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# Stress, Coping, Social Support, and Prostate Cancer Risk Among Older African American and Caucasian Men

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# STRESS, COPING, SOCIAL SUPPORT, AND PROSTATE CANCER RISK AMONG OLDER AFRICAN AMERICAN AND CAUCASIAN MEN

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**Objectives:** While psychosocial stress and high effort coping have been associated with reduced immune function, no epidemiologic study has addressed psychological stress and risk of prostate cancer. The purpose of this analysis was to investigate the association between stress, coping, social support, and risk of prostate cancer among older men (age 65–79 years).

**Design:** Population-based case-control study in South Carolina.

**Participants:** Cases were 400 incident, histologically confirmed prostate cancer cases identified through the South Carolina Central Cancer Registry between 1999 and 2001 (70.6% response rate). Controls were 385 men identified through the 1999 Health Care Financing Administration Medicare beneficiary file for South Carolina (63.8% response rate).

**Main Outcome Measures:** Consenting participants completed telephone interviews addressing demographics (age, race, income, education, marital status, body mass index), medical and prostate cancer screening history, stress (Global Perceived Stress), coping (John Henryism Scale), and social support.

**Results:** After adjusting for age, race, and South Carolina region, higher John Henryism scores (>24) were modestly associated with prostate cancer risk relative to lower scores (<24) (adjusted odds ratio 1.63, 95% confidence interval 1.11–2.40). This effect is somewhat more pronounced among those perceiving some stress, yet the effect of John Henryism on prostate cancer risk was reduced among those with high levels of social support. Neither higher stress nor social support alone was associated with prostate cancer risk.

**Conclusions:** Higher John Henryism scores indicating high-effort coping may be associated with an increase in prostate cancer risk. (*Ethn Dis*. 2006;16:978–987)

**Key Words:** Coping, Epidemiology, Prostate Neoplasms, Psychological Stress, Race

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## INTRODUCTION

Most studies addressing the effect of chronic stress on health find that chronic stress is associated with an increased risk of infectious diseases[1–7] including HIV,[8,9] cardiovascular disease,[10–14] and cancer.[15–17] Hilakivi-Clark and Dickson[16] found that male transgenic mice overexpressing transforming growth factor-alpha (TGF-alpha) who lived in stressful environments with aggressive non-siblings developed hepatocellular tumors earlier and had greater tumor burden than did mice housed in less stressful environments. Ben-Eliyahu et al[17] found that stress-induced suppression of natural killer cell activity (NKA) was sufficient to cause enhanced tumor development. Byrnes et al[18] proposed a causal model for the association between stress, depression, and cancer. Stress and depression are associated with a deregulation of inflammatory cytokines; stress is associated with increased expression of interleukin (IL)-1β and down-regulation of IL-2, interferon (IFN)-gamma (Interferon), NKA, and major histocompatibility complex (MHC) class II molecules.[19] Stress and depression can foster tumor progression by inhibiting expression of MHC class I and II molecules and through NKA reduction. Although several recent studies have identified the negative effect of chronic stress on health,[1,20] we found no published epidemiologic studies that have addressed psychological stress and risk of prostate cancer development. Epidemiologic studies have investigated psychological stress predominately by using stressful life events measures and cancers of the cervix,[21,22] lung,[23] breast,[24–30] and colon.[31–34]

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Coping characteristics of the individual and social support from family and friends can modify the association between stress and disease. Among caregivers, Esterling et al[35] found evidence that social support may modulate the effect of chronic stress on immune function. Social support may be a key moderator of the effect of psychosocial stress on cancer development. In a meta-analysis, Suls and Fletcher[36] found that coping style (cognitive avoidance vs attentive-confrontive) was more favorably associated with acute stress; however, information-seeking was associated with better long-term adjustment to stress. James et al[37,38] developed the construct of John Henryism as a measure

of high-effort active coping, defined as an individual's self-perception that environmental and psychosocial demands can be met through hard work and determination. Prolonged high-effort coping with chronic psychosocial stressors may result in adverse health effects, particularly for those with limited social or economic resources[39] who, in the United States, may be disproportionately African American. James et al[37] found that higher John Henryism Scale (JHS) scores were associated with hypertension among low-income African Americans. In a recent review, Bennett et al[39] reported that 9 of 16 studies evaluating John Henryism and hypertension found an association; many of these positive studies reported interactions between John Henryism, lower socioeconomic status, and stress. Like hypertension, prostate cancer is a chronic disease that African Americans are significantly more likely than Whites to experience. The high-effort coping that contributes to the racial difference in blood pressure may be relevant to the racial disparity in prostate cancer incidence.

Ellison et al[40] proposed a conceptual model for the role of stress, coping, and social support on prostate cancer development; this model was adapted from the work of Adler and Matthews.[41] Ellison's model hypothesizes that psychological stress may lead to prostate cancer through physiologic responses to environmental stressors.[40] The physiologic response to environmental stress is a function of the individual's perception of the stress and his ability to cope with the stress. Those who perceive life stressors as threatening and lack effective coping strategies and resources to address these environmental stressors may be at greater risk of cancer because of their inability to mount an effective immunologic response to carcinogenesis.[40]

The purpose of this analysis was to investigate whether higher perceived stress, high-effort coping, and lower social support may interact to increase the risk of prostate cancer among African American and Caucasian men in a population-based case-control study.

# METHODS

## Cases and Controls

Details of this population-based case-control study have been reported elsewhere.[42] Briefly, patients aged 65–79 years who were diagnosed with primary, invasive, histologically confirmed prostate cancer between October 1999 and September 2001 were identified through the South Carolina Central Cancer Registry (SCCCR). During the study period, 551 Caucasian men and 245 African American men with localized disease (stages I and II) and 98 Caucasian men and 70 African American men with advanced disease (stages III and IV) who met the eligibility criteria were reported to the SCCCR. All eligible cases with advanced disease and a random sample of men with localized disease within five-year age groups (42% of Caucasian cases and 83% of African American cases) were selected. A total of 426 prostate cancer cases (70.6% of eligible cases) completed a standardized telephone interview. Of potentially eligible cases, 90 physicians refused (13.0%), 71 patients refused (10.3%), 24 died before the interview (3.5%), 59 were not located (8.5%), and 23 were too sick to participate (3.3%). After eliminating seven prevalent prostate cancer patients and 19 patients who did not provide complete interview data, 400 cases remained for analyses.

Control subjects were South Carolina residents aged 65–79 who were randomly sampled from the 1999 Health Care Financing Administration (HCFA) Medicare beneficiary file. Controls were frequency matched to cases on age (five-year age groups), race (Caucasian, African American), and geographic region (western, middle, and eastern third of the state). A total of 482 control subjects (63.8%) completed the interview. Of potentially eligible controls, 108 refused (14.3%), 22 died before the interview (2.9%), 112 were not located (14.8%), and 32 were too sick to participate (4.2%). After eliminating 52 controls with prevalent prostate cancer and 45 controls whose interviews were incomplete, 385 controls remained for analyses.

Cases and controls were recruited through mailings that described the study and informed the potential participant that an interviewer would contact them. Since the HCFA file does not contain telephone numbers, controls whose phone numbers could not be located through directory assistance, telephone directories, or reverse directories were sent an additional letter asking for a preferred contact number. Trained interviewers from the University of South Carolina Survey Research Laboratory conducted computer-assisted telephone interviews with subjects who provided verbal consent with the understanding that written consent would be obtained. Telephone interviews of 30–40 minutes in length collected information on demographic characteristics, socioeconomic status, alcohol and tobacco use, and medical history (including diabetes, stroke, myocardial infarction, cirrhosis or other liver disease, hypertension and hypercholesterolemia, and family history of cancer). Most exposures pertained to the period before a reference date: the date of diagnosis for cases and an assigned date for controls. For psychosocial factors, this time frame was the one-year period before the diagnosis or reference date. Institutional review boards of the University of South Carolina, the Centers for Disease Control and Prevention, and the National Cancer Institute approved this project's data collection procedures.

## Stress, Coping, and Social Support Measurement

We used seven items from the 10-item Global Perceived Stress (GPS)[43]

scale as a measure of self-perceived stress. Respondents were asked to think about how they usually felt before the reference date. Response options were as follows: never (1), almost never (2), sometimes (3), fairly often (4), and very often (5). When assessing the psychometric properties of the scale, we identified two factors within this scale. Factor I, which generally measures stress (hereafter, perceived stress), included the following three items: How often: 1) did you feel nervous and stressed (correlation within the factor=.76); 2) were you angered because of things that happened that were outside your control (correlation=.75); and 3) did you feel difficulties were piling up so high that you could not overcome them (correlation=.68). Factor II generally measured perceived ability to cope or control life stressors (hereafter, control stress) and included the following four items: How often: 1) did you feel that you were effectively coping with important changes that were occurring in your life (correlation=.50); 2) did you feel confident about your ability to handle your personal problems (correlation=.75); 3) were you able to control irritations in your life (correlation=.72); and 4) did you feel that you were on top of things (correlation=.72). The four items in the control stress subscale were reverse coded such that a higher score indicated less perceived control over stress. The higher the total GPS score, the greater the perceived stress and the lower the perceived control over stress. Cronbach alpha $\alpha$ for our 7-item scale was .50, which indicates limited internal consistency of the scale, yet the alphas for the perceived stress (.60) and control stress (.61) subscales were higher than the alpha for the GPS scale. We created cut-points based on the distribution in the controls to indicate three levels of the continuous scores. The highest category includes those answering most items as sometimes to fairly often (scores >20), the intermediate category includes those who answered questions in general as almost never to sometimes (scores 14–20), and the lowest category includes those answering the seven items as generally never to almost never (scores 7–13).

We used a shortened version of the 12-item JHS as a measure of high-effort coping.[37] This 12-item scale includes three main themes: efficacious mental and physical vigor, a strong commitment to hard work, and a single-minded determination to succeed. We included two of the four items for each theme to create our reduced six-item scale. Respondents were instructed to think about how they saw themselves as a person living and doing things in the real world before the referent date. The five response options for each statement ranged from strongly agree (5) to strongly disagree (1). Higher scores indicated higher effort coping. The following six items were used: "I always felt I could make my life pretty much what I wanted to make of it"; "Once I made up my mind to do something I stayed with it until the job was completely done"; "When things didn't go the way I wanted them to, that just made me work even harder"; "Sometimes I felt that if anything was going to be done right, I had to do it myself"; "I didn't let my personal feelings get in the way of doing a job"; and "Hard work really helped me to get ahead in life." The Cronbach's alpha for this six-item scale, ranging from 6–30, was .64, similar to the .67 reported by James et al.[37] Note that the JHS does not assess coping in response to stress but is a generalized approach to one's work life. Cut-points were created to reflect meaningful differences in scores. The highest group included those who consistently answered strongly agree on almost all items (scores 29–30), intermediates included those answering agree to strongly agree on most items (scores 25–28), and the lowest category included those answering strongly disagree to agree on some items (scores 6–24).

We used three items based on the measure developed by Sarason et al to assess social support.[44] Again, respondents were instructed to think about their social networks before the referent date. The following three items were used to measure social support: "There was someone: 1) who accepted me totally including both my worst and best points; 2) I could count on to care about me, regardless of what was happening to me; and 3) I could count on to help me feel better when I was feeling down in the dumps." Five response options ranged from strongly agree (5) to strongly disagree (1). Higher scores indicated greater perceived support; scores ranged from 3 to 15 with a Cronbach's alpha coefficient of .68. Again, because this scale was skewed toward the majority who reported high social support, we created cut-points to reflect meaningful comparisons. The highest cut-point included those who answered all items as strongly agree (scores=15), the intermediate included those answering agree to strongly agree (scores 13–14), and the lowest category included those answering strongly disagree to agree (scores 3–11).

## STATISTICS

We used unconditional logistic regression to estimate the relative risk of prostate cancer associated with 1) high stress, 2) high-effort coping, and 3) social support, while controlling for potential confounding factors.[45] Potential confounding factors included age, race, educational level, marital status, family history of prostate cancer, body mass index, alcohol and tobacco use, and number of prostate cancer screenings (digital rectal exam [DRE] or prostate-specific antigen [PSA] test) in the five years before the reference date. Since screening by DRE and PSA test were highly correlated ($r$=.61, $P<$ .0001), we created a variable to combine the number of prostate cancer screenings in the past five years by DRE or PSA test. Most studies addressing John Henryism have performed analyses by race; there-

fore, we followed this pattern in Tables 2–4. Body mass index, defined as self-reported weight (kg) before reference date divided by the square of self-reported height ($m^2$), was categorized as normal weight ($<25.0$ kg/$m^2$), overweight (25.0–29.9 kg/$m^2$), or obese ($\geq 30.0$ kg/$m^2$). Dummy variables based on the cut-points for each measure were included in the logistic regression model. Odds ratios (ORs) for psychological factors and prostate cancer are presented by race and adjusted for age and South Carolina region. No other confounding factors materially affected the ORs for stress, coping, or social support and prostate cancer.

## RESULTS

The final sample included 400 prostate cancer patients (160 African American and 240 Caucasian men) and 385 controls (161 African American and 224 Caucasian men). Crude ORs and 95% confidence intervals (CIs) for prostate cancer were presented for the risk factors of interest. Because of frequency matching, cases and controls were, in general, comparable in age and race. Having had benign prostatic hyperplasia (BPH) or a family history of prostate cancer was associated with incident prostate cancer (data not presented). Annual PSA tests or DREs over the past five years before the referent date were also associated with prostate cancer ($P$ value for trend $<.0001$). No other risk factors were associated with prostate cancer risk in these data.

Table 1 presents the mean scores with standard deviations for GPS, JHS, and social support by levels of risk factors among controls. Factors associated with having higher stress scores included African American race, less education, and lower income. Higher John Henryism scores were observed among African American men and those with less education, yet these differences were not statistically significant. Lastly, the following factors were associated with higher social support scores: Caucasian race, higher education, higher income, being married or living as married, and having annual prostate cancer screening.

Presented in Table 2 are the multivariate ORs for categories of each stress, coping, and social support scale (full GPS scale, perceived stress subscale, ability to control stress subscale, JHS, and social support scales) for cases and controls. Neither the full GPS measure nor the ability to control stress subscale were associated with prostate cancer. The perceived stress subscale may be associated with prostate cancer risk among African American men; however, the association does not follow a dose-dependent pattern. Higher John Henryism scores may be associated with prostate cancer risk, yet again the pattern did not reflect a dose-dependent pattern. The association was only statistically significant for African American men when comparing intermediate-to-low JH scores. Statistically nonsignificant ORs in the same direction were observed for all the other race-specific associations with John Henryism. Social support was not associated with prostate cancer risk. No evidence of interaction was found with the Breslow-Day test for homogeneity of the odds ratios for prostate cancer risk and psychosocial measures across race; therefore, subsequent analysis will include both race groups in one model.

We also addressed the potential for variables to interact with stress (Table 3) and coping (Table 4) to modify prostate cancer risk. These factors include prostate cancer stage, social support, stress, occupation, education, race, and income. We conducted these subanalyses to be consistent with the conceptual model proposed by Ellison,[40] which suggests that men who experience stress, but are high-effort coping either because of coping styles or social or economic support, are at the greatest risk of cancer.

Table 3 addresses the association between stress scores (as two dummy variables and a comparison of high and middle with low scores) and prostate cancer risk while adjusting for potentially modifying factors. In general, higher perceived stress scores were not consistently associated with prostate cancer risk in any subgroup investigated.

Table 4 presents the parallel analysis to that presented in Table 3. Higher and intermediate levels of JHS scores relative to lower scores were associated with an increased prostate cancer risk (OR 1.63, 95% CI 1.11–2.40). This association was similar among African American and Caucasian men. The effect of higher JHS scores on prostate cancer risk was somewhat more pronounced when perceived stress was intermediate or high. The effect of John Henryism on prostate cancer appears to be reduced among those with high social support. Neither education nor income modified the association between John Henryism and prostate cancer.

## DISCUSSION

These results provide limited support for the hypothesis presented by Ellison et al[40] that high-effort coping, as measured by the JHS, may be associated with a modest increase in risk of prostate cancer, particularly among those with lower social support. No racial differences in the effect of John Henryism on prostate cancer risk were noted. Neither social support nor higher perceived stress was associated with an increased prostate cancer risk.

The literature addressing psychological stress and breast cancer is perhaps most relevant to interpreting first study of stress and prostate cancer, since breast cancer is epidemiologically similar to prostate cancer.[46] Results from several recent cohort studies addressing perceived stress or stressful events and risk of subsequent breast cancer development are mixed. Of nine studies with at least five years of followup before breast cancer development, five found an

association with perceived stress or stressful events[27–29,47,48] while four did not.[49–52] We did not find that perceived stress was associated with prostate cancer risk. Since most studies that found an association between stress and breast cancer used stressful life events as a measure of stress, future studies assessing prostate cancer risk may also opt to measure stressful life events as well as perceived stress. No studies addressing John Henryism and breast cancer risk have been conducted.

As noted by Ellison et al,[40] chronic stress may affect prostate cancer risk. In response to stress, corticosteroid hormones, which have immunosuppressive properties,[53] including lower natural killer cell cytotoxicity,[17] are released. Prolonged stress may impair immune function, which may increase risk of carcinogenesis. In contrast to prior studies with other adverse outcomes,[54] we did not find that chronic perceived stress, unmitigated by high-effort coping or social support, increased the risk of prostate cancer.

South Carolina has one of the highest incidence rates of prostate cancer,[55] and African American men are at significantly greater risk than their Caucasian counterparts.[56] In this study, African American men had higher perceived stress, higher John Henryism, and lower social support scores than did Caucasian men. African American men are well known to have higher prostate cancer rates than do Caucasians. This study adds to the literature as the first study to address perceived stress, coping, social support, and prostate cancer among both African American and Caucasian men in a region with high prostate cancer rates.

Our study has several limitations to consider in interpreting these results. While we attempted to frame the subject's recall of stress, coping, and social support to experiences before prostate cancer development (eg, before the referent date), patients may have difficulty recalling feelings and experi-

**Table 1. Comparison of controls ($N=385$) on stress, coping, and social support scores**

| Risk Factor | Global Perceived Stress Score (Mean ± SD) | John Henryism (Coping) Score Mean (± SD) | Social Support Score (Mean ± SD) |
|---|---|---|---|
| Age (years)* | | | |
| 65–69 ($n=169$) | 14.88 (3.70) | 27.10 (3.12) | 13.70 (2.14) |
| 70–74 ($n=112$) | 15.29 (4.17) | 27.06 (3.43) | 13.77 (2.00) |
| 75–79 ($n=104$) | 15.40 (4.34) | 27.54 (2.81) | 13.62 (2.11) |
| *P* value for trend | .27 | .32 | .78 |
| Race† | | | |
| African American ($n=161$) | 15.88 (4.71)‡ | 27.46 (3.37) | 13.30 (2.41)‡ |
| Caucasian ($n=224$) | 14.61 (3.35) | 27.02 (2.95) | 13.98 (1.77) |
| Education† | | | |
| Less than high school graduate ($n=142$) | 16.36 (4.63) | 27.60 (3.29) | 13.31 (2.40) |
| High school graduate ($n=90$) | 15.19 (3.75) | 27.09 (3.44) | 13.81 (2.00) |
| Some college or technical school ($n=153$) | 13.98 (3.15) | 26.89 (2.75) | 14.01 (1.74) |
| *P* value for trend | <.0001 | .06 | .004 |
| Annual income | | | |
| <$20,000 ($n=104$) | 16.63 (4.43) | 27.30 (3.45) | 13.32 (2.24) |
| $20,000–$29,999 ($n=57$) | 15.46 (3.74) | 27.33 (2.67) | 13.84 (1.64) |
| $30,000–$39,999 ($n=54$) | 14.87 (3.20) | 27.33 (2.95) | 13.85 (1.74) |
| $40,000–$49,999 ($n=36$) | 13.61 (3.54) | 27.63 (2.97) | 13.86 (1.96) |
| ≥$50,000 ($n=77$) | 13.51 (2.55) | 27.08 (2.68) | 14.25 (1.76) |
| Missing ($n=57$) | | | |
| *P* value for trend | <.0001 | .77 | .002 |
| Marital status | | | |
| Single§ ($n=77$) | 15.83 (4.38) | 27.36 (3.07) | 13.29 (2.46) |
| Married‖ ($n=308$) | 14.93 (3.86) | 27.21 (3.06) | 13.83 (1.92)¶ |
| Body mass index (mg/kg$^2$) | | | |
| ≤24.9, normal weight ($n=111$) | 15.17 (3.68) | 27.81 (2.68) | 13.82 (2.00) |
| 25.0–29.9, overweight ($n=173$) | 14.91 (3.80) | 27.01 (3.10) | 13.82 (1.77) |
| ≥30.0, obese ($n=94$) | 15.32 (4.65) | 26.97 (3.32) | 13.54 (2.42) |
| *P* value for trend | .84 | .05 | .36 |
| History of benign prostatic hyperplasia | | | |
| No ($n=280$) | 15.00 (4.15) | 27.35 (3.07) | 13.70 (2.10) |
| Yes ($n=105$) | 15.40 (3.52) | 26.87 (3.25) | 13.66 (2.07) |
| Family history of prostate cancer | | | |
| No ($n=325$) | 15.21 (4.04) | 27.19 (3.18) | 13.65 (2.12) |
| Yes ($n=60$) | 14.70 (3.85) | 27.20 (2.94) | 13.97 (1.90) |
| History of hypertension | | | |
| No ($n=182$) | 15.27 (4.04) | 27.27 (3.07) | 13.81 (1.96) |
| Yes ($n=203$) | 14.99 (4.00) | 27.11 (3.22) | 13.61 (2.19) |
| Annual prostate cancer screening# | | | |
| No ($n=206$) | 15.46 (4.19) | 25.75 (2.84) | 13.49 (2.26) |
| Yes ($n=169$) | 14.71 (3.78) | 27.25 (2.87) | 13.99 (1.77)¶ |
| Ever drank alcohol | | | |
| No ($n=109$) | 14.92 (4.11) | 27.56 (3.33) | 13.69 (2.11) |
| Yes ($n=276$) | 15.20 (3.98) | 27.08 (3.04) | 13.71 (2.08) |
| Cigarette smoking history | | | |
| Never smoker ($n=118$) | 14.85 (4.31) | 27.06 (3.49) | 13.86 (2.20) |
| Former smoker ($n=204$) | 15.07 (3.83) | 27.25 (3.05) | 13.75 (1.96) |
| Current smoker ($n=63$) | 15.74 (4.03) | 27.23 (2.77) | 13.60 (2.28) |

**Table 1. Continued**

| Risk Factor | Global Perceived Stress Score (Mean ± SD) | John Henryism (Coping) Score Mean (± SD) | Social Support Score (Mean ± SD) |
|---|---|---|---|
| *P* value for trend | .19 | .67 | .89 |

SD=standard deviation
* Adjusted for South Carolina region (three areas).
† Adjusted for age (categorical variable), South Carolina region (three areas).
‡ $P<.01$.
§ Single includes single, never married, divorced, separated, widowed.
‖ Married includes currently married and living as married.
¶ $P=.01–.05$.
# Annual digital rectal exam or PSA screening received during the past five years.

ences before a prostate diagnosis. Thus, the measure of stress, coping, and social support among cases may be biased to reflect: 1) feelings that are a consequence of prostate cancer, or 2) feelings that did not change with prostate cancer diagnosis. Relative to controls, cases may have recalled social support after diagnosis. The measures of stress and coping are generalized measures of behaviors that are less likely to be affected by a specific recent health threat and, therefore, less likely to be mis-

**Table 2. Odds ratios for prostate cancer and stress, coping, and social support among men aged 65–79 by race**

| | African American Men (*n*=321) | | | Caucasian Men (*n*=464) | | |
|---|---|---|---|---|---|---|
| | Case *n*=160 | Control *n*=161 | Adjusted* OR (95% CI) | Case *n*=240 | Control *n*=224 | Adjusted* OR (95% CI) |
| Full global perceived stress scale (GPS)† | | | | | | |
| High (GPS score >20) | 21 (13.3%) | 28 (17.8%) | 1.18 (.47–2.63) | 14 (5.9%) | 10 (4.5%) | .85 (.43–1.68) |
| Intermediate (GPS score 14–20) | 88 (55.7%) | 74 (46.8%) | .76 (.52–1.12) | 123 (51.4%) | 129 (53.5%) | 1.33 (.81–2.18) |
| Low (GPS score 7–13) | 49 (31.0%) | 56 (35.4%) | 1.00 REF | 102 (42.7%) | 82 (34.0%) | 1.00 REF |
| *P* value for trend | | | .53 | | | .41 |
| Missing | 2 | 3 | | 1 | 3 | |
| Perceived stress subscale (of GPS) | | | | | | |
| Higher (score 9–15) | 60 (37.7%) | 56 (34.8%) | 1.40 (.85–2.31) | 75 (31.3%) | 80 (35.7%) | .67 (.43–1.06) |
| Intermediate (score 7–8) | 38 (23.9%) | 27 (16.8%) | 1.80 (.99–3.28) | 79 (32.9%) | 80 (35.7%) | .71 (.46–1.12) |
| Lower (score 3–6) | 61 (38.4%) | 78 (48.5%) | 1.00 REF | 85 (35.8%) | 64 (28.6%) | 1.00 REF |
| | | | .17 | | | .09 |
| Ability to control stress subscale (of GPS) | | | | | | |
| Higher (score >10) | 54 (34.0%) | 59 (37.3%) | .80 (.47–1.38) | 30 (12.5%) | 32 (14.5%) | .88 (.50–1.55) |
| Intermediate (score 7–9) | 51 (32.0%) | 51 (32.3%) | .85 (.49–1.48) | 95 (39.8%) | 85 (38.5%) | 1.02 (.68–1.51) |
| Lower (scores 4–6) | 54 (34.0%) | 48 (30.4%) | 1.00 REF | 114 (47.7%) | 104 (47.1%) | 1.00 REF |
| | | | .43 | | | .74 |
| Active coping (John Henryism [JH])‡ | | | | | | |
| High (JH score 29–30) | 83 (52.5%) | 86 (53.4%) | 1.69 (.86–3.30) | 98 (41.4%) | 90 (40.5%) | 1.44 (.85–2.44) |
| Intermediate (JH score 25–28) | 58 (36.7%) | 46 (28.6%) | 2.19 (1.07–4.48) | 104 (43.9%) | 85 (38.3%) | 1.61 (.52–1.36) |
| Low (JH score 12–24) | 17 (10.8%) | 29 (18.0%) | 1.00 REF | 35 (14.8%) | 47 (21.2%) | 1.00 REF |
| *P* value for trend | | | .40 | | | .31 |
| Missing | 3 | 0 | | 3 | 2 | |
| Social support (SS)§ | | | | | | |
| High (SS score: 15) | 89 (56.3%) | 75 (46.2%) | 1.30 (.96, 1.76) | 156 (65.7%) | 131 (58.8%) | 1.27 (.94, 1.73) |
| Intermediate (SS score 12–14) | 49 (16.5%) | 58 (17.7%) | .87 (.66, 1.15) | 69 (15.1%) | 75 (17.2%) | .87 (.69, 1.10) |
| Low (SS score 3–11) | 20 (27.2%) | 28 (36.1%) | 1.00 REF | 12 (19.2%) | 16 (24.0%) | 1.00 REF |
| *P* value for trend | | | .09 | | | .12 |
| Missing | 2 | 3 | | 3 | 2 | |

OR= odds ratio; CI= confidence interval.
* Adjusted for age (categorical variable), South Carolina region (three areas).
† Global Perceived Stress Scale: 7 items, range 7–29, Cronbach's alpha=.51.
‡ John Henryism Scale: 6 items, range 10 to 30, Cronbach's alpha=.66.
§ Social Support: 3 items, range 4–15, Cronbach's alpha=.69.

**Table 3. Global perceived stress and prostate cancer risk by social support, perceived and control stress, and socioeconomic status indicators**

| | | Global Perceived Stress (GPS) Score Comparing | | |
|---|---|---|---|---|
| | ***n* in Strata** | **Highest (GPS>20) with Lowest (GPS<14) OR (95%CI)** | **Middle (GPS 14–20) with Lowest (GPS<14) OR (95%CI)** | **Highest and Middle (≥14) with Lowest GPS (<14) OR (95%CI)** |
| All men | 777 | .82 (.48–1.38) | .95 (.70–1.29) | .93 (.69–1.24) |
| African American men | 318 | 1.18 (.47–2.63) | .85 (.43–1.68) | 1.20 (.75–1.92) |
| Caucasian men | 459 | .76 (.52–1.12) | 1.33 (.81–2.18) | .79 (.54–1.14) |
| GPS‡ by John Henryism (JH)* | | | | |
| High (JH score 29–30) | 355 | .85 (.36–2.02) | .92 (.59–1.43) | .91 (.59–1.40) |
| Intermediate (JH score 25–28) | 294 | 1.33 (.52–3.43) | 1.18 (.73–1.93) | 1.16 (.72–1.89) |
| Low (JH score 12–24) | 128 | .58 (.19–1.83) | .69 (.29–1.64) | .67 (.29–1.53) |
| GPS‡ by Social Support (SS)† | | | | |
| High (SS score 15) | 445 | .95 (.46–1.94) | 1.23 (.83–1.81) | 1.18 (.81–1.72) |
| Intermediate (SS score 12–14) | 250 | .87 (.29–2.55) | .66 (.38–1.13) | .68 (.40–1.15) |
| Low (SS score 3–11) | 75 | .96 (.20–4.70) | 1.11 (.25–4.88) | 1.05 (.26–4.31) |
| GPS‡ by stage at diagnosis | | | | |
| Stage I–II / controls | 295/383 | .97 (.56–1.67) | .96 (.69–1.33) | .96 (.70–1.31) |
| Stage III–IV / controls | 99/383 | .55 (.20–1.53) | .89 (.56–1.43) | .85 (.54–1.34) |
| GPS‡ by education level | | | | |
| Less than high school graduate | 287 | 1.12 (.55–2.28) | 1.78 (1.04–3.06) | 1.56 (.93–2.62) |
| High school graduate | 188 | .36 (.12–1.12) | .66 (.35–1.23) | .60 (.33–1.10) |
| College or technical school | 298 | 2.76 (.54–14.18) | .74 (.47–1.18) | .80 (.50–1.26) |
| GPS‡ by income | | | | |
| <$40,000 | 443 | .90 (.48–1.69) | .99 (.65–1.50) | .97 (.65–1.46) |
| ≥$40,000 | 230 | 2.61 (.26–26.26) | .74 (.44–1.26) | .77 (.46–1.30) |
| Missing | 104 | .51 (.13–2.03) | 1.23 (.51–2.92) | 1.04 (.45–2.37) |

Adjusted for age (categorical variable), South Carolina region (three areas), and race (African American or Caucasian).
* John Henryism Scale: 6 items, range 10 to 30, Cronbach's alpha=.66.
† Social Support: 3 items, range 4–15, Cronbach's alpha=.69; 7 missing.
‡ Global Perceived Stress Scale: 7 items, range 7–29, Cronbach's alpha=.51.

classified based on case status. We used a measure of global perceived stress that does not measure the frequency and magnitude of specific stressful life events. The GPS scale requires a significant self-knowledge and ability to disclose individual vulnerability; this ability to disclose may be associated with higher education and greater social support. This measure of stress may not be an appropriate measure of stresses experienced but rather of stresses perceived. Life experiences may be a more germane factor to assess. All measures of stress, coping, and social support were self-reported because the individual is the best barometer of perceived stress, coping, and support. We used abbreviated measures for stress, coping, and social support, which may lead to some misclassification; however, the Cronbach's $\alpha$ values for our measures were comparable to those reported for the full measures.[37,43] The Cronbach's $\alpha$ values were lower than optimal, and this finding indicates the potential for misclassification, which may reduce ORs toward the null. Our measure of perceived stress, social support, and John Henryism in the year before the interview may cause the exposure measure to not reflect the etiologically relevant time period. However, determining that relevant time period is difficult as it may range from experiences in childhood through adulthood. Other limitations include a lower response rate among African Americans than Caucasians. The refusal rates did not differ by race, but the proportion that could not be located was higher among African American (19.3%) than Caucasian (6%) men. Finally, this study had limited power to adequately evaluate several interactions.

This is the first population-based case-control study to address stress, coping, and social support and prostate cancer risk among both African American and Caucasian men. African American men may have higher prostate cancer rates because of genetic factors and environmental exposures, which may include environmental and individual stress, reactions to stress, and social support to buffer the effects of stress. We found that high-effort coping was more important than perceived stress as a correlate of prostate cancer risk, particularly among those with less social support. While the biologic effect of coping and support may be similar by race, the distribution of these risk

**Table 4. John Henryism Scores and prostate cancer risk by social support, perceived and control stress, and socioeconomic status indicators**

| | | John Henryism Scale (JHS) Score* comparing | | |
|---|---|---|---|---|
| | ***n* in Strata** | **Highest (JHS 29–30) with Lowest scores (JHS 12–24) OR (95%CI)** | **Middle (JHS 25–28) with Lowest scores (JHS 12–24) OR (95%CI)** | **High and Middle (JHS >24) with Lowest (JHS 12–24) OR (95%CI)** |
| All men | 777 | 1.51 (1.00–2.29) | 1.79–(1.17–2.73) | 1.63 (1.11–2.40) |
| African American men | 318 | 1.69 (.86–3.30) | 2.19 (1.07–4.48) | 1.52 (.94–2.48) |
| Caucasian men | 459 | 1.44 (.85–2.44) | 1.61 (.52–1.36) | 1.86 (.97–3.55) |
| John Henryism* by Global Perceived Stress (GPS)‡ | | | | |
| High (GPS score >20) | 73 | 1.47 (.42–5.13) | 1.79 (.49–6.55) | 1.61 (.53–4.87) |
| Intermediate (GPS score 14–20) | 416 | 1.52 (.77–2.68) | 1.93 (1.09–3.42) | 1.71 (1.01–2.89) |
| Low (GPS score 7–13) | 288 | 1.21 (.55–2.64) | 1.18 (.53–2.65) | 1.20 (.57–2.54) |
| John Henryism* by Social Support (SS)† | | | | |
| High (SS score 15) | 445 | .62 (.32–1.18) | .75 (.39–1.47) | .67 (.36–1.25) |
| Intermediate (SS score 12–14) | 250 | 2.98 (1.42–6.25) | 3.69 (1.80–7.55) | 3.35 (1.71–6.55) |
| Low (SS score 3–11) | 75 | 2.10 (.60–7.33) | 2.06 (.63–6.78) | 2.08 (.71–6.07) |
| John Henryism* by stage at diagnosis | | | | |
| Stage I–II / controls | 290/383 | 1.45 (.93–2.28) | 1.86 (1.18–2.94) | 1.63 (1.07–2.49) |
| Stage III–IV / controls | 99/383 | 1.72 (.88–3.38) | 1.56 (.77–3.14) | 1.65 (.87–3.13) |
| John Henryism* by education | | | | |
| Less than high school graduate | 287 | 1.14 (.56–2.33) | 1.71 (.81–3.63) | 1.34 (.68–2.65) |
| High School graduate | 188 | 1.65 (.71–3.84) | 1.68 (.69–4.09) | 1.67 (.74–3.71) |
| College or technical school | 298 | 1.67 (.87–3.21) | 1.82 (.97–3.42) | 1.75 (.98–3.15) |
| John Henryism* by income | | | | |
| <$40,000 | 443 | 1.57 (.92–2.67) | 1.56 (.89–2.73) | 1.56 (.94–2.59) |
| ≥$50,000 | 230 | 1.34 (.59–3.03) | 2.32 (1.04–5.19) | 1.81 (.85–3.86) |
| Missing | 104 | 1.46 (.48–4.44) | 1.90 (.62–5.90) | 1.65 (.59–4.63) |

Adjusted for age (categorical variable), South Carolina region (three areas), and race (African American or Caucasian).
* John Henryism Scale: 6 items, range 10 to 30, Cronbach =.66.
† Social Support: 3 items, range 4–15, Cronbach =.69; 7 missing.
‡ Global Perceived Stress Scale: 7 items, range 7–29, Cronbach =.51.

factors, and particularly economic support, may differ markedly by race and possibly explain part of the racial difference in prostate cancer incidence. Further research is needed to explore the interactions between stress, coping, and forms of support and prostate cancer risk. These studies need to include sufficient numbers of African American men to explore interactions in this high-risk group. Additional research with multiple measures of stress, coping, and support, including biologic measures, could further explore any biologic mechanisms by which stress, coping, and support may be etiologically linked with prostate cancer.

*Neither social support nor higher perceived stress was associated with an increased prostate cancer risk.*


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## REFERENCES


1. Cohen S, Frank E, Doyle WJ, et al. Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychol.* 1998;17:214–223.
2. Cohen S, Tyrrell DA, Smith AP. Psychological stress and susceptibility to the common cold. *N Engl J Med.* 1991;325:606–612.
3. Glaser R. Plasma cortisol levels and reactivation of latent Epstein-Barr virus in response to examination stress. *Psychoneuroendocrinology.* 1994;19:765–772.
4. McKinnon W, Weisse CS, Reynolds CP, et al. Chronic stress, leukocyte subpopulations, and humoral response to latent viruses. *Health Psychol.* 1989:389–402.
5. Kiecolt-Glaser J, Glaser R, Cacioppo J, et al. Marital stress: immunologic, neuroendocrine, and autonomic correlates. *Ann N Y Acad Sci.* 1998;840:656–663.
6. Zorrilla E, McKay J, Luborsky L, Schmidt K. Relation of stressors and depressive symptoms to clinical progression of viral illness. *Am J Psychiatry.* 1996;153:626–635.
7. Noisakran S, Halford W, Veress L, et al. Role of the hypothalamic pituitary adrenal axis and IL-6 in stress-induced reactivation of latent herpes simplex virus type 1. *J Immunol.* 1998;160:5441–5447.
8. Evans E, Leserman J, Perkins D, et al. Stress-associated reductions of cytotoxic T lymphocytes and natural killer cells in asymptomatic HIV infection. *Am J Psychiatry.* 1995;152:543–550.

9. Lesserman J, Petitto J, Perkins D, et al. Severe stress, depressive symptoms, and changes in lymphocyte subsets in human immunodeficiency virus-infected men: a 2 year follow-up study. *Arch Gen Psychiatry*. 1997;54:279–285.
10. Greenwood DC, Muir KR, Packham CJ, et al. Coronary heart disease: a review of the role of psychosocial stress and social support. *J Public Health Med*. 1996;18:221–231.
11. Gullette ECD, Blumenthal JA, Babyak M, et al. Effects of mental stress on myocardial ischemia during daily life. *JAMA*. 1997;277:1521–1526.
12. Rosengren A, Tibblin G, Wilhelmsen L. Self-perceived psychological stress and incidence of coronary artery disease in middle-aged men. *Am J Cardiol*. 1991;68:1171–1175.
13. Iso H, Date C, Yamamoto A, et al. Perceived mental stress and mortality from cardiovascular disease among Japanese men and women: the Japanese collaborative cohort study for evaluation of cancer risk sponsored by Monbusho (JACC study). *Circulation*. 2002;106: 1229–1236.
14. Schwartz A, Gerin W, Davidson KW, et al. Toward a causal model of cardiovascular response to stress and the development of cardiovascular disease. *Psychosom Med*. 2003;65:22–35.
15. Andersen B, Kiecolt-Glaser J, Glaser R. A biobehavioral model of cancer stress and disease course. *Am Psychol*. 1994;49:389–404.
16. Hilakivi-Clarke L, Dickson R. Stress influence on development of hepatocellular tumors in transgenic mice overexpressing TGR alpha. *Acta Oncol*. 1995;34:907–912.
17. Ben-Eliyahu S, Page GG, Yirmiya R, et al. Evidence that stress and surgical interventions promote tumor development by suppressing natural killer cell activity. *Int J Cancer*. 1999;80:880–888.
18. Byrnes DM, Antoni MH, Goodkin K, et al. Stressful events, pessimism, natural killer cell cytotoxicity, and cytotoxic/suppressor T cells in HIV+ Black women at risk for cervical cancer. *Psychosom Med*. 1998;60:714–722.
19. Dhabhar RS, McEwen BS. Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: a potential role for leukocyte trafficking. *Brain Behav Immun*. 1997;11:286–306.
20. Theorell T, Kaasek RA. Current issues relating to psychosocial job strain and cardiovascular disease research. *J Occup Health Psychol*. 1996;1:9–26.
21. Goodkin K, Antoni MH, Blaney PH. Stress and hopelessness in the promotion of cervical intraepithelial neoplasia to invasive squamous cell carcinoma of the cervix. *J Psychosom Res*. 1986;30:67–76.
22. Coker AL, Bond SM, Madeleine MM, et al. Psychosocial stress and cervical neoplasia risk. *Psychosom Med*. 2003;54:644–651.
23. Horne RL, Picard RS. Psychosocial risk factors for lung cancer. *Psychosom Med*. 1979;41: 503–514.
24. Cooper CL, Cooper R, Faragher EB. Incidence and perceptions of psychosocial stress: the relationship with breast cancer. *Psychol Med*. 1989;19:415–422.
25. Cooper CL, Faragher EB. Psychosocial stress and breast cancer: the inter-relationship between stress events, coping strategies and personality. *Psychol Med*. 1993;23:653–662.
26. Forsen A. Psychological stress as a risk for breast cancer. *Psychother Psychosom*. 1991;55: 176–185.
27. Chen CC, David AS, Nunnerley H. Adverse life events and breast cancer: a case-control study. *BMJ*. 1995;311:1527–1530.
28. Helgesson O, Cabrera C, Lapidus L, et al. Self-reported stress levels predict subsequent breast cancer in a cohort of Swedish women. *Eur J Cancer Prev*. 2003;12:377–381.
29. Lillberg K, Verkasalo PK, Kaprio J, et al. Stressful life events and risk of breast cancer in 10,808 women: a cohort study. *Am J Epidemiol*. 2003;157:415–423.
30. Ginsberg A, Price S, Ingram D, et al. Life events and the risk of breast cancer: a case control study. *Eur J Cancer*. 1996;32A: 2049–2052.
31. Courtney JG, Longnecker MP, Theorell T, et al. Stressful life events and the risk of colorectal cancer. *Epidemiology*. 1993;4:407–414.
32. Courtney JG, Longnecker MP, Peters RK. Psychosocial aspects of work and the risk of colon cancer. *Epidemiology*. 1996;7: 175–181.
33. Kune S, Kune GA, Watson LF, et al. Recent life change and large bowel cancer. *J Clin Epidemiol*. 1991;44:57–68.
34. Lehrer S. Life change and gastric cancer. *Psychosom Med*. 1980;42:499–502.
35. Esterling BA, Kiecolt-Glaser JK, Glaser R. Psychosocial modulation of cytokine-induced natural killer call activity in older adults. *Psychosom Med*. 1996;58:264–272.
36. Suls J, Fletcher B. The relative efficacy of avoidant and nonavoidant coping strategies: a meta-analysis. *Health Psychol*. 1985;4: 247–288.
37. James SA, Strogatz DS, Wing SB, et al. Socioeconomic status, John Henryism, and hypertension in Blacks and Whites. *Am J Epidemiol*. 1987;126:664–673.
38. James S, Hartnett S, Kalsbeek W. John Henryism and blood pressure differences among Black men. *J Behav Med*. 1983;6: 259–278.
39. Bennett GG, Merritt MM, Sollers JJ, et al. Stress, coping, and health outcomes among African Americans: a review of the John Henryism hypothesis. *Psychol Health*. 2004; 19:369–383.
40. Ellison GL, Coker AL, Hebert JR, et al. Psychosocial stress and prostate cancer: a theoretical model. *Ethn Dis*. 2001;11:484–495.
41. Adler N, Matthews K. Health psychology: why do some people get sick and some stay well? *Ann Rev Psychol*. 1994;45:229–259.
42. Sanderson M, Coker AL, Logan P, et al. Lifestyle and prostate cancer among older African American and Caucasian men in South Carolina. *Cancer Causes Control*. 2004;15: 647–655.
43. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav*. 1983;24:385–396.
44. Sarason BR, Shearin EN, Pierce GR, et al. Interrelations of social support measures: theoretical and practical implications. *J Pers Soc Psychol*. 1987;52:813–832.
45. Breslow NE, Day NE. *Statistical Methods in Cancer Research, Volume 1 -The Analysis of Case-Control Studies*. Lyon, France: WHO International Agency for Research on Cancer; 1980. IARC Scientific Publication No. 32.
46. Grover PL, Martin FL. The initiation of breast and prostate cancer. *Carcinogenesis*. 2002;23: 1095–1102.
47. Geyer S. Life events, chronic difficulties, and vulnerability factors preceding breast cancer. *Soc Sci Med*. 1993;37:1545–1555.
48. Price MA, Tennant CC, Butow PN, et al. The role of psychosocial factors in the development of breast carcinoma: Part II. *Cancer*. 2001;91: 686–697.
49. Lillberg K, Verkasalo PK, Kaprio J, Teppop L, Helenius H, Koskenvuo M. Stress of daily activities and risk of breast cancer: a prospective cohort study in Finland. *Int J Cancer*. 2001; 91:888–893.
50. Jacobs JR, Bovacco GB. Early and chronic stress and their relation to breast cancer. *Psychol Med*. 2000;30:669–678.
51. Achat H, Kawachi I, Byrne C, et al. A prospective study of job strain and risk of breast cancer. *Int J Epidemiol*. 2000;29:622–628.
52. Kroenke CH, Hankinson S, Schernhammer ES, et al. Caregiving stress, endogenous sex steriod hormone levels, and breast cancer incidence. *Am J Epidemiol*. 2004;159: 1019–1027.
53. McEwen BS, Stellar E. Stress and the individual. *Arch Intern Med*. 1993;153:2093–2101.
54. James S. John Henryism and the health of African Americans. *Cult Med Psychiatry*. 1994;18:163–182.
55. US Cancer Statistics Working Group. *United States Cancer Statistics: 1999 Incidence*. Atlanta, Ga: Department of Health and Human Services, Centers for Disease Control and Prevention, and National Cancer Institute; 2002.
56. Ries LAG, Eisner MP, Kosary CL, et al. *SEER Cancer Statistics Review, 1975–2000*. Bethesda, Md: National Cancer Institute; 2003.

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