Obesity Is Negatively Associated with Prostate-Specific Antigen in U.S. Men, 2001-2004

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Abstract

Background: Recent studies have shown a negative association between body mass index (BMI) and prostate-specific antigen (PSA), a commonly used serum marker for the detection and diagnosis of prostate cancer. We have examined the association between several anthropometric measures and PSA in a nationally representative sample of men.

Methods: We analyzed data from the 2001-2004 National Health and Nutrition Examination Survey. Participants in this study were men ages ≥40 years without previously diagnosed prostate cancer who had PSA measured. Height, weight, waist circumference, BMI, triceps skinfold, subscapular skinfold, and calculated total body water were examined categorically by quintiles using multiple linear regression models. All tests of significance were two sided.

Results: Among white men, we report a trend for decreasing PSA with increasing weight, BMI, waist circumference, triceps skinfold thickness, and calculated total body water. Among Mexican American men, we found a trend for decreasing PSA with increasing BMI, and among black men we found a trend for decreasing PSA with increasing triceps thickness. None of the interaction terms between race/ethnicity and any of the anthropometric measures were statistically significant. Controlling for age and race/ethnicity in the multiple linear regression model, we found moderate declines in PSA with a 1 SD increase in BMI [5.9% decrease (95% confidence interval, -9.0% to -2.8%) in geometric mean PSA per 5.2-unit increase], weight [5.9% decline (-8.8% to -2.8%) per 17.7-kg increase], waist circumference [6.6% decline (-9.4% to -3.6%) per 13.4-cm increase], triceps skinfold [5.4% decline (-8.9% to -1.8%) per 6.4-mm increase], and calculated total body water [5.7% decline (-8.9% to -2.4%) per 6.5-liter increase].

Conclusion: Our population-based, nationally representative results expand the validity of previous studies on obesity and PSA. Higher weight, BMI, waist circumference, triceps skinfold, and total body water are associated with moderately lower PSA values. A prospective study is needed to verify whether this association affects the accuracy of the PSA test in obese men. (Cancer Epidemiol Biomarkers Prev 2007;16(1):70–6)

Introduction

The prevalence of obesity in the United States has sharply increased over the last 30 years, particularly among men ages 60 to 75 years, in whom the absolute prevalence has more than tripled to 35.8% (1). This rapid increase in obesity is expected to greatly increase the future incidence and mortality rate of several chronic diseases (2). Prostate cancer is the second leading cause of cancer death among U.S. men and it is primarily diagnosed in men ages ≥50 years (3). Several cancers have been strongly linked with obesity, but the relationship between prostate cancer and obesity seems to be more complex (4). Studies have been unable to consistently show an association between body mass index (BMI) and prostate cancer incidence (5) but instead found a mixture of positive (6, 7), negative (8), and null associations (9-12).

On the other hand, several studies have found that a higher BMI is associated with an increased risk of prostate cancer death (9), higher Gleason scores at diagnosis (13-15), and worse outcomes after treatment (14-16). Of note are the large increases in the prevalence of obesity in the United States. This rapid increase in obesity is expected to greatly increase the future incidence and mortality rate of prostate cancer, and in the management of disease after diagnosis (22). Although specific recommendations vary, several organizations suggest that doctors begin discussing PSA screening when their male patients reach the age of 50 years (23-25). Yet many men with “normal” PSA values have cancer, and many investigators have commented on the PSA test’s lack of specificity (26). PSA values increase with age and prostate volume, and they may vary across racial groups. Some researchers have attempted to improve the accuracy of the test by controlling for these factors in the interpretation of PSA results (27). As a part of this strategy, several cross-sectional studies have examined the association between BMI and PSA levels. Investigators have shown that PSA levels are lower in obese men (28-30), and some have proposed this as a mechanism for later detection of prostate cancer. This article must therefore be hereby marked advertisement in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

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Note: The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

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cancer in obese men (31, 32). The National Health and Nutrition Examination Survey (NHANES) provides nationally representative data on PSA and a number of anthropometric measures including height, weight, BMI, triceps and subscapular skinfolds, and waist circumference. We have analyzed data from the 2001-2004 surveys to examine the magnitude of the association between PSA and obesity and to discuss the implications of this association for PSA screening.

### Materials and Methods

#### Study Population and Sample Design.

The NHANES 2001-2004 is a population-based cross-sectional survey of the civilian noninstitutionalized U.S. population conducted by the Centers for Disease Control and Prevention National Center for Health Statistics. Details of the procedures involved in sampling and data collection have been published elsewhere (33). Briefly, this survey used a complex, multistage probability sample based on a selection of counties, blocks, households, and persons within households. Mexican Americans, non-Hispanic blacks, and adults ages ≥40 years were oversampled. From 2001 to 2004, 68.6% of men ages ≥40 years who were contacted by National Center for Health Statistics were interviewed, examined, and eligible for phlebotomy. All procedures were approved by the National Center for Health Statistics institutional review board; written, informed consent was obtained from all participants.

Men ages ≥40 years were eligible for PSA testing. After these men received general information about the test from the examining NHANES physician, they were offered the opportunity to be tested. Men were excluded from the test if they refused or reported having any of the following procedures or conditions because these may have altered the results: current infection or inflammation of the prostate gland, digital rectal exam in the past week, prostate biopsy in the past 30 days, cystoscopy in the past 30 days, or history of prostate cancer. Our methods for collecting and handling the PSA samples have previously been published (34).

#### Anthropometric Measures.

NHANES data continue to provide national growth charts for the United States. National Center for Health Statistics trains NHANES technicians to follow rigorous standardized procedures for the collection of anthropometric data. Electronic measuring devices automatically enter data to minimize error due to parallax. The National Center for Health Statistics anthropometry protocol for the NHANES can be found online (35). Participants were weighed wearing underwear, a disposable paper gown, and a pair of foam slippers. Standing height was measured with a fixed stadiometer with a vertical backboard and a movable headboard. Waist circumference was measured using a tape around the narrowest part of the waist. Subscapular skinfold thickness was measured on the back of the right arm, midway between the acromion and olecranon processes. Subscapular skinfold thickness was measured on the inferior angle of the scapula.

#### Statistical Analysis.

We categorized self-reported race/ethnicity as non-Hispanic white, non-Hispanic black, Mexican American, and other (other Hispanics and all others). Because the number of participants was relatively limited in the “other” category, we restricted this analysis to non-Hispanic whites, non-Hispanic blacks, and Mexican Americans. Of the 3,126 eligible men in the three race/ethnicity groups ages ≥40 years, 2,930 (93.7%) men participated in the NHANES examination. Of these 2,930 men, 76 (2.6%) refused the PSA test, refused to answer the consent question, or responded “don’t know”; 222 (7.6%) were considered ineligible because of the other exclusion criteria; an additional 158 (5.4%) were missing information on at least one of the exclusion criteria; and 78 (2.7%) had a missing PSA result but were otherwise fine by any of the exclusion criteria. The final study population contained 2,396 men, or 81.8% (2,396 of 2,930) of all men ages ≥40 years who participated in the NHANES examination. Comparing the men excluded or with missing exclusion criteria that had PSA measured to the men in our analysis, we found that the excluded men were older, more likely to be black, and had lower mean height, weight, and BMI. The two groups did not differ by mean PSA value when PSA was considered continuously; however, those excluded were more likely to have a PSA >4.0 ng/mL. The number of men with missing anthropometric measures varied by the measure; subscapular skinfold had the most missing observations (n = 491) and height had the fewest (n = 45).

For the unadjusted results (Tables 1 and 2), the distribution of continuous variables is presented as medians with 25th and 75th percentiles, and discrete variables are presented as weighted percentages. Statistical testing for differences in continuous variables was done using linear regression models with the ranks of the continuous variable of interest as the dependent variable and the comparison groups as the independent variable. Statistical testing for discrete variables was done using the Pearson χ2 test.

In Tables 2 and 3, we examined the associations between PSA and categories of the anthropometric measures. Measures were categorized into quintiles with the exception of BMI, which was categorized according to WHO classifications (<25, 25-25.9, 30-34.9, 35-39.9, >40; ref. 33). An estimator of plasma volume, total body water was calculated using a formula from the literature [2.447 − 0.09516 × age + 0.1074 × height (cm) + 0.3362 × weight (kg); ref. 34].

Race/ethnicity-specific predictive margins were computed from multiple linear regression models for each of the anthropometric measures controlling for age. The predictive margin for a given group represents the average predicted response if everyone in the sample had been in that group (36). Statistical testing for a trend over each anthropometric

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**Table 1. Anthropometric measures for U.S. men ≥40 years of age, NHANES 2001-2004**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>White (n = 1,476)</th>
<th>Black (n = 435)</th>
<th>Mexican American (n = 485)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>52.0 (46.0, 63.0)</td>
<td>50.0 (44.0, 58.0)</td>
<td>49.0 (44.0, 58.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>177.3 (172.1, 182.2)</td>
<td>176.5 (172.5, 181.4)</td>
<td>170.3 (165.7, 174.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>88.6 (78.9, 99.4)</td>
<td>86.1 (75.3, 100.4)</td>
<td>81.2 (72.2, 90.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total body water (liter)</td>
<td>28.3 (25.5, 31.2)</td>
<td>27.3 (24.6, 31.6)</td>
<td>28.2 (25.7, 31.0)</td>
<td>0.110</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>103.3 (95.7, 111.5)</td>
<td>97.8 (89.7, 108.7)</td>
<td>100.4 (93.3, 106.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triceps skinfold (mm)</td>
<td>14.2 (11.0, 19.0)</td>
<td>12.5 (8.4, 17.3)</td>
<td>12.3 (9.6, 16.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Subscapular skinfold (mm)</td>
<td>19.4 (15.0, 24.2)</td>
<td>19.0 (13.3, 27.0)</td>
<td>20.1 (16.2, 24.4)</td>
<td>0.301</td>
</tr>
<tr>
<td>Total body water (liter)</td>
<td>46.2 (42.4, 50.3)</td>
<td>45.5 (41.5, 50.4)</td>
<td>43.2 (39.9, 46.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Obese, BMI ≥30 kg/m² (%)</td>
<td>33.5</td>
<td>33.2</td>
<td>33.1</td>
<td>0.988</td>
</tr>
<tr>
<td>PSA &gt;4 ng/mL (%)</td>
<td>5.9</td>
<td>7.8</td>
<td>3.5</td>
<td>0.050</td>
</tr>
</tbody>
</table>

**NOTE:** Continuous variables are presented as median (25th percentile, 75th percentile); discrete variables are presented as weighted percentages.

*P* value for testing the association between race and the ranks for the continuous variable; Pearson χ2 test for discrete variables.
measure was done by testing the hypothesis that the β coefficient for the continuous linear variable was equal to zero. A logistic regression model was fit to determine the effect of obesity (BMI ≥30 kg/m²) on elevated PSA (PSA ≥4.0 ng/mL) after controlling for age and race/ethnicity. Results are presented as predictive margins.

Linear regression models were fit to determine the continuous relationship between each anthropometric measure and PSA. We used restricted cubic spline functions to assess the linearity assumption between each continuous independent variable and PSA (36). Only age had a significant nonlinear relationship and was modeled with splines in the final models. In all models, we adjusted for race/ethnicity and an interaction variable and PSA (36). Only age had a significant nonlinear relationship and was modeled with splines in the final models.

Results

Distributions of the anthropometric measures for the study population are presented by race/ethnicity in Table 1. Distributions of height, weight, waist circumference, triceps skinfold thickness, and total body water varied by race/ethnicity. The percentage of men classified as obese did not vary by race/ethnicity, but the percentage of men with a PSA ≥4.0 ng/mL did vary.

Some of the anthropometric measures exhibited a high degree of correlation with each other. Waist circumference, BMI, and total body water were all highly correlated with weight (r = 0.89, r = 0.91, and r = 0.98, respectively). Also of note, waist circumference and BMI were very highly correlated with each other (r = 0.91). Triceps skinfold, subscapular skinfold, BMI, and waist circumference were not correlated with height (r = 0.08, r = 0.05, r = -0.02, and r = 0.13, respectively). The rest of the measures were moderately correlated with each other (r = 0.41-0.84).

All statistics were generated using SUDAAN version 9.0 (Research Triangle Institute, Research Triangle Park, NC) and SAS version 9.1 (SAS Institute, Cary, NC). All data were weighted to account for the complex survey design and nonresponse to household interview and exam.

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Unadjusted, median PSA values by race/ethnicity and anthropometric measure are presented in Table 2. Among white men, distributions of PSA differed by quintile of total body water, weight, and BMI category. Associations between BMI and triceps skinfold thickness and PSA among Mexican American men were suggested by low P values, but they did not attain statistical significance (P = 0.084 and P = 0.056), perhaps due to smaller sample sizes.

Predicted marginal PSA adjusted for age and stratified by race/ethnicity showed a decreasing trend by quintile for waist circumference, weight, triceps skinfold thickness, and calculated total body water for white men (Table 3). PSA decreased as the BMI category increased for both white and Mexican American men. Among black men, a trend for decreasing PSA with increasing triceps skinfold thickness was statistically significant, although, when presented by quintile, there was no clear pattern to the association. A trend for decreasing PSA with increasing waist circumference was suggested among Mexican American men, but again this was not statistically significant, perhaps due to a smaller sample size.

We also calculated the predicted margin percentage of men with a PSA ≥4.0 ng/mL by obesity classification. After controlling for age and race/ethnicity, the predicted margin percentage of nonobese men (BMI <30 kg/m²) with a PSA ≥4.0 was 6.6%, and that for obese men was 3.8% (P = 0.001; data not shown).

In a multivariate linear regression analysis controlling for age, race/ethnicity, and the age-by-race/ethnicity interaction, BMI had a decreasing linear relationship with PSA (Table 4). We found moderate declines in PSA with a 1 SD increase in BMI [5.9% decrease (95% confidence interval, −9.0% to −2.8%) in geometric mean PSA per 5.2-unit increase], weight [5.9% decline (−8.8% to −2.8%) per 17.7-kg increase], waist circumference [6.6% decline (−9.4% to −3.6%) per 13.4-cm increase], triceps skinfold [5.4% decline (−8.9% to −1.8%) per 6.4-mm increase], and calculated total body water [5.7% decline (−8.9% to −2.4%) per 6.5-liter increase]. None of the interactions between race/ethnicity and an anthropometric measure were statistically significant. The interaction term between race/ethnicity and BMI was most suggestive of a significant effect (P = 0.07).
Using nationally representative data, we have shown a
likely to cause delayed detection. We believe that other factors
test (39). Therefore, we do not believe that this difference is
lower PSA level for a man with a BMI of 40 kg/m² relative to a
BMI and PSA is not a local phenomenon but is present in the
appropriate rate of diagnosis, rather than underdiagnosis, for
PSA. Data from the Prostate Cancer Prevention Trial may be
high-grade disease (15), the type of prostate cancer for which
prevalence of benign prostatic hyperplasia and prostatic
access to screening. The diagnosis and treatment of prostate cancer in the PSA era
complicated by a number of factors, including access to
surgery success rate. Obese men may be slightly more likely
to be screened with PSA (40) and this may be due to a higher
of benign prostatic hyperplasia and prostatic symptoms (41). However, obese men often present with
and PSA have used convenience samples either from a referral
biopsies on its participants and the likely presence of
population or from surrounding communities. We feel that our
and PSA modeled continuously, U.S. men 2001-2004
BMI and PSA

<table>
<thead>
<tr>
<th>BMI (kg/m²)</th>
<th>% Change* in</th>
<th>SD of</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>PSA geometric mean (95% confidence interval for % change)</td>
<td>anthropometric measure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-5.9 (-9.0 to -2.8)</td>
<td>5.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>-5.9 (-8.8 to -2.8)</td>
<td>17.7</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>-6.6 (-9.4 to -3.6)</td>
<td>13.4</td>
</tr>
<tr>
<td>Triceps skinfold (mm)</td>
<td>-5.4 (-8.9 to -1.8)</td>
<td>6.4</td>
</tr>
<tr>
<td>Total body water (liter)</td>
<td>-5.7 (-8.9 to -2.4)</td>
<td>6.5</td>
</tr>
</tbody>
</table>

NOTE: Controlling for a race by age interaction.
*Percent change associated with an increase of 1 SD in the anthropometric characteristic of interest.

Unlike other investigators, we did not detect a significant interaction between age and BMI (31). However, when we assumed age had a linear relationship with PSA, we did detect a significant interaction between age and the dichotomous BMI variable used by Barqawi et al. (obese versus nonobese). When we allowed for a nonlinear relationship between age and PSA, this interaction was no longer significant.

Discussion

Using nationally representative data, we have shown a negative association between several anthropometric measures and PSA. Weight, BMI, waist circumference, triceps skinfold thickness, and calculated total body water all exhibited negative trends with PSA levels. When stratified by race/ethnicity, all of these trends were seen among white men; a trend for BMI was found in Mexican American men; and a trend for triceps skinfold thickness was found in black men.

Our overall negative association between BMI and PSA is consistent with those found in several recent studies (28-32). Baillargeon et al. found similar geometric mean PSA values using the WHO obesity classifications in a population-based San Antonio sample containing a much higher proportion (37%) of Hispanic men than our weighted NHANES sample (5%). Our findings are very similar to the statistically significant moderate decline in PSA values with increasing BMI reported by Kristal et al. (30). The nationally representative data reported here expand the validity of these previous results to a national scale. We were able to verify the categorical effect modification reported by previous authors (31).

The PSA test is a commonly used test (22) in older men and any factors that influence its accuracy may have important consequences for the diagnosis of prostate cancer. Some authors have suggested that lower PSA values in obese men may decrease the test sensitivity, leading to a later diagnosis with a less favorable prognosis in this population (31, 32).

Supporting this assertion are studies showing that obese men with prostate cancer present with later stages, have a poorer prognosis, and have a higher biochemical failure rate than healthy-weight men (14-16, 37, 38). When comparing men at extremes of the BMI scale, our model predicts only a 16.1% lower PSA level for a man with a BMI of 40 kg/m² relative to a man with a BMI of 25 kg/m² (predicted margin geometric means, 0.98 and 0.83 ng/mL, respectively). The predicted magnitude of the BMI effect is well within the range of the normal analytic and biological day-to-day variation of the PSA test (39). Therefore, we do not believe that this difference is likely to cause delayed detection. We believe that other factors are at least partially responsible for the poorer outcomes observed for obese men.

The diagnosis and treatment of prostate cancer in the PSA era is complicated by a number of factors, including access to screening, the presence of comorbidities, prostate size, and surgery success rate. Obese men may be slightly more likely to be screened with PSA (40) and this may be due to a higher prevalence of benign prostatic hyperplasia and prostatic symptoms (41). However, obese men often present with comorbidities, and such men are less likely to be screened (42) in accordance with many PSA screening guidelines. Prostate volume is another potentially important confounder of disease detection. Obese men have larger prostates than nonobese men, which may decrease the sensitivity of prostate biopsy, perhaps leading to delayed detection of disease (32, 43). An additional factor to consider is the increased difficulty of surgical intervention in obese men. Obese men undergoing radical prostatectomy are more likely to experience incomplete removal of the prostate because of capsular incision (44), and this may be adversely associated with posttreatment survival (45). It seems plausible that a combination of these factors explains the association between obesity and poor prostate cancer outcomes, but currently the relative importance of each factor is unclear.

Still, we believe that the potential for a harmful delay in prostate cancer detection due to obese men’s lower PSA values should be more fully investigated. If such a bias exists, obese men face a lower likelihood of cancer detection by PSA and biopsy. Furthermore, obese men may have an increased risk of high-grade disease (15), the type of prostate cancer for which early detection and treatment may be most important (45). However, these effects must be considered within the larger debate of PSA screening. If randomized screening trials find that PSA screening overdiagnoses prostate cancer in normal-weight men, lower PSA levels in obese men may support a proper rate of diagnosis, rather than underdiagnosis, for this portion of the population. We suggest that prospective data that include biopsy results for men regardless of PSA level could comment on the amount of missed cancer due to low PSA. Data from the Prostate Cancer Prevention Trial may be appropriate for such a study.

Whereas our analysis of PSA as a continuous variable did not suggest a clinically relevant association between PSA and BMI, our categorical analysis showed a reasonably large relative difference in the percentage of men with a PSA >4.0 ng/mL in the obese and nonobese groups (6.6% compared with 3.8%). Whereas some may believe these results to be contradictory, we do not feel that this is necessarily the case. BMI had a similar effect statistically in the continuous and categorical analyses. However, we do not feel that the size of this effect is clinically relevant for the continuous analysis. For the categorical analysis, <6% of the men in our study population had a PSA greater than the commonly used clinical cutpoint of 4.0 ng/mL. As a result, the modest absolute percent difference at this cutpoint translates to a somewhat large relative percent difference. Previous investigations into BMI and PSA have used convenience samples either from a referral population or from surrounding communities. We feel that our use of nationally representative data is an important strength of this study. These results verify that the association between BMI and PSA is not a local phenomenon but is present in the overall U.S. adult male population.

Baillargeon et al. and Ochiai et al. biopsied men with a PSA >4.0 ng/mL and removed those with positive results from their analysis, lessening the likelihood of confounding by cancer status (32). However, there may have been a significant number of tumors in men with a PSA <4.0 ng/mL, suggesting that the presence of undiagnosed tumors may still have confounded the results (26). NHANES does not conduct biopsies on its participants and the likely presence of undiagnosed tumors in our population prevents the authors from concluding that the results described here are independent of prostate cancer status.
We did not find a statistically significant interaction term between race/ethnicity and any of the anthropometric measures, but most of the significant trends shown in Table 4 are among white men. NHANES does not collect data that would allow us to comment on other possible causes for the discrepancy in significant trends by race. For example, previous studies have hypothesized that differences in sex hormone levels may explain racial variation in PSA values (47). In addition, the ability of various anthropometric measures to estimate obesity may differ across racial groups because of assumptions about stature or body density (48).

Our intention was to measure the association between adiposity and PSA; however, attempts to estimate adiposity from anthropometric measures, BMI, and waist circumference, and skinfolds are inherently subject to both measurement error and misclassification error. We believe that the influence of measurement error on our results is minimal because of the rigorous NHANES quality control mechanisms. Regardless, our study may be subject to misclassification error. BMI correlates well with weight and poorly with height and, hence, is a measure of excess weight relative to height, but it is unable to discriminate between fat body mass and fat-free body mass. Higher BMI does not necessarily indicate more adiposity, weakening our ability to see the true association between adiposity and PSA. The accuracy of BMI as an indicator of body fatness is also influenced by age, relative leg length, body density, body build, lean body mass, and perhaps race/ethnicity (49). Indeed, the actual relationship between PSA and adiposity may be more pronounced than has been described here or elsewhere. Similar misclassification error is also possible in the other measures. Triceps skinfold thickness can predict body fat percentage in adults, albeit imperfectly, with a SE of 3% to 5% (50).

Waist circumference is a better predictor than BMI of the specific portion of total body fat known as visceral body fat (51). The accumulation of fat in this region of the body is believed to be an important part of the link between obesity and sex hormone alterations (52). Therefore, if the association between body size and PSA was driven by sex hormone alterations, we would have expected waist circumference to be a stronger predictor of PSA than BMI. In our analysis, they were essentially equal predictors, and collinearity prevented us from modeling them simultaneously. A hypothesis to explain these results is that a less specific mechanism may be behind the association, perhaps serum dilution from an increase in total body size.

The consistency across recent studies suggests that the association of low PSA with BMI is not due to chance, although it underlies obese men’s poorer prostate cancer outcomes is unclear. Cross-sectional studies like the current one and those before it cannot directly implicate the association between PSA and obesity as a factor in these outcomes. A number of other factors, including access to screening, comorbidities, prostate size, and treatment effectiveness, are likely to play a significant role in prostate cancer detection and mortality (18). We agree with other researchers (30) that the magnitude of decrease in PSA associated with obesity is not likely to affect the interpretation of a PSA test result, but this assertion can only be proved prospectively.

Given the potential for increased mortality if prostate cancer is diagnosed later in obese men, we believe the effects of the described association on prostate cancer detection should be evaluated fully in a prospective study.

References
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