Abstract: Saw palmetto is an herb used to treat the symptoms of benign prostatic hyperplasia. In vitro studies have found that saw palmetto inhibits growth of prostatic cancer cells and may induce apoptosis. To evaluate whether saw palmetto supplements are associated with a reduced risk of prostate cancer, we conducted a prospective cohort study of 35,171 men aged 50–76 yr in western Washington state. Subjects completed questionnaires between 2000 and 2002 on frequency of use of saw palmetto supplements and saw palmetto–containing multivitamins over the previous 10 yr in addition to other information on supplement intake, medical history, and demographics. Men were followed through December 2003 (mean of 2.3 yr of follow-up) via the western Washington Surveillance, Epidemiology, and End Results cancer registry, during which time 580 developed prostate cancer. Ten percent of the cohort used saw palmetto at least once per week for a year in the 10 yr before baseline. No association was found between this level of use of saw palmetto and risk of prostate cancer development [hazard ratio (HR) = 0.95; 95% confidence interval = 0.74–1.23] or with increasing frequency or duration of use. In this free-living population, use of commercial saw palmetto, which varies widely in dose and constituent ratios, was not associated with prostate cancer risk.

Introduction

Prostate cancer is the most common non-skin cancer and has the second highest cancer death rate in men (1). Age, family history of prostate cancer, and race are the only well-established risk factors for the disease. Identifying modifiable risk factors could help decrease the incidence and mortality rates of prostate cancer.

Saw palmetto’s use in the treatment of benign prostatic hyperplasia (BPH) (2,3) and its purported mechanism of action have prompted researchers to consider its possible efficacy in decreasing the risk of prostate cancer. Saw palmetto supplements are derived from the berries of the plant Serenoa repens, which is native to the South Atlantic coast of the United States and other areas. About 2% of American men use saw palmetto supplements (4). Popular saw palmetto supplements are available as encapsulated, powdered berries in doses of 900 mg or above or as a lipid and sterol extract in doses of about 160 mg (5).

Although BPH is not considered a precursor for prostate cancer pathologically, 83% of prostate cancers occur in men with BPH (6), and both conditions respond to anti-androgen treatment and share common risk factors (7). Saw palmetto is thought to treat BPH by inhibiting the 5-α-reductase enzyme (8–17), which irreversibly converts testosterone to the more potent androgen dihydrotestosterone (DHT), although conflicting evidence exists (18–20). The Prostate Cancer Prevention Trial found that treatment with finasteride, a strong 5-α-reductase inhibitor also used to treat BPH, decreased the risk of developing prostate cancer (21). In vitro studies have described other mechanisms of action for saw palmetto, such as inhibiting DHT from binding to androgen receptors (22,23), diminishing estrogen (24), and decreasing inflammation (25–27). These associations have prompted scientists to investigate whether saw palmetto is active against prostate tumors in vitro studies. Saw palmetto lipidosterolic extract appears to inhibit the growth of LNCaP (27–31), PC-3 (29,31), 267B-1 (27), and BRFF-41T (27) cancerous human prostatic cell lines in vitro when used in physiologically plausible doses (32). This extract has been found to inhibit cancerous prostate cell growth through several different mechanisms, including apoptosis (27,30), necrosis (30), and growth inhibition (27–29). Saw palmetto also appears to target prostate tissue. Active compounds in saw palmetto extract, when taken orally, accumulate in the prostate gland preferentially over other organs in rats (33), although no studies have examined saw palmettos activity against prostate cancer in rodent models.

The only human studies involving saw palmetto and prostate cancer have been on the use of saw palmetto for treatment of prostate cancer, including clinical studies of the saw palmetto–containing formula PC-SPES (34–36), and two case reports of aggressive prostate cancer treated with a combination of saw palmetto and other supplements in the absence of conventional treatment (37,38). PC-SPES includes eight herbs and was withdrawn from the market in 2002 due to contamination with synthetic estrogens (39), thus limiting our ability to speculate on the role saw palmetto may play rel-
ative to the other compounds in the formula. The subjects treated with saw palmetto in the case reports improved markedly, but the use of additional compounds, including lycopene, and the positive bias in the publication of case reports limit the conclusions we can draw from them about saw palmetto and prostate cancer.

Given the fairly common use of saw palmetto and the evidence that it has activity in the prostate, it is important to understand whether saw palmetto supplement use decreases, increases, or has no effect on the risk of developing prostate cancer. We examined this association in a large cohort of older U.S. men that specifically recruited supplement users. To our knowledge, this is the first epidemiological study of saw palmetto use and prostate cancer risk.

Methods

Study Population and Design

Subjects included men who enrolled in the Vitamins and Lifestyle (VITAL) study, a longitudinal cohort study of 50- to 76-yr-old individuals living in the 13 western counties of Washington state. Details of recruitment, data collection, and follow-up procedures can be found in Ref. 40. Briefly, recruitment began in October 2000 and was completed in December 2002. Questionnaires (195,465) with a cover letter targeting supplement users were sent to men whose names were obtained from a commercial mailing list; 37,382 men (19.1%) returned questionnaires that passed eligibility and quality-control checks. Subjects with a self-reported history (n = 2,013), who developed noninvasive prostate cancer (n = 2), or with incomplete answers regarding history of prostate cancer (n = 128) or saw palmetto use (n = 68) were excluded, leaving 35,171 men for analysis.

Exposure Assessment

Subjects provided detailed information about demographic characteristics, supplement use, medications, medical history, and diet in a 24-page mark-sense questionnaire.

Assessment of saw palmetto from supplements and multivitamins: Respondents were asked about regular intake (defined as at least once a week for a year) of saw palmetto both as part of a multivitamin (defined as containing 10 or more vitamins and/or minerals) and as a single supplement or a part of other mixtures (referred to from here on as “single supplements” for simplicity). Respondents were asked to report the brand name of their multivitamin and whether it contained saw palmetto. We only asked about herbs in multivitamins taken currently because multivitamins contain too many compounds for subjects to reliably identify previous herbal constituents. For the single-supplement and multivitamin forms of saw palmetto, respondents were asked to report the number of times per week (1–3, 4–6, or 7) and the number of years in the past 10 (1–2, 3–5, or 6+) that they had taken it and if they were currently taking it. We did not ask about dosage per day because of the variation in constituent ratios and the disparity between the stated and actual dose of many saw palmetto supplements (41,42). We computed the average use of saw palmetto over the 10 yr prior to baseline as

$$\frac{\text{days per week} \times \text{years}}{7}$$

summed over saw palmetto single supplements and saw palmetto in multivitamins. We weighted saw palmetto-containing multivitamins as one-tenth of the value of saw palmetto single supplements to account for the average difference in dose between multivitamins and saw palmetto single supplements that we found on the market. Respondents were asked to look at their supplement bottles when completing the form. The multivitamin portion of the VITAL study questionnaire was found to be valid and reliable (43).

Assessment of covariates: Information about possible confounders and effect modifiers was collected via the questionnaire. These factors included whether no, one, or two or more first-degree relatives [father and/or brother(s)] had had prostate cancer; use of testosterone or medications for BPH (finasteride, doxazosin, and tamsulosin) in the previous 2 wk; and medical tests and conditions, including a prostate specific antigen (PSA) test in the previous 2 yr, prostate biopsy, or transurethral resection of the prostate, and physician-diagnosed BPH. Additional potential confounders, including age, ethnicity, marital status, income, education, anthropometrics, physical activity, and usual diet and supplement intake, were also recorded in the questionnaire.

Follow-up for Cancer and Censoring

We identified men diagnosed with prostate cancer through December 2003 through linkage to the western Washington Surveillance, Epidemiology, and End Results (SEER) cancer registry, which records new diagnoses of cancer, including their stage and grade, in the 13 counties of the Puget Sound. Information is obtained from all hospitals in the area and from pathologists, oncologists, radiotherapists, and state death certificates. During the approximately 2-yr follow-up period (75,755 person-years), 580 men developed invasive prostate cancer.

Cancer grade was classified as Gleason score of 2–6 (low/moderate grade) vs. Gleason score of 7–10 (high grade). This classification scheme was used by SEER beginning in January 2003, and data on Gleason scores from cancers in earlier years were reabstracted and recoded into these categories.

We obtained information on deaths by linkage to Washington state death records and, for moves out of the SEER registry catchment area, by linkage to the U.S. National Change of Address System. Men were censored at the earli-
likely to take saw palmetto supplements.

a family history of prostate cancer were only slightly more
lenium, vitamin E, and lycopene (data not shown). Men with
baseline as well as other dietary supplements, especially se-
more likely to have taken drugs for BPH in the 2 wk before
prostate biopsy. Furthermore, men taking saw palmetto were
men in the cohort (\(n = 3,625\)) used saw palmetto at least once
post-baseline PSA testing. Finally, a limitation of this study
misclassification.

results of date of prostate cancer diagnosis (1.7%), date with-
drawn from the study (0.02%), date moved out of the SEER
coverage area (3.0%), date died (2.0%), or date of last cohort
follow-up (December 31, 2003; 93.3%).

Statistical Analyses

We used Cox proportional hazard models to calculate the
hazard ratios and 95% confidence intervals (CIs) of prostate
cancer by categories of saw palmetto use. Age was used as
the time variable, effectively controlling for age in all analy-
ses. We evaluated as potential confounders PSA testing, fam-
ily history of prostate cancer, BPH, prostate biopsy, dietary
and supplemental lycopene and selenium intake, testosterone
and medications for BPH, education level, race/ethnicity, and
servings of fruit and vegetables. We controlled for history of
BPH (yes or no) and selenium supplement intake (quartiles)
because only inclusion of these factors changed the hazard
ratio coefficient by more than 10%. We chose to control for
family history of prostate cancer (none, one first-degree rela-
tive, and two or more first-degree relatives) a priori because
of its well-established relationship with the risk of prostate
cancer. To explore whether the association between saw pal-
metto and prostate cancer differed by another variable (for
example, BPH, age, or PSA testing), we conducted analyses
stratified on each variable and also evaluated the coefficients
for the cross-products term (saw palmetto \(\times\) effect modifier)
using the likelihood ratio test. The association between PSA
testing and prostate cancer differed by age; therefore, we
controlled for PSA testing as a stratification variable in the
Cox model. To test for a trend across categories, we modeled
a single linear variable. Additional models looked at the
associations of saw palmetto use by disease subgroups (Gleason
score of 2–6 vs. Gleason score of 7–10).

We examined Schoenfeld residuals to evaluate the propor-
tional hazards assumption. In the models presented, there
was no evidence that this assumption was violated. The
two-tailed \(P\) value of 0.05 was considered statistically signif-
ica. All statistical analyses were conducted using STATA
version 8.2 (College Station, TX).

Results

Table 1 gives distributions of the cohort overall and of the
men who used saw palmetto by demographic characteristics,
family history, and medical history. Overall, 10.3% of the
men in the cohort (96) used saw palmetto at least once
a week for a year was associated with a 16% increased risk
(HR = 1.03; 95% CI = 0.80–1.30) and quartiles of sele-
nium use (HR = 0.95; 95% CI = 0.74–1.23), the hazard ratios
were attenuated. There was no indication of a lower relative
risk with increasing years of use, days per week of use, aver-
age 10-yr use categorized as above and below the median
(Table 2), or average 10-yr use categorized into quartiles
(data not shown).

There was no clear difference in the association of saw pal-
metto with prostate cancer by grade of cancer (Table 3). Two other subgroups, PSA testing within the previous 2 yr
and BPH at baseline, were analyzed similarly. Neither showed significant effect modification (data not shown).

Discussion

In this large cohort of older men, we found no evidence of
an association between use of saw palmetto at any level and
risk of prostate cancer. Neither increasing duration nor fre-
quency of saw palmetto use was related to the risk of prostate
cancer.

We evaluated subgroups by PSA testing and BPH at base-
line because PSA testing is associated with diagnosis of both
prostate cancer and BPH (44), and BPH is often the indica-
tion for use of saw palmetto. In each case, we found the asso-
ciation to be similar among both groups. In addition, saw pal-
metto does not appear to be more strongly related to high-grade (Gleason score of 7–10) or low/moderate-grade
(Gleason score of 2–6) prostate cancer.

There are limitations to the conclusions we can draw from
these analyses. The dose and purity of commercial saw pal-
metto supplements vary widely (41,42) and were not re-
corded. Therefore, the results of this study address the effec-
tiveness of saw palmetto supplementation as the public uses it. This lack of information on the dose and purity of the herb
may have obscured the relationship and increased the chance
of incorrectly accepting the null hypothesis. Another limita-
tion is that we may have had too few cases to detect a small
increase or decrease in risk, especially in the analyses by sub-
groups of the population. Residual confounding by factors
such as PSA screening and other prostate cancer screening is
another concern. Men with symptoms of BPH are more
likely to both take saw palmetto and have their PSA levels
tested, and PSA testing is strongly associated with prostate
cancer diagnosis (45). We only knew if subjects had a PSA
test within the 2 yr pre-baseline, and we used it as a proxy for
post-baseline PSA testing. Finally, a limitation of this study
is that the supplementation and medical history data were
self-reported, which could have resulted in nondifferential
misclassification.
This study has several strengths. The cohort design minimizes recall bias. The study population was recruited in such a way as to encourage supplement users to join. Thus, over 10% of men in our cohort, compared with an estimated 2% of men in the United States (4), had used saw palmetto. Subjects reported up to 10 yr of saw palmetto use in the questionnaire, which should be a sufficient amount of time to observe a treatment effect. Furthermore, the large majority of the non-user comparison group were users of other supplements; thus, the unmeasured characteristics of supplement-taking behavior were somewhat controlled. The VITAL questionnaire also provided detailed information on use of other supplements and other potential confounders and effect modifiers. Data collected on the grade of the prostate cancer cases allowed us to evaluate whether saw palmetto was associated with the severity of prostate cancer.

Table 1. Demographic and Health-Related Characteristics in the VITAL Cohort and Among Saw Palmetto Usersa

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Cohort (n = 35,171)</th>
<th>Users of Single Saw Palmetto Supplement in the Past 10 yr (n = 3,625)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age at baseline (yr)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50–54</td>
<td>8,386</td>
<td>23.8</td>
</tr>
<tr>
<td>55–59</td>
<td>8,065</td>
<td>22.9</td>
</tr>
<tr>
<td>60–64</td>
<td>6,616</td>
<td>18.8</td>
</tr>
<tr>
<td>65–69</td>
<td>5,881</td>
<td>16.7</td>
</tr>
<tr>
<td>70–76</td>
<td>6,223</td>
<td>17.7</td>
</tr>
<tr>
<td><strong>Race/ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>32,354</td>
<td>93.2</td>
</tr>
<tr>
<td>Hispanic</td>
<td>294</td>
<td>0.8</td>
</tr>
<tr>
<td>Black</td>
<td>437</td>
<td>1.3</td>
</tr>
<tr>
<td>American Indian/Alaska native</td>
<td>520</td>
<td>1.5</td>
</tr>
<tr>
<td>Asian or Pacific Islander</td>
<td>854</td>
<td>2.5</td>
</tr>
<tr>
<td>Other</td>
<td>265</td>
<td>0.8</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade school/some high school</td>
<td>1,110</td>
<td>3.2</td>
</tr>
<tr>
<td>High school graduate or equivalent</td>
<td>4,420</td>
<td>12.7</td>
</tr>
<tr>
<td>Some college/technical school</td>
<td>12,179</td>
<td>35.0</td>
</tr>
<tr>
<td>College graduate</td>
<td>9,516</td>
<td>27.4</td>
</tr>
<tr>
<td>Advanced degree</td>
<td>7,550</td>
<td>21.7</td>
</tr>
<tr>
<td><strong>Number of first-degree relatives with prostate cancer</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>30,148</td>
<td>87.0</td>
</tr>
<tr>
<td>1</td>
<td>4,239</td>
<td>12.2</td>
</tr>
<tr>
<td>2 or more</td>
<td>287</td>
<td>0.8</td>
</tr>
<tr>
<td><strong>PSA test in the last 2 yr</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>24,999</td>
<td>72.0</td>
</tr>
<tr>
<td>No</td>
<td>9,738</td>
<td>28.0</td>
</tr>
<tr>
<td><strong>Benign prostatic hyperplasia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>5,589</td>
<td>15.9</td>
</tr>
<tr>
<td>No</td>
<td>29,573</td>
<td>84.1</td>
</tr>
<tr>
<td><strong>Used finasteride in the last 2 wk</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>201</td>
<td>0.6</td>
</tr>
<tr>
<td>No</td>
<td>34,963</td>
<td>99.4</td>
</tr>
<tr>
<td><strong>Used terazosin, doxazosin, or tamsulosin in the last 2 wk</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1,259</td>
<td>3.6</td>
</tr>
<tr>
<td>No</td>
<td>33,912</td>
<td>96.4</td>
</tr>
<tr>
<td><strong>Transurethral resection of the prostate</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1,161</td>
<td>3.3</td>
</tr>
<tr>
<td>No</td>
<td>34,010</td>
<td>96.7</td>
</tr>
<tr>
<td><strong>Prostate biopsy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2,973</td>
<td>8.5</td>
</tr>
<tr>
<td>No</td>
<td>32,198</td>
<td>91.6</td>
</tr>
<tr>
<td><strong>Selenium supplementation, 10-yr average intake (mcg/day)b</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>14,047</td>
<td>40.1</td>
</tr>
<tr>
<td>0.2–8.9</td>
<td>5,066</td>
<td>14.5</td>
</tr>
<tr>
<td>9–19.9</td>
<td>5,130</td>
<td>14.6</td>
</tr>
<tr>
<td>20–29.9</td>
<td>5,480</td>
<td>15.6</td>
</tr>
<tr>
<td>30–400</td>
<td>5,304</td>
<td>15.1</td>
</tr>
</tbody>
</table>

a: Abbreviations are as follows: VITAL, Vitamins and Lifestyle; PSA, prostate specific antigen.
b: Includes selenium from multivitamins and individual supplements.
No other epidemiological study of saw palmetto use and risk of prostate cancer has been published to our knowledge. However, several lines of evidence suggest that saw palmetto may be protective against prostate cancer. In vitro studies found that saw palmetto inhibits the growth (27–29) of prostatic cancer cells and induces apoptosis (27,28,30). The drug finasteride, which is thought to decrease levels of DHT by the same mechanism as saw palmetto, appears to decrease the risk of developing prostate cancer (21). Nevertheless, we did not find evidence that saw palmetto decreases the risk of developing prostate cancer.

Daily use of commercial saw palmetto supplements for several years was not associated with the risk of developing prostate cancer in this free-living cohort of 35,171 men ages 50–76 living in western Washington. No association between frequency or duration of use and the severity of cancer was detected. Thus, despite a biologically plausible hypothesis that saw palmetto may reduce prostate cancer risk, this study was not able to detect such an association.

Table 2. Association of Saw Palmetto Supplementation With Risk of Prostate Cancer

<table>
<thead>
<tr>
<th>Cases</th>
<th>Cohort</th>
<th>Incidence Rate per 10,000 Person-Years</th>
<th>Adjusted HR&lt;sup&gt;b&lt;/sup&gt;</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Single Saw Palmetto Supplement</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ever used</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>502</td>
<td>86.6</td>
<td>31,546</td>
<td>89.7</td>
</tr>
<tr>
<td>Yes</td>
<td>78</td>
<td>13.4</td>
<td>3,625</td>
<td>10.3</td>
</tr>
<tr>
<td>Years of use in past 10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>502</td>
<td>86.6</td>
<td>31,546</td>
<td>89.7</td>
</tr>
<tr>
<td>1–2</td>
<td>37</td>
<td>6.4</td>
<td>1,709</td>
<td>4.9</td>
</tr>
<tr>
<td>3–5</td>
<td>22</td>
<td>3.8</td>
<td>1,261</td>
<td>3.6</td>
</tr>
<tr>
<td>≥6</td>
<td>19</td>
<td>3.3</td>
<td>655</td>
<td>1.9</td>
</tr>
<tr>
<td>P for trend</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency of use (days/week) in the years used</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>502</td>
<td>86.9</td>
<td>31,546</td>
<td>90.0</td>
</tr>
<tr>
<td>&lt;7</td>
<td>11</td>
<td>1.9</td>
<td>915</td>
<td>2.6</td>
</tr>
<tr>
<td>7</td>
<td>65</td>
<td>11.2</td>
<td>2,589</td>
<td>7.4</td>
</tr>
<tr>
<td>P for trend</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined Single Supplement and Saw Palmetto–Containing Multivitamin</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average 10-yr use</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>499</td>
<td>86.0</td>
<td>31,294</td>
<td>89.0</td>
</tr>
<tr>
<td>Below median&lt;sup&gt;c&lt;/sup&gt;</td>
<td>39</td>
<td>6.7</td>
<td>1,977</td>
<td>5.6</td>
</tr>
<tr>
<td>Equal to/above median&lt;sup&gt;c&lt;/sup&gt;</td>
<td>42</td>
<td>7.2</td>
<td>1,900</td>
<td>5.4</td>
</tr>
<tr>
<td>P for trend</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>: Abbreviations are as follows: HR, hazard ratio; CI, confidence interval; —, no data.

<sup>b</sup>: Adjusted for age, history of benign prostatic hyperplasia, quartiles of selenium intake, and number of first-degree relatives with prostate cancer and stratified by history of prostate specific antigen testing (yes/no) in the 2 yr before baseline.

<sup>c</sup>: Median equivalent to daily use for 1–2 yr.

Table 3. Association of Saw Palmetto Supplementation With Grade of Prostate Cancer

<table>
<thead>
<tr>
<th>Grade</th>
<th>Low/Moderate Grade (Gleason score of 2–6)</th>
<th>High Grade (Gleason score of 7–10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>Person-Years</td>
<td>Adjusted HR&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Average 10-yr use</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>341</td>
<td>67,120</td>
</tr>
<tr>
<td>Below median&lt;sup&gt;c&lt;/sup&gt;</td>
<td>26</td>
<td>4,286</td>
</tr>
<tr>
<td>Equal to/above median&lt;sup&gt;c&lt;/sup&gt;</td>
<td>31</td>
<td>4,009</td>
</tr>
</tbody>
</table>

<sup>a</sup>: Abbreviations are as follows: HR, hazard ratio; CI, confidence interval; —, no data.

<sup>b</sup>: Adjusted for age, history of benign prostatic hyperplasia, quartiles of selenium intake, and number of first-degree relatives with prostate cancer and stratified by prostate specific antigen screening.

<sup>c</sup>: Median equivalent to saw palmetto taken 7 days/wk for 1–2 yr.

Acknowledgments and Notes

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