2.6–7.4)	-0.2(6.9)	-4.1(9.1)	0.4(0.9)
Q4 (7.5)	1.9(6.3)	-1.3(10.2)	0.1(0.7)