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Why is Cancer More Depressing for Men than Women among Older White Adults?

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Abstract

Using data from two waves of the Wisconsin Longitudinal Study ($N = 8,054$), I examine gender differences in psychological adjustment to cancer among older White adults. Results from different types of longitudinal models reveal that cancer has more adverse psychological implications for men than women. Men's higher levels of depression are reduced after adjustment for adherence to masculinity ideals of strength, independence, and invincibility. Cancer poses a threat to the masculine identity because it entails lack of control over one's body and other consequences incompatible with traditional masculinity. This study contributes to sociological knowledge of the ways in which gender shapes psychological resilience and vulnerability to cancer through meanings people attach to gender roles.

Life expectancy of cancer patients has been rising in recent decades because of advances in diagnosing cancer at an earlier stage and improvements in treatment. Over 10 million Americans with a history of cancer were alive in 2003 (National Cancer Institute 2008). Given increasing numbers of long-term cancer survivors, the question about the quality of life after cancer becomes fundamentally important to researchers and clinicians (Stanton, Revenson, and Tennen 2007). Do longer lives of cancer patients bring more years of distress or are cancer patients leading happy and fulfilling lives?

The answer to this question is likely to be gender-specific because existing research indicates that the experience of cancer differs for men and women. Although men face a greater lifetime risk of cancer, women are confronted with cancer earlier in life (National Cancer Institute 2008). Women in their 30s, 40s, and 50s have a greater risk of cancer than their male peers, whereas the most common male cancers, including prostate cancer, typically occur after age 60 (Chapple and Ziebland 2002; National Cancer Institute 2008). Younger age at cancer diagnosis, especially before 40–45 years old, is associated with higher levels of distress compared to older ages (Kroenke et al. 2004; Schnittker 2005). A diagnosis of cancer may be a profound shock for younger people, whereas older adults are likely to anticipate the onset of chronic illness with advanced age (Mosher and Danoff-Burg 2005). These findings suggest that cancer may be more disruptive for women's than men's roles. Younger female cancer patients confront the possibility of jeopardized reproductive function and an early menopause, which may challenge their self-concept (Avis, Crawford,

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and Manuel 2004). Younger women with cancer may feel isolated from their healthy peers, and may experience unmet childcare needs and financial problems due to their illness (Mor, Malin, and Allen 1994; Mosher and Danoff-Burg 2005). Yet, although a cancer diagnosis happens, on average, later in life for men, older men may also be confronted with a disruption in family and work roles. In a study of prostate cancer survivors aged 50 and older, one of the men's primary concerns was whether they would be able to provide financially for their wives and children. Some men had to retire earlier than planned, and this forced early retirement was distressing for them and challenged their self-concept as providers and heads of the household (Chapple and Ziebland 2002).

These gender differences in the experience of cancer suggest that men and women may differ in their psychological reactions to the disease. Extant research on psychological adjustment to cancer provides some evidence of differential adjustment, although findings are mixed. Some studies show that among cancer patients, women report higher levels of distress than men (Bultz and Carlson 2006; Stommel et al. 2004), whereas others document that male cancer patients exhibit more depressive symptoms than their female peers (Goldzweig et al. 2009; Reisine et al. 2005). Finally, a number of studies report no significant gender differences in psychological distress of cancer patients (Gustavsson-Lilius et al. 2007; Rabin et al. 2007).

Our understanding of psychological resilience and vulnerability to cancer is hampered by limitations of previous research. Most importantly, psychological implications of cancer have been overwhelmingly explored within clinical psychology and nursing fields. As a result, psychological adjustment to cancer has been viewed as an individual accomplishment or, at best, as a dyadic effort of patients and their spouses. Whereas proximate individually-based mechanisms of coping with chronic illness are well documented (see Stanton et al. 2007 for review), little attention has been given to the social origins of psychological distress among cancer patients. From a sociological perspective, it is important to contextualize psychological reactions to cancer by identifying the ways in which individual processes and experiences are constrained or facilitated by societal norms and structures (Link and Phelan 1995; Schnittker and McLeod 2005). I examine gender differences in psychological adjustment to cancer within a framework that links men's and women's individual experiences of cancer and depression to the social and cultural context of gendered stress processes.

Gendered Patterns of Psychological Adjustment to Cancer

A central premise of this study is that psychological adjustment to cancer is likely to depend on the enactment of meanings and ideals attached to gender roles. To explore the ways in which social and cultural expectations associated with gender roles shape psychological reactions to cancer, I develop a theoretical framework combining two perspectives: the *cost of caring* and the *cost of dominance*.

The *cost of caring* perspective emphasizes traditional ideals of femininity and prevailing cultural images of women as nurturing, caring, and compassionate (Bottorff et al. 2008). Women, especially older cohorts, were socialized to put the needs of their families first and

to focus on nurturing others rather than advancing their own accomplishments. Women perform carework more often than men and are more responsive to others' emotional and physical needs as well as to events in other people's lives (England 2005; Kessler and McLeod 1984).

In the context of a chronic illness, women are more likely than men to be confronted with continuing demands in the household and to neglect their own health care needs because of family responsibilities (Boogaard 1984). For example, after a heart attack, male patients tend to reduce work and are nurtured by their wives, whereas female patients quickly resume household responsibilities (King 2000; Stanton et al. 2007). Women are typically responsible for juggling more roles than men, and when women must simultaneously deal with their chronic illness, their resources may become exhausted, leading to adjustment problems and distress (Northouse et al. 2000). A study of breast cancer (Sulik 2007) suggests that effective management of cancer requires that women become protective of themselves and prioritize their needs. Yet, the process of breaking the gender norms of compliance, nurturance, and putting others first may create stress and contribute to identity crisis. Moreover, women's distress may be exacerbated by the guilt about burdening others with their illness (Sulik 2007).

The *cost of dominance* perspective is derived from the social-constructionist gender framework (Courtenay 2000). The socially dominant gender construction is "hegemonic masculinity" (Connell 1995; Oliffe 2006), which represents power, tradition, and authority as well as emphasizes dominance, competitiveness, independence, control, and stoic display of emotions (Oliffe 2006). In the contemporary United States, dominant masculinity is embodied in heterosexual middle-class European American men and is explicitly constructed as distinct from White middle-class femininity (Courtenay 2000; Kimmel 1996). The enactment of masculinity is influenced by cultural beliefs that men are more powerful and resilient than women, that men's bodies are structurally superior to women's bodies, and that seeking help is feminine (Courtenay 2000). A two-dimensional view consistent with the institutional dominance is that men are strong, competitive, independent, and ambitious, whereas women are nurturant, warm, sensitive, expressive, and happiest caring for others (Bem 1974).

Social constructionist research has consistently drawn a link between men's health and prevailing cultural constructs of masculine stoicism and invulnerability (Courtenay 2000; Oliffe 2006). Adherence to traditional masculinity ideals becomes particularly problematic in the context of older age and chronic health conditions. A chronic debilitating illness is a threat to masculinity because it entails weakness, lack of control over one's body, and other consequences that are incompatible with invincibility, dominance, and independence (Charmaz 1995). The sick role and its ensuing needs may threaten men's selfconceptions and raise their self-doubts about masculinity (Arrington 2003; Charmaz 1995).

The cost of caring and the cost of dominance perspectives yield different predictions about psychological adjustment to cancer among men and women. From the cost of caring perspective, women are expected to be more adversely affected by cancer because women assume more caregiving and family responsibilities, and cast a wider net of concerns than

men do (Stanton et al. 2007). This emotional cost of caring may increase women's vulnerability to stressors compared to men (Kessler and McLeod 1984). In contrast, the cost of dominance perspective suggests that men may be more depressed by cancer than women because cancer is a more powerful threat to masculine than feminine identities. Cancer is incompatible with the dominant discourses of masculinity. Despite (or because of) their greater institutional power and a lifetime of achievement-orientation, older White men may have less effective coping skills and resources to adjust to dependency, vulnerability, and lack of control associated with a life-threatening illness.

This study is based on the 1993–1994 and 2004–2005 waves of the Wisconsin Longitudinal Study, a large community-based survey that started in 1957 when most participants were adolescents. I examine gender differences in depressive reactions to cancer in late midlife and old age when the incidence of cancer rises dramatically compared to earlier life stages. I compare change and continuity in depressive symptoms over approximately a ten-year period among recent and long-term cancer survivors as well as persons without cancer. Because participants were *not* selected into the study based on their cancer diagnosis as in most previous research on psychological adjustment to cancer, a wide range of pre-cancer characteristics is available to clarify the causal relationship between cancer and distress. In addition, I use fixed-effects models and propensity score matching to further elucidate the question of how much distress can be ascribed directly to the cancer experience of men and women.

DATA AND METHODS

Data

The Wisconsin Longitudinal Study (WLS) is a long-term study of a random sample of 10,317 men and women who graduated from Wisconsin high schools in 1957 and of their randomly selected siblings. The main participants (“graduates”) were interviewed at ages 17–18 (in 1957), 36 (in 1975), 53–54 (in 1993), and 64–65 (in 2004). Survey data were also collected from a selected sibling in 1977, 1994, and 2005. The overwhelming majority of the WLS participants are non-Hispanic White because very few members of racial or ethnic minority groups graduated from Wisconsin high schools in the 1950s. Detailed information on the WLS design, implementation, and sample characteristics is provided in Hauser (2009).

My analysis is based on the 1993–1994 and 2004–2005 data for graduates and siblings. The 1993–1994 surveys included a one-hour telephone interview and a self-administered questionnaire. Phone interviews were completed with 8,493 graduates, which constitute 82% of the original 10,317 participants and 87% of the 9,741 surviving members of the original sample. In addition, 6,875 graduates completed mail questionnaires in 1993. Moreover, 3,478 siblings completed phone interviews and returned mail questionnaires. The 2004–2005 round of data collection included a one-hour telephone interview and a 48-page mail survey. A telephone survey was completed by 7,265 graduates in 2004, which constitute 80.5% of the 9,025 living graduates. In addition to phone interviews, mail questionnaires were returned by 6,378 graduates. Additionally, 3,572 siblings completed both phone interviews and mail questionnaires in 2005. My analytic sample is based on graduates and

siblings who participated in phone interviews and returned self-administered mail questionnaires in both 1993–1994 and 2004–2005. The pooled sample of graduates and siblings includes 8,054 participants (3,656 men and 4,398 women).

Sample attrition—Depressive symptoms at baseline do not affect the probability of participating in the follow-up ($OR = 1.004$, $p = .846$). Although there is no evidence of outcome-dependent attrition bias, I adjust for the hazard of attrition in all models as an additional precaution. Further, I conducted a propensity score matching analysis of patterns of sample attrition among cancer patients (available upon request). I estimated the likelihood of being retained in the sample by comparing persons who were similar on a wide variety of characteristics in 1993–1994 but differed with respect to their cancer status. Women cancer patients were significantly more likely than non-cancer controls to drop out of the study due to death, whereas this difference is not significant among men. Conversely, the likelihood of nonparticipation for reasons other than death was *lower* among cancer survivors compared to non-cancer controls. In other words, persons who had cancer at baseline and survived to the follow-up were somewhat more likely to participate in the study than individuals without cancer, although this coefficient reaches statistical significance only among women. Finally, logistic regression models (not shown) comparing participants and nonparticipants at Time 2 with respect to characteristics *other than cancer* revealed that persons who were not retained in the study reported worse self-rated health, had lower levels of education, were less likely to be employed, had fewer financial assets, and were less likely to be married. Moreover, women and younger people were somewhat more likely to be retained in the study than men and older people.

All attrition analyses combined suggest the ways in which sample attrition may potentially bias my findings. Results from this study may be more likely to reflect experiences of higher-SES, married, and healthier individuals. Moreover, because cancer patients with the most advanced illness were likely to have died, persons who participated in the follow-up were robust cancer survivors. To the extent that higher SES, marriage, and physical health are associated with lower levels of distress, and long-term cancer survivors experienced fewer depressive symptoms than would have experienced cancer patients who had died, my findings may underestimate the levels of mental health problems in the population of cancer patients.

Measures

All models include both baseline (Time 1) and follow-up (Time 2) measures of *depressive symptoms* ($\alpha = .88$ in both waves). Depressive symptoms were evaluated using sixteen items reflecting negative feelings from the 20-item Center for Epidemiologic Studies Depression (CES-D) Scale. Participants were asked to indicate the number of days in the past week that they experienced depressive symptoms, such as feeling sad, depressed, unable to “get going,” or feeling that one’s life has been a failure. Response categories range from 0 to 7 days. Responses to all items are averaged to create an index. To reduce the positive skew, I took natural log of the depressive symptoms scale.

The focal predictor variable is the *presence or absence of a cancer diagnosis*. Three mutually exclusive dummy variables represent change and continuity in cancer status in the ordinary least squares (OLS) models: cancer at Time 1 and Time 2; no cancer at Time 1, cancer at Time 2; no cancer at Time 1 and Time 2 (reference group). There were also a small number of people who reported having cancer at Time 1 but then said at Time 2 that they had never had cancer. I treat these persons as a separate category, and an exploratory analysis showed that most of these people had nonmelanoma skin cancer at Time 1 and reported it, although they were explicitly asked to exclude minor skin cancers. Further, persons who had cancer at both waves and cancer patients diagnosed between Time 1 and Time 2 were categorized according to the *cancer type*: breast; genitourinary; colon; lymphatic and hematopoietic; digestive, respiratory, bone, skin, and connective tissue; and other cancer. In fixed-effects and propensity score matching models, a dichotomous indicator of cancer is used for each wave, coded 0 if a participant never had cancer, and 1 for persons ever diagnosed with cancer. *Age at cancer diagnosis* is included in the OLS models as a linear and squared term. *Time since diagnosis* is measured as a continuous variable in years (both linear and quadratic) and, alternatively, represented with four mutually exclusive dummy variables: 0–2 years, 2–4 years, 4–8 years, and over 8 years. Moreover, 33 persons reported diagnoses of two different cancers, and 3 persons had three cancers. I use a dummy indicator of *multiple cancers* as a control in all OLS models.

Physical characteristics were assessed in 1993 and 2004. Propensity score matching and OLS models include baseline (1993) physical characteristics, whereas fixed-effects models adjust for the change in physical characteristics between Time 1 and Time 2. Comorbidity is assessed as the *number of chronic illnesses other than cancer* diagnosed by a physician in the 12 months prior to the 1993 interview (Time 1) and 12 months prior to the 2004 interview (Time 2). A measure of *physical symptoms* is the sum of five symptoms experienced in the past six months, including dizziness/faintness, excessive sweating, fatigue/exhaustion, lack of energy, and shortness of breath, with a value of 0 assigned to those reporting none of these symptoms. A measure of *pain* is based on a one-item question: “During the past 4 weeks, how much did pain interfere with your normal work, including both work outside the home and housework?” Response categories range from 1 = “not at all” to 5 = “extremely.” I include binary indicators of whether the participant *needed help with the activities of daily living*, whether the participant *received care*, and whether the participant *accomplished less* with work or other daily activities as a result of physical health. In addition, the models adjust for the *number of days stayed in bed* during the last year because of illness or injury and an indicator of *limited physical activity* because of health.

Sexual activity was assessed in 2004 with the questions about the frequency and enjoyment of a sexual relationship in the 12 months prior to the interview. Participants were asked how often they had sex with their spouse or partner and how physically pleasurable they found their sexual relationship. In addition, participants answered whether they decreased or stopped sexual activity with their spouse or partner due to their illness, their physical changes, or their losing interest.

Masculinity beliefs—Men and women were asked about their extent of agreement with the following statements: “A man should always try to project an air of confidence even if he really doesn’t feel confident inside.” “When a man is feeling pain he should not let it show.” “Being larger, stronger-looking, and more muscular makes men more attractive to women.” “It bothers me when a man does something that I consider ‘feminine’.” “When a husband and wife make decisions about buying major things for the home, the husband should have the final say.” “Men have greater sexual needs than women.” “In some kinds of situations a man should be ready to use his fists.” Response categories range from 1 = “strongly agree” to 5 = “strongly disagree.” Responses were averaged to create the masculinity scale ($\alpha = .62$) that ranges from 1 to 5 with a mean of 2.7 and a standard deviation of 0.42. The distribution of masculinity beliefs differs markedly by gender. For example, 11% of men and 28% of women reported masculinity levels in the first (lowest) quintile, whereas 30% of men and 10% of women had scores in the fifth (highest) quintile.

Sociodemographic characteristics—*Gender* is coded 1 for women and 0 for men. *Age at the time of the interview* is measured in years. I include both linear and squared terms. Age range was between 37 and 75 years at Time 1 and between 48 and 86 years at Time 2. Only 10% of participants were younger than 47 years old at Time 1 and younger than 60 years old at Time 2. The mean age was 52.8 years in 1993–1994 and 64.6 years in 2003–2005.

Control variables were assessed in 1993 and 2004. The measure of *education* reflects the years of schooling one has completed; categories include fewer than 12 years, 12 years (reference category), 13 to 15 years, 16 years, and 17 or more years. *Net worth* reflects the participant’s total household assets. *Employment status* is coded 1 if a participant was working for pay at the time of the interview. Further, I include indicators of *pension plan* and *health insurance* as well as occupational income and occupational education of current or last occupation. *Occupational education* is a natural log of the proportion of persons in the respondent’s occupation that completed at least some college as of 1990, whereas *occupational income* is a natural log of the proportion of persons earning at least \$14.30 per hour in 1990. Five mutually exclusive categories represent *marital status and marital history*: currently married, married only once (reference category); currently married, married more than once; divorced/separated; widowed; and never married. Parental status is assessed with the *total number of children* (0 for nonparents) and the presence of *at least one child under 18* at Time 1.

Propensity score matching covariates—Cancer survivors and controls without cancer are matched on the following variables: time in months between the baseline and follow-up interviews, baseline depressive symptoms, age, age², gender, gender $\times$ age, family history of cancer, early-life characteristics (family structure, number of siblings, father’s education, farm origin, parental income, and religious affiliation), and a number of Time 1 predictors, including education, net worth, employment status, occupation, occupational education, occupational income, marital status, number of children, smoking status, alcohol consumption, body mass index, and physical activity.

Analytic Plan

I start with descriptive statistics summarizing distribution of cancer among men and women at Time 1 and Time 2. For multivariate analyses, I use three types of regression models. First, an *ordinary least squares model (OLS) with a lagged dependent variable* estimates the association between cancer and change in depressive symptoms between Time 1 and Time 2. Adjusting for baseline levels of depressive symptoms, the model compares two groups of cancer survivors to the reference group of individuals without cancer:

$$DS_{i2} + \beta_0 + \beta_1 \text{Cancer1}_i + \beta_2 \text{Cancer2}_i + \beta_3 DS_{i1} + \beta_4 X_i + \varepsilon_i \quad (1)$$

where DS_{i2} is depressive symptoms at Time 2, β_0 is a constant, β_1 is a regression coefficient estimating the difference in depressive symptoms between individuals who had cancer at both waves and the reference group, β_2 is a regression coefficient estimating the difference in depressive symptoms between individuals who had cancer only at Time 2 and the reference group, β_3 is a regression coefficient for baseline depressive symptoms, $\beta_4 X_i$ is a vector of potential baseline confounders, and ε_i is the error term. I use robust standard errors to account for nonindependence of observations for siblings within families. The advantage of this OLS model is that I can explicitly compare depressive symptoms of persons who had cancer at baseline, persons who were diagnosed with cancer between the waves, and persons who have never been diagnosed with cancer.

Second, a *fixed-effects pooled time-series model (FE)* estimates the effect of change in cancer between the two waves on change in depressive symptoms while taking into account unobserved characteristics of individuals that remain stable between the waves and may confound the association between cancer and mental health. The fixed-effects specification eliminates the part of the association between cancer and mental health that is spurious due to unmeasured time-invariant individual characteristics. This model can be represented with the equation:

$$Y_{it} - Y_{.i} = (\alpha_i - \alpha_i) + \beta_1 (X_{it1} - X_{.t1}) + \beta_2 (X_{it2} - X_{.t2}) + \dots + \beta_n (X_{itn} - X_{.tn}) \quad (2)$$

where Y_{it} is the psychological outcome for individual i at time t ; α_i is a fixed term capturing the influence of unobserved time-invariant factors related to individual i ; X_{itm} denotes the values of independent variables for individual i at time t ; $Y_{.i}$ and $X_{.in}$ are overall means that are subtracted from a specific value at time t . The nonindependence of observations between main participants and their siblings is taken into account by using standard errors robust to clustering of observations within families.

Third, I use *propensity score matching* to examine the effect of cancer on depressive symptoms among cancer survivors and matched “non-cancer” controls. This analysis applies to all persons who did *not* have cancer at baseline. The purpose of propensity score matching estimators is to approximate the conditions of an experiment. Because cancer is not randomly distributed in the population, estimates from regression models are likely to be biased by the existence of confounding factors. Propensity score matching is a way to correct for this potential bias by comparing persons with cancer and non-cancer controls who are as similar as possible (Becker and Ichino 2002). Propensity score matching is a

two-step procedure. The first step is to estimate propensity score – each individual's propensity to be diagnosed with cancer by the follow-up based on observed baseline characteristics. The second step involves matching cancer survivors and non-cancer controls on their propensity scores. Matched individuals in the cancer and control groups are then compared in terms of depressive symptoms.

In the first step, I summarize baseline (pre-diagnosis) characteristics of each individual into a single composite propensity score. The propensity score, which represents the conditional probability of being diagnosed with cancer, can be defined with the following equation:

$$p(\text{cancer}) = \Pr(C_i = 1 | X_i) \quad (3)$$

where $C_i = 1$ if individual i has ever been diagnosed with cancer, and X_i is a vector of covariates that predict cancer or may confound the association between cancer and mental health.

In the second step, I estimate the average treatment effect for the treated (ATT), i.e. group differences in depressive symptoms between cancer survivors and non-cancer controls that have similar propensities of experiencing cancer on the basis of observable characteristics. For a given propensity score, cancer can be viewed as though it had occurred at random and, therefore, individuals in the cancer and control groups should be on average homogeneous except for cancer. The average treatment effect for the treated (ATT) can be estimated as follows (Becker and Ichino 2002):

$$ATT = E[E\{Y_{1i} | C_i = 1, p(\text{cancer})\} - E\{Y_{0i} | C_i = 0, p(\text{cancer})\} | C_i = 1] \quad (4)$$

where Y_{1i} and Y_{0i} are the levels of depressive symptoms in the cancer and control groups, respectively. I compare results from two propensity score matching methods: the nearest neighbor and kernel matching.

A potential weakness of propensity score matching is a hidden bias arising from unobserved variables. For example, if one person has an unobserved characteristic U while the other does not, these two persons have different propensities of being diagnosed with cancer, yet this difference will not be captured when the persons are matched on observed characteristics. I address this problem with a sensitivity analysis based on the bounding approach proposed by Rosenbaum (Rosenbaum 2002). The sensitivity analysis allows to determine how strongly an unmeasured variable must influence selection into treatment in order to undermine conclusions of propensity score matching. If γ is the effect of U on the risk of cancer, and $P_i/(1 - P_i)$ and $P_j/(1 - P_j)$ are the odds of being diagnosed with cancer for a matched pair of individuals i and j , we can estimate the following bounds on the odds ratio that either of the two matched individuals will receive treatment (Rosenbaum 2002):

$$\frac{1}{e^\gamma} \leq \frac{P_i(1 - P_j)}{P_j(1 - P_i)} \leq e^\gamma \quad (5)$$

Let us denote $e\gamma$ as Γ . Both matched persons have the same probability of being diagnosed with cancer only if $\Gamma = 1$. In this sense, Γ is a measure of the degree of departure from a study that is free of hidden bias (Becker and Caliendo 2007). I use this sensitivity analysis to simulate how changing the values of Γ alters the inferences of propensity score matching based on observed variables (Becker and Caliendo 2007; Rosenbaum, 2002). The bounding approach provides evidence of how sensitive the results are to unobserved influences on selection into treatment.

RESULTS

Cancer Prevalence among Men and Women

Table 1 indicates that in 1993–1994, 2.56% of all men and nearly 4% of all women had ever been diagnosed with cancer. Whereas women were more likely to be diagnosed with cancer earlier in life ($p < .001$), this gender difference became nonsignificant later in life, and by 2004–2005, roughly 11% of men and women had ever received a cancer diagnosis. The predominant cancer types were genitourinary and colon cancer among men and women, and breast cancer among women.

Multivariate Analyses

Table 2 shows regression coefficients from OLS and within-individual fixed-effects models. Model 1 reveals that, adjusting for baseline levels of distress and a wide range of sociodemographic characteristics, individuals who did not have cancer at Time 1 but developed new cancer by Time 2 report significantly more depressive symptoms than their peers without cancer ($b = .167$, $se = .052$, $p < .001$). Similarly, long-term cancer survivors who had cancer both at baseline and follow-up exhibited higher levels of distress than persons without cancer ($b = .302$, $se = .097$, $p < .01$). Because the depression scale was converted to natural log, exponentiated coefficients reveal that compared to individuals without cancer, people with relatively recent cancer diagnosed between the waves report $e^{.167} = 1.18$ additional days per week of experiencing depressive symptoms, and people with long-term cancer diagnosed before 1993–1994 report $e^{.302} = 1.35$ more days of depressive symptoms per week. In contrast, individuals who reported cancer at Time 1 but not Time 2 did not differ from persons without cancer in terms of depressive symptoms.

Within-individual fixed-effects estimates are shown in Model 4 of Table 2. This model adjusts for unobserved time-invariant characteristics, such as genetic predispositions, unhealthy lifestyle, or chronic environmental strains that may affect *both* cancer and mental health and, thus, produce a spurious association between the two. Fixed-effects models estimate within-individual change in cancer status and mental health while taking into account all characteristics that remain stable over time. Compared to the OLS Model 1, the effect of cancer on depressive symptoms declines by 30% $(.167 - .117)/.167 = .299$, although it remains statistically significant at the .05 level. Thus, the adverse mental health effect of cancer persists net of unmeasured time-invariant confounders. Individuals who were diagnosed with cancer between the waves report more depressive symptoms compared not only to persons without cancer but also to their own pre-cancer levels of distress.

Interaction terms shown in Models 2, 3, and 4 of Table 2 reveal significant gender differences in psychological adjustment to cancer. The direction and magnitude of these differences is consistent across OLS and fixed-effects models. Figure 1 illustrates interaction terms in Model 2. *Between-gender* comparisons indicate that men without cancer report fewer depressive symptoms than women without cancer, yet this pattern is reversed among cancer survivors. Men who had cancer at Time 1 and Time 2 exhibit considerably more depressive symptoms than their female peers. Similarly, men who were diagnosed with cancer between the waves exhibit significantly higher levels of distress than women in the same category, although this difference is small in magnitude. *Within-gender* comparisons reveal that the effect of cancer on depressive symptoms is particularly pronounced among men. Men diagnosed with cancer, especially long-term cancer survivors, report much higher distress than men without cancer, whereas women who have cancer are more similar in terms of distress to women without cancer. Both within- and between-gender comparisons suggest that men are more adversely affected by cancer than women. With respect to effect magnitude, the difference between predicted depressive symptoms of men and women with long-term cancer is $e^{.376} = 1.46$ days, and with relatively recent cancer is $e^{.058} = 1.06$ days. In other words, men who had cancer at Time 1 and Time 2 report, on average, 1.5 more days per week of being depressed than their female peers, and men who developed new cancer by Time 2 report, on average, one additional day of depressive symptoms.

Finally, I include the masculinity scale in Model 3 and examine the extent to which adherence to the ideals of traditional masculinity explains more adverse psychological implications of cancer among men. After adjustment for masculinity beliefs, the coefficients for gender $\times$ new cancer and gender $\times$ old cancer interaction terms decline by 22–23%, although they remain close to marginal significance at the .10 level. Within- and between-gender differences in psychological adjustment to cancer that persist net of a wide array of sociodemographic characteristics are substantially reduced when masculinity is taken into account. It is notable that masculinity is associated positively with depressive symptoms. Both men and women who strongly adhere to dominant masculinity beliefs report more depressive symptoms than individuals with lower scores on the masculinity scale; yet, men on average are more supportive of masculinity ideals than women.

Propensity Score Matching

I use propensity score matching techniques to estimate the effect of cancer on depressive symptoms by comparing cancer patients (the treatment group in experimental terminology) to “noncancer” controls matched on a wide variety of characteristics listed in the note to Table 3. The treatment and control groups are well matched, and the balancing property has been satisfied. A comparison between the treatment and control groups with respect to variables used for matching (available upon request) did not reveal any significant differences, which suggests that the two groups are indeed similar to each other with the exception of a cancer diagnosis. Both nearest neighbor and kernel matching estimates shown in Table 3 consistently indicate that men who have developed cancer between the waves report 1.4 additional days per week of depressive symptoms compared to their controls (ATT = .313, $e^{.313} = 1.4$, $p < .001$ for the kernel matching estimator). In contrast, women who have cancer are similar to their controls in terms of depressive symptoms. A

comparison of ATT estimates between men and women shows that the effect of cancer on depressive symptoms differs significantly by gender: $t = 2.23$, $p < .05$ for the nearest neighbor matching estimator and $t = 3.42$, $p < .01$ for the kernel matching estimator. This significant between-gender difference suggests that not only are men with cancer more depressed than men without cancer but also men with cancer are more depressed than women with cancer. As for effect magnitude, men with cancer report, on average, 1.27 more days per week of depressive symptoms than women with cancer, as shown both by nearest neighbor ($e^{[.333 - .097]} = 1.27$) and kernel ($e^{[.313 - .072]} = 1.27$) estimators. Thus, results from propensity score matching analysis confirm findings from OLS and fixed-effects models that cancer appears to elevate depressive symptoms among men but not women.

The sensitivity analysis shows that the effect of cancer on depressive symptoms among men becomes nonsignificant at the .05 level when $\Gamma = 1.5$. In other words, if there is an unobserved variable that increases the odds of being diagnosed with cancer by 1.5 net of all observed variables used for matching, then my ATT may overestimate the true ATT. For comparison, the effect of having a first-degree relative with cancer is 1.38, the effect of smoking at baseline on cancer 10 years later is 1.33, and the effect of being 65 or older versus younger than 65 is 1.37. Therefore, to wipe out the effect of cancer on depressive symptoms, the effect of this hypothetical unobserved covariate on selection into treatment should be stronger than the effect of age, smoking, and family history of cancer *net* of all these and other characteristics used for matching. Because such a scenario is highly unrealistic, I conclude that my propensity score matching estimates are robust to bias from unobserved characteristics.

Differential Psychological Adjustment by Gender and Cancer Type

Table 4 presents analyses separately by gender. All models compare change in mental health of cancer survivors to mental health of persons without cancer while adjusting for baseline levels of distress.

As shown in Model 1, regardless of cancer site, women who developed new cancer between the waves or had cancer both at Time 1 and Time 2 are similar in terms of depressive symptoms to women who have never had cancer. In contrast, men who were diagnosed with genitourinary cancer (including prostate) report significantly more depressive symptoms than men without cancer. This elevated distress is evident for long-term genitourinary cancer ($b = .762$, $se = .241$, $p < .01$) as well as more recent cancer diagnosed between the waves ($b = .331$, $se = .090$, $p < .001$). Further, men in the broad group comprising survivors of digestive, respiratory, bone, and skin cancers report elevated distress if this cancer was diagnosed between the waves but not if it was present at baseline. Men with long-term colon cancer report more depressive symptoms than men without cancer, yet there is no evidence of elevated distress among survivors of relatively recent colon cancer. In addition, men diagnosed with rare cancers in the “other” category have significantly higher levels of distress compared to men without cancer.

Model 2 in Table 4 includes comorbid conditions, physical symptoms, and functional limitations. Among men, these variables explain the associations between all cancer types and depressive symptoms – with two exceptions. First, although the coefficient for

genitourinary cancer diagnosed between the waves declines by 52% and the coefficient for long-term genitourinary cancer declines by 36%, both effects remain statistically significant at the .05 level and, thus, are not fully accounted for by health conditions and limitations. Second, the coefficient for less common cancers in the “other” category is reduced almost by 40%, although it retains statistical significance.

Model 3 in Table 4 adjusts for sexual activity among men only. Because genitourinary cancers often disrupt sexual functioning (Chapple and Ziebland 2002), it is possible that sexual impairment mediates the adverse psychological effect of genitourinary cancers. As shown in Model 3, frequency of sexual activity and decreased sexual activity because of illness, physical changes, or losing interest are not related to depressive symptoms, whereas finding sex physically pleasurable is associated negatively with depression ($b = -.136$, $se = .026$, $p < .001$). Yet, none of the sexuality variables explain the association between genitourinary cancer and depression. The coefficients for cancers remain virtually unchanged in Model 3 compared to Model 2.

The masculinity scale is added in Model 4 of Table 4. The adverse implications of long-term genitourinary cancer for men's mental health are reduced to nonsignificance after adjustment for masculinity. Men who strongly espouse traditional masculinity are more adversely affected by genitourinary cancer than men with low scores on the masculinity scale. Once the differences in masculinity beliefs are taken into account, the genitourinary cancer patients become similar in terms of mental health to men without cancer. In contrast, the effect of genitourinary cancer diagnosed between Time 1 and Time 2 *increases* by nearly 30% after adjustment for masculinity. This suppression effect occurs because men with relatively recent genitourinary cancer report lower levels of masculinity than their peers without cancer, and masculinity is positively associated with depressive symptoms. If the levels of masculinity among cancer patients in this group had been higher, they would have reported even more depressive symptoms than we actually observe, as indicated by the increase in the effect size from .159 in Model 2 to .218 in Model 3.

Age at Diagnosis and Time Elapsed Since Diagnosis

I estimated OLS regression models (not shown) to examine whether psychological adjustment to cancer depends on age at which the illness was diagnosed. Findings reveal that among women, the psychological implications of cancer do not depend on age at diagnosis (net of current age). In contrast, among men who were diagnosed with cancer between the waves, younger age at diagnosis is associated with more depressive symptoms, and this effect is significant at the .10 trend level – net of current age, sociodemographic characteristics, and various health indicators and symptoms. In addition, once participants are categorized based on the presence or absence of cancer at baseline, more detailed measures of the duration since cancer diagnosis are not related to psychological adjustment.

DISCUSSION

Using data from the 1993–1994 and 2004–2005 waves of the Wisconsin Longitudinal Study, I examine psychological adjustment to cancer among middle-aged and older White men and women. Results from different types of longitudinal models consistently reveal that cancer

has more adverse psychological implications for men than women. Among individuals without cancer, men report significantly *fewer* depressive symptoms than women, consistent with well-documented patterns in social stress research (Kessler and Zhao 1999; Ross and Mirowsky 2008). Yet, this gender difference is reversed among cancer survivors: men with cancer report *more* depressive symptoms than both women with cancer and persons without cancer, and men's greater distress is not explained by the disease characteristics, such as age at diagnosis, duration since diagnosis, symptoms, functional limitations, and decreased sexual activity. Even when cancer patients are matched to non-cancer controls on a wide variety of adult characteristics, early-life family influences, and family history of cancer in the propensity score matching models, men who have developed cancer between the waves report 1.4 more days per week of depressive symptoms than their matched controls. With respect to specific cancer types, genitourinary cancers (including prostate) appear to have particularly adverse implications for depressive symptoms among men, which are not explained by debilitating physical symptoms or sexual impairment.

Higher levels of distress among men with cancer are reduced substantially after adjustment for adherence to the traditional masculinity ideals. Cancer survivors holding strong beliefs that men should be stoic, confident, muscular, and in control report elevated depressive symptoms compared to men without cancer who have similar levels of masculinity beliefs. Men who strongly espouse masculinity ideals are more adversely affected by cancer than men with low scores on the masculinity scale. Once the differences in masculinity beliefs are taken into account, male cancer survivors become similar to their peers without cancer in terms of mental health. Moreover, among men with relatively recent genitourinary cancer, the disease appears to reduce the level of masculinity beliefs. This finding is consistent with qualitative studies revealing that prostate cancer challenges survivors' self-conceptions of manhood (Arrington 2003; Oliffe 2006). Men with genitourinary cancer diagnosed between the waves report lower levels of masculinity than their peers without cancer, and masculinity is positively associated with depressive symptoms. If the levels of masculinity among cancer patients in this group had been higher, they would have reported even more depressive symptoms than we actually observe.

From the cost of caring perspective, women's roles as nurturers, comforters, and selfless helpers may increase their vulnerability to stressors because "the inability to act according to one's own interests also predisposes individuals to feelings of helplessness and hopelessness that characterize depressive reactions" (Rosenfield 1999:216). Women encounter more severe role strains than men in paid work and family as a consequence of economic, institutional, and cultural devaluation of carework (England 2005; Rosenfield 1999). Based on the cost of caring perspective, I predicted that women with cancer would report more depressive symptoms than their male peers. Contrary to this hypothesis, my analysis revealed men's greater vulnerability to the adverse mental health implications of cancer, despite the fact that in these cohorts characterized by the traditional and clearly demarcated gender roles, men have had more institutional power and resources than women over the life course.

Conversely, the finding of men's higher levels of cancer-related distress is consistent with the cost of dominance perspective derived from the social constructionist studies of

masculinities. While privileged in terms of institutional power and status attainment, men may be disadvantaged in relation to health, health behaviors, and emotional reactions to illness by the pervading discourses of invincible masculinity (Courtenay, 2000; Wall and Kristjanson 2004). Men who face difficulties in complying with the demands of traditional masculinity are likely to experience masculine gender-role stress, which in turn compromises men's mental health (Eisler, Skidmore, and Ward 1988). Men are more depressed by cancer than women largely because cancer is incompatible with the ideals of dominant masculinity and, thus, poses a threat to the masculine identity. Dependence, vulnerability, and lack of control associated with cancer can undermine the performance of culturally defined masculinity but not femininity (Arrington 2003; Korda 1997). Moreover, because older cohorts of men and women experienced gender-typed division of labor, most men in this study have performed the breadwinner role and assumed the primary economic responsibility in the public sphere, whereas women have been primary caretakers for their families. The breadwinner role associated with achievement, dominance, and control appears to be less resilient to cancer than nurturance, sensitivity, and emotional expressiveness of women who have spent a lifetime of caring for others (Carr 2004; Rosenfield, Phillips, and White 2006).

Several limitations of this study should be mentioned. Although the WLS is one of the longitudinal social surveys with the most detailed measures of physical and mental health over time, information on certain disease characteristics is not available, including stage of cancer at diagnosis, recurrence, and the type of cancer treatment. It is likely, however, that some of observed variables in my analysis as well as fixed-effects models have indirectly captured the effects of these omitted variables. Moreover, cancer process and cancer treatment can elevate depression via endocrinologic pathways or debilitating side effects (Schnoll and Harlow 2001; Stommel et al. 2004). I cannot control directly for these physiological effects, although they can confound my findings. This potential confounding would have been particularly problematic for my main argument if men's mental health was more strongly than women's influenced by direct physiological effects of cancer. Yet, I have found no evidence in the clinical literature suggesting a greater likelihood of depression related to cancer sequelae among men compared to women. Thus, there is little reason to believe that sex differences in physiological pathways have produced gender differences in psychological reactions to cancer observed in my study.

CONCLUSION

Psychological and clinical studies have overwhelmingly viewed cancer-related distress as an individual response that can be explained by the characteristics of the illness and treatment or by patients' psychosocial resources. I argue that sociological perspectives can enhance our understanding of psychological adjustment to cancer because mental health trajectories of cancer survivors unfold within socio-historical and cultural contexts. Psychological reactions to cancer may appear highly individualized and intimate, yet they are socially and culturally patterned. Gender differences in depression reflect the dominant discourses of masculinity and femininity. My finding that older White men with cancer may be particularly vulnerable to depression underscores the importance of depression screening

among this group of cancer patients and designing psychosocial interventions tailored specifically to their needs.

Moreover, studying chronic illness can make an important contribution to sociology by enriching sociological understanding of the interplay of private bodies and public discourses. Sociologists have long established that social statuses are “fundamental causes of disease” (Link and Phelan 1995). Yet, an important task facing sociology today is bridging the gap between macro-level social structures and individual well-being via meso-level social psychological processes of self, identity, and meaning (Schnittker and McLeod 2005). This study expands sociological knowledge of the ways in which gender shapes psychological resilience and vulnerability to cancer through the meanings people attach to their gender roles and identities. A focus on psychological implications of physical changes in the body entailed by cancer enables me to explore how social statuses “get under the skin” and become powerful influences on survivors’ well-being.

This study suggests some directions for future research. Because the WLS comprises older White men and women, an important direction for future research is to compare gendered patterns of cancer-related distress among racial and ethnic groups, especially given that the ideals of masculinity and femininity vary by race (Courtenay 2000; Rosenfield, Phillips, and White 2006). Further, it would have been interesting to include a measure of femininity beliefs equivalent to the measure of masculinity. Are White older adults who believe that women should comply with the ideals of traditional femininity (for example, to be emotionally expressive, nurturing, and happiest caring for others) more depressed than their peers with more gender egalitarian views? It is possible that adherence to the traditional ideals of both masculinity and femininity is associated with elevated depressive symptoms and poorer adjustment to cancer, and it is an interesting question to be explored further.

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Figure 1. Gender Differences in Depressive Symptoms

Notes: The figure is based on Model 2 of Table 2.

Table 1

Numbers and Percentages of Cancer Survivors by Gender and Cancer Site

Cancer	Men n = 3,656	Women n = 4,398
Cancer at Time 1 (all sites)	93 2.56% ***	174 3.98%
Cancer at Time 2 (all sites)	390 10.67%	485 11.03%
<i>Cancer at Time 2 by Cancer Site:</i>		
Breast	—	276 6.28%
Prostate	199 2.47%	—
Genitourinary	46 1.26% ***	99 2.25%
Colon	39 1.07% **	25 .57%
Lymphatic and hematopoietic	24 .66% *	14 .32%
Digestive	14 .38%	9 .20%
Respiratory	16 .44%	14 .32%
Bone, skin, connective tissue	15 .41%	8 .18%
Other cancer	30 .82%	37 .84%
<i>Change in Cancer Between Waves:</i>		
No cancer at Time 1 and Time 2	3,204 88.05%	3,832 87.71%
Cancer at Time 1 and Time 2	43 1.18% ***	119 2.72%
Cancer at Time 1, no cancer at Time 2	50 1.26%	55 1.31%
No cancer at Time 1, cancer at Time 2 (all sites)	342 9.40%	363 8.31%
<i>No Cancer at Time 1, Cancer at Time 2, by Site:</i>		
Breast	—	204 4.67%
Prostate	181 4.97%	—
Genitourinary	36 .99% **	80 1.83%
Colon	34 .93% **	17 .39%
Lymphatic and hematopoietic	21 .58% *	11 .25%
Digestive, respiratory, bone, skin, connect. tissue	41 1.13% **	25 .57%

Cancer	Men <i>n</i> = 3,656	Women <i>n</i> = 4,398
Other cancer	22 .60%	23 .53%

Notes:

*
p < .05

**
p < .01

p < .001.

Asterisks denote significant differences between men and women in the distribution of cancer. Each cell contains the number of cancer survivors and the percentage of cancer survivors among all men or women.

Table 2

Unstandardized Coefficients from Longitudinal Ordinary Least Squares (OLS) and Fixed-Effects (FE) Models of the Association between Cancer and Depressive Symptoms

Variables	OLS				FE	
	Model 1	Model 2	Model 3	Model 4	Model 5	
<i>Change in Cancer:</i>						
No cancer at T1 and T2 (reference group)						
No cancer at T1, cancer at T2	.17*** (.05)	.28*** (.08)	.34*** (.09)	.12* (.05)	.18* (.08)	
Cancer at T1 and T2	.30*** (.09)	.72*** (.19)	.53* (.25)			
Cancer at T1, no cancer at T2	.13 (.13)	-.08 (.19)	-.14 (.19)			
Depressive symptoms at T1	.47*** (.01)	.47*** (.01)	.47** (.02)			
Gender (Female = 1)	.17*** (.04)	.19*** (.04)	.27*** (.05)			
Gender × No cancer at T1 and T2 (reference group)						
Gender × No cancer at T1, cancer at T2		-.25* (.10)	-.19 (.12)		-.15# (.11)	
Gender × Cancer at T1 and T2		-.57*** (.22)	-.44 (.28)			
Gender × Cancer at T1, no cancer at T2		.37 (.25)	.42# (.25)			
Masculinity Scale			.25*** (.05)			
Age at Time 2	-.002 (.004)	-.003 (.004)	-.003 (.004)	-.02*** (.00)	-.02*** (.00)	
Age ² at Time 2				.0004*** (.0001)	.0004*** (.0001)	
Intercept	-.61	-.60	-.60	-1.36	-1.36	
Adjusted R ²	.21	.22	.24			
σ_u				1.28	1.28	
σ_e				1.11	1.11	

Variables	OLS				FE	
	Model 1	Model 2	Model 3	Model 4	Model 4	Model 5
ρ					.57	.57
N of observations	8,054	8,054	8,054	16,108	16,108	16,108
N of groups				8,054	8,054	8,054

Notes:

- # $p < .10$
- * $p < .05$
- ** $p < .01$
- *** $p < .001$.

Standard errors are robust to clustering of observations within families. All models adjust for education, employment, pension plan, health insurance, occupation, occupational education, occupational income, net worth, marital status, number of children, and minor children.

Table 3

Propensity Score Matching Estimates of the Relationship between Cancer and Depressive Symptoms at T2

	Propensity Score Matching Methods			
	Nearest Neighbor		Kernel	
	Men	Women	Men	Women
The effect of being diagnosed with cancer compared to not having cancer (Average treatment effect for the treated)	.33** (.11)	.09 (.09)	.31*** (.06)	.07 (.07)
Treatment observations (persons with cancer)	426	527	426	527
Control observations (matched controls without cancer)	355	447	3,594	4,203

Notes:

*
p < .05**
p < .01***
p < .001.

Cancer patients and controls are matched on the following Time 1 variables: graduate or sibling status, time in months between the baseline and follow-up interviews, baseline depressive symptoms, age, age², gender, gender × age, family history of cancer, education, employment status, net worth, occupation, occupational education, occupational income, marital status, number of children, the presence of children 18 or younger, smoking, alcohol consumption, body mass index, physical activity level, and early-life characteristics (family structure, number of siblings, father's education, farm origin, parental income, and religious affiliation).

Unstandardized Regression Coefficients from Longitudinal Ordinary Least Squares (OLS) Models Estimating the Effects of Cancer on Mental Health, by Cancer Site and Gender

Table 4

Variables	Model 1		Model 2		Model 3		Model 4	
	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398
<i>Change in Cancer:</i>								
No cancer at T1 and T2 (reference group)								
Cancer at T1, no cancer at T2	-.06 (.19)	.29 (.16)	-.15 (.18)	.16 (.15)	-.16 (.18)		-.17 (.18)	
No cancer at T1, cancer at T2, by site:								
Breast	—	-.01 (.09)	—	-.05 (.09)	—		—	
Genitourinary	.33*** (.09)	.23 (.14)	.17* (.08)	.21 (.13)	.17* (.08)		.22* (.11)	
Colon	-.19 (.27)	.22 (.31)	-.32 (.24)	.12 (.27)	-.32 (.24)		-.36 (.32)	
Digestive, respiratory, bone, skin, connective tissue	.46* (.21)	.46 (.25)	.22 (.20)	.23 (.22)	.23 (.20)		.23 (.26)	
Lymphatic and hematopoietic tissue	.20 (.33)	.18 (.36)	.04 (.32)	-.27 (.35)	.04 (.32)		.21 (.36)	
Other cancer	.41* (.18)	-.31 (.36)	.31 (.17)	-.45 (.32)	.28 (.16)		.34 (.20)	
Cancer at T1 and T2, by site:								
Breast	—	.12 (.14)	—	.07 (.13)	—		—	
Genitourinary	.76** (.24)	-.03 (.31)	.48* (.25)	-.13 (.29)	.48* (.25)		.25 (.31)	
Colon	1.24* (.65)	.38 (.50)	.37 (.29)	.19 (.56)	.39 (.32)		.33 (.46)	
Digestive, respiratory, bone, skin, connective tissue, lymphatic tissue	.23 (.54)	.15 (.22)	-.07 (.44)	-.12 (.17)	-.06 (.38)		-.34 (.47)	
Other cancer	.78* (.32)	.29 (.29)	.49* (.24)	.22 (.25)	.46* (.22)		.44 (.29)	

Variables	Model 1		Model 2		Model 3		Model 4	
	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398	Men n = 3,656	Women n = 4,398
Depressive symptoms at T1	.45*** (.02)	.49*** (.03)	.35*** (.02)	.37*** (.01)	.35*** (.02)	.36*** (.02)		
Age at T2	-.01 (.01)	.01 (.01)	-.01* (.01)	.01 (.01)	-.02* (.01)	-.01* (.00)		
<i>Comorbidity and Symptoms at T2:</i>								
Number of chronic illnesses (excluding cancer)			.05** (.01)	.04*** (.01)	.02 (.01)	.02 (.02)		
Number of physical symptoms			.30*** (.02)	.24*** (.02)	.27*** (.02)	.27*** (.02)		
<i>Pain and Functional Limitations at T2:</i>								
Pain interfered with activities			.35*** (.03)	.11*** (.02)	.25*** (.03)	.28*** (.04)		
Needed help with ADLs			.08 (.19)	.10 (.08)	.16 (.18)	.13 (.22)		
Received care			-.17 (.13)	.07 (.08)	-.13 (.12)	-.12 (.15)		
Number of days in bed			.01* (.00)	.01 (.01)	.01* (.00)	.01 (.01)		
Limited physical activity			.10* (.05)	.03 (.04)	.02 (.05)	-.01 (.06)		
Accomplished less			.17* (.07)	.13* (.06)	-.06 (.07)	-.05 (.08)		
<i>Sexuality:</i>								
Sex frequency					.02 (.02)			
Sex physically pleasurable					-.14*** (.02)			
Decrease in sexual activity					.01 (.03)			
Masculinity Scale						.32*** (.07)		
Constant	.10	-.97	-.95	-1.79	-.47	-1.83		
Adjusted R ²	.20	.22	.29	.30	.31	.33		

Notes:

* $p < .05$,
 ** $p < .01$,
 *** $p < .001$.

Standard errors robust to clustering of observations within groups are given in parentheses. All models adjust for baseline education, employment, pension plan, health insurance, occupation, occupational education and occupational income, SEI, net worth, marital status, number of children under 18. Model 3 includes flags for men who refused to answer at least one of the sexuality items.