Higher-Status Occupations and Breast Cancer: A Life-Course Stress Approach

Tetyana Pudrovska
Department of Sociology & Crime, Law, and Justice, Population Research Institute,
Pennsylvania State University,
tup3@psu.edu

Deborah Carr
Department of Sociology, Rutgers University
carrds@sociology.rutgers.edu

Michael McFarland
Center for Research on Child Wellbeing, Office of Population Research, Princeton University
mjm11@exchange.princeton.edu.

Caitlyn Collins
Department of Sociology, University of Texas-Austin
caitlyn.collins@utexas.edu.

Higher-Status Occupations and Breast Cancer: A Life-Course Stress Approach

ABSTRACT

Using the 1957-2011 data from 3,682 White non-Hispanic women (297 incident breast cancer cases) in the Wisconsin Longitudinal Study, United States, we explore the effect of occupation in 1975 (at age 36) on breast cancer incidence up to age 72. Our study is motivated by the paradoxical association between higher-status occupations and elevated breast cancer risk, which presents a challenge to the consistent health advantage of higher social class. We found that women in professional occupations had 72%-122% and women in managerial occupations had 57%-89% higher risk of a breast cancer diagnosis than housewives and women in lower-status occupations. We explored an estrogen-related pathway (reproductive history, health behaviors, and life-course estrogen cycle) as well as a social stress pathway (occupational experiences) as potential explanations for the effect of higher-status occupations. The elevated risk of breast cancer among professional women was partly explained by estrogen-related variables but remained large and statistically significant. The association between managerial occupations and breast cancer incidence was fully explained by job authority defined as control over others' work. Exercising job authority was related to higher breast cancer risk (HR = 1.57, 95% CI: 1.12, 2.18), which accumulated with longer duration in authority positions. We suggest that the assertion of job authority by women in the 1970s involved stressful interpersonal experiences that may have promoted breast cancer development via prolonged dysregulation of the glucocorticoid system and exposure of the breast tissue to adverse effects of chronically elevated cortisol. Our study emphasizes complex biosocial pathways through which women's gendered occupational experiences become embodied and drive forward physiological repercussions.

Higher-Status Occupations and Breast Cancer: A Life-Course Stress Approach

INTRODUCTION

Lower morbidity associated with higher socioeconomic status (SES) is one of the most consistent findings in social epidemiology (Elo, 2009), yet one paradox persists: the elevated risk of breast cancer among women holding higher-status occupations. Higher-status occupations are defined as professional and managerial occupations that are at the top of the U.S. Census classification system and are characterized by the highest levels of socioeconomic prestige indexes (Stevens & Cho, 1985). Women in professional and managerial occupations have 1.4-2.0 times greater risk of breast cancer diagnosis than women in lower-status occupations (Danø et al., 2003; Larsen et al., 2011; Pukkala et al., 2009). Moreover, the effect of higher-status occupations on breast cancer risk is only partly explained by reproductive histories, exogenous hormones, health behaviors, and socioeconomic differences in screening mammography (Danø et al., 2004; Larsen et al., 2011; Sprague, Trentham-Dietz, & Burnside, 2010).

Because women with more socioeconomic resources are advantaged in terms of nearly all other health outcomes (Elo, 2009), the breast cancer risk associated with higher-status occupations presents a paradox that calls for particular attention to this disease. Existing research, however, is limited in several respects. Most studies were based on cross-sectional analyses of breast cancer rates across occupations or had a relatively short follow-up that did not capture the long latency period of breast cancer. Further, past studies considered a limited set of mediators potentially linking occupation to breast cancer and focused overwhelmingly on estrogen-related pathways, such as reproductive histories. Because estrogen-related factors explain only part of the excess risk for breast cancer associated with higher-status occupations (Danø et al., 2004; Larsen et al., 2011), researchers need to continue the search for explanatory mechanisms, especially psychosocial stressors to which women in higher-status occupations are exposed because of the structural and cultural constraints of a gendered workplace (Ridgeway, 2001).

Using the 1957-2011 data from the Wisconsin Longitudinal Study (WLS), we explore the effect of occupation in 1975 (at age 36) on breast cancer incidence up to age 72. Our study extends previous research in several important ways. A long follow-up captures a time lag between exposures in higher-status occupation and breast cancer onset. Because the WLS includes women's lifetime occupational histories, we explore not only the effect of occupation at a given time point but also the effect of the duration of occupational exposures. In addition, the WLS collected extensive information on job characteristics in 1975, which enables us to uncover specific aspects of higher-status occupations that are related to breast cancer.

Life-Course Mechanisms Linking Occupation and Breast Cancer

Biopsychosocial factors affecting the development of chronic conditions operate across the life course (Ben-Shlomo & Kuh, 2002). Consequently, the etiology of chronic diseases cannot be fully understood without incorporating earlier circumstances, in particular, exposures to health-related stressors (Pearlin, Schieman, Fazio, & Meersman, 2005). We adopt a life-course approach and explore how occupational experiences in young adulthood are related to breast cancer incidence over a 36-year period. To understand this relationship, we focus on two mechanisms – estrogen-related processes and social stress – that are not mutually exclusive and may supplement each other in explaining the effect of occupation.

A traditional approach to the etiology of breast cancer focuses on ovarian hormones, especially, estrogen (Kelsey, 1993). Influences that increase cumulative lifetime exposure to estrogen are considered important risk factors for breast cancer. Among these factors are reproductive history (later age at first birth and lower parity), health behaviors (regular alcohol use, sedentary lifestyle, and obesity for post-menopausal cancers), and components of the life-course estrogen cycle, including early age at menarche, late age at menopause, and hormone replacement therapy (Boyle & Boffetta, 2009; Friedenreich & Cust, 2008; Kelsey, 1993; Reeves et al., 2007; Vogel, 2008). Empirical studies highlight the importance of adopting a life-course approach to estrogen-related factors. For example, obesity increases the risk of post-menopausal breast cancer while decreasing the risk of pre-menopausal breast cancer (Reeves et al., 2007). Moreover, adiposity in early life has a long-term effect on breast cancer risk and is *inversely* related to the disease risk decades later (Sangaramoorthy et al., 2011).

Recently researchers have become interested in the social stress pathway to breast cancer and explored the prolonged exposure to steroid hormones produced by the adrenal cortex – glucocorticoids (GCs) – as an underlying physiological mechanism (Antonova et al., 2011; McClintok et al., 2005). The effect of chronically elevated GCs, such as cortisol, is mediated by the activation of the glucocorticoid receptor (GR). GR is ubiquitously expressed in human breast tissue both in normal epithelium and cancerous cells (Antonova et al., 2011; McClintok et al., 2005). GR activation can directly promote mammary cell proliferation and inhibit apoptosis, which increases the risk of malignant transformations (Hermes et al., 2009).

Our study focuses on a cohort of women for whom particular types of employment presented exposure to a range of daily stressors. Participants in the WLS were born in 1939, launched their work and family trajectories in the 1950s and 1960s, and were the first cohort of White educated women to join the labor force in fairly large numbers (U.S. Census, 1970). U.S. women in professional and managerial occupations in the 1960s and 1970s faced socially structured stressors associated with gender stratification and cultural scripts of gender-appropriate behaviors (Kanter, 1977; Roussell, 1974). We consider the stress of female authority in managerial occupations and the stress of caring in professional occupations as gendered stress processes that can increase breast cancer risk via prolonged exposure of breast tissue to the anti-apoptotic and proliferative effects of chronically elevated cortisol.

The Stress of Female Authority

Women of the WLS cohort who entered managerial occupations in the 1970s experienced prejudice and discrimination due to prevailing cultural attitudes that men made better leaders than women (Bartol, 1974; Kanter, 1977; Roussell, 1974). Neither men nor women preferred to work for a woman because women were seen as “temperamentally unfit” for management, which was consistent with the cultural stereotype of the woman boss as petty, controlling, and interfering (Bartol, 1974; Kanter, 1977). Roussell (1974) showed that high school departments headed by men were perceived as high in morale, whereas departments headed by women were perceived as high in “hindrance” – an indicator that the leader was seen as getting in the way of subordinates’ interests. Women in authority positions across a range of workplace settings found themselves socially isolated from subordinates and superiors and were more likely than men to report lack of communication and support from superiors and co-workers (Kanter, 1977; Roussell, 1974). Taken together, these findings suggest that authority positions exposed women to interpersonal tension and negative social interactions in the workplace (Korabik, 1995; Roussell, 1974).

The Stress of Caring

Traditional gender expectations in the 1950s and 1960s constrained career choices of highly educated women to primarily gender-appropriate areas, mostly teaching and nursing. In the U.S. in 1970, 25% of all professional women were nurses and 39% were teachers (U.S.

Census, 1970). The proportions in our study are very similar, with 31% of professional women employed as nurses and 39% as teachers in 1975. Employees in caring occupations are required to act in their clients' best interests and work in close contact with care recipients (Barron & West, 2007). Many workers feel responsible for clients' well-being, which may lead to emotional and physical exhaustion, distress, and the inability to withdraw from work obligations (Barron & West, 2007). Moreover, workers in caring occupations perform emotional labor, which involves an expression of empathy and comfort as well as the suppression of negative feelings (Barron & West, 2007; Hochschild, 1983). Not only is emotional labor one of the major causes of occupational stress (Pugliesi, 1999), but also women are more psychologically and physically vulnerable than men to the adverse consequences of emotional labor and suppression (Barron & West, 2007).

In sum, the central argument of our study is that, in addition to estrogen-related factors, higher incidence of breast cancer among higher-status women may be explained by gendered occupational experiences. To evaluate the salience of the social stress pathway, we explore (a) whether women in professional and managerial occupations at age 36 had a higher breast cancer risk over the next 36 years relative to women in lower-status occupations and housewives, (b) whether the risk associated with higher-status occupations accumulated with longer duration of psychosocial workplace exposures, and (c) the relative importance of the estrogen pathway and the social stress pathway as explanations for the effect of higher-status occupations on breast cancer risk.

METHODS

The Wisconsin Longitudinal Study (WLS) is a long-term study of a random sample of men and women who graduated from Wisconsin high schools in 1957. Participants were interviewed in 1957 (5,326 women), 1975 (4,808 women), 1993 (4,513 women), and 2004 (3,792 women). The WLS sample retention is very high: 71% of women from the baseline sample participated in the 2004 wave, with vital status known for 94% of the original sample. Deceased participants were matched to the National Death Index (NDI) to ascertain the cause of death and age at death. Because the type of cancer was reported only in 2004, our analytic sample comprises 3,682 women including (1) women who participated in 1975, 1993, and 2004 (both alive and deceased as of 2011), and (2) women who died of breast cancer any time after 1975 as established via the NDI. We conducted a detailed analysis of sample attrition and created two selection instruments based on the propensity score approach to adjust for potential selection bias, as described in the Methodological Appendix, Part A.

Measures

The binary indicator of *breast cancer incidence* is coded 1 for all women diagnosed with breast cancer (alive and deceased) and 0 for women without breast cancer. We used two sources of information about breast cancer. First, women reported whether they had ever been diagnosed with breast cancer by a medical professional. Second, incident breast cancer cases include women who died of breast cancer before reporting it in the study. Out of the 297 incident breast cancer cases, 222 women were alive and 75 women were deceased as of 2011.

We considered two types of bias with respect to our measure of breast cancer. Type 1 bias may arise if some women who dropped out of the study were diagnosed with breast cancer but died of another cause. In this scenario, these breast cancer cases were neither reported in the study nor reflected in women’s death certificates. This proportion is likely to be very small and should be largely accounted for by our analysis of sample selection bias described in the Methodological Appendix, Part A. Type 2 bias may arise if a woman’s occupation affected her self-report of breast cancer. Results from Monte Carlo simulations described in the Methodological Appendix, Part B, suggest that our results are unlikely to be affected by this bias.

Occupation in 1975. Women reported their occupation and employer for every job spell held over the life course; these open-ended reports were assigned a three-digit Census occupational code. About 995 unique occupations were combined into major occupation categories following the U.S. Census classification system. Some of these major categories were further combined into broader groups to produce the final set of categories used in our analysis based on three-digit 1970 Census codes: housewife (985); professional (001-195); managerial (201-245); clerical, sales, service (260-395, 901-954, 980-984); crafts, operatives, laborers (401-824). Women reported the start and end dates of each job held between high school and retirement. A measure of *job duration* reflects a number of years that a woman spent in the job reported during the 1975 interview.

Job characteristics in 1975. The number of *hours worked per week* is represented with four dummy variables: fewer than 20, 20-29, 30-39, and 40 or more. The frequency of working under *time pressure* is coded from 1 = never to 5 = always. Women's *responsibility for things at work outside of her control* is coded as 1 = never, 2 = rarely, 3 = some, 4 = frequently. High *job autonomy* is coded 1 if a woman reported not being supervised at work. An indicator of *job satisfaction* reflects the extent to which a woman was satisfied with her 1975 job coded from 1 = very dissatisfied to 4 = very satisfied. *Job authority* is measured with four items: "Do you have authority to hire and fire others?" "Can you influence pay received by others?" "Do you supervise the work of others?" "Do you decide what others do and how they do it?" This variable is dichotomized into high job authority (2+ tasks) and low job authority (0-1 tasks). All measures of job characteristics have been widely used in social sciences, and their validity and reliability are well-documented (Jencks, Perman, & Rainwater, 1988; Wolf & Fligstein, 1979).

Estrogen-related variables. Reproductive history in 1975 and 1993 is represented with a variable coded 1 if a woman *had at least one child*, *age at first birth*, and the *number of biological children*. Facial *adiposity in adolescence* was coded from pictures in 1957 high school yearbooks by six coders on a scale ranging from 1 (the lowest) to 11 (the highest). The scale has very high reliability and validity (Reither et al., 2009). Body mass index (BMI) in 1993 is categorized as *healthy weight* (BMI < 25), *overweight* (BMI 25 - 29.9), and *obese* (BMI ≥ 30). Alcohol use in 1993 reflects the *number of days on which a woman consumed alcohol* in a month prior to the interview and the *number of drinks for each day* on which alcohol was consumed. Participation in *light exercise* (such as walking) and *vigorous exercise* (such as jogging) in 1993 are coded from 1 = less than once a month to 4 = 3 or more times a week. The *life-course estrogen cycle* is reflected with ages at menarche and menopause, hysterectomy/oophorectomy (1 = yes), and hormone replacement therapy ever (1 = yes).

Control variables include *education* (in years), logged household annual *income,* and *marital status* (1 = married). *Family history of breast cancer* is coded 1 for women who reported that their mother or sister was diagnosed with breast cancer.

Our variables have 2%-3% of missing values on average. Multiple imputation analysis was carried out in Stata 12.1 using two imputation models. The first model (for the analysis presented in Table 2) included all variables from Table 2. The second model (for the analysis presented in Table 3) included all variables from Table 3. Both imputation models also included a binary indicator of a breast cancer diagnosis, the cumulative baseline hazard, and characteristics of family background in 1957 described in the Methodological Appendix, Part A.

Five completed data sets were generated under each imputation model, the survival analysis was conducted separately on each data set, and five sets of results were pooled into a single multiple-imputation inference.

Analytic Plan

We begin by comparing means/proportions for all study variables by 1975 occupation categories (Table 1). To estimate the effect of occupation on breast cancer, we use a semi-parametric Cox survival model. The hazard function for woman *i* at time *j* is modeled as:

$$h(t_{ij}) = h_0(t_j)exp(\boldsymbol{X}_{ij}'\boldsymbol{\beta} + \boldsymbol{Z}_i'\boldsymbol{\gamma}) \qquad (1)$$

where $h(t_{ij})$ is the hazard of breast cancer incidence evaluated at exact age t, h_0 is a nonparametric baseline hazard, $\boldsymbol{\beta}$ and $\boldsymbol{\gamma}$ are vectors of parameters containing the effects of variables on the breast cancer hazard, $\boldsymbol{X}_{ij}$ is a vector of occupation categories, and $\boldsymbol{Z}_i$ is a vector of mediating and control variables. The test of the proportionality assumption indicates that the effect of each occupation category is constant over time, thus, satisfying the assumption.

Table 2 shows results from models predicting breast cancer incidence for women diagnosed after 1975. Table 3 presents findings for women diagnosed after 1993 because health behaviors and certain other estrogen-related variables were reported for the first time only in 1993. To explore a potential accumulation of the effects of occupational exposures with duration, we centered the number of years that each woman spent in her 1975 job at the median (8 years), limited our sample to women in professional and managerial occupations, and estimated an interactive effect of job authority and job duration. To illustrate a significant interaction term, we plot predicted hazard functions (Figure 1).

Finally, we apply a decomposition technique for a formal test of mediation in a survival setting (VanderWeele, 2011) to examine the extent to which estrogen-related and social stress variables mediate the effect of occupation. If we denote the focal predictor as X, the mediator as M, and a vector of control variables as **Z**, then

$$log\ h(t|X, M, \mathbf{Z}) = log(h_0) + \gamma_1 X + \gamma_2 M + \gamma_3 \mathbf{Z} \qquad (2)$$

Further, if we denote the effect of *X* on *M* as β_1, the total effect of the focal predictor reflecting a change in the log hazard of breast cancer with a one-unit change in the predictor from X to X* can be decomposed into direct and indirect effects as follows:

$$\underbrace{\log h(t, X, M) - \log h(t, X^*, M^*)}_{\text{Total effect}} = \underbrace{[\gamma_1(X - X^*)]}_{\text{Direct effect}} + \underbrace{[\gamma_2\beta_1(X - X^*)]}_{\text{Indirect effect}} \qquad (3)$$

RESULTS

Descriptive Analysis

Table 1 shows that compared to housewives, a significantly greater proportion of women in professional and managerial occupations were diagnosed with breast cancer (.095 and .145, respectively, $p < .001$). In contrast, women in blue-collar occupations had a significantly lower prevalence of breast cancer (.051, $p < .05$), whereas women in clerical, sales, and service occupations were similar to housewives in terms of breast cancer.

With respect to job characteristics in 1975, women in managerial occupations had the highest job autonomy but also worked longer hours and were more frequently held responsible for things outside their control. Professional and managerial women worked under time pressure more frequently but also reported higher job satisfaction than lower-status women. Women in professional occupations were less likely than managerial women but more likely than lower-status women to supervise others and decide what/how others did at work. Women in managerial occupations were much more likely than all other women to have high job authority (72%), although job authority was still more prevalent among professional women (27%) than women in lower-status occupations. Finally, lower-status women spent about two fewer years in their 1975 job than professional and managerial women.

With respect to estrogen-related variables, women in higher-status occupations had several characteristics of an unfavorable profile of breast cancer risk: later age at first birth, lower parity, more regular use of alcohol, lower adiposity in adolescence, and, among professional women only, later age at menopause and higher use of hormone replacement therapy. Yet, two characteristics reflected a favorable risk profile: higher physical activity (professional women only) and a lower risk of obesity in midlife.

[TABLE 1 about here]

Survival Analysis

Model 1 in Table 2 reveals that, compared to housewives, women in professional occupations had 72% higher risk (HR = 1.72, 95% CI: 1.25, 2.36) and women in managerial occupation had 57% higher risk (HR = 1.57, 95% CI: 1.02, 2.42) of a breast cancer diagnosis after 1975. In contrast, women in lower-status occupations were similar to housewives in terms of breast cancer risk.

[TABLE 2 about here]

Models 2 and 3 explore the estrogen-related pathway. As indicated in Model 2, higher adiposity in adolescence was associated with a lower breast cancer risk (HR = .82, 95% CI: .71, .95), yet the effect of higher-status occupation changed only trivially compared to Model 1. Model 3 includes reproductive history and shows that nulliparity and later age at first birth increased the risk of breast cancer. The mediation decomposition analysis reveals that reproductive variables mediate 23% of the association between professional occupations and breast cancer hazard, and this mediating effect is significant at the .01 level. Yet, the elevated breast cancer risk of professional women remained statistically significant and large in magnitude (HR= 1.59, 95% CI: 1.15, 2.20). In contrast, adjustment for reproductive variables does not change the effect of managerial occupations.

Models 4 and 5 evaluate the social stress pathway. Weekly work hours, time pressure, responsibility for things at work outside own control, job autonomy, and job satisfaction are not significantly related to breast cancer incidence (Model 4). In a sensitivity analysis we estimated the effect of each job characteristic separately and found that none of them was significant even without other variables in the model. Interestingly, the effect of higher-status occupations even increases compared to Model 3 because responsibility for things outside one's own control, time pressure, and job satisfaction are more prevalent among higher-status women and are related negatively (although not significantly) to breast cancer risk.

When job authority is added in Model 5, breast cancer risk associated with managerial occupations declines and becomes not significant (HR = 1.42, 95% CI: .87, 2.30). Women in managerial occupations had higher job authority than other women. In turn, high job authority is associated with a 1.57 greater hazard of breast cancer than low authority (95% CI: 1.12, 2.18). The decomposition analysis indicates that job authority mediates 55% of the relationship between managerial occupations and breast cancer incidence. This mediating effect of job authority is significant at the .001 level. In contrast to women in managerial occupations, job authority does not explain the elevated risk of breast cancer among professional women likely because their jobs involved less authority. Only 27% of professional women reported high levels of job authority compared to 72% of women in managerial occupations (Table 1). Job authority mediates 12% of the effect of professional occupations on breast cancer hazard, and this indirect effect is not statistically significant.

We explored the accumulation of breast cancer risk with duration of occupational exposures and found a significant interactive effect between job authority and duration in the 1975 job. Figure 1 shows that the effect of job authority on the breast cancer hazard accumulates with longer duration among professional and managerial women who had high authority ($HR_{job\ authority \times duration}$ = 1.15, 95% CI: 1.06, 1.27). For example, women with high job authority who worked for 15 years in this job had a significantly greater risk of breast cancer than women with similar duration but low job authority. Women with low job authority and short job duration (5 years) had the lowest hazard.

[FIGURE 1 about here]

Table 3 presents results from models based on women diagnosed with breast cancer after 1993, thus, excluding 36 women. Model 1 indicates that the effect of professional (HR = 2.22, 95% CI: 1.55, 3.19) and managerial (HR = 1.89, 95% CI: 1.18, 3.05) occupations becomes even stronger when women with a diagnosis before age 54 are excluded. Model 2 confirms that, consistent with Model 5 of Table 2, the effect of managerial occupations is reduced substantially and becomes not significant after adjustment for job authority, whereas the effect of professional occupations does not change.

Model 3 includes reproductive history and characteristics of life-course estrogen cycle. Later age at first birth, later age at menopause, and hormone replacement therapy are related to a higher risk of breast cancer. The decomposition analysis indicates that 20% of the effect of professional occupations is conveyed indirectly via these estrogen-related variables ($p < .01$). The effect of managerial occupations, already not significant in Model 2, was not altered after adjustment for estrogen-related variables. Job authority and estrogen-related variables are both significant predictors of breast cancer net of each other, which emphasizes the additive effects of these two mechanisms. Finally, Model 4 indicates that higher adiposity in 1957 reduces breast cancer risk, whereas health behaviors in 1993 are unrelated to post-1993 breast cancer. The effects of higher-status occupations changed only trivially after adjustment for all estrogen-related health behaviors in Model 4.

[TABLE 3 about here]

DISCUSSION

Drawing on a life-course biosocial stress framework, our study documents long-term effects of higher-status occupations on women's elevated risk of a breast cancer diagnosis. We find that women who were in professional and managerial occupations in 1975 at age 36 had a substantially higher risk of a breast cancer diagnosis up to age 72 compared to housewives and women in lower-status occupations. To explain the long-reaching effect of occupation in 1975, we assess estrogen-related and social stress explanations and find that these mechanisms have additive effects, which points to their complementary nature in explaining the elevated breast cancer risk of professional and managerial women.

With respect to the estrogen-related pathway, about 20% of the elevated breast cancer risk among professional women was explained by their later age at first birth, lower parity, more regular alcohol use, higher use of hormone replacement therapy, and later menopause. Yet, the effect of professional occupations remains large and significant net of estrogen-related variables. Moreover, estrogen variables have little effect on the association between managerial occupations and breast cancer risk. These patterns are consistent with other studies that show that the effect of higher-status occupation decreases only modestly and remains large in magnitude and statistically significant after adjustment for reproductive histories and other estrogen-related variables (Danø et al., 2004; Larsen et al., 2011).

With respect to the social stress pathway, our findings point to women's job authority as a potentially important source of job-related stress (Schieman & Reid, 2009). Job authority mediated 55% of the effect of managerial occupations on breast cancer risk, which reduced the direct effect of managerial occupations to non-significance. Women in managerial occupations had higher job authority than other women. In turn, high job authority is associated with a 1.55 higher risk of breast cancer than low authority, net of all estrogen-related and control variables. Further, our findings reveal that the risk of breast cancer associated with job authority accumulates with longer duration. This pattern points to the importance of *chronic* stress resulting in gradual accumulation of deleterious exposures and incremental transformations of bodily systems (Ben-Shlomo & Kuh, 2002; Pearlin et al., 2005). Consistent with the life course perspective, we emphasize the concept of duration (Pearlin et al., 2005) and show how social risk factors for breast cancer operate over extended periods of time resulting in the compounding of cumulative damage with the longer exposure to stressors.

Exercising job authority was particularly stressful for women in the context of gender inequality embedded in the occupational structure of the 1960s and 1970s, when women in managerial positions often faced prejudice, tokenism, discrimination, social isolation, and resistance from subordinates, colleagues, and superiors (Rousell, 1974; Kanter, 1977). This chronic stress may have been an important long-term link between higher-status jobs in young adulthood and the risk of breast cancer. Animal models suggest potential mechanisms through which interpersonal stress of higher status is related to chronic hyperactivity of glucocorticoid responses. Among female rats, social isolation and disruption of supportive ties increase the risk of developing mammary tumors fourfold, with the primary mechanism being dysregulation of the GC system (McClintok et al., 2005). In young adulthood, months before tumor initiation, socially isolated rats developed an enhanced GC response to stressors with markedly delayed reduction of corticosterone to normal levels. Both aspects of this stress reactivity process were related to an increased risk of mammary tumors in middle and old age (Hermes et al., 2009).

Further, studies among baboons suggest that in some contexts, dominant individuals exhibit an unfavorable profile of GC hyperactivity. Cortisol dysregulation among higher-status primates is associated with low social control, lack of social support, and situations where dominant individuals have to repeatedly reassert their rank (Gesquiere et al., 2011). This scenario is consistent with interpersonal tension and resistance experienced by incumbents of authority positions who are not perceived as legitimate (Ridgeway, 2001). Thus, the GC-related effects of higher status may have also extended to this cohort of women in managerial occupations. These mechanisms remain speculative in our study because we do not have direct measures of women's perceptions of gender discrimination or biomarkers of the GC system. We view our findings as the first step suggesting that it is worthwhile to explore women's experiences in higher-status occupations with respect to breast cancer and a call for more attention to stressful occupational exposures as explanations for the puzzling effect of managerial occupations.

Our findings provide indirect support for the stress of caring model among professional women. The effect of professional occupations on the elevated risk of breast cancer was not explained by all estrogen-related variables and job characteristics available in our study. Because professional women were predominantly employed in caring occupations, mostly teaching and nursing, characteristics reflecting the stressful side of carework may be an important underlying mechanism. Emotional suppression and emotional exhaustion are two aspects of caring higher-status occupations that are not measured in our study but can be related to breast cancer through the GC pathway. Women who regularly engage in emotion suppression as an emotion regulation strategy in their everyday lives exhibit heightened GC response to social stress compared to women low on emotional suppression (Lam, Dickerson, Zoccola, & Zaldivar, 2009). Similarly, emotional exhaustion among women teachers is associated with higher plasma cortisol levels (Wolfram et al., 2012). These findings suggest that chronic strains of caring occupations are likely to be associated with systematically elevated GCs, and the cortisol mechanism in human breast cancer etiology is a promising direction for future research (McClintock et al., 2005).

Limitations and Future Directions

Our study has several important limitations. The analyses are based on one cohort of women who were born in 1939; thus, our findings may be most relevant to women born in the first half of the 20th century. Yet, because of the persistence of gender inequality and dominant gender beliefs, our study is still likely to reflect experiences of women in current and future cohorts (Ridgeway, 2001). Further, the WLS contains only White non-Hispanic participants. Conditions of minority women in higher-status positions may be even more stressful than those of White women. Recent biological research suggests that the cortisol-mediated effects of social stress on breast cancer may be stronger among Black women compared to White women (McClintok et al., 2005). An important step for future research is to explore race differences in health implications of higher-status occupations.

Some estrogen-related variables were not measured in our study, in particular, oral contraceptives and breastfeeding. Evidence for the role of these two variables in breast cancer etiology is mixed and varies by context and duration (Kelsey, 1993; Nichols et al., 2007). Moreover, breastfeeding rates were very low in the 1970s among women of all social classes (Wolf, 2003). Although it is unlikely that inclusion of these variables would substantially alter our findings, it is still important to incorporate a wide range of indicators of life-course estrogen cycle to assess more precisely their relative importance compared to the social stress pathway. Further, the WLS did not collect information on health behaviors in 1975. Therefore, our conclusions about the lack of explanatory power of health behaviors in midlife (in 1993) are limited because lifestyle assessed contemporaneously with occupation in 1975 may have been more consequential for breast cancer than behaviors in midlife.

In this study we focus on chronic stress and do not include acute stressful life events, such as loss of loved ones or disease in the family, which can have potential – albeit modest – effects on breast cancer (Kruk & Aboul-Enein, 2004). For a more nuanced understanding of the social stress pathway, future research should distinguish between chronic strains and stressful events (Pearlin et al., 2005). Moreover, a myriad of other aspects of higher-status jobs are not measured in the WLS. We could not include measures of emotional labor, emotional suppression, gender discrimination, women's actual relationships with co-workers and superiors, and women's perceptions of social support and social strain at work in 1975. Yet, we are not aware of any data set that combines the strengths of the WLS with more comprehensive measures of stressful experiences in higher-status occupations. Moreover, it is impossible to document all pathways linking social stress to breast cancer in one study because these stress processes operate on different levels: social, psychological, physiological, and molecular. Therefore, important directions for future research include developing interdisciplinary collaborations, integrating multiple levels of analysis, and collecting longitudinal data on chronic strains and biomarkers among women in higher-status occupations.

REFERENCES:

Antonova, L., Aronson, K., & Mueller, C. (2011). Stress and breast cancer: From epidemiology to molecular biology. *Breast Cancer Research, 13,* 208.

Barron, D. N., & West, E. (2007). The emotional costs of caring incurred by men and women in the British labor market. *Social Science & Medicine, 65,* 2160-2171.

Bartol, K. (1974). Male versus female leaders: The effect of leader need for dominance on follower satisfaction. *The Academy of Management Journal, 17,* 225-233.

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology. *International Journal of Epidemiology, 31,* 285-293.

Boyle, P., & Boffetta, P. (2009). Alcohol consumption and breast cancer risk. *Breast Cancer Research, 11,* S3.

Danø, H., Andersen, O., Ewertz, M., Petersen, J., & Lynge, E. (2003). Socioeconomic status and breast cancer in Denmark. *International Journal of Epidemiology, 32,* 218-224.

Danø, H., Hansen, K. D., Jensen, P., Petersen, J. H., Jacobsen, R., Ewertz, M., & Lynge, E. (2004). Fertility pattern does not explain social gradient in breast cancer in Denmark. *International Journal of Cancer, 111,* 451-456.

Doescher, M. P., & Jackson, J. E. (2009). Trends in cervical and breast cancer screening practices among women in rural and urban areas of the United States. *Journal of Public Health Management and Practice, 15,* 200-209.

Elo, I. T. (2009). Social class differentials in health and mortality: Patterns and explanations in comparative perspective. *Annual Review of Sociology, 35,* 553-572.

Fox, M. P., Lash, T. L., & Greenland, S. (2005). A method to automate probabilistic sensitivity analyses of misclassified binary variables. *International Journal of Epidemiology, 34,* 1370-1376.

Friedenreich, C. M., & Cust, A. E. (2008). Physical activity and breast cancer risk: Impact of timing, type and dose of activity and population subgroup effects. *British Journal of Sports Medicine, 42,* 636-647.

Gesquiere, L. Learn, N., Simao, M., Onyango, P., Alberts, C., & Altmann, J. (2011). Life at the top: Rank and stress in wild male baboons. *Science, 333,* 357-360.

Gierisch, J. M., O'Neill, S., Rimer, B., DeFrank, J., Bowling, J. M., & Skinner, C. S. (2009). Factors associated with annual-interval mammography for women in their 40s. *Cancer Epidemiology, 33,* 72-78.

Hermes, G., Delgado, B., Tretiakova, M., Cavigelli, S., Krausz, T., Conzen, S., & McClintock, M. (2009). Social isolation dysregulates endocrine and behavioral stress while increasing malignant burden of spontaneous mammary tumors. *PNAS*, *106*, 22393–22398.

Hochschild, A. R. (1983). *The managed heart: The commercialization of human feeling*. Berkeley, CA: University of California Press.

Jencks, C., Perman, L., & Rainwater, L. (1988). What is a good job? A new measure of labor-market success. *American Journal of Sociology, 93,* 1322-1357.

Kanter, R. (1977). *Men and women of the corporation*. New York: Basic Books.

Kelsey, J. L. (1993). Breast cancer epidemiology. *Epidemiologic Reviews, 15,* 256-263.

Korabik, K. (1995). Gender, social support, and coping with work stressors among managers. *Journal of Social Behavior and Personality, 10,* 135-148.

Kruk, J., & Aboul-Enein, H. (2004). Psychological stress and the risk of breast cancer: A case-control study. *Cancer Detection and Prevention, 28,* 399-408.

Lam, S., Dickerson, S. S., Zoccola, P., & Zaldivar, F. (2009). Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology, 34,* 1355-1362.

Lash, T. L., Fox, M. P., & Fink, A. K. (2009). *Applying quantitative bias analysis to epidemiologic data*. New York: Springer.

Larsen, S. B., Olsen, A., Lynch, J., Christensen, J., Overvad, K., Tjønneland, A., Johansen, C., & Dalton, S. O. (2011). Socioeconomic position and lifestyle in relation to breast cancer incidence among postmenopausal women: A prospective cohort study, Denmark, 1993-2006. *Cancer Epidemiology, 35,* 438-441.

McClintock, M. K., Conzen, S. D., Gehlert, S., Masi, C., & Olopade, F. (2005). Mammary cancer and social interactions. *Journal of Gerontology: Social Sciences, 60B,* 32-41.

Nichols, H. B., Trentham-Dietz, A., Egan, K. M., Titus-Ernstoff, L., Hampton, J. M., & Newcomb, P. A. (2007). Oral contraceptive use and risk of breast carcinoma *in situ*. *Cancer Epidemiology, Biomarkers, & Prevention, 16,* 2262-2268.

Pearlin, L., Schieman, S., Fazio, E., & Meersman, S. (2005). Stress, health, and the life course. *Journal of Health and Social Behavior, 46,* 205-219.

Pugliesi, K. (1999). The consequences of emotional labor: Effects on work stress, job satisfaction, and well-being. *Motivation and Emotion, 23,* 125-154.

Pukkala, E., Martinsen, J. I., Lynge, E., Gunnarsdottir, H. K., Sparén, P., Tryggvadottir, L., Weiderpass, E., & Kjaerheim, K. (2009). Occupation and cancer - follow-up of 15 million people in five Nordic countries. *Acta Oncologica, 48*, 646-790.

Reeves, G. K., Pirie, K., Beral, V., Green, J., Spencer, E., & Bull, D. (2007). Cancer incidence and mortality in relation to body mass index in the Million Women Study: Cohort study. *British Medical Journal, 335,* 1134.

Reither, E, Hauser, R., & Swallen, K. (2009). Predicting adult health and mortality from adolescent facial characteristics in yearbook photographs. *Demography*, *46,* 27-41.

Ridgeway, C. (2001). Gender, status, and leadership. *Journal of Social Issues, 57,* 637-655.

Roussell, C. (1974). Relationship of sex of department head to department climate. *Administrative Science Quarterly, 19,* 211-220.

Sangaramoorthy, M., Phipps, A. I., Horn-Ross, P. L., Koo, J., & John, E. M. (2011). Early-life factors and breast cancer risk in Hispanic women: The role of adolescent body size. *Cancer Epidemiology, Biomarkers & Prevention, 20,* 2572-2582.

Schieman, S., & Reid, S. (2009). Job authority and health: Unraveling the competing suppression and explanatory influences. *Social Science and Medicine, 69,* 1616-1624.

Schueler, K., Chu, P., & Smith-Bindman, R. (2008). Factors associated with mammography utilization. *Journal of Women's Health, 17,* 1477-1498.

Sprague, B. L., Trentham-Dietz, A., & Burnside, E. S. (2010). Socioeconomic disparities in the decline in invasive breast cancer incidence. *Breast Cancer Research and Treatment, 122,* 873-878.

Stevens, G., & Cho, J. (1985). Socioeconomic indexes and the new 1980 Census occupational classification scheme." *Social Science Research, 14*, 142-168.

VanderWeele, T. (2011). Causal mediation analysis with survival data. *Epidemiology, 22,* 582-585.

Vogel, V. G. (2008). Epidemiology, genetics, and risk evaluation of postmenopausal women at risk of breast cancer. *Menopause, 15,* 782-789.

Wolf, J. H. (2003). Low breastfeeding rates and public health in the United States. *American Journal of Public Health, 93,* 2000-2010.

Wolf, W., & Fligstein, N. (1979). Sex and authority in the workplace. *American Sociological Review, 44,* 235-252.

Wolfram, M., Bellingrath, S., Feuerhahn, N., & Kudielka, B. M. (2012). Emotional exhaustion and overcommitment to work are differentially associated with hypothalamus-pituitary-adrenal (HPA) axis responses to a low-dose ACTH(1-24) and dexamethasone-CRH test in healthy school teachers. *Stress and Health*. doi: 10.1002/smi.2439.

Table 1. Summary Statistics for the Study Variables by Occupation in 1975: The Wisconsin Longitudinal Study, 1957-2011, N = 3,682

Variables	Occupation in 1975				
	Housewife	Professional	Managerial	Clerical, sales, service	Crafts, operatives, laborers
Breast cancer diagnosis	.069	.095***	.149***	.061	.051*
Job Characteristics in 1975:					
Hours worked per week		31.26	39.09 c	29.76 a	35.01 c
Long hours (40+)		.47	.65 c	.37 c	.58 c
Frequency of work under pressure of time [d]		2.74	2.78	2.26 c	2.59 c
Responsible for things outside own control [e]		2.43	2.72 c	2.18 c	2.12 c
High job autonomy		.29	.56 c	.32 a	.22 b
Job satisfaction [f]		3.64	3.63	3.34 b	3.45 b
Job authority:					
Hire and fire people		.12	.60 c	.09 b	.11
Influence others' pay		.17	.65 c	.13 a	.17
Supervise others		.58	.77 c	.25 c	.25 c
Decide what/how others do		.53	.73 c	.21 c	.22 c
High job authority (2+ tasks)		.27	.72 c	.15 c	.17 c
Duration in the 1975 job (years)		8.75	8.83	6.51 c	6.75 c
Estrogen-Related Variables:					
Adiposity in 1957 (standardized)	-.01	-.05***	-.04**	.04***	.15***
Reproductive history in 1975:					
At least one birth	.96	.71***	.75***	.83***	.91
Age at first birth	23.01	25.13***	24.38***	22.35***	21.83***
Two or more children	.89	.66***	.65***	.72**	.85
Reproductive history in 1993:					
At least one birth	.98	.79***	.83**	.90	.93
Age at first birth	23.13	26.05***	25.33***	23.39	21.91***
Three or more children	.617	.43***	.48***	.51**	.67
Health behaviors in 1993:					
Healthy weight (BMI < 25)	.37	.43***	.39*	.32*	.29**
Overweight (BMI 25-29.9)	.43	.35***	.39	.48*	.48**
Obese (BMI ≥ 30)	.19	.20	.21	.19	.21
Daily number of drinks	.84	.85	.86	.91*	.83
Days of the month when drinks	5.09	6.11 **	6.58**	4.41*	3.55*
Light physical activity [g]	3.13	3.27**	3.14	3.07*	3.09
Vigorous physical activity [g]	1.82	1.98*	1.80	1.79	1.78

Table 1 (*cont'd*)

Variables	Housewife	Professional	Managerial	Clerical, sales, service	Crafts, operatives, laborers
Life-course estrogen cycle:					
Age at menarche	12.70	12.59	12.65	12.71	12.79
Age at menopause	48.21	48.97***	48.32	47.42***	47.43***
Hysterectomy/Oophorectomy	.32	.28*	.31	.36*	.35
Hormone replacement therapy	.40	.46**	.40	.40	.35*
Family history of breast cancer	.08	.08	.08	.07	.07
Control Variables:					
Education in 1975	12.93	15.02***	14.40***	12.32***	12.27***
Household income in 1975 (in $100's)	152.08	175.16***	181.27***	153.05	126.49***
Married in 1975	.95	.75***	.76***	.84***	.87

Note: Each cell contains means or proportions. Asterisks denote a statistically significant difference between women in a given occupation and housewives: *p < .05. **p < .01. ***p < .001. Significantly different from women in professional occupations: [a] .05 level. [b].01 level. [c].001 level.
[d] 1 = never, 2 = rarely, 3 = some, 4 = frequently, 5 = always.
[e] 1 = never, 2 = rarely, 3 = some, 4 = frequently.
[f] 1 = very dissatisfied, 2 = somewhat dissatisfied, 3 = fairly satisfied, 4 = very satisfied.
[g] 1 = less than once a month, 2 = 1-3 times a month, 3 = once or twice a week, 4 = 3 or more times a week.

Table 2. Cox Hazard Models Predicting Breast Cancer Incidence Based on Occupation in 1975 among Women Diagnosed after 1975 (*N* = 3,682)

Variables	Model 1	Model 2	Model 3	Model 4	Model 5
Occupation in 1975:					
Housewife [a]	1.00	1.00	1.00	1.00	1.00
Professional	1.72*** (1.25, 2.36)	1.74*** (1.26, 2.38)	1.59** (1.15, 2.20)	1.88*** (1.30, 2.72)	1.82*** (1.26, 2.63)
Managerial	1.57* (1.02, 2.42)	1.58* (1.03, 2.44)	1.53* (1.01, 2.37)	1.73** (1.09, 2.74)	1.42 (.87, 2.30)
Clerical, sales, service	.95 (.70, 1.29)	.95 (.71, 1.29)	.92 (.67, 1.25)	1.06 (.75, 1.50)	1.06 (.75, 1.50)
Crafts, operatives, laborers	.83 (.53, 1.29)	.85 (.54, 1.32)	.87 (.56, 1.37)	.97 (.61, 1.57)	.98 (.61, 1.57)
Estrogen-Related Variables:					
Adiposity in 1957		.82** (.71, .95)	.82** (.71, .95)	.82** (.71, .95)	.82** (.71, .95)
Reproductive history in 1975:					
At least one birth			.29* (.08, .95)	.36 (.10, 1.15)	.31 (.10, 1.13)
Birth × Age at first birth			1.06** (1.02, 1.11)	1.05** (1.01 1.09)	1.05** (1.01, 1.09)
Number of children			.95 (.62, 1.46)	.92 (.59, 1.42)	.92 (.59, 1.41)
Job Characteristics in 1975:					
Hours worked per week:					
Less than 20 [a]				1.00	1.00
20-29				1.29 (.83, 2.03)	1.29 (.83, 1.99)
30-39				.94 (.68, 1.31)	.98 (.70, 1.37)
40+				1.04 (.76, 1.43)	1.01 (.73, 1.38)
Work under pressure of time				.94 (.82, 1.06)	.93 (.82, 1.05)
Responsibility outside control				.98 (.85, 1.12)	.96 (.83, 1.09)
High job autonomy				.97 (.73, 1.45)	.98 (.79, 1.59)
Job satisfaction				.95 (.93, 1.01)	.95 (.93, 1.01)
High job authority					1.57** (1.12, 2.18)
Attrition propensity score	1.08 (.90, 1.29)	1.08 (.91, 1.30)	1.08 (.89, 1.29)	1.06 (.89, 1.28)	1.07 (.89, 1.29)

Table 2 (*cont'd*)

Variables	Model 1	Model 2	Model 3	Model 4	Model 5
Model fit:					
Log likelihood (df) [b]	-2448 (7)	-2445 (8)	-2438 (12)	-2434 (20)	-2431 (21)
AIC [c]	4911	4906	4900	4909	4904
BIC [d]	4955	4957	4975	5037	5035

Note: Each cell contains hazard ratios and 95% confidence intervals (in parentheses). All models control for education, household income, and marital status. *p < .05. **p < .01. ***p < .001 (two-tailed test).

[a] Reference group.

[b] df = degrees of freedom.

[c] AIC = Akaike information criterion.

[d] BIC = Bayesian information criterion.

Table 3. Cox Proportional Hazard Models Predicting Breast Cancer Incidence Based on Occupation in 1975 among Women Diagnosed after 1993 (*N* = 3,646)

Variables	Model 1	Model 2	Model 3	Model 4
Occupation in 1975:				
Housewife [a]	1.00	1.00	1.00	1.00
Professional	2.22*** (1.55, 3.19)	2.07*** (1.43, 2.99)	1.89*** (1.30, 2.75)	1.91*** (1.31, 2.77)
Managerial	1.89** (1.18, 3.05)	1.50 (.89, 2.53)	1.49 (.88, 2.52)	1.50 (.88, 2.55)
Clerical, sales, service	.90 (.63, 1.29)	.870 (.60, 1.25)	.895 (.62, 1.29)	.883 (.61, 1.27)
Crafts, operatives, laborers	.82 (.49, 1.39)	.78 (.46, 1.33)	.84 (.50, 1.43)	.87 (.51, 1.48)
High job authority in 1975		1.45* (1.02, 2.07)	1.49* (1.05, 2.13)	1.49* (1.05, 2.13)
Estrogen-Related Variables:				
Adiposity in 1957				.81* (.67, .97)
Reproductive history in 1993:				
At least one birth			.32 (.09, 1.05)	.30* (.09, .99)
Birth × Age at first birth			1.05* (1.01, 1.09)	1.05* (1.01, 1.09)
Number of children			.95 (.69, 1.30)	.94 (.69, 1.29)
Health behaviors in 1993:				
Healthy weight (BMI < 25) [a]				1.00
Overweight (BMI 25-29.9)				1.06 (.70, 1.61)
Obese (BMI ≥ 30)				1.19 (.87, 1.63)
Daily number of drinks				1.00 (.98, 1.02)
Days of the month when drinks				1.07 (.93, 1.23)
Light physical activity				1.07 (.94, 1.22)
Vigorous physical activity				.97 (.91, 1.05)

Table 3 (*cont'd*)

Variables	Model 1	Model 2	Model 3	Model 4
Life-course estrogen cycle:				
Age at menarche			.93 (.85, 1.03)	.93 (.85, 1.03)
Age at menopause			1.03* (1.01, 1.05)	1.03* (1.01, 1.05)
Hysterectomy/Oophorectomy			.81 (.57, 1.15)	.82 (.57, 1.16)
Hormone replacement therapy			1.36* (1.03, 1.79)	1.30* (1.00, 1.73)
Family history of breast cancer	1.12 (.57, 1.15)	1.12 (.57, 1.15)	1.12 (.57, 1.15)	1.13 (.57, 1.16)
Attrition propensity score	.84 (.64, 1.09)	.84 (.64, 1.09)	.83 (.63, 1.09)	.87 (.66, 1.15)
Model fit:				
Log likelihood (df) [b]	-1772 (7)	-1770 (8)	-1760 (16)	-1755 (23)
AIC [c]	3559	3557	3552	3557
BIC [d]	3608	3604	3653	3702

Note: Each cell contains hazard ratios and 95% confidence intervals (in parentheses). All models control for education, household income, and marital status. *p < .05. **p < .01. ***p < .001 (two-tailed test).

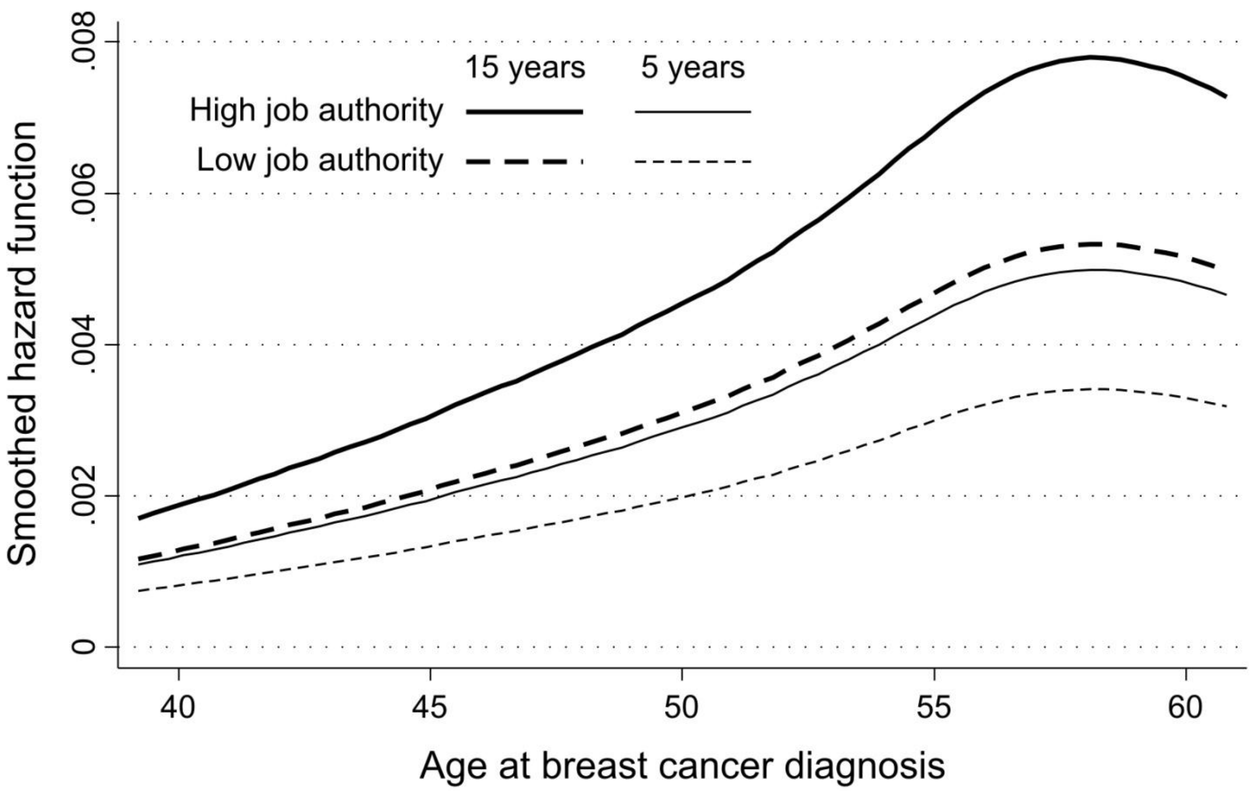
[a] Reference group.

[b] df = degrees of freedom.

[c] AIC = Akaike information criterion.

[d] BIC = Bayesian information criterion.

Figure 1. The Risk of a Breast Cancer Diagnosis among Professional and Managerial Women by the Number of Years with High and Low Job Authority between 1975 and 1993 (N = 939)



METHODOLOGICAL APPENDIX

A. Sample Selection Bias

Sample attrition presents a problem in all longitudinal panel studies. In our case, it may be problematic if sample selection is nonrandom with respect to women's occupation and factors that affect women's occupation (e.g., family background). An unequal sample selection by socioeconomic status may create an illusion of a positive association between higher-status jobs and breast cancer risk if women in professional and managerial occupations are more likely to be retained in the study to report their breast cancer diagnosis, whereas women in lower-status occupations are more likely to be lost to the follow-up without their breast cancer being registered in the study. Although the WLS has excellent sample retention during the fifty years of the follow-up, we conducted an extensive analysis to understand how our findings may be biased by sample selection and to adjust for these potential sources of selection bias.

We explored how women's family background at baseline in 1957 affects their propensity to drop out of the study by 1975. Significant predictors that are related to the likelihood of retention are mother's education, family income, the number of siblings, two-parent family, and high school grades. In contrast, rural versus urban residence and father's education and occupation are unrelated to women's sample selection between 1957 and 1975. Further, we analyzed how women's characteristics in 1975 affected their participation in 1993. Women's occupation, job characteristics, age at first birth, and the number of children were not significantly related to sample selection, whereas higher education and income as well as being married increased the likelihood of participation.

Although her own occupation is not a significant predictor of women's selection out of the study, characteristics that affect occupation (socioeconomic family background and women's own education) and characteristics that are related to occupation (women's income and marital status) are related to sample selection bias. To account for this bias, we created two selection instruments based on the propensity score approach. A propensity score represents a conditional probability of selection out of the sample:

$$p(selection) = Pr(P_i = 1|\boldsymbol{X}_i) \tag{1}$$

where $P_i = 1$ for women who dropped out of the study and $\mathbf{X}_i$ is a vector of covariates that predict attrition. The strength of propensity score approach is that each woman's observed characteristics in 1957 and 1975 that affect sample attrition are summarized into a single composite propensity score reflecting a predicted probability to be lost to follow-up.

First, we obtained a propensity score reflecting each woman's predicted likelihood to drop out of the study by 1975 based on the 1957 family background characteristics: father's and mother's education measured in years, family income measured in $100's, father's occupation (unskilled worker, farmer, skilled worker, white-collar worker, and professional/executive), rural residence in childhood, intact family structure while growing up, and high school percentile rank based on grades. This propensity score variable is included in all models in Table 2
Second, we estimated a propensity score reflecting the predicted likelihood of dropping out between 1975 and 1993 based on women's characteristics in 1975: education, occupation, job characteristics, marital status, age at first birth, the number of children, and household income. The second propensity score variable is included in all models in Table 3.

B. Breast Cancer Reporting Bias

Because a breast cancer diagnosis in the WLS is self-reported, it is important to evaluate the extent to which women's occupation might have affected their knowledge and reporting of the disease. Early breast cancer is often asymptomatic, and it is possible that women may have breast cancer but do not report it simply because they are unaware of it. At the early asymptomatic stage, the major source of knowledge of the diagnosis is preventive screening. Higher-SES women are more likely to have regular screening mammograms (Sprague et al., 2010) and, thus, may be more likely to be diagnosed with breast cancer and more likely to report it than lower-SES women. This kind of misclassification bias was shown to affect the results dramatically under certain conditions; therefore, a careful analysis is required to quantify the possible bias effect (Lash, Fox, & Fink, 2009). In an approach similar to Fox, Lash, & Greenland (2005), we use Monte Carlo simulations to evaluate which proportion of women in lower-status occupations in 1975 should underreport a breast cancer diagnosis to wipe out the observed effects of professional and managerial occupations. Our simulations were based on two assumptions. First, we assumed that a fraction of professional/managerial women and women in other occupations that had breast cancer did not report it simply because they were unaware of it. Second, we assumed that the two groups of women differed in their underreporting of a breast cancer diagnosis (fraction f_1 for professional/managerial women and fraction f_2 for other women). Fractions f_1 and f_2 were assumed to follow a normal distribution with the mean equal zero and standard deviations σ_1 and σ_2. The distributions were truncated at zero, so that only positive values of f_1 and f_2 were allowed to exclude over-reporting of breast cancer. Results of the Monte Carlo simulations (details available upon request) reveal that for all the simulated combinations of σ_1 and σ_2, the minimum fraction of women in lower-status occupations that should underreport their diagnosis to produce the observed hazard ratio of 1.72 for professional women and 1.57 for managerial women is 0.503. In other words, if 50.3% of women in lower-status occupations had cancer but did not report it, the observed effect of higher-status occupations on breast cancer could be driven entirely by differential reporting of a breast cancer diagnosis (Type I error). This appears unrealistically high, especially compared to much smaller SES differences in screening mammography (Doescher & Jackson, 2009; Gierisch et al., 2009; Schueler, Chu, & Smith-Bindman, 2008), which is the main source of differential knowledge of a breast cancer diagnosis. We conclude that our findings are unlikely to reflect reporting bias.