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BENIGN PROSTATE-SPECIFIC ANTIGEN DISTRIBUTIONS AND ASSOCIATIONS WITH UROLOGIC OUTCOMES IN COMMUNITY-DWELLING BLACK AND WHITE MEN

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Abstract

Purpose—To describe the cross-sectional associations of benign prostate-specific antigen (BPSA) with clinical urologic measures, and to examine the risk of future urologic outcomes in two population-based cohorts of black and white men.

Materials and Methods—Two population-based, cohort studies were established to characterize the natural history and risk factors for progression of prostate disease in white and black male residents of Olmsted County, Minnesota and Genesee County, Michigan, respectively.

Results—The distribution of BPSA levels was similar in blacks (median (25th, 75th percentiles) =32.9 (17.3, 68.0) pg/mL) and whites (median=32.2 (16.6,68.9) pg/mL), $p=0.71$, but much lower than previous reports. For Olmsted County men in the upper quartile of BPSA, there was a 15-fold increased risk of prostate cancer (CaP; hazard ratio (HR): 14.6 95% confidence interval (CI): 3.1, 68.6) and a 2-fold higher risk of treatment for benign prostatic hyperplasia (BPH; HR: 2.2, 95% CI: 1.2, 4.2) after adjusting for age. After additional adjustment for baseline PSA, the association between BPSA and CaP risk was attenuated, but remained nearly 9-fold higher for men in the upper BPSA quartile (HR: 8.7, 95% CI: 1.8, 42.4). The 2-fold higher risk of treatment for BPH also remained after adjustment for baseline PSA for men in the upper BPSA quartile (HR: 1.9, 95% CI: 0.9, 4.0).

Conclusions—These results suggest that elevated BPSA level may help to identify men at risk of treatment for BPH and identify men with prostate cancer.

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Keywords

prostate enlargement; prostate cancer; biomarkers; BPSA

INTRODUCTION

Prostate-specific antigen (PSA) is a widely used serum marker for the early detection of prostate cancer (CaP) and is recommended in men over 50 years or at high risk of CaP by the National Cancer Institute/American Urological Association guidelines.[National Cancer Institute. Prostate cancer screening/Prostate-specific Antigen (PSA) test 2011 1/04/2011 [cited; Available from: <http://www.cancer.gov/cancertopics/factsheet/Detection/PSA>]; American Urologic Association. Clinical Guidelines/Prostate Specific Antigen 2011 1/04/2011 [cited; Available from: <http://www.auanet.org/content/media/psa09.pdf>] However, elevated PSA levels are not specific to CaP, since PSA can also be elevated in benign conditions such as benign prostatic hyperplasia (BPH) or prostatitis.

Benign prostate-specific antigen (BPSA) is an inactive form of PSA that has been cleaved at lysine residues 145 and 182.¹² This form of free PSA has been shown to be elevated in nodular BPH tissue and is also correlated with transition zone volume and prostatic enlargement.^{1, 3} Additionally, BPSA was found to be significantly higher in men with an elevated PSA level and a percent free PSA level less than 15% who were also biopsy negative for CaP.⁴ Together, these results suggest that BPSA may be associated with benign disease.

The characteristics of BPSA have been described in clinical and convenience samples.^{1, 3–5} These studies provide initial support for the usefulness of BPSA as a marker for benign prostate disease; however, they may not reflect the full spectrum of men with and without benign prostate disease. The objective of this paper is to provide normative data in a population-based, biracial sample of men and to describe the cross-sectional associations between baseline values of BPSA and common clinical urologic measures. In addition, we examined associations between baseline values of BPSA and risk of clinically relevant urologic outcomes such as acute urinary retention (AUR), CaP, and treatment for BPH.

METHODS

Details on subject selection for both the Olmsted County Study of Urinary Symptoms and Health Status among Men (OCS) and Flint Men's Health Study (FMHS) have been published previously.^{6, 7} Briefly, the OCS and FMHS are population-based, cohort studies established to characterize the natural history and risk factors for progression of prostate disease in white and black male residents of Olmsted County, Minnesota and Genesee County, Michigan, respectively. In the OCS, 2,115 of 3,874 (55%) 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, completed the self-administered American Urological Association Symptom Index (AUASI).⁸ A detailed urologic examination that included uroflowmetry, digital rectal exam, transrectal ultrasound (TRUS), and serum PSA measurement was performed in a 25% random subsample (476 participants of 537 sampled, 89%).^{6, 9} The cohort was actively followed on a biennial basis for seventeen years using a protocol similar to the initial examination.

Applying similar criteria and procedures as the OCS, the FMHS recruited 730 of 943 (77%) eligible black men to complete an interview-administered questionnaire in 1996.^{7, 10} Of these, 369 (51%) men were free of prostate cancer and completed a comprehensive urologic

examination that included, as in the OCS, uroflowmetry, TRUS, a serum PSA measurement, and a self-administered AUASI.⁷ The selective participation in the clinical examination and potential resulting selection bias have been addressed previously.^{11, 12}

Since the FMHS urologic measurements were collected in 1996, BPSA measures were obtained from serum samples collected in 1996 for both studies and the corresponding 1996 OCS measurements were used as the initial/baseline values. Levels of BPSA were measured in 420 OCS men and 329 FMHS men. This study received approval from the Mayo Clinic, Olmsted Medical Center, and University of Michigan Medical School Institutional Review Boards.

Urinary flow rates

Urinary flow rates were measured using a Dantec 1000 uroflowmeter calibrated by trained study assistants.¹³ Electronically measured maximum urinary flow rates, average flow rates, and voided volume were determined. Measurements were repeated if voided volume was less than 150 mL. The highest rate with a voided volume ≥ 150 mL was used in analyses where multiple measurements existed.

Prostate volume

Total prostate volume for the OCS and FMHS participants were measured via transrectal ultrasound (TRUS).^{7, 14} In addition to assessing the echogenic pattern of the prostate gland, three measurements were made to calculate the total volume of the prostate assuming a prolate ellipsoid shape.¹⁵

Prostate cancer and acute urinary retention

Information on use of medical and surgical LUTS/BPE treatments was obtained through self-report and through passive follow-up of the community medical records for the OCS men. Dates of biopsy-confirmed prostate cancer and acute urinary retention were abstracted from community medical records. Acute urinary retention was defined as medical record documentation of both the inability to urinate and catheterization to empty the bladder. Catheterizations done perioperatively were excluded, but episodes in which a catheter was needed after surgery, secondary to acute retention were included. Data on CaP, AUR and treatment were not available for the FMHS men; therefore analyses examining associations between BPSA and these outcomes were confined to the OCS cohort.

Assays

For both studies, serum samples were obtained prior to any prostatic manipulations, including digital rectal examination and transrectal ultrasound, and were frozen at -70°C for latter assays. Stored blood samples from the OCS fourth biennial round (1996) of follow-up ($n=42$) and from the FMHS baseline (1996) visit ($n=329$) were used to measure BPSA levels. Serum PSA levels for men in the OCS were assayed using the Tandem-E PSA assay (Hybritech, Incorporated, San Diego, CA). Serum PSA levels for men in the FMHS were assayed using the Abbott AxSYM polyclonal-monoclonal immunoassay (Abbott Diagnostics, Abbott Park, IL). The lower limit of detection for this Abbott assay is reported to be 0.1 ng/mL.¹⁶ We have previously reported that PSA determinations were consistent across different assays and laboratories.^{17,18} For both studies, BPSA levels were assessed using an automated, sequential, two-step immunoenzymatic (“sandwich”) assay developed for use on the Beckman Coulter (Brea, CA) Access® instrument. The BPSA measurements were run on an Access 2 Immunoassay analyzer using research-use-only two-site immunoenzymatic reagents provided by Beckman Coulter, Inc. In our hands, the intra-assay

variation ranged from 4.3% to 8.1% while the inter-assay variation ranged from 5.1% to 5.2%.

Statistical analysis

Descriptive statistics and cumulative distribution function plots were used to describe the distributions of BPSA. Spearman correlations (r_s) and logistic regression were used to investigate the relationships between BPSA and other baseline urologic measures. Multivariable logistic regression was used to describe the association of BPSA with clinically meaningful diagnostic measures, which are presented as odds ratios (ORs) and 95% confidence intervals (CIs). Moderate-to-severe LUTS was defined as AUASI score >7 while depressed urinary flow rate was defined as maximum urinary flow rate <12 mL/s.¹⁹ Prostatic enlargement was defined as prostate volume >30 cc.¹⁹ Due to availability of follow-up data, Cox proportional hazards regression was used in the OCS cohort only to investigate the association of baseline values of BPSA with the risk of a subsequent diagnosis of AUR, biopsy confirmed CaP, and treatment for BPH which are presented as hazard ratios (HRs) and 95% CIs.

RESULTS

Baseline characteristics of the participants in each cohort are shown in Table 1. Urological characteristics were similar for FMHS and OCS men. The two cohorts were also similar with respect to distribution of BPSA levels. BPSA percentile levels increased across age decade almost uniformly for each percentile shown (Figure 1). The 5th, 10th, 25th, 50th, 75th, 90th, and 95th percentiles of the combined BPSA distributions were 7.3, 10.1, 16.9, 32.5, 68.8, 157.0, and 267.9 pg/mL, respectively.

BPSA was significantly correlated with age and all urologic measures examined, with Spearman correlations (r_s) ranging from 0.11–0.82 (all P values <0.05 , Table 2). BPSA was most strongly correlated with PSA levels (r_s : 0.82 and 0.80 for OCS and FMHS, respectively; $p<0.0001$) as well as age ($r_s=-.58$, $r_s=0.4$) and prostate volume ($r_s=0.52$, $r_s=0.5$).

In multivariable models for both cohorts, men in the upper quartile of BPSA were more likely to have moderate-to-severe LUTS, a depressed urinary flow rate, and an enlarged prostate compared to men in the lower quartile (supplementary table available on journal website). The magnitude of the association between baseline values of BPSA and the urologic measures increased with increasing quartile of BPSA; however, it was most pronounced for an enlarged prostate (tests for trend: all P values <0.05).

Overall 46 (11.3%) men in the OCS had AUR, 111 (27.8%) of men were treated for BPH, and 41 (9.8%) men were diagnosed with CaP after the baseline BPSA measurements. Men with higher baseline levels of BPSA (upper quartile) were more likely to have a subsequent event of AUR (age-adjusted HR: 2.1, 95% CI: 0.7, 6.5), biopsy-confirmed CaP (age-adjusted HR: 14.6, 95% CI: 3.1, 68.6), and treatment for BPH (age-adjusted HR: 2.2, 95% CI: 1.2, 4.2) than men in the lowest quartile level of BPSA (Table 3). For all three outcomes, the magnitude of the association increased with increasing BPSA quartile (tests for trend, age-adjusted P values: 0.17, <0.0001 , and 0.003 for AUR, treatment for BPH, and CaP, respectively). **The majority of the CaP tumors were low grade (78% Gleason score of 6 or lower).**

After further adjusting for baseline PSA, the associations between BPSA and all outcomes were attenuated (Table 3). However, the association between BPSA and CaP remained nearly 9-fold higher for men in the upper quartile relative to men in the lowest quartile (HR:

8.7, 95% CI: 1.8, 42.4). **Further adjustment for prostate volume did not change the results (results not shown).** Additionally, the association between the highest quartile of BPSA and a two-fold increased risk of BPH treatment also remained after adjustment for baseline PSA (Table 3; HR: 1.9, 95% CI: 0.9, 4.0), however it was no longer statistically significant.

DISCUSSION

These results suggest that black and white men have similar distributions of BPSA. Men with higher levels of BPSA were more likely to have a depressed urinary flow rate and an enlarged prostate. For the OCS, men with higher baseline levels of BPSA were also more likely to receive subsequent treatment for BPH (i.e., medical therapy, procedure or surgery) and were more likely to be subsequently diagnosed with biopsy-confirmed CaP even after adjusting for age and baseline PSA.

The median (25th, 75th percentiles) BPSA levels in the OCS and FMHS cohorts were 32.2 (16.6, 68.9) and 32.9 (17.3, 68.0) pg/mL, respectively. These levels are much lower than levels previously reported in patients receiving a biopsy or patients who had a diagnosis of prostate cancer. Canto, et al. in a study of 91 consecutive patients without prostate cancer who underwent a 10-core biopsy, reported a median BPSA level of 220 pg/mL.³ This observed value of BPSA falls at approximately the 93rd percentile in the combined FMHS and OCS distribution. In another study, de Vries, et al. reported data from 61 men participating in the screening arm of a large cancer screening study in which men with less aggressive prostate cancer had a median BPSA value of 172 pg/mL and those men with more aggressive prostate cancer had a median BPSA value of 264 pg/mL, again, falling into approximately the 91st and 95th percentile of the BPSA distribution observed for the combined cohorts.⁵ Linton, et al. reported median BPSA levels of 220 pg/mL and 170 pg/mL in 79 men with BPH (defined as elevated PSA with negative biopsy) and 91 men with prostate cancer, respectively.²⁰ These median values reported by Linton for BPSA were both above the 90th percentile of the BPSA distribution for this study. The higher levels observed in these other studies could be due to differences in the underlying populations or to laboratory measurement/calibration.

BPSA has been associated with clinically significant prostatic enlargement in the transition zone and Canto referred to this result as evidence of the potential of BPSA as a novel predictor of outcomes or response to therapy in patients with BPH.³ Results from phase II and III studies of the Medical Therapy of Prostatic Symptoms Prostatic Samples Analysis Consortium suggested that BPSA is predictive of BPH progression after accounting for prostate volume.²¹ The current study found that BPSA was correlated with prostate volume and PSA level in both black and white men. BPSA was also modestly correlated with AUASI score and maximum urinary flow rate in both cohorts. In the OCS, men with higher baseline levels of BPSA were also more likely to receive subsequent treatment for BPH. These data suggest that BPSA may have utility in predicting LUTS severity and prostate enlargement.

Based on an artificial neural network model, Stephan, et al. also found that BPSA may improve prostate cancer detection.²² In this study, higher levels of BPSA were associated with risk of biopsy-confirmed CaP after adjustment for age and baseline PSA level. This result seems to support the potential utility of this marker in further distinguishing men with or without prostate cancer in the presence of a PSA test. It has been suggested that BPSA may improve the sensitivity and specificity of detecting prostate cancer in men who have PSA levels in the 4–10 ng/mL range or when percent free PSA is less than 15%.⁴

Due to the restriction in the range of values in both the benign prostate disease group and the prostate cancer group, the study samples from these previous reports may not necessarily be the most appropriate for investigating assay performance in differentiating benign prostate disease or prostate cancer. It appears that both the benign prostate disease group and the prostate cancer groups from the previous reports have high levels of BPSA when compared to the general population. Thus, the previous reports may actually be a conservative estimate of the utility of distinguishing diseased men from healthy men if the higher levels observed in these other studies are due to the underlying study populations and the true differences between “diseased” and “non-diseased” men are greater.²³

The generalizability of the results to other races or ethnicities may be limited; however, few differences were seen between black and white men in cross-sectional analyses.

Furthermore, the wide confidence intervals for some of the parameter estimates may be a result of higher variability due to the small number of cases. In addition, the stability of BPSA in frozen samples is currently unknown, however free and total PSA have appeared to be stable in serum samples frozen two years at -70°C .²⁴

CONCLUSIONS

These population-based data provide useful reference ranges to researchers for future studies examining the utility of BPSA, with similar distributions of BPSA for white and black men. Elevated levels suggest some prostate disease, benign or malignant. Further work is needed to develop our understanding of how this might be used in a diagnostic workup.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

PSA	prostate-specific antigen
CaP	prostate cancer
BPSA	benign prostate-specific antigen
AUR	acute urinary retention
TRUS	transrectal ultrasound
OR	odds ratios
CI	confidence intervals
HR	hazard ratios
AUASI	American Urological Association Symptom Index

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FIGURE 1. Cumulative distribution function plots of BPSA by age decade in the combined Olmsted County Study and Flint Men's Health Study Sample.

TABLE 1

Baseline characteristics of Olmsted County Study of Urinary Symptoms and Health Status among Men and Flint Men's Health Study.

Characteristic	OCS N=420	FMHS N=329	P value*
	N (%) [†]	N (%) [†]	
Age			
40–49	112 (26.7)	91 (27.7)	0.64
50–59	143 (34.0)	104 (31.6)	
60–69	92 (21.9)	83 (25.2)	
70+	73 (17.4)	51 (15.5)	
AUASI score (>7)	183 (43.6)	128 (39.6)	0.28
Maximum urinary flow rate (<12 mL/s)	67 (18.9)	28 (14.4)	0.18
Prostate volume (>30 cc)	153 (40.7)	111 (35.8)	0.19
PSA (>1.4 ng/mL)	146 (34.8)	109 (33.1)	0.64
	Median (Q1, Q3)	Median (Q1, Q3)	
BPSA (pg/mL)	32.2 (16.6, 68.9)	32.9 (17.3, 68.0)	0.71

* Rank-sum P value reported for continuous variables, chi-square P value reported for dichotomous variables

[†] Percentages based on non-missing observations

AUASI: American Urological Association Symptom Index; Q1: 25th percentile, Q3: 75th percentile

TABLE 2

Spearman correlations between baseline BPSA and baseline clinical urologic measures.

Characteristic	BPSA	
	OCS	FMHS
	r_s	r_s
Age	0.58 [*]	0.40 [*]
Maximum flow rate	-0.18 [*]	-0.21 [*]
Prostate volume	0.52 [*]	0.50 [*]
AUASI score	0.28 [*]	0.11 [*]
PSA	0.82 [*]	0.80 [*]

*
P<0.05

TABLE 3

Crude and age- and PSA-adjusted association of baseline BPSA levels with occurrence of urologic outcomes in the Olmsted County Study.

BPSA quartile (range, pg/mL)	Acute Urinary Retention (N=46)			Biopsy-Confirmed Prostate Cancer (n=41)			Treatment for BPH (N=111)		
	Crude HR (95% CI)	Age-adj. HR (95% CI)	Age-, PSA-adj. HR (95% CI)	Crude HR (95% CI)	Age-adj. HR (95% CI)	Age-, PSA-adj. HR (95% CI)	Crude HR (95% CI)	Age-adj. HR (95% CI)	Age-, PSA-adj. HR (95% CI)
Quartile 1 (min-16.9)	reference	reference	reference	reference	reference	reference	reference	reference	reference
Quartile 2 (17.0-32.5)	1.7 (0.5, 5.1)	1.5 (0.5, 4.5)	1.4 (0.5, 4.4)	3.7 (0.8, 17.9)	3.8 (0.8, 18.4)	3.7 (0.8, 17.6)	0.8 (0.4, 1.5)	0.7 (0.4, 1.4)	0.7 (0.4, 1.4)
Quartile 3 (32.6-68.8)	2.6 (0.9, 7.3)	1.8 (0.6, 5.3)	1.7 (0.6, 4.9)	5.5 (1.2, 25.2)	5.8 (1.3, 27.0)	5.2 (1.1, 24.1)	1.6 (0.9, 2.9)	1.5 (0.8, 2.6)	1.4 (0.8, 2.5)
Quartile 4 (68.9-max)	4.6 (1.7, 12.2)	2.1 (0.7, 6.5)	1.5 (0.4, 5.4)	12.9 (3.0, 54.8)	14.6 (3.1, 68.6)	8.7 (1.8, 42.4)	2.9 (1.7, 4.9)	2.2 (1.2, 4.2)	1.9 (0.9, 4.0)
P trend	0.0004	0.17	0.48	<0.0001	<0.0001	0.004	<0.0001	0.003	0.04

HR: hazard ratio, CI: confidence interval