Management Science

Publication details, including instructions for authors and subscription information:
http://pubsonline.informs.org

The Relationship Between Workplace Stressors and Mortality and Health Costs in the United States

Joel Goh, Jeffrey Pfeffer, Stefanos A. Zenios

To cite this article:
Joel Goh, Jeffrey Pfeffer, Stefanos A. Zenios (2015) The Relationship Between Workplace Stressors and Mortality and Health Costs in the United States. Management Science

Published online in Articles in Advance 13 Mar 2015

. http://dx.doi.org/10.1287/mnsc.2014.2115

MANAGEMENT SCIENCE
Articles in Advance, pp. 1–21
ISSN 0025-1909 (print) | ISSN 1526-5501 (online)

http://dx.doi.org/10.1287/mnsc.2014.2115

The Relationship Between Workplace Stressors and Mortality and Health Costs in the United States

Joel Goh
Harvard Business School, Boston, Massachusetts 02163, jgoh@hbs.edu

Jeffrey Pfeffer, Stefanos A. Zenios
Graduate School of Business, Stanford University, Stanford, California 94305
{pfeff@stanford.edu, stefzen@stanford.edu}

Even though epidemiological evidence links specific workplace stressors to health outcomes, the aggregate contribution of these factors to overall mortality and health spending in the United States is not known. In this paper, we build a model to estimate the excess mortality and incremental health expenditures associated with exposure to the following 10 workplace stressors: unemployment, lack of health insurance, exposure to shift work, long working hours, job insecurity, work–family conflict, low job control, high job demands, low social support at work, and low organizational justice. Our model uses input parameters obtained from publicly accessible data sources. We estimated health spending from the Medical Expenditure Panel Survey and joint probabilities of workplace exposures from the General Social Survey, and we conducted a meta-analysis of the epidemiological literature to estimate the relative risks of poor health outcomes associated with exposure to these stressors. The model was designed to overcome limitations with using inputs from multiple data sources. Specifically, the model separately derives optimistic and conservative estimates of the effect of multiple workplace exposures on health, and uses optimization to calculate upper and lower bounds around each estimate, which accounts for the correlation between exposures. We find that more than 120,000 deaths per year and approximately 5%–8% of annual healthcare costs are associated with and may be attributable to how U.S. companies manage their work forces. Our results suggest that more attention should be paid to management practices as important contributors to health outcomes and costs in the United States.

Keywords: occupational health; health costs; mortality; applied optimization
History: Received August 16, 2012; accepted October 7, 2014, by Dimitris Bertsimas, optimization. Published online in *Articles in Advance*.

1. Introduction

The United States leads the world in per capita health spending, and does so by a large margin (OECD 2011, p. 147). However, by a variety of measures (e.g., life expectancy at birth, premature mortality, cancer incidence), health outcomes in the United States are either on par with or even poorer than those in other industrialized nations (OECD 2011, p. 23). There is a large body of research that explores the various contributing factors to these high costs and poor outcomes, which to date has primarily focused on three broad topic areas: (1) the way the overall U.S. healthcare system is organized and paid for, and the consequent large administrative burden (e.g., Woolhandler and Himmelstein 1991, Woolhandler et al. 2003); (2) the differences in efficiency and quality that derive from variation in the operation, organization, and management of healthcare delivery organizations (e.g., Ozcan and Luke 1993, Wennberg et al. 2005); and (3) the effect of individual behavioral choices such as diet and exercise on healthcare costs and mortality (Cardarelli et al. 2009, Keeney 2008).

Without denying the importance of these three factors, we argue that a critical topic that has thus far been ignored in discussions of health costs and morbidity is the role of stressors in the workplace that affect employee health. Specifically, we focus on 10 workplace stressors: Layoffs and unemployment, lack of health insurance, shift work, long working hours, job insecurity, work–family conflict, low job control, high job demands, low social support at work, and low organizational justice. We use an expansive definition of the workplace to include stressors that are primarily attributable to managerial practices in an organization (e.g., shift work, overtime, job control, and demands) as well as stressors that result from a combination of managerial practices and prevailing socioeconomic factors (e.g., layoffs and unemployment, health insurance). A large body of epidemiological evidence, which we briefly summarize in §2, has robustly demonstrated the health consequences of these workplace stressors. Moreover, the observed associations make intuitive sense: Stress has a direct effect on health and it also induces

unhealthy choices and behaviors, ranging from alcohol abuse, smoking, and drug consumption to suicide (e.g., Harris and Fennell 1988, Piazza and Le Moal 1998, Kouvonen et al. 2005). Research has also begun to uncover specific behavioral and physiological pathways for these associations (e.g., von Känel et al. 2001, Chandola et al. 2008). The wealth of this evidence suggests that the workplace could be an important contributor to the high healthcare spending and poor health outcomes in the United States.

Our primary contribution in this paper is to estimate the annual *workplace-associated* healthcare expenditures (i.e., costs) and mortality in the United States, which we define as the difference between the annual healthcare costs and mortality that are presently observed in the United States and these corresponding quantities in a counterfactual world where these work stressors are absent. These quantities represent the overall contribution of the workplace toward health outcomes and costs, and are important for at least two reasons. First, healthcare costs are not just an issue of public policy, but also have important financial implications for employers. For example, in recent years, General Motors spent more on healthcare, including providing health insurance, than it did on steel (e.g., Levine 1992, Appleby and Carty 2005). Many employers have also proactively moved to control their healthcare costs, in some instances dropping health insurance coverage, and in many others raising the proportion of costs that employees pay (Kaiser Family Foundation 2011). Second, because these workplace stressors are affected by managerial practices at least to some degree, substantially large estimates for these quantities would suggest that employers have a potentially strong influence over their employees' health outcomes and costs. It would also suggest that employers can potentially take measures to improve employee health by engaging in managerial practices that mitigate or reduce these stressors.

The ideal data source from which to obtain these estimates would be a single nationally representative data set of the U.S. labor force that records their exposure to workplace stressors as well as health outcomes and spending, preferably using a panel design. Standard statistical methods (e.g., regression analyses) could then be used to estimate the contribution of these workplace stressors to costs and mortality, and control for the contribution of other variables such as sociodemographic factors. To the best of our knowledge, such a data source does not exist. Nonetheless, it is our premise that the question considered here is far too important to remain unanswered because the perfect data do not exist.

In this paper, we adopt a model-based approach to estimate these quantities. Specifically, the model (described in §3) relates workplace stressors to health outcomes and spending, and its input parameters can be estimated from existing data sources (described in §4). The desired estimates for workplace-associated healthcare cost and mortality are then obtained as outputs of the model. Our model-based approach does not obviate the problem of imperfect data and has certain limitations because it necessitates the use of simplifying assumptions that abstract from reality. However, one advantage of this approach is that it enables us to convey our assumptions transparently and rigorously. Furthermore, it provides us with a platform upon which we can apply various analytical techniques and numerical sensitivity analyses to address these limitations as best as we can. Our approach follows in the footsteps of other published studies that consider questions of important public interest where available data were limited. For example, Keeney (2008) also used a model that involved simplifying assumptions to examine the effect of personal decisions on death. As another example, cost-effectiveness studies of medical technologies (e.g., Ladabaum et al. 2001, Hutton et al. 2007) employed simplifying models based on Markov chains. In both examples, data were drawn from multiple sources to estimate model parameters, which were then used as inputs into the models to obtain estimates of output measures of interest. Also, in both examples, sensitivity analyses were used to study the effects of varying some of the underlying assumptions of the model. Our current approach shares these two traits.

2. Background

We do not attempt to cover every possible stressor faced by employees in the workplace. Instead, we focus on the 10 stressors listed in §1, which were chosen because there is broad support for their health consequences from the epidemiological literature, and because there are data sources that allow us to produce sound estimates of their prevalence and the sizes of their health effects. We proceed to briefly review the epidemiological evidence on the health effects of each stressor, grouping related stressors in our review for expedience. Because this encompasses a vast body of literature, our review is not comprehensive. Instead, our review focuses on presenting representative epidemiological findings and discussing evidence that suggests that these stressors occur frequently in the workplace.

Provision of Health Insurance. There are two pathways through which an absence of health insurance affects mortality and costs. First, not having health insurance increases financial stress. That is because a significant fraction of personal bankruptcies derive from healthcare bills (e.g., Himmelstein

et al. 2009, Zywicki 2005), and not having insurance also increases the effort required to obtain healthcare for oneself and one's family. Second, an absence of insurance can also directly increase mortality and increase costs because treatment of health conditions is delayed by an absence of preventive screenings and treatment until the disease state becomes more severe (Franks et al. 1993, Wilper et al. 2009, Woolhandler and Himmelstein 1988, Sudano and Baker 2003).

Unemployment and Layoffs. Layoffs, job loss, and unemployment adversely affect physical and mental health and mortality. There is the financial stress resulting from the loss of income, and also separation from the social identity of being productively employed and social isolation from coworkers. Strully (2009) found that job loss increased the odds of reporting fair or poor health by 80%. Dooley et al. (1994) reported that people who become unemployed were at twice the risk of experiencing increased depression. Job loss has also been linked to an increased mortality risk between 44% and 100% in the year following job loss (Eliason and Storrie 2009, Sullivan and Von Wachter 2009).

Job Insecurity. Even among the employed, the insecurity associated with the prospect of losing one's job contributes to stress and therefore to morbidity and mortality. For example, Lee et al. (2004) reported that female nurses who experienced job insecurity were about 89% more likely to develop nonfatal myocardial infarction, whereas Kivimäki et al. (2000) found that there was a more than twofold increase in sickness absence among employees who worked in downsizing firms. Further evidence for the relationships between job insecurity and health is reviewed by Sverke et al. (2002).

Work Hours and Shift Work. Although it is possible that employees have some discretion in choosing the amount of time spent working and their work schedules, for the most part, working times and the amount of work are under the substantial control of employers. The evidence shows that decisions about work hours and shift work have profound health consequences, possibly through their effects on work stress, sleep, and the conflict between work and other roles. Yang et al. (2006) reported that long work hours were associated with self-reported hypertension. Vegso et al. (2007) found that the number of hours worked in the preceding week significantly predicted the incidence of acute occupational injury. Another study reported that 20% of employees who reported high levels of overwork said that they made a lot of mistakes at work compared to none who experienced low levels of overwork (Galinsky et al. 2005). In addition, summaries of the literature consistently report that both shift work and long work hours lead to poor health and also to more unhealthy behaviors such as smoking (e.g., Barnett 2006, Johnson and Lipscomb 2006, Sparks et al. 1997).

Work–Family Conflict. Work–family conflict refers to the situation "in which the role pressures from the work and family domains are mutually incompatible in some respect" (Greenhaus and Beutell 1985, p. 77). Using a cohort of 2,700 employed individuals who were either married or the parent of a child under the age of 18, Frone (2000) found that such work–family conflict was positively related to having clinically significant mental conditions and problems with substance abuse. Other studies show that work–family conflict produces physical health problems and also leads to excessive use of alcohol (Frone et al. 1996). As a significant source of stress, work–family conflict is an important contributor to both poor mental and physical health. Moreover, longitudinal panel studies show that it is work–family conflict that produces bad health, and not the reverse (Frone et al. 1997).

Job Control and Job Demands. The Karasek–Theorell model of job strain associates low job control and high job demands to poor health such as cardiovascular disease (Karasek et al. 1981, 1988). This model has been extensively investigated since its introduction, and the association between job strain and poor physical and mental health has been robustly supported in empirical studies (e.g., Shields 2006, Tsutsumi et al. 2009). The famous Whitehall studies of British civil servants (Marmot et al. 1997, 1978) not only documented the negative association between hierarchical rank and the risk of cardiovascular disease, but also determined that it was the level of job control that explained this relationship.

Social Support. In addition to doing things that increase workplace stress and decrease access to healthcare, work organizations can also make decisions that increase the social support available to their workforce to cope with various stressors. As one example, continuity in employment facilitates the formation of social networks and informal ties, which can be helpful in coping with stress. Company-organized social events and formal mentorship programs are other ways of increasing the social support available to people at work. There is good evidence for the beneficial effect of social support on health. Cohen and Wills (1985) noted that there were two possible effects of social support: a direct, main effect of social support and also a buffering effect, so that the presence of social support reduces the harmful effects of stress. Their review of the literature found support for both mechanisms. The comprehensive review by Broadhead et al. (1983) also provided support for a direct effect of social support on health and the buffering role of social support on the consequences of psychosocial and physical stressors.

Organizational Justice. Recent research also suggests that organizational injustice, i.e., the perception of unfairness at work, is an important job stressor that potentially affects employees' psychological health, and ultimately even their physical health, through deleterious health behaviors. The empirical evidence for this association is reviewed by Robbins et al. (2012).

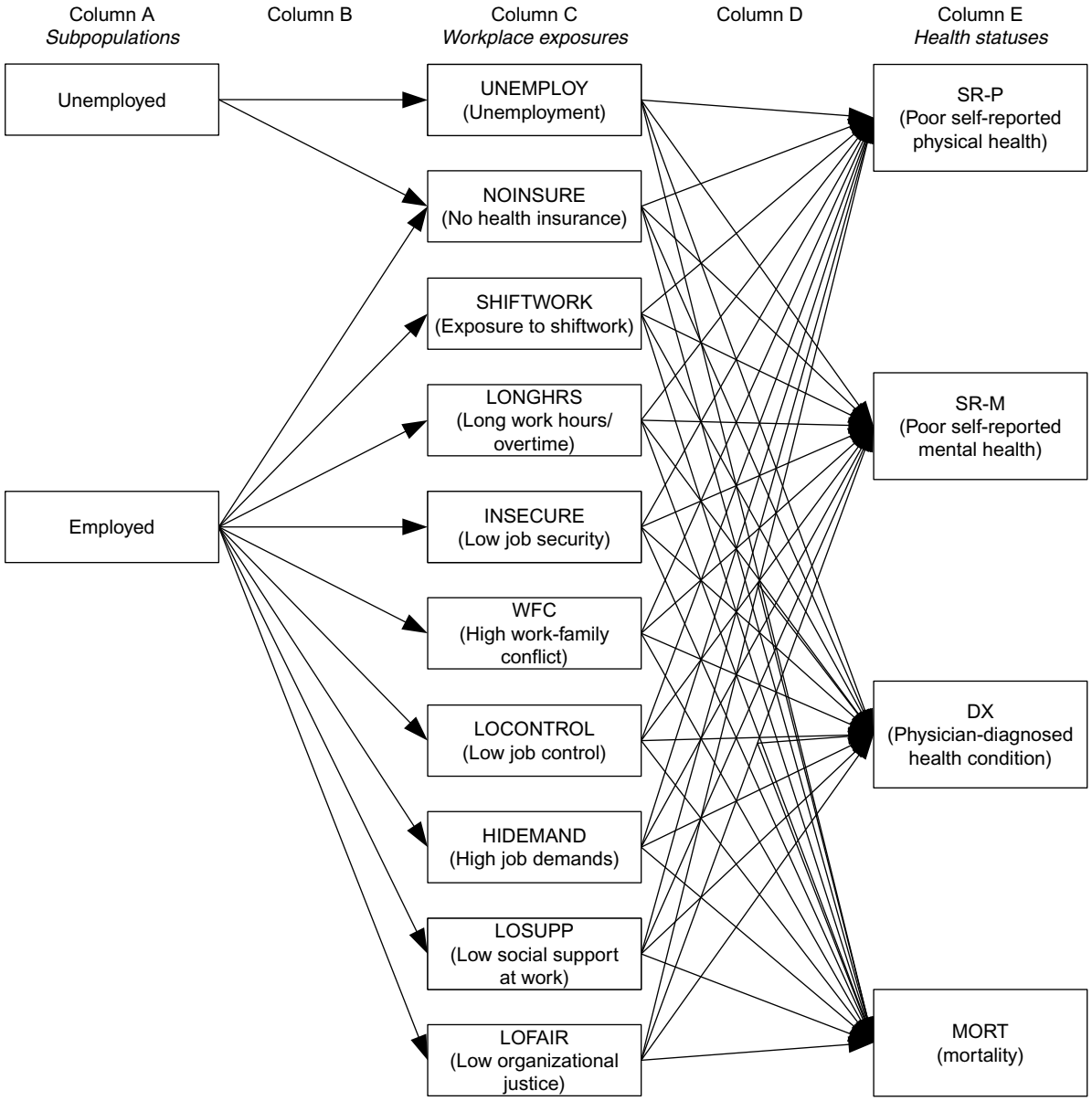
3. Model

In this section we present our model for estimating the effect of workplace practices on healthcare costs and outcomes. To do this we proceed in five steps: §3.1 provides a high-level overview of the model, §3.2 introduces the key notation, §3.3 presents all the input parameters for the model, §3.4 shows how these input parameters can be combined to calculate healthcare spendings and mortality associated with workplace practices and exposure, and §3.5 presents our methodology for computing confidence intervals on all our model estimates.

3.1. Preliminaries

An outline of the model is presented in Figure 1. The model focuses on the U.S. civilian labor force in 2010 and divides the analysis according to four subpopulations: (men, women) × (employed, unemployed).

Figure 1 **Graphical Representation of the Model of Workplace Stressors (Exposures) and Categories of Negative Health (Outcomes)**



Notes. An arrow on the left represents an exposure that a subpopulation (employed or unemployed) may possibly experience. An arrow on the right represents a potential association between an exposure and the increased risk of a particular outcome.

The unemployed are assumed to be exposed to only two stressors: unemployment and no insurance. The employed are exposed to all the stressors defined in §2 except unemployment.

The model estimates the increased prevalence of four categories of poor health (henceforth termed *outcomes*) associated with the 10 workplace stressors (henceforth termed *exposures*) and then combines them with separate estimates of the increase in health spending associated with each of the categories of poor health. The four outcomes that we consider are those that are commonly measured in the medical literature: poor self-rated physical health, poor self-rated mental health, presence of physician-diagnosed health conditions, and mortality. Self-rated health measures are included because (a) they have been shown to be excellent proxies for actual health and mortality (e.g., Marmot et al. 1995, Idler and Benyamini 1997); (b) they are easy to assess in surveys, including surveys of healthcare costs; and (c) they are commonly used in epidemiological studies.

Before we provide more details about the model, it is important to highlight two key structural assumptions: First, we assume each of the four subpopulations considered to be statistically homogeneous. This allows us to focus our analysis on a characteristic individual within each subpopulation and estimate for that individual the annual healthcare spending and probability of mortality associated with workplace stressors. The corresponding population-level estimates are obtained by scaling the individual-level estimates (by the subpopulation's size) and summing across the four subpopulations. Second, we assume that exposures to the 10 stressors and outcomes are binary; that is, we do not account for a more nuanced interaction between stressors and outcomes that takes into account the duration of the exposure to a stressor. This is because most of the studies used to obtain the parameters in our model also employ a binary model of exposures and outcomes.

3.2. Notation

Let $j = 1, \ldots, n := 4$ index outcomes and $i = 1, \ldots, m := 10$ index exposures. Let $\mathbf{Y} = (Y_1, \ldots, Y_4)$ be a binary random vector where $Y_j = 1$ represents that the individual has health outcome j, and where outcome 4 represents mortality. Let $\mathbf{X} = (X_1, \ldots, X_{10})$ be a binary random vector where $X_i = 1$ represents that the individual is exposed to stressor i. For a given realization $\mathbf{x}$ of the random vector $\mathbf{X}$, let $p_j(\mathbf{x}) := \mathbb{P}(Y_j = 1 \mid \mathbf{X} = \mathbf{x})$ represent the probability that the individual has health outcome j, conditional on exposure to the nonzero components of $\mathbf{x}$. Furthermore, letting $\mathbf{0}$ represent the zero vector, let $f_j(\mathbf{x}) := p_j(\mathbf{x})/p_j(\mathbf{0})$ represent the relative risk of outcome j from the multiple exposures in vector $\mathbf{x}$. In addition, Δ_{cost} and Δ_{mort} refer to the incremental costs and mortality associated with exposure to all 10 stressors.

3.3. Input Parameters

The model relies on four input parameters to estimate the healthcare cost and mortality associated with workplace stressors. Here we introduce these parameters and describe the data sources used for their estimation. The estimation methods and all assumptions made are described in §4.

1. *Joint probability distribution of exposures,* $g(\mathbf{x}) := \mathbb{P}(\mathbf{X} = \mathbf{x})$. This parameter captures the average prevalence of (and correlations between) stressors faced by workers in the United States. We use the General Social Survey (GSS) (National Opinion Research Center 2011) as the primary data source for this estimate and supplement it with data on health insurance coverage from the Current Population Survey (CPS) Annual Social and Economic Supplement (U.S. Census Bureau 2012). Recognizing that estimating the full joint distribution g can be error prone, we develop an optimization-based technique for estimating the workplace-associated health cost and mortality that requires estimating only the second moments of $\mathbf{X}$ instead of the full joint distribution.

2. *Relative risk for each exposure–outcome pair,* r_{ij}. This parameter quantifies the extent to which workplace stressors affect health outcomes. For exposure i and outcome j, we estimate the incremental probability (i.e., the relative risk) of individuals having outcome j for individuals that were exposed to i, relative to nonexposed individuals. We obtain these estimates by conducting a meta-analysis of the relevant epidemiological literature.

3. *Status quo prevalence of each outcome,* p_j. This parameter captures the observed prevalence of each category of poor health in the United States. We use the Medical Expenditure Panel Survey (MEPS) Household Component (Agency for Healthcare Research and Quality 2011a) as the primary data source and supplement it with mortality data published by the Center for Disease Prevention and Control (CDC) (Kochanek et al. 2011).

4. *Incremental cost of each outcome per year,* c_j. This parameter quantifies the excess healthcare spending per year associated with each category of poor health in the United States; that is, c_j represents the average increase in healthcare spending for individuals with outcome j compared to individuals without outcome j. For each outcome, we use the MEPS to estimate the average excess (direct) medical cost of individuals with that outcome, compared to individuals without the outcome. Our estimation method in this step controls for the overlapping healthcare cost contributions from multiple health outcomes.

Figure 2 Flow Diagram Representation of the Estimation Procedure

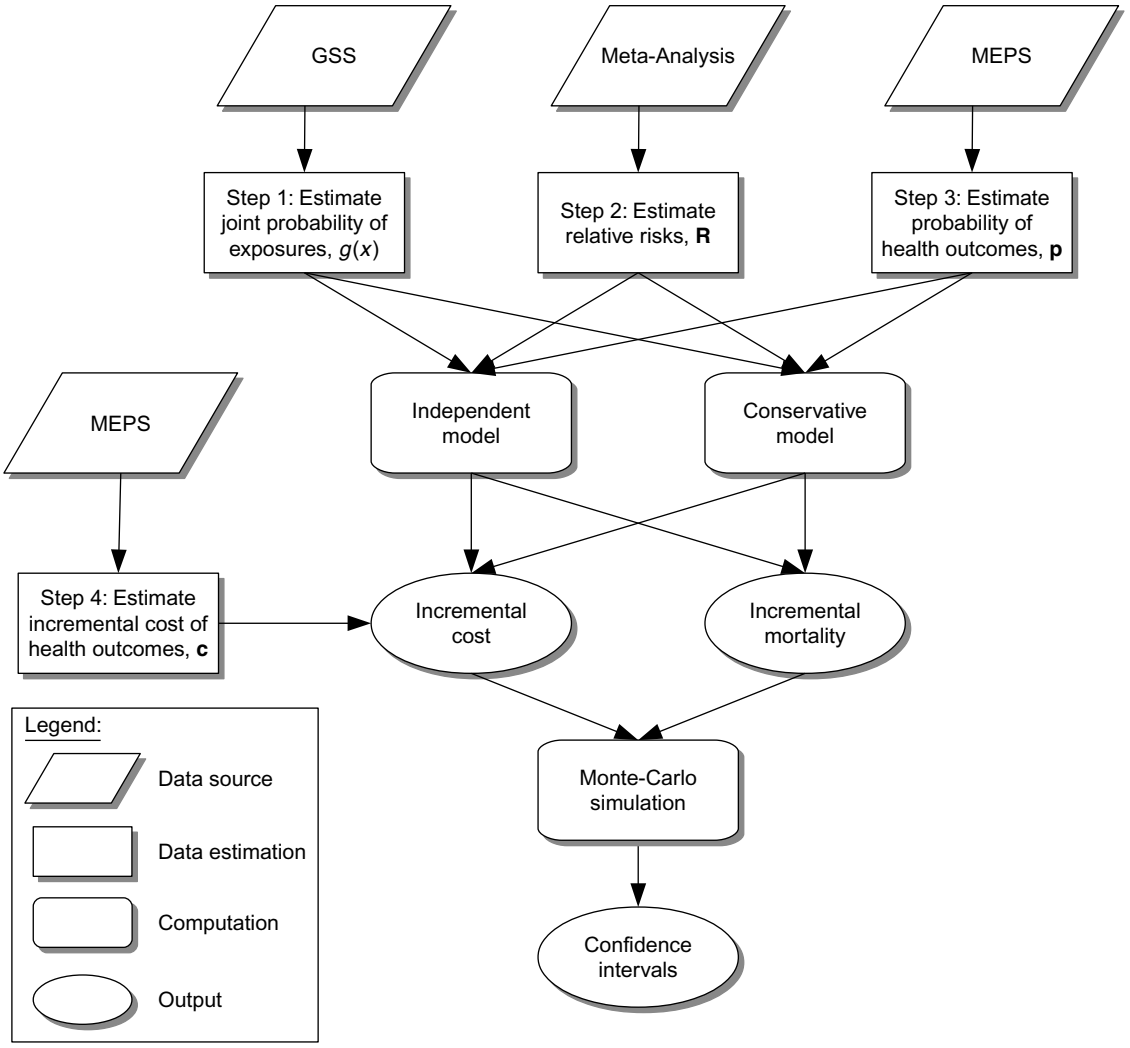


Figure 2 summarizes the notation and data sources for the input parameters, and also illustrates the estimation and computation steps for the model.

3.4. Workplace-Associated Healthcare Spending and Mortality

The purpose of our model is to estimate the following two quantities directly from the input parameters defined in §3.3:

$$\Delta_{\text{cost}} := \sum_{j=1}^{n} c_j(p_j - p_j(\mathbf{0})) \quad \text{and} \quad \Delta_{\text{mort}} := p_n - p_n(\mathbf{0}). \tag{1}$$

We proceed to derive mathematical expressions for Δ_{cost} and Δ_{mort} only in terms of the input parameters (this is presented in Proposition 1 below). However, we need to digress first to address a key challenge: the definitions in (1) contain the terms $p_j(\mathbf{0}) = \mathbb{P}(Y_j = 1 \mid \mathbf{X} = \mathbf{0})$, the probability of outcome j occurring in an *unobservable* counterfactual world where all exposures are absent, and therefore cannot be directly estimated from data. Deriving $p_j(\mathbf{0})$ from the data is not possible: we do not have any data from this counterfactual world, and it is intractable to estimate the probabilities $p_j(\mathbf{x})$ for all $\mathbf{x} \neq \mathbf{0}$. To circumvent this problem, we consider the following two models that use different assumptions to relate $p_j(\mathbf{0})$ to $p_j(\mathbf{x})$.

Multiplicative Model. This model assumes that when multiple exposures are present, their overall effect is the aggregate of the effect of the individual exposures:

$$p_j(\mathbf{x}) = p_j(\mathbf{0}) \prod_{i:\, x_i = 1} r_{ij} \quad \text{for all } \mathbf{x} \in \{0, 1\}^m. \tag{2}$$

We note that the multiplicative model can be rewritten in the following form:

$$\log p_j(\mathbf{x}) = \log p_j(\mathbf{0}) + \sum_{i=1}^{m} (\log r_{ij}) x_i, \tag{3}$$

which shows that it is a type of generalized linear model.

Conservative Model. This model assumes that the aggregate effect from multiple exposures is the maximal effect of the individual exposures:

$$p_j(\mathbf{x}) = p_j(\mathbf{0}) \max_{i:x_i=1}\{r_{ij}\} \quad \text{for all } \mathbf{x} \in \{0,1\}^m. \tag{4}$$

Informally, the multiplicative model assumes that the total effect of multiple exposures is a simple accumulation of their individual effects. On the other hand, the conservative model assumes that exposures are not ameliorative: If exposure i, on its own, raises the probability of outcome j by a multiple of r_{ij}, then the presence of some other exposure i' alongside i does not decrease the effect of i on j and vice versa. For a given outcome j, the multiplicative model yields more aggressive estimates than the conservative model when r_{ij} are greater than 1 for every exposure i, which is what we generally expect. Even though other theoretical models are possible, we think that these two models represent a simple way to obtain a reasonable range of the overall effect of multiple exposures, and they have the added advantage that they have modest data requirements.

With these models in place, we now derive Δ_{cost} and Δ_{mort} in terms of input parameters, under both multiplicative and conservative models.

Proposition 1. *For the multiplicative model,*

$$\Delta_{\text{cost}} = \sum_{j=1}^{n} c_j p_j \left[1 - \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} \prod_{i:x_i=1}\{r_{ij}\} g(\mathbf{x})}\right] \quad \textit{and}$$

$$\Delta_{\text{mort}} = p_n \left[1 - \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} \prod_{i:x_i=1}\{r_{in}\} g(\mathbf{x})}\right]. \tag{5}$$

For the conservative model,

$$\Delta_{\text{cost}} = \sum_{j=1}^{n} c_j p_j \left[1 - \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} \max_{i:x_i=1}\{r_{ij}\} g(\mathbf{x})}\right] \quad \textit{and}$$

$$\Delta_{\text{mort}} = p_n \left[1 - \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} \max_{i:x_i=1}\{r_{in}\} g(\mathbf{x})}\right]. \tag{6}$$

The proof is straightforward and omitted for brevity.

3.5. Estimating Confidence Intervals

To assess the effect of estimate sampling error on our cost and mortality estimates, we calculate confidence intervals using Monte Carlo simulation. Our method is as follows: For three of the input parameter categories, incremental cost, status quo prevalence, and relative risks, we draw 10,000 independent samples of the input parameters using standard distributional assumptions. We then feed these simulated inputs into our model to obtain corresponding sampled values of Δ_{cost} and Δ_{mort} and construct 95% confidence intervals using their 2.5% and 97.5% quantile points. The standard distributional assumptions are as follows: the incremental costs of outcomes $(c_1, \ldots, c_4)$ and status quo prevalence of outcomes $(p_1, \ldots, p_4)$ are sample estimates derived from large samples; therefore, we can assume that they are normally distributed. The mean of the distribution is the observed sample mean, and the variance is the squared standard error of the sample estimates. For the relative risks r_{ij}, we also utilize a distributional assumption that is common in the literature: lognormal with sample parameters given by their empirical estimates (Fleiss and Berlin 2009). For the fourth parameter category, the joint probability distribution of exposures, the Monte Carlo simulation approach was not feasible because of the large number of parameters that needed to be estimated for this distribution. Instead, we used mathematical optimization to find a range of estimates that were consistent with the data used to estimate this distribution (see §4.1).

3.6. Alternative Models

We also considered the possibility of using two alternative models. The first was a linear model that assumes

$$p_j(\mathbf{x}) = \beta_{0j} + \sum_{i=1}^{m} \beta_{ij} x_i. \tag{7}$$

However, the principal obstacle that prevented us from using this model was that we were not able to reliably estimate the coefficients for such a model from the literature, because most papers in the literature use logistic regression as their statistical approach when outcomes are binary, instead of a pure linear model such as (7).

The second alternative approach was a nonparametric model based on convex optimization, where the decision variable was the joint probability distribution of the *combined* vector of outcomes and exposures. The data on exposures, relative risks, and outcomes were incorporated into the model as linear constraints. However, this approach produced values that were far too conservative (i.e., the estimated ranges of mortality and costs were too large to be meaningful). Intuitively, this was ultimately because it was too flexible, as it did not constrain the effect of multiple exposures.

4. Estimation Procedures

In this section, we describe the methods used to estimate the model's input data parameters (introduced in §3.3). Our estimation methods were designed to overcome two major challenges. First, estimating the joint distribution of exposures (§4.1) is prone to excessive sampling error because of the large number of parameters to be estimated. To control for

these sampling errors, we use a robust optimization approach to calculate upper and lower bounds for our estimates. Second, the four health outcomes incorporated in our model can be correlated, and this may cause a double-counting problem when translating these outcomes to total costs. To avoid double counting, we use a group-matching estimation procedure, detailed in §4.4.

4.1. Estimating the Joint Probability Distribution of Exposures, $g(\mathbf{x})$

Our estimation procedure separates employed from unemployed people. It then estimates the probability of each exposure and their correlations. We first present the methodology for estimating the exposure probabilities for employed and then for unemployed people (see Table 1). We then present the methodology for estimating the correlation between the exposures.

For the subpopulation of employed persons, we used the GSS as our primary data source. The GSS includes survey responses on a wide variety of work-related conditions. This allows us to estimate the joint distribution for all the workplace exposures faced by employed people in our model, with the exception of the only condition not covered by the GSS, *NOINSURE*. Because of that, we assumed that *NOINSURE* was independent of the other variables and we estimated its probability using data from the 2011 CPS (U.S. Census Bureau 2012). To assess the sensitivity of our conclusions to this assumption, we also considered a model without this independence assumption in §5.3.

To estimate the joint distribution of the other eight exposures, we pooled data from the three years in the GSS that included responses to workplace exposure questions: 2002, 2006, 2010 ($N = 4{,}086$ respondents; 1,969 men, 2,117 women). Table 1 lists the GSS exposures.

Because for six of the eight exposure variables in the survey the responses were on a four-point scale, we used the cutoff of 2.5 points as the dividing line between exposed (values >2.5) and not exposed (value <2.5). For exposures measured by multiple variables in the GSS, we used a cutoff of 2.5 times the number of variables. For the remaining two exposures, *SHIFTWORK* and *LONGHRS*, which were each measured by one GSS variable, we classified respondents as *SHIFTWORK* cases as long as they did not work the day shift, and respondents as *LONGHRS* cases if they had to work extra hours for more than seven days per month (about one-quarter of a month).

For the subpopulation of unemployed persons, the estimation problem focuses on the estimation of two quantities: the probability of *NOINSURE* among the unemployed for men and for women. These estimates were derived from the 2011 CPS (U.S. Census Bureau 2012).

We now proceed to describe our approach for estimating the probability $g(\mathbf{x})$. Given that the vector $\mathbf{x}$ is m dimensional and each dimension can take two values, 0 or 1, $g(\mathbf{x})$ involves $2^m - 1$ parameters. Estimating all these parameters from data on 4,086 responses would introduce excessive estimation errors for most of the entries. Instead we use an approach from robust optimization that proceeds as follows. First, we estimate the covariance matrix of the joint exposures. This requires the estimation of a total of $m(m+1)/2$ entries. Second, we formulate the estimation problem as a mathematical program. This mathematical program will be shown to be a linear program (LP) and generates lower and upper bounds for the cost and mortality estimates that are consistent with the data.

We now present the formulation of this mathematical program using notation that captures both the cost and mortality formulations. First, let $\mathbf{V} = [v_{ik}] \in \mathbb{R}^{m \times m}$ represent the second-moment matrix obtained from

Table 1 GSS Variables Used to Estimate Each Exposure and Their Estimated Marginal Probabilities for Men and Women

			Marginal probabilities	
Population	Exposure	GSS variables	Men	Women
Unemployed	*UNEMPLOY*	*	1.0000	1.0000
	NOINSURE	*	0.4419	0.3448
Employed	*NOINSURE*	*	0.2091	0.1675
	SHIFTWORK	*WRKSCHED*	0.1735	0.1426
	LONGHRS	*MOREDAYS*	0.3153	0.2239
	INSECURE	*JOBSECOK*	0.1364	0.1422
	WFC	*FAMWKOFF*, *WKVSFAM*	0.1950	0.2047
	LOCONTROL	*WKDECIDE*, *WKFREEDM*	0.0785	0.0930
	HIDEMAND	*WORKFAST*, *OVERWORK*, *TOOFEWWK*, *WRKTIME*	0.3095	0.3447
	LOSUPP	*SUPCARES*, *COWRKINT*, *SUPHELP*, *COWRKHLP*	0.0811	0.0971
	LOFAIR	*PROMTEFR*	0.2603	0.3327

Note. Entries with asterisks represent probabilities estimated from the 2011 CPS.

the empirical distribution of $\mathbf{X}$. Second, for some fixed $\mathbf{w} \in \mathbb{R}^n_+$, define

$$\Delta_{\mathbf{w}} := \sum_{j=1}^{n} w_j p_j \left(1 - \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} f_j(\mathbf{x}) g(\mathbf{x})}\right). \tag{8}$$

When $\mathbf{w} = \mathbf{c}$, then $\Delta_{\mathbf{w}}$ is Δ_{cost}, and when $\mathbf{w} = (0, 0, \ldots, 1)$, then $\Delta_{\mathbf{w}}$ is Δ_{mort}. Third, for any $q \in \mathbb{R}^{\{0,1\}^m}$ and any $i, k \in \{1, \ldots, m\}$, define the linear functional $\mathscr{L}_{ik}\colon \mathbb{R}^{\{0,1\}^m} \to \mathbb{R}$ as $\mathscr{L}_{ik} q := \sum_{\mathbf{x}\in\{0,1\}^m, x_i=1, x_k=1} q(\mathbf{x})$. Using this notation, the constraint that the second-moment matrix for $g(\mathbf{x})$ is equal to the empirical matrix can be written as $\mathscr{L}_{ik} g = v_{ik}$ for all $i, k \in \{1, \ldots, m\}$.

The following theorem demonstrates how to construct bounds for $\Delta_{\mathbf{w}}$ for any $\mathbf{w}$ by solving two linear programs.

Theorem 1. *For a fixed $\mathbf{w} \in \mathbb{R}^n_+$, define $\underline{\Gamma}_{\mathbf{w}}$ to be optimal value of the LP*

$$\underline{\Gamma}_{\mathbf{w}} := \min_{q_j, \mathbf{t}} \quad \sum_{j=1}^{n} w_j p_j t_j \tag{9}$$

$$\text{s.t.} \quad \sum_{\mathbf{x}\in\{0,1\}^m} f_j(\mathbf{x}) q_j(\mathbf{x}) = 1 \quad j \in \{1, \ldots, n\}, \tag{10}$$

$$\sum_{\mathbf{x}\in\{0,1\}^m} q_j(\mathbf{x}) - t_j = 0 \quad j \in \{1, \ldots, n\}, \tag{11}$$

$$\mathscr{L}_{ik} q_j - v_{ik} t_j = 0 \quad j \in \{1, \ldots, n\},\ i, k \in \{1, \ldots, m\}, \tag{12}$$

$$\mathbf{t} \geq \mathbf{0}, q_j(\mathbf{x}) \geq 0 \quad \mathbf{x} \in \{0,1\}^m,\ j \in 1, \ldots, n, \tag{13}$$

and define $\bar{\Gamma}_{\mathbf{w}}$ as optimal value of the LP (9)–(13) *but with the* min *replaced by* max. *Then,*

$$\sum_{j=1}^{n} w_j p_j - \bar{\Gamma}_{\mathbf{w}} \leq \Delta_{\mathbf{w}} \leq \sum_{j=1}^{n} w_j p_j - \underline{\Gamma}_{\mathbf{w}}. \tag{14}$$

We now provide some intuition behind the theorem and the LP formulation in (9)–(13). First, the LP is a relaxation of an optimization problem that looks for a density function that minimizes or maximizes $\sum_{j=1}^{n} w_j p_j - \Delta_{\mathbf{w}}$ (recall that this represents either cost or mortality depending on the vector $\mathbf{w}$ used), subject to the constraint that this density is consistent with the empirical second-moment matrix. Second, the decision variables $q_j(\mathbf{x})$ represent scaled versions of the density function, and t_j are auxiliary variables that linearize the optimization problem and are linked to the main decision variables, $q_j(\mathbf{x})$, through constraints (11) and (12). Third, the objective function (9) and the first constraint (10) together represent a linearization of $\sum_{j=1}^{n} w_j p_j - \Delta_{\mathbf{w}}$. Fourth, the constraint (12) enforces the requirement that the density is consistent with the empirical second-moment matrix. The remaining constraints (13) are standard nonnegativity constraints.

Theorem 1 can also be used even when there exist pairs of exposures i and k for which the correlation is unknown because of limited data. To do this, we use the Boole–Fréchet inequalities (Boole 1854), which imply that $\max\{v_{ii} + v_{kk} - 1, 0\} \leq v_{ik} \leq \min\{v_{ii}, v_{kk}\}$, where v_{ii}, v_{kk} are the marginal probabilities of exposures i and k. Then, the two bounds are obtained by solving (9)–(13) with the third constraint (12) modified to

$$t_j \max\{v_{ii} + v_{kk} - 1, 0\} \leq \mathscr{L}_{ik} q_j \leq t_j \min\{v_{ii}, v_{kk}\} \quad j = 1, \ldots, n. \tag{15}$$

4.2. Estimating the Relative Risks of Each Exposure–Outcome Pair, r_{ij}

We estimated the relative risk matrix, $\mathbf{R} = [r_{ij}]$, through a meta-analysis. A meta-analysis is a method of research synthesis commonly used in medicine and the social sciences to quantify the average size of an effect by summarizing the results of multiple empirical studies. We outline our approach below and refer readers to the online supplement (available at http://dx.doi.org/10.1287/mnsc.2014.2115) for details.

Our meta-analytic sample comprised 228 studies. These studies were identified through a systematic review of the literature from an initial number of 6,452 studies found through a computerized reference search. We obtained the final meta-analytic sample by applying formal inclusion criteria to ensure impartiality in selecting studies. Our inclusion criteria ensured that the selected studies were germane to our research question, used consistent statistical methods, and were not double counted. At this stage, these criteria were not designed to be too exclusive because we wanted to ensure that we had enough studies covering as many entries of $\mathbf{R}$ as possible. Later, we use sensitivity analyses to study the effect of having more restrictive criteria. For example, we were primarily interested in studies that used logistic regressions because the health outcomes were usually measured as dichotomous values (present/absent), but we also included studies that used Cox proportional hazards regressions (henceforth, *Cox regressions*) because they were also commonly used in the literature and because they can be viewed as approximations to logistic regressions. In Appendix B, we discuss details of these two econometric models and provide a derivation of this approximation. We did not include studies that used other econometric models because we would have had to make additional assumptions to include these other models, which would compromise the consistency of our methods. We also note that similar restrictions are common in meta-analyses in this domain (see, e.g., Virtanen et al. 2013). More complex econometric techniques

(e.g., instrumental variables, structural models) are uncommon in this literature, and were not used by any of the final 228 studies in the sample, even though we did not explicitly exclude studies based on these criterion. Matching methods such as propensity score weighting were only used by one study (Eliason and Storrie 2009) in the final sample.

All 228 studies were observational, which is highly representative of the literature on this subject because ethical concerns generally prevent randomized experiments from being conducted. We refer to a study as using *longitudinal data* if it assesses exposures at a time point that precedes the assessment of outcomes; otherwise, we say that it uses *cross-sectional data*. We note that the studies that we classify as using longitudinal data are not strictly *longitudinal studies* as is traditionally defined in the literature because they are not required to assess exposures repeatedly over time. However, these studies are not cross-sectional studies either, because there are at least two observations that are made at two distinct times: one observation that captures the exposure and a second that captures the outcome of interest at a later time. Studies that use cross-sectional data are prone to bias stemming from reverse causation, where an observed correlation between workplace stressors and poor health outcomes could be a consequence of poor health causing these stressors, and not vice versa. On the other hand, studies that use longitudinal data are protected against this type of bias since health outcomes are measured after exposures are assessed, and therefore the estimates from these studies are generally viewed as more reliable than estimates from studies using cross-sectional data. Of our sample, 115 studies used longitudinal data (these included panel studies), 115 studies used cross-sectional data, and 2 studies used both types of data. In our base model, the estimates for the entries of $\mathbf{R}$ are generated from all 228 studies, and in §5.3, we conduct sensitivity analyses to investigate the impact of using only longitudinal data.

For each study, we recorded the effect sizes that were adjusted for the largest number of covariates. We note that the actual covariates used differed across studies, which is a well-recognized issue with this method. Nonetheless, we did this because it is regarded as best practice in meta-analyses (Higgins and Green 2011), it minimized the chance of omitted variable bias, and these effect sizes were usually conservative. We recorded separate effects by gender if the study permitted it; otherwise, we recorded a common effect for both men and women for the study. Finally, we assessed the research methodology of the study and determined an overall quality score for each study. Each study was initially awarded score of 1, which was reduced according to a scoring rubric (with a minimum score of zero) that penalized the study for methodological limitations. We investigated the effect of modifying the scoring rubric through sensitivity analyses in §5.3. The rubrics used in our base model and for our sensitivity analyses are listed in Table 2.

Table 2 Scoring Rubrics Used to Penalize Studies for Methodological Flaws in the Base Model (P0) and the Sensitivity Analyses (P1–P3)

Methodological limitations	P0	P1	P2	P3
1 Mean age too small or too large[a]	−0.2	0.0	−1.0	−0.5
2 Few adjustments for confounders[b]	−0.2	0.0	−1.0	−0.5
3 Very few or no adjustments for confounders[b]	−0.5	0.0	−1.0	−0.5
4 No confidence interval reported and an approximation used instead	−0.5	0.0	−1.0	−0.5
5 No number or percentage of males/females reported	−0.5	0.0	−1.0	−0.5
6 Outcome variable not directly related to value of interest	−0.5	0.0	−1.0	−0.5
7 Workplace exposure indirectly measured by occupation	−0.5	0.0	−1.0	−0.5

[a]Less than 30 or more than 60 years of age.

[b]We assessed a study to have limitation 3 if it had adjustments for only a few (approximately less than three) basic demographic confounders, e.g., age, sex, education, marital status, or race. The study was assessed to have adequate adjustments (i.e., neither limitation 2 nor 3) if it adjusted for what we deemed were comprehensive coverage of relevant factors: demographic factors, other relevant workplace factors, and health factors (e.g., existing conditions, health behaviors). The study was assessed to have limitation 2 instead if it was somewhere in between: if it considered basic demographic confounders as well as a few additional confounders, but did not do so comprehensively.

For each exposure–outcome pair, we used the quality-weighted random effects model by Shadish and Haddock (2009) to average the results of the different studies to estimate an average odds ratio for that exposure–outcome pair. The resulting odds ratios and the number of studies used to form each estimate are reported in Table 3. An in-depth discussion of the results and policy implications of these results is beyond the scope of this paper, and we refer interested readers to Goh et al. (2015) for such a discussion.

Finally, we (a) applied a standard transformation to convert the odds ratios from Table 3 into relative risks (Zhang and Yu 1998) and (b) excluded entries that were estimated by only a small number of studies (which we defined as two studies or fewer) because these estimates were more likely to be unreliable. For such entries, we set their relative risks to a default value of 1. This effectively excludes the results of these studies from our analysis. Since most of the odds ratio (and therefore, relative risk) estimates are above 1, our approach to replace these entries by 1 is conservative.

Table 3 Results of Meta-Analysis

		SR-P		SR-M		DX		MORT	
Exposure	Gender	*n*	OR (95% CI)	*n*	OR (95% CI)	*n*	OR (95% CI)	*n*	OR (95% CI)
UNEMPLOY	Men	8	1.71 (1.27, 2.30)*	15	1.92 (1.61, 2.29)*	8	1.38 (1.05, 1.82)*	17	1.42 (1.28, 1.58)*
	Women	8	1.69 (1.26, 2.31)*	14	1.78 (1.50, 2.46)*	6	1.29 (0.89, 2.17)	14	1.43 (1.19, 1.69)*
NOINSURE	Men	1	1.43 (1.27, 1.61)*	0	—	3	1.86 (1.22, 2.85)*	8	1.17 (1.10, 1.24)*
	Women	1	1.43 (1.27, 1.61)*	0	—	5	2.31 (1.64, 2.11)*	10	1.29 (1.17, 1.17)*
SHIFTWORK	Men	3	1.03 (0.92, 1.15)	5	1.30 (0.89, 1.91)	14	1.27 (1.11, 1.45)*	4	0.98 (0.89, 1.07)
	Women	2	1.16 (0.88, 1.20)	5	1.14 (0.97, 1.75)	16	1.38 (1.24, 1.30)*	3	1.21 (0.88, 1.09)
LONGHRS	Men	4	0.98 (0.80, 1.21)	12	1.17 (1.05, 1.30)*	8	1.20 (1.07, 1.35)*	2	1.17 (1.01, 1.35)*
	Women	4	1.03 (0.63, 1.54)	12	1.23 (1.10, 1.24)*	7	1.17 (1.01, 1.44)*	2	1.41 (0.89, 1.53)
INSECURE	Men	13	1.53 (1.22, 1.91)*	19	1.45 (1.15, 1.83)*	9	1.22 (1.03, 1.45)*	3	1.32 (0.77, 2.27)
	Women	13	1.39 (1.14, 2.04)*	18	1.39 (1.12, 1.89)*	10	1.12 (1.02, 1.47)*	3	1.03 (0.84, 2.08)
WFC	Men	6	1.90 (1.67, 2.17)*	10	2.40 (1.91, 3.01)*	1	1.57 (1.20, 2.05)*	1	1.20 (1.03, 1.40)*
	Women	6	1.91 (1.51, 2.39)*	10	2.68 (2.04, 2.81)*	2	1.28 (0.82, 3.00)	0	—
LOCONTROL	Men	17	1.48 (1.23, 1.78)*	36	1.39 (1.22, 1.59)*	36	1.23 (1.12, 1.36)*	4	1.40 (1.21, 1.62)*
	Women	16	1.41 (1.21, 1.82)*	33	1.38 (1.25, 1.55)*	35	1.27 (1.16, 1.31)*	3	1.31 (1.12, 1.76)*
HIDEMAND	Men	14	1.46 (1.19, 1.78)*	37	1.65 (1.42, 1.92)*	39	1.42 (1.27, 1.60)*	6	0.99 (0.85, 1.14)
	Women	13	1.49 (1.23, 1.72)*	33	1.59 (1.38, 1.98)*	37	1.38 (1.23, 1.65)*	3	0.95 (0.83, 1.17)
LOSUPP	Men	12	1.34 (1.12, 1.60)*	28	1.41 (1.30, 1.53)*	21	1.21 (1.10, 1.33)*	3	1.06 (0.84, 1.32)
	Women	13	1.40 (1.18, 1.52)*	26	1.36 (1.24, 1.59)*	23	1.22 (1.12, 1.31)*	2	1.13 (0.67, 1.67)
LOFAIR	Men	4	1.35 (1.23, 1.47)*	6	1.61 (1.08, 2.39)*	1	1.55 (1.11, 2.17)*	0	—
	Women	4	1.38 (1.19, 1.53)*	6	1.66 (1.14, 2.28)*	1	1.55 (1.11, 2.17)*	0	—

Note. Numbers of studies used for each estimate (*n*), odds ratios (ORs), and 95% confidence intervals (CIs) for each exposure and outcome in men and women are shown.

*$p < 0.05$.

4.3. Estimating the Status Quo Prevalence of Health Outcomes, p_j

To estimate the status quo prevalence of three of the health outcomes considered, poor self reported physical and mental health and physician diagnosed medical conditions, we used data from the MEPS, a nationally representative survey of families and individuals within the civilian noninstitutionalized U.S. population (Agency for Healthcare Research and Quality 2011a). We formed our estimates by pooling the most recent five years of MEPS data that were available (2004–2008). To estimate the probability of death, we computed mortality rates using the CDC Vital Statistics Reports for 2009 (see Kochanek et al. 2011). All the estimates are summarized in Table 4.

We now present details for the estimation procedure that used the MEPS data. For each health outcome, we classified each subject in the data into "cases" and "controls." For self-reported physical health (SR-P), we used responses to a single variable (RTHLTH31), which asked respondents to assess their physical health status on a five-point scale (1, excellent; 2, very good; 3, good; 4, fair; and 5, poor). We classified respondents that reported 4 or 5 as "cases," whereas we classified respondents that reported 1–3 as "controls." We used an identical approach for self-reported mental health (SR-M) on the variable (MNHLTH31).

For physician-diagnosed conditions (DX), the MEPS contains information on whether each subject was ever diagnosed with one the following major disease categories: coronary heart disease, angina, myocardial infarction, other heart disease, stroke, emphysema, asthma, high cholesterol, diabetes, and arthritis. These diseases comprise almost all of the "Priority Conditions" considered in the MEPS, with two exceptions: cancer diagnoses were excluded because such data were only collected in 2008; diagnoses of high blood pressure were excluded because it is usually a symptom of other diseases and inclusion could cause confounding. We classified respondents as "cases" of DX if they reported one or more diseases and "controls" otherwise. Through sensitivity analyses, we studied the effect of varying the threshold

Table 4 Estimates (and Standard Errors) of p, the Occurrence Probability of Negative Health Outcomes

Gender	SR-P	SR-M	DX	MORT
Men	0.1016 (0.0018)	0.0537 (0.0013)	0.3531 (0.0039)	0.0089 (0.0000)
Women	0.1182 (0.0021)	0.0586 (0.0013)	0.3940 (0.0037)	0.0063 (0.0000)

number of diseases that determines DX cases and controls.

4.4. Estimating the Incremental Cost of Poor Health, c_j

To estimate the incremental healthcare cost of poor health, we also used MEPS data. A simplistic method of estimating c_j is to take the difference of average cost between cases and controls for each j. However, such a method double counts costs, because of the correlation between various health outcomes.

To deal with this double-counting problem, we estimated c_j for each outcome j by comparing subject groups that differed *only* by health outcome j. Specifically, for each outcome j, we split the subjects into groups corresponding to all of the eight possible combinations of the remaining three non-j health outcomes. Then, for each of the eight groups, we computed the difference in average costs between the subjects that had the j outcome and the subjects that did not. The cost c_j was then the weighted average of the cost differences computed for each of the eight groups (the weight was given by the number of respondents in each group). The estimated costs are reported in Table 5. To ensure the robustness of our approach, we also considered an alternative in which we estimated all components of $\mathbf{c}$ simultaneously using a linear regression model with linear and two-way interaction terms. This also yielded qualitatively similar results, which provides us with further confidence in the validity of our estimates.

Before we conclude this section, we must report on a possible shortcoming of the MEPS data and our way to overcome it. Specifically, the MEPS is an excellent data source on person-level expenditures, but tends to underestimate total healthcare expenditures when aggregated. The major reasons for this are that (a) the MEPS undersamples the most seriously ill people in the population (Sing et al. 2006, Garrett et al. 2008), and (b) it omits certain costs that survey respondents cannot recall accurately (Selden and Sing 2008). In particular, the MEPS estimate for total healthcare expenditure in 2008 was \$1.15 trillion (Agency for Healthcare Research and Quality 2011b), which is slightly less than half of the \$2.39 trillion in 2008 estimated from the National Health Expenditure Accounts (NHEA) by the Centers for Medicare and Medicaid Services (2011). A simple way to correct for this problem is to uniformly adjust all costs by the adjustment factor 2.39/1.15.

The introduction of this adjustment can be problematic. More sophisticated adjustment methods that apply higher adjustments to more expensive cases and lower adjustments to less expensive cases have been used elsewhere (see Selden and Sing 2008). Our approach's advantage lies in its simplicity and on the fact that applying a uniform adjustment generates conservative cost estimates (i.e., estimates that are on the low side).

4.5. Summary of Key Assumptions

The estimation procedures described here involved several assumptions that are necessary because of data limitations. We summarize the key assumptions for the convenience of the reader and describe sensitivity analyses we will perform in §5.3 to address the effect of these assumptions on our aggregate estimates.

1. The meta-analysis computed pooled relative risks by combining study populations from various countries. The assumption is that these estimates are relevant to our U.S.-based target population. To test this assumption, we performed sensitivity analyses in which we restricted the studies for the meta-analysis calculations to populations drawn from G8 countries and high-income Organisation for Economic Co-operation and Development (OECD) countries (Sensitivity Analysis 2).

2. To generate relative risk estimates for the mortality outcome in our meta-analysis, we pooled studies that estimated the risks of all-cause mortality and cause-specific mortality. To test the effect of this assumption, we repeated our analysis but excluded studies with cause-specific mortality (Sensitivity Analysis 3).

3. We pooled studies using longitudinal and cross-sectional data to estimate the relative risks in the base model. Because cross-sectional data have limitations as outlined before, we conducted Sensitivity Analysis 4 to study how only our final estimates change if only studies that use longitudinal data are included in the meta-analytic sample.

4. In our base model, the meta-analytic sample contains studies that use either logistic regressions or Cox regressions. We test this assumption in Sensitivity Analysis 5, where we excluded studies that use Cox regressions.

5. To derive the relative risk estimates for *NOINSURE*, we included studies that group respondents with public insurance (Medicaid) together with the

Table 5 Estimates (and Standard Errors) of c, the Annual Incremental Cost of Poor Health Outcomes (in USD)

Gender	SR-P	SR-M	DX	MORT
Men	\$11,012 (\$1,155)	\$2,291 (\$476)	\$7,909 (\$309)	\$36,575 (\$13,012)
Women	\$9,564 (\$605)	\$3,075 (\$1,014)	\$7,436 (\$258)	\$24,130 (\$7,383)

uninsured. In Sensitivity Analysis 6, we excluded studies that performed this pooling.

6. Uninsurance was assumed to be independent of the remaining exposures for the employed subgroup. In Sensitivity Analysis 7, we extended the robustness analysis to allow correlation between these exposures and no insurance.

7. Our definition of physician-diagnosed medical condition included any respondent in the MEPS data who had one or more health conditions within a list of conditions. To test sensitivity to that assumption, we repeated our estimation, but varied the threshold of conditions present needed to determine whether someone had a physician-diagnosed medical condition (Sensitivity Analysis 8).

8. We pooled exposure prevalence data from 2002, 2006, and 2010 in our base model, which assumes that the exposures were similar in those years. We tested this assumption in Sensitivity Analysis 9, where we repeated the analysis separately for each year 2002, 2006, and 2010 by using exposure data that were specific to that year.

5. Results

In this section, we present the final estimates (and 95% confidence intervals) of the annual workplace-associated healthcare expenditure and mortality in the United States. Results are presented for both the multiplicative and conservative models. For each model, we present estimates using the empirical distribution of g as well as upper and lower bounds computed using the robustness analysis from §4.1. We then present results on the individual contribution of each of these exposures on mortality and costs and finally summarize the results from our sensitivity analysis.

5.1. Overall Estimates

Table 6 reports our estimates. There are some differences across the different methods we used to estimate the effects of workplace practices, but these differences are small. In all instances there are more than 120,000 excess deaths each year associated with the various workplace factors. There is more variation in the cost estimates, but once again the incremental costs are substantial, comprising 5%–8% of the total national healthcare expenditure in 2008.

5.2. Marginal Estimates

To estimate the healthcare expenditure and mortality associated with *each* workplace exposure, we proceeded as follows: For each $i = 1, \ldots, m$, we repeated our calculations with a new relative risk matrix $\mathbf{R}_{(i)}$ that replaced the entries to all rows except i with 1 and retained the original values for row i. Table 7 reports our estimates. Unlike the aggregate estimates, for the estimates of individual effects there is no need for multiple models and multiple estimates because there is no concern about double counting.

There are several observations that follow from these results.

1. Estimates generated by our model are consistent with estimates reported previously in the literature. In particular, our results show that not having insurance is associated with about 50,000 excess deaths per year, a number quite close to the 45,000 reported by Wilper et al. (2009). This provides some confidence that our other estimates, derived and presented here for the first time, are likely to be reliable.

2. Absence of insurance contributes the most toward excess mortality, followed closely by unemployment. Low job control is, however, also an important factor contributing an estimated 31,000 excess deaths annually.

3. Not having health insurance, being in jobs with high demands, and work–family conflict are the major exposures that contribute to healthcare expenditures.

4. The exposures that contribute the most to healthcare expenditures differ from the highest contributors to mortality. This is because incremental costs stop when someone dies, so exposures with higher deaths are not necessarily associated with higher costs.

5. Although each of the exposures contributes to healthcare expenditure, not all of them contribute, at least from our estimates, to incremental deaths. This is partly due to data limitations: our analysis excluded relative risk estimates that were generated only by two or fewer studies. From Table 3, we observe that several exposures for mortality fall into this category.

6. Because of the nonlinear manner in which each workplace exposure contributes to the final estimate of either expenditure or mortality, the sum of the marginal contributions from each exposure does not add up to the totals reported in Table 6.

Table 6 **Point Estimates (and 95% Confidence Intervals) of Incremental Healthcare Cost and Mortality in the United States, Expressed in Billions of USD and Thousands of People, Respectively, Associated with Workplace Exposures**

Factor	Model	Optimization (min)	Empirical	Optimization (max)
Cost	Conservative	\$117 (\$104, \$138)	\$125 (\$111, \$145)	\$134 (\$119, \$153)
	Multiplicative	\$186 (\$164, \$209)	\$187 (\$166, \$211)	\$190 (\$168, \$214)
Mortality	Conservative	122 (89, 193)	127 (97, 199)	132 (103, 203)
	Multiplicative	141 (74, 224)	141 (74, 224)	142 (74, 225)

Table 7 Point Estimates (and 95% Confidence Intervals) of Incremental Healthcare Cost and Mortality in the United States, Expressed in Billions of USD and Numbers of People, Respectively, Associated with Each Workplace Exposures

Exposure	Cost (in billions)	Mortality (in thousands)
UNEMPLOY	\$15 (\$9, \$20)	34 (26, 41)
NOINSURE	\$40 (\$25, \$57)	49 (35, 64)
SHIFTWORK	\$12 (\$8, \$16)	12 (−12, 36)
LONGHRS	\$13 (\$2, \$24)	—
INSECURE	\$16 (\$11, \$22)	29 (−21, 97)
WFC	\$24 (\$19, \$30)	—
LOCONTROL	\$11 (\$9, \$14)	31 (20, 44)
HIDEMAND	\$48 (\$38, \$58)	−2 (−45, 25)
LOSUPP	\$9 (\$7, \$12)	3 (−9, 17)
LOFAIR	\$16 (\$12, \$21)	—

Note. A dash indicates insufficient relative risk data to estimate the entry.

The reader should also observe that the relative magnitudes of the effects presented in Table 7 are interesting in their own right and can provide some guidance to where employers and public policy might effectively focus attention to reduce healthcare costs and unnecessary deaths that derive from workplace practices and the decisions that produce them. For example, one specific implication of our results is that providing universal health insurance, which is one of the objectives of the Affordable Care Act, could potentially reduce excess mortality in the United States. Although our model does not indicate the mechanism through which this mortality reduction occurs, there are many other studies in the literature that do, and we reviewed two of these mechanisms in §2.

5.3. Sensitivity Analyses

We conducted nine sensitivity analyses to investigate our model's robustness to its implicit modeling assumptions.

Sensitivity Analysis 1: *Varying the meta-analysis scoring rubric*. To investigate the effect of varying the scoring rubric used to assess study quality, we repeated the analyses using the scoring rubrics P1, P2, and P3 listed in Table 2.

Sensitivity Analysis 2: *Excluding countries from the meta-analytic sample*. We performed two analyses: one in which we included only countries in the "group of eight" (G8), and one in which we include countries that belong to the 31 high-income OECD economies, as classified by the World Bank (2012).

Sensitivity Analysis 3: *Excluding cause-specific mortality*. For this analysis, we retained only studies that measured all-cause mortality.

Sensitivity Analysis 4: *Excluding cross-sectional data*. For this analysis, we excluded studies that used cross-sectional data. This exclusion causes many entries of the relative risk matrix to have either zero or too few studies for estimation, resulting in the default (conservative) value of 1 being used. Therefore, we also performed an additional analysis that substitutes the missing values with estimates from the original relative matrix (that were estimated by using both longitudinal and cross-sectional data).

Sensitivity Analysis 5: *Excluding Cox regression models*. For this analysis, we investigated the effect of retaining only studies that used logistic regression for their analysis. Because this exclusion caused several entries of the relative risk matrix to have too few studies to be estimated, we performed an additional analysis by substituting these missing entries in the same manner as in Sensitivity Analysis 4.

Sensitivity Analysis 6: *Excluding public insurance*. For this analysis, we excluded studies where the respondents had public (Medicaid) insurance from the meta-analytic sample.

Sensitivity Analysis 7: *Allowing NOINSURE to correlate with other exposures*. We relaxed this assumption through a modified robustness analysis (in §4.1) that models unknown correlations between exposures.

Sensitivity Analysis 8: *Varying the DX threshold*. For this analysis, we investigated the effect of increasing the value of the threshold used to define when someone has physician-diagnosed medical conditions. As the threshold increases, fewer people are classified as "cases," i.e., the status quo prevalence of DX decreases, but these "cases" are more expensive.

Sensitivity Analysis 9: *Stratifying by exposure years*. For this analysis, we investigated the effect of repeating our analysis with separate exposure distributions for the years 2002, 2006, and 2010.

Tables 8 and 9 respectively present the differences in estimated workplace-associated healthcare expenditure and mortality from these analyses, relative to the base model estimates. Most of the results show only modest changes to our estimates relative to the base, which suggests that our base model is robust to variations in its modeling assumptions.

For the expenditure estimates, the largest difference occurs when the meta-analytic sample is restricted to studies using longitudinal data (Sensitivity Analysis 4). This is because this criterion substantially shrinks the meta-analytic sample size, and many entries of the relative risk matrix are therefore estimated by two studies or less and substituted with the default conservative value of 1. Specifically, out of the 80 entries of the relative risk matrix (10 exposures × 4 outcomes × 2 genders), 37 entries are substituted with the default value under this restriction compared with 16 entries in the base model. However, even in this case, the healthcare cost that is associated with workplace exposures is still significant: \$48 billion in the conservative model with minimum bound. The next row of the table (longitudinal with substitution)

Table 8 Estimated Annual Expenditures for Each Sensitivity Analysis, Expressed as a Percentage (%) of the Estimated Annual Expenditure from the Base Model

	Expenditure					
	Conservative optimization (min)	Conservative empirical	Conservative optimization (max)	Multiplicative optimization (min)	Multiplicative empirical	Multiplicative optimization (max)
Sensitivity Analysis 1: Varying the meta-analysis scoring rubric						
Penalty P1	94 (88, 101)	94 (90, 101)	94 (90, 101)	94 (89, 100)	94 (89, 100)	94 (89, 100)
Penalty P2	102 (99, 104)	102 (99, 104)	102 (99, 104)	103 (100, 105)	103 (101, 105)	103 (101, 105)
Penalty P3	101 (100, 101)	101 (100, 101)	101 (100, 101)	101 (101, 101)	101 (101, 101)	101 (101, 101)
Sensitivity Analysis 2: Excluding countries from the meta-analytic sample						
Only G8	79 (72, 85)	77 (70, 83)	75 (68, 81)	68 (61, 74)	67 (60, 74)	67 (60, 73)
Only high-income OECD	103 (101, 105)	103 (101, 105)	103 (101, 104)	103 (101, 104)	103 (101, 104)	103 (101, 104)
Sensitivity Analysis 3: Excluding cause-specific mortality						
Only all-cause mortality	100 (99, 100)	100 (99, 100)	100 (99, 100)	100 (99, 100)	100 (99, 100)	100 (99, 100)
Sensitivity Analysis 4: Excluding cross-sectional data						
Only longitudinal	40 (31, 51)	39 (30, 49)	38 (29, 48)	33 (24, 42)	33 (24, 41)	32 (24, 41)
Longitudinal with substitution	92 (87, 96)	90 (86, 94)	89 (85, 93)	85 (79, 89)	84 (79, 89)	84 (79, 89)
Sensitivity Analysis 5: Statistical method used						
Only logistic	93 (83, 102)	93 (84, 101)	92 (84, 99)	91 (84, 97)	91 (84, 97)	91 (84, 97)
Logistic with substitution	104 (103, 105)	103 (102, 105)	103 (102, 104)	102 (100, 103)	102 (100, 103)	102 (100, 103)
Sensitivity Analysis 6: Excluding public insurance						
Exclude public insurance	91 (81, 101)	91 (82, 100)	91 (82, 99)	91 (84, 98)	91 (84, 98)	91 (84, 98)
Sensitivity Analysis 7: Allowing *NOINSURE* to correlate with other exposures						
Nonindependent insurance	84 (76, 91)	†	103 (102, 104)	85 (79, 90)	†	104 (102, 105)
Sensitivity Analysis 8: Varying the DX threshold						
Threshold = 2	100 (98, 102)	100 (98, 101)	99 (97, 101)	99 (97, 100)	99 (97, 101)	99 (97, 101)
Threshold = 3	87 (85, 89)	87 (85, 89)	86 (84, 88)	86 (84, 89)	87 (85, 89)	87 (85, 89)
Threshold = 4	78 (75, 82)	78 (75, 81)	77 (74, 81)	79 (75, 82)	79 (75, 82)	79 (76, 83)
Sensitivity Analysis 9: Only using exposures from year						
Year 2002	97 (96, 98)	97 (96, 98)	97 (96, 98)	98 (98, 99)	99 (98, 99)	98 (98, 99)
Year 2006	98 (97, 99)	98 (97, 98)	98 (97, 98)	96 (95, 96)	95 (95, 96)	95 (95, 96)
Year 2010	101 (100, 102)	101 (100, 101)	100 (100, 101)	102 (102, 103)	102 (102, 103)	102 (102, 103)

†There is no entry because this sensitivity analysis does not have a well-defined empirical estimate for g.

shows a much smaller reduction. This points to the possibility that the large reduction could be a result of insufficient studies that use longitudinal data. The next largest difference occurs for the analysis that restricts the meta-analytic sample to only G8 countries (Sensitivity Analysis 2), which is also a consequence of substitution of the default value for several entries. All other sensitivity analyses generate much smaller changes in the cost estimates.

For the mortality estimates, we note that the greatest different occurs when the meta-analytic sample excludes studies that use Cox regression models. As above, this difference is driven by a small sample size and default value substitution because many mortality studies in our base sample use Cox regression models as their statistical method of choice instead of logistic regression. These studies are removed from the sample by this restriction. Specifically, out of the 20 entries for the mortality outcome in the relative risk matrix, 17 entries are substituted with the default value under this restriction compared with 7 entries in the base model. Because this restriction results in such a small sample size, we do not think that the estimate under the restriction is reliable. The next largest differences are associated with (a) changing the scoring rubric for the meta analyses that led to the exclusion of a large number of studies; (b) exclusion from the analysis of data from studies on less economically advanced countries; and (c) assuming correlation between lack of insurance and other exposures. Among these analyses, even in the worst case, there were still a total of 93,000 deaths associated with workplace exposures. We note that Sensitivity Analyses 4 and 8 generate results identical to those of the base model. This is because all the studies that measure the outcome of mortality in our sample use longitudinal data, and because the mortality estimates are not affected by the DX threshold.

Finally, we observe some interesting results when exposures from specific years are used (Sensitivity

Table 9 Estimated Annual Mortality for Each Sensitivity Analysis, Expressed as a Percentage (%) of the Estimated Annual Mortality from the Base Model

	Mortality					
	Conservative optimization (min)	Conservative empirical	Conservative optimization (max)	Multiplicative optimization (min)	Multiplicative empirical	Multiplicative optimization (max)
Sensitivity Analysis 1: Varying the meta-analysis scoring rubric						
Penalty P1	106 (98, 126)	105 (95, 127)	104 (95, 127)	106 (86, 129)	106 (85, 129)	106 (85, 129)
Penalty P2	71 (42, 107)	69 (42, 106)	68 (42, 104)	66 (22, 115)	66 (22, 115)	66 (22, 115)
Penalty P3	100 (100, 100)	100 (100, 100)	100 (100, 100)	100 (100, 100)	100 (100, 100)	100 (100, 100)
Sensitivity Analysis 2: Excluding countries from the meta-analytic sample						
Only G8	80 (58, 98)	76 (56, 91)	73 (53, 88)	78 (52, 110)	78 (52, 110)	78 (52, 109)
Only high-income OECD	84 (57, 100)	85 (59, 102)	84 (59, 101)	82 (52, 124)	82 (52, 124)	82 (52, 124)
Sensitivity Analysis 3: Excluding cause-specific mortality						
Only all-cause mortality	91 (74, 108)	91 (75, 105)	90 (75, 104)	87 (66, 109)	87 (66, 109)	86 (66, 109)
Sensitivity Analysis 4: Excluding cross-sectional data						
Only longitudinal	No change					
Longitudinal with substitution	No change					
Sensitivity Analysis 5: Statistical method used						
Only logistic	38 (23, 55)	41 (25, 57)	41 (26, 58)	28 (12, 50)	28 (12, 50)	28 (12, 50)
Logistic with substitution	107 (98, 115)	109 (100, 116)	109 (101, 116)	100 (89, 112)	100 (89, 112)	100 (89, 112)
Sensitivity Analysis 6: Excluding public insurance						
Exclude public insurance	99 (97, 100)	99 (98, 100)	99 (98, 100)	99 (97, 100)	99 (97, 100)	99 (97, 100)
Sensitivity Analysis 7: Allowing *NOINSURE* to correlate with other exposures						
Nonindependent insurance	77 (60, 89)	†	103 (101, 106)	71 (46, 85)	†	106 (103, 109)
Sensitivity Analysis 8: Varying the DX threshold						
Threshold = 2	No change					
Threshold = 3	No change					
Threshold = 4	No change					
Sensitivity Analysis 9: Only using exposures from year						
Year 2002	97 (91, 101)	98 (94, 100)	98 (95, 101)	98 (91, 101)	98 (91, 101)	98 (91, 101)
Year 2006	95 (91, 98)	95 (94, 97)	95 (94, 98)	93 (91, 96)	93 (91, 96)	93 (91, 96)
Year 2010	101 (96, 104)	101 (98, 103)	100 (98, 103)	101 (96, 104)	101 (96, 104)	101 (96, 104)

†There is no entry because this sensitivity analysis does not have a well-defined empirical estimate for g.

Analysis 9). Our model estimates significantly lower workplace-associated expenditures and mortality for 2006, which is when the U.S. economy was doing well, relative to estimates in the base model and for 2010, when the U.S. economy was bruising from the global financial crisis. The estimates for 2002 were moderately lower, which was around the time of an economic recession in many developed countries. Overall, these results corroborate the intuition that people experience greater workplace stressors during times of economic turbulence, and that these can have significant impact on health costs and outcomes. This suggests that workplace exposures could be used to better understand how the economic climate affects health, which is a subject that is an interesting direction for future research.

6. Discussion

We have seen that employer decisions about work and the workplace are associated with excess deaths and healthcare costs in the United States. To put our results in perspective, our model's estimate of workplace-associated mortality is comparable to the fourth (cerebrovascular diseases) and fifth (accidents) largest causes of death in the United States in 2009 (Kochanek et al. 2011, Table B), and exceeds the number of deaths from diabetes, Alzheimer's, or influenza. Our model also estimates that the workplace-associated healthcare cost is comparable to the estimated cost of diabetes in the United States in 2007 (Centers for Disease Control and Prevention 2011), which was $174 billion.

Our analysis is conservative in several ways. First, it only estimates the costs and morbidity for the individual in the workplace who faces the actual exposure, and does not account for any health consequences to the individual's social network. For example, a stressed worker might abuse alcohol or tobacco, which are well-known risk factors for detrimental psychological and physical health in his or her family

members (e.g., Wand et al. 1998, Graham 1987). Second, our present analysis only considers the direct association between workplace practices and healthcare costs and morbidity. Specifically, we have not yet attempted to model or estimate the association of these employer practices to other costly outcomes such as reduced employee productivity (e.g., Burton et al. 1999, Burton et al. 2005), absenteeism and its costs (e.g., Wright et al. 2002), or worker compensation expenses (Musich et al. 2001). The existing literature suggests that these additional costs are likely to be substantial. Further research that account for these factors would provide a more accurate estimate of the actual association of workplace stressors and health and health costs.

Our analysis is not without limitations. Some of these limitations could cause our final estimates to be artificially inflated. The first and overarching limitation stems from the lack of a comprehensive, longitudinal data set of employees, their workplace exposures, medical outcomes, and costs. Many of the leading biopsychosocial surveys have measures of important life transitions, social relationships, demographics, health behaviors, and health outcomes, but do not collect measures of respondents' work environments (Mikulak 2011). We attempted to overcome this data limitation through analytical modeling and numerical sensitivity analyses, but cannot completely eliminate the possibility of model misspecification and its associated biases. A second limitation is that effect sizes estimated by our meta-analysis could be overly optimistic (i.e., too large). The epidemiological studies that we used are observational by design, and we cannot conclusively rule out the possibility of selection biases that could affect the estimates. Adjustment for covariates can partially mitigate this problem, and we attempted to overcome this limitation by decreasing the quality scores for studies that did not adjust for sufficient covariates. A third limitation relates to our model's handling of layoffs, an important workplace exposure. Evidence suggests that the adverse health effects of job loss persist even after people find new jobs (Eliason and Storrie 2009, Strully 2009). Because the effect of layoffs persists over time, we approximated it by incorporating "unemployment" as one of the exposures. Unemployment partly captures the effect of layoffs, but it ignores a possible persistence of the layoff effect after the employees regain employment. On the other hand, unemployment also includes the effect of structural unemployment that is caused by macroeconomic conditions and is not solely the consequence of what happens to people in the workplace. Therefore, unemployment is an imperfect proxy for the effect of layoffs. Additional work on developing better proxies is a worthwhile research endeavor.

Nonetheless, despite these limitations, it seems difficult to ignore the overall implication of our findings—stressors in the work environment are closely connected to health outcomes and costs. Moreover, the estimated effect of these workplace stressors is substantially large, with the number of deaths associated with such stressors exceeding the number of deaths from diabetes, for instance, and with a reasonable estimate of the total costs incurred in excess of $180 billion. Therefore, our analysis suggests that these stressors could potentially be fruitful avenues for policy attention to improve health outcomes and costs.

It is important to note that we do not claim that an ideal stress-free workplace (i.e., one where the prevalence of all of these workplace exposures is zero) is realistically or economically achievable, even though some of these exposures (e.g., related to job control, demands, social support, organizational fairness) are elements of an organization's work environment that seem like they could reasonably be improved (e.g., by better management of human resources) without significant detriment to the organization's functions. Instead, we pursue our analysis in the same spirit as Keeney (2008), who argued that about half of all deaths for people in the United States (aged 15–64) stem from personal decisions. He did not claim that there is a realistic way to achieve an ideal society with perfect decision making that can avoid these deaths. Rather, he pointed to personal decisions as an important factor that policy attention could target, and he further highlighted specific avenues (e.g., educating people about drunk driving) that seem promising for effecting change. Similarly, we do not claim that the ideal workplace is attainable, but rather, our analysis highlights the workplace as an important source of stressors that are associated with poor health, and also suggests specific stressors that can be targeted to improve health. Even though it is likely that these stressors cannot be completely eradicated in practice, our analysis suggests that even reducing their prevalence could potentially go a long way in improving health outcomes and cost, and we hope that this will encourage further research into specific management practices that can be put in place to mitigate these stressors.

7. Managerial and Policy Implications

Our models can potentially be used to understand other healthcare issues and also to develop policy recommendations for affecting employer behavior. First, consider the recent attention to the large inequalities in health indicators (e.g., life expectancy, infant mortality) that are known to exist between individuals

with high and low levels of income and education in the United States (e.g., Geronimus et al. 2001, Singh and Siahpush 2006). Addressing this inequality in health outcomes has been a growing focus of public attention (e.g., Deaton and Paxson 1998). Our results suggest that exposure to workplace stressors is a plausible pathway for these observed inequalities: Poor, less educated people not only suffer health consequences because of the direct relationship of health status to education and income (e.g., Marmot 2004), but also because these people tend to work in jobs that have higher exposure to these stressors. For instance, jobs with higher levels of discretion and control are more likely to be held by more educated individuals. Workers that are more educated and more highly paid are more likely to receive health insurance benefits and avail themselves to employer-offered health insurance. Consequently, to understand inequalities in health status, one needs to consider the different working conditions confronted by people with various characteristics as part of the explanatory story.

Second, the distribution and incidence of these workplace exposures, which undoubtedly vary across industries and also by the degree of union coverage of the work force, may help explain the variation in health outcomes across different geographies. And this variation would include the potential for partially explaining the differences between the United States and other industrialized countries in terms of the amount spent compared to the health outcomes obtained. In general, other OECD countries have more regulated labor markets and working conditions, and also afford stronger social safety nets. So, although economic insecurity and job conditions including work hours would undoubtedly have similar effects regardless of where they were experienced, the incidence of long hours, layoffs, and job control could vary in ways that would help explain the difference in health outcomes per dollar between the United States and other countries. This is a subject clearly worthy of further empirical examination, as cross-national variations in health status constitute an important topic of public policy discussion.

Third, for employees working in organizations where they are covered by employer-provided health plans and subject to cost shifting from employers to workers, the cost of management practices that harm health (and drive up expenditures) are borne by the employers, particularly to the extent that they are either self-insured or subject to ratings that reflect the health cost experience of their employees. Nevertheless, employers may not take appropriate decisions concerning workplace management if they are unaware of the link between management decisions and employee health and healthcare costs. Our analysis suggests that for such organizations, paying attention to the structure of the workplace and the associated job stressors experienced by their employees may be a fruitful way to reduce unnecessary healthcare costs. However, as the 2011 Kaiser Family Foundation survey of health benefits noted, some 42% of workers are not covered by healthcare plans offered by their employers. In these instances, and also in the case of laid-off employees who suffer adverse health effects but are no longer employed and whose costs post-layoff therefore do not fall on any employer, there is little economic incentive for employers to take the cost or mortality implications of their decisions into account. Simply put, some considerable fraction of the adverse health costs and mortality caused by workplace practices are undoubtedly externalized and borne by the larger society, but not reflected in the costs incurred by the employers who actually make the workplace choices. As in the case of air or water pollution, when costs are externalized, decision makers do not have accurate prices for the consequences of their decisions and are therefore likely to underinvest in actions that would reduce real costs that they do not bear. In the case of the physical environment, both pricing and regulatory regimes have been developed so that decision makers confront prices and information that more completely and accurately reflect the costs of various alternatives. A similar situation would seem to apply to employee health. Unless and until employers see and incur the costs of their workplace decisions that affect employee health, it is unlikely that such decisions will be societally optimal.

8. Conclusion

People spend a lot of their waking hours at work. Therefore, work environments are consequential not just for stress and feelings of competence and control, but also as a locus of a person's identity and status. It is, therefore, scarcely surprising that the work environments created by employer decisions can have profound effects on mental and physical well-being and, consequently, morbidity, mortality, and healthcare costs. In both the analysis of healthcare outcomes and policies to affect healthcare costs and population health status, employer actions have thus far been largely missing from the picture. The results reported in this paper suggest that the association between employer actions and healthcare outcomes and costs is strong. Although we stop short of claiming that employer decisions have a definite effect on these outcomes and costs, denying the possibility of an effect is not prudent either. Analyzing how employers affect health outcomes and costs through the workplace decisions they make is incredibly important if we are to more fully understand the landscape of health and well-being.

Supplemental Material

Supplemental material to this paper is available at http://dx.doi.org/10.1287/mnsc.2014.2115.

Acknowledgments

The authors are grateful to department editor Dimitris Bertsimas, the anonymous associate editor, and four anonymous referees for their insightful comments and suggestions. The authors also thank Edward Kaplan and Scott Wallace for their helpful comments on an earlier version of this paper. This paper greatly improved as a result of their feedback.

Appendix A. Proof of Theorem 1

We only prove the case of the upper bound. The lower bound follows by an identical argument. Define $\underline{\Theta}_{\mathbf{w}}$ as

$$\begin{aligned}\underline{\Theta}_{\mathbf{w}} := \min_{u} \quad & \sum_{j=1}^{n} \frac{w_j p_j}{\sum_{\mathbf{x}\in\{0,1\}^m} f_j(\mathbf{x})u(\mathbf{x})} \\ \text{s.t.} \quad & \mathcal{L}_{ik} u = v_{ik} \quad i,k \in \{1,\dots,m\}, \\ & \sum_{\mathbf{x}\in\{0,1\}^m} u(\mathbf{x}) = 1, \\ & u(\mathbf{x}) \geq 0.\end{aligned} \tag{A1}$$

Clearly, $\underline{\Theta}_{\mathbf{w}} \leq \langle \mathbf{w}, \mathbf{p}\rangle - \Delta_{\mathbf{w}}$, because the true distribution g is a feasible choice of the decision variable u in problem (A1), and their objectives coincide. Rearranging this inequality yields $\Delta_{\mathbf{w}} \leq \langle \mathbf{w}, \mathbf{p}\rangle - \underline{\Theta}_{\mathbf{w}}$.

It remains to show that $\underline{\Gamma}_{\mathbf{w}} \leq \underline{\Theta}_{\mathbf{w}}$. We will now reformulate (A1) in two steps. First, we construct an auxiliary decision variable $\mathbf{t} := (t_j)_{j=1}^n$, $\mathbf{t} \geq \mathbf{0}$, where, for each $j \in \{1,\dots,n\}$,

$$t_j = \frac{1}{\sum_{\mathbf{x}\in\{0,1\}^m} f_j(\mathbf{x})u(\mathbf{x})}. \tag{A2}$$

We may rearrange (A2) and write it as

$$\sum_{\mathbf{x}\in\{0,1\}^m} t_j f_j(\mathbf{x}) u(\mathbf{x}) = 1.$$

Second, we introduce auxiliary decision variables u_j that are all equal to u. Without loss, we may set $u = u_1$.

Putting both these constructs together yields the optimization problem

$$\begin{aligned}\underline{\Theta}_{\mathbf{w}} = \min_{u_j,\, \mathbf{t}\geq\mathbf{0}} \quad & \sum_{j=1}^{n} w_j p_j t_j \\ \text{s.t.} \quad & \sum_{\mathbf{x}\in\{0,1\}^m} f_j(\mathbf{x}) t_j u_j(\mathbf{x}) = 1 \quad j \in \{1,\dots,n\}, \\ & \mathcal{L}_{ik} u_j = v_{ik} \quad j \in \{1,\dots,n\}, \\ & \qquad\qquad i,k \in \{1,\dots,m\}, \\ & \sum_{\mathbf{x}\in\{0,1\}^m} u_j(\mathbf{x}) = 1 \quad j \in \{1,\dots,n\}, \\ & u_j(\mathbf{x}) \geq 0 \quad j \in \{1,\dots,n\},\ \mathbf{x} \in \{0,1\}^m, \\ & u_j(\mathbf{x}) = u_{j'}(\mathbf{x}) \quad j,j' \in \{1,\dots,n\},\ \mathbf{x} \in \{0,1\}^m.\end{aligned} \tag{A3}$$

From (A3), we define a new decision variable q_j, with $q_j = t_j u_j$ pointwise, and we remove the final constraint that all the u_j have to be equal. We note that the relaxed problem is exactly the required LP (9)–(13). Hence, the LP (9)–(13) is indeed a relaxation of (A3), and consequently $\underline{\Gamma}_{\mathbf{w}} \leq \underline{\Theta}_{\mathbf{w}}$ holds.

Appendix B. Econometric Models for Meta-Analysis

Our meta-analytic sample primarily contains studies that use logistic regression as their statistical model, which we proceed to describe. Suppose we have a binary adverse health outcome of interest, denoted by H; a binary exposure of interest, denoted by X_1; and $p-1$ controls, denoted by $X_2,\dots,X_p$. We collect studies that use a logistic regression model; that is, it assumes that the conditional probability $\mathbb{P}(H \mid X_1,\dots,X_p)$ has a parametric structure given by

$$\frac{\mathbb{P}(H \mid X_1,\dots,X_p)}{1-\mathbb{P}(H \mid X_1,\dots,X_p)} = \exp(\beta_0 + \beta_1 X_1 + \cdots + \beta_p X_p). \tag{B1}$$

The odds ratio for the exposure–outcome pair is $\exp(\beta_1)$, which is what we extract from the study. The same structure applies for studies using either cross-sectional or longitudinal data. However, for the latter, the predictor variables are assessed at a time point called *baseline*, whereas the health outcome H is assessed at a later time point called *follow-up*.

Our meta-analytic sample also contains studies using longitudinal data that apply the Cox proportional hazards model as their statistical model, which we presently describe. Let τ be a random variable that represents the random occurrence time of the adverse health outcome. We assume that τ is positive with probability 1 and has a density, which is denoted by $f\colon \mathbb{R}_+ \to \mathbb{R}_+$. Let $\lambda\colon \mathbb{R}_+ \to \mathbb{R}_+$ represent the hazard rate function for τ, which is defined as $\lambda(t) := f(t)/\mathbb{P}(\tau > t)$ for all $t \geq 0$, and is related to the survival function, $\mathbb{P}(T > t)$, through the expression $\mathbb{P}(\tau > T) = \exp(-\int_0^T \lambda(t)\,dt)$. The Cox proportional hazards model assumes that there exists a positive-valued function $\lambda_0(t)$, whose structure is unspecified, such that

$$\lambda(t) = \lambda_0(t)\exp(\beta_1 X_1 + \cdots + \beta_p X_p). \tag{B2}$$

We now derive the connection between the Cox and logistic models. Given the setup of the Cox model, further suppose that baseline is defined as time 0, and that follow-up is defined as a constant time point T. Then, we can define event H in terms of τ by as $H = \{\tau \leq T\}$, and the odds ratio on the left-hand side of Equation (B1) can be written in terms of the hazard function of τ as

$$\frac{\mathbb{P}(\tau \leq T \mid X_1,\dots,X_p)}{1-\mathbb{P}(\tau \leq T \mid X_1,\dots,X_p)} = \exp\left(\int_0^T \lambda(t)\,dt\right) - 1,$$

by (B2). Next, by the first-order approximation $e^x - 1 \approx x$, we get $\exp(\int_0^T \lambda(t)\,dt) - 1 \approx \int_0^T \lambda(t)\,dt = (\int_0^T \lambda_0(t)\,dt)\exp(\beta_1 X_1 + \cdots + \beta_p X_p)$. Finally, by defining $\beta_0 = \log(\int_0^T \lambda_0(t)\,dt)$, we obtain

$$\frac{\mathbb{P}(\tau \leq T \mid X_1,\dots,X_p)}{\mathbb{P}(\tau > T \mid X_1,\dots,X_p)} \approx \exp(\beta_0 + \beta_1 X_1 + \cdots + \beta_p X_p).$$

Therefore, the Cox proportional hazards model may be viewed (at least to the first order) as an approximation to the logistic regression model.

References

Agency for Healthcare Research and Quality (2011a) Medical expenditure panel survey. Accessed November 7, 2011, http://www.meps.ahrq.gov/.

Agency for Healthcare Research and Quality (2011b) Total health services—Mean and median expenses per person with expense and distribution of expenses by source of payment: United States, 2008. Medical Expenditure Panel Survey, Household Component data. Generated interactively. Accessed November 7, 2011, http://meps.ahrq.gov/mepsweb/data_stats/quick_tables.jsp.

Appleby J, Carty SS (2005) Ailing GM looks to scale back generous health benefits. *USA Today* (June 23), http://usatoday30.usatoday.com/money/autos/2005-06-22-gm-healthcare-usat_x.htm.

Barnett RC (2006) Relationship of the number and distribution of work hours to health and quality-of-life (QOL) outcomes. *Research in Occupational Stress and Well-Being*, Vol. 5 (Emerald Group Publishing, New York), 99–138.

Boole G (1854) *An Investigation of the Laws of Thought: On Which Are Founded the Mathematical Theories of Logic and Probabilities*, Vol. 2 (Walton and Maberly, London).

Broadhead WE, Kaplan BH, James SA, Wagner EH, Schoenbach VJ, Grimson R, Heyden S, Tibblin G, Gehlbach SH (1983) The epidemiologic evidence for a relationship between social support and health. *Amer. J. Epidemiology* 117(5):521–537.

Burton WN, Conti DJ, Chen CY, Schultz AB, Edington DW (1999) The role of health risk factors and disease on worker productivity. *J. Occupational Environ. Med.* 41(10):863–877.

Burton WN, Chen CY, Conti DJ, Schultz AB, Pransky G, Edington DW (2005) The association of health risks with on-the-job productivity. *J. Occup. Environ. Med.* 47(8):769–777.

Cardarelli KM, Low MD, Vernon SW, Preacely N, Baumler ER, Tortolero S (2009) Critical review of the evidence for the connection between education and health: A guide for exploration of the causal pathways. *Research in the Sociology of Health Care* (Emerald Group Publishing, New York), 137–159.

Centers for Disease Control and Prevention (2011) National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011. Accessed November 28, 2011, http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011.pdf.

Centers for Medicare and Medicaid Services (2011) NHE web tables. Accessed November 21, 2011, https://www.cms.gov/NationalHealthExpendData/downloads/tables.pdf.

Chandola T, Britton A, Brunner E, Hemingway H, Malik M, Kumari M, Badrick E, Kivimaki M, Marmot M (2008) Work stress and coronary heart disease: What are the mechanisms? *Eur. Heart J.* 29(5):640–648.

Cohen S, Wills TA (1985) Stress, social support, and the buffering hypothesis. *Psychol. Bull.* 98(2):310–357.

Deaton AS, Paxson CH (1998) Aging and inequality in income and health. *Amer. Econom. Rev.* 88(2):248–253.

Dooley D, Catalano R, Wilson G (1994) Depression and unemployment: Panel findings from the Epidemiologic Catchment Area study. *Amer. J. Commun. Psychol.* 22(6):745–765.

Eliason M, Storrie D (2009) Does job loss shorten life? *J. Human Resources* 44(2):277–302.

Fleiss JL, Berlin JA (2009) Effect sizes for dichotomous data. *The Handbook of Research Synthesis*, 2nd ed. (Russell Sage Foundation, New York), 237–253.

Franks P, Clancy CM, Gold MR (1993) Health insurance and mortality: Evidence from a national cohort. *J. Amer. Med. Assoc.* 270(6):737–741.

Frone MR (2000) Work–family conflict and employee psychiatric disorders: The National Comorbidity Survey. *J. Appl. Psych.* 85(6):888–895.

Frone MR, Russell M, Barnes GM (1996) Work–family conflict, gender, and health-related outcomes: A study of employed parents in two community samples. *J. Occup. Health Psych.* 1(1):57–69.

Frone MR, Russell M, Cooper ML (1997) Relation of work–family conflict to health outcomes: A four-year longitudinal study of employed parents. *J. Occup. Organ. Psych.* 70(4):325–335.

Galinsky EJ, Bond T, Kim SS, Backon L, Brownfield E, Sakai K (2005) *Overwork in America: When the Way We Work Becomes Too Much* (Families and Work Institute, New York).

Garrett AB, Clemans-Cope L, Masi P, Bovbjerg RR (2008) The Urban Institute's microsimulation model for reinsurance: Model construction and state-specific application. Technical report, Urban Institute, Washington, DC.

Geronimus AT, Bound J, Waidmann TA, Colen CG, Steffick D (2001) Inequality in life expectancy, functional status, and active life expectancy across selected black and white populations in the United States. *Demography* 38(2):227–251.

Goh J, Pfeffer J, Zenios SA (2015) Workplace stressors and health outcomes: Health policy for the workplace. *Behavioral Sci. Policy.* Forthcoming.

Graham H (1987) Women's smoking and family health. *Soc. Sci. Medicine* 25(1):47–56.

Greenhaus JH, Beutell NJ (1985) Sources of conflict between work and family roles. *Acad. Management Rev.* 10(1):76–88.

Harris MM, Fennell ML (1988) A multivariate model of job stress and alcohol consumption. *Soc. Quart.* 29(3):391–406.

Higgins JPT, Green S (2011) *Cochrane Handbook for Systematic Reviews of Interventions*, version 5.1.0 (The Cochrane Collaboration, London).

Himmelstein DU, Thorne D, Warren E, Woolhandler S (2009) Medical bankruptcy in the United States, 2007: Results of a national study. *Amer. J. Med.* 122(8):741–746.

Hutton DW, Tan DW, So SK, Brandeau ML (2007) Cost-effectiveness of screening and vaccinating Asian and Pacific Islander adults for Hepatitis B. *Ann. Internal Medicine* 147(7):460–469.

Idler EL, Benyamini Y (1997) Self-rated health and mortality: A review of twenty-seven community studies. *J. Health Soc. Behav.* 38(1):21–37.

Johnson JV, Lipscomb J (2006) Long working hours, occupational health and the changing nature of work organization. *Amer. J. Industrial Medicine* 49(11):921–929.

Kaiser Family Foundation (2011) Employer health benefits: 2011 annual survey. Kaiser Family Foundation, Menlo Park, CA, and Health Research and Educational Trust, Chicago. Last accessed March 5, 2015, https://kaiserfamilyfoundation.files.wordpress.com/2013/04/8225.pdf.

Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T (1981) Job decision latitude, job demands, and cardiovascular disease: A prospective study of Swedish men. *Amer. J. Public Health* 71(7):694–705.

Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL (1988) Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Amer. J. Public Health* 78(8):910–918.

Keeney R (2008) Personal decisions are the leading cause of death. *Oper. Res.* 56(6):1335–1347.

Kivimäki M, Vahtera J, Pentti J, Ferrie JE (2000) Factors underlying the effect of organisational downsizing on health of employees: Longitudinal cohort study. *British Medical J.* 320(7240):971–975.

Kochanek KD, Xu J, Murphy SL, Miniño AM, Kung H-C (2011) Deaths: Preliminary data for 2009. Technical report, National Center for Health Statistics, Hyattsville, MD.

Kouvonen A, Kivimäki M, Virtanen M, Pentti J, Vahtera J (2005) Work stress, smoking status, and smoking intensity: An observational study of 46 190 employees. *J. Epidemiology and Community Health* 59(1):63–69.

Ladabaum U, Chopra CL, Huang G, Scheiman JM, Chernew ME, Fendrick AM (2001) Aspirin as an adjunct to screening for prevention of sporadic colorectal cancer. A cost-effectiveness analysis. *Ann. Internal Medicine* 135(9):769–781.

Lee S, Colditz GA, Berkman LF, Kawachi I (2004) Prospective study of job insecurity and coronary heart disease in us women. *Ann. Epidemiology* 14(1):24–30.

Levine DP (1992) G.M. orders staff to pay part of health-care cost. *New York Times* (August 26), http://www.nytimes.com/1992/08/26/business/company-news-gm-orders-staff-to-pay-part-of-health-care-cost.html.

Marmot M (2004) *The Status Syndrome: How Social Standing Affects Our Health and Longevity* (Times Books, New York).

Marmot MG, Rose G, Shipley M, Hamilton PJ (1978) Employment grade and coronary heart disease in British civil servants. *J. Epidemiology Community Health* 32(4):244–249.

Marmot M, Feeney A, Shipley M, North F, Syme SL (1995) Sickness absence as a measure of health status and functioning: From the UK Whitehall II study. *J. Epidemiology Community Health* 49(2):124–130.

Marmot MG, Bosma H, Hemingway H, Brunner E, Stansfeld S (1997) Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 350(9073):235–239.

Mikulak A (2011) What keeps adults well? Using biopsychosocial surveys to understand well-being. *Observer* 24(8), http://www.psychologicalscience.org/index.php/publications/observer/2011/october-11/what-keeps-adults-well.html.

Musich S, Napier D, Edington DW (2001) The association of health risks with workers' compensation costs. *J. Occup. Environ. Med.* 43(6):534–541.

National Opinion Research Center (2011) General Social Survey. Accessed November 7, 2011, http://www3.norc.org/GSS+Website/.

OECD (Organization for Economic Cooperation and Development) (2011) Health at a glance 2011: OECD indicators (OECD Publishing, Paris).

Ozcan YA, Luke RD (1993) A national study of the efficiency of hospitals in urban markets. *Health Services Res.* 27(6):719–739.

Piazza PV, Le Moal M (1998) The role of stress in drug self-administration. *Trends Pharmacol. Sci.* 19(2):67–74.

Robbins JM, Ford MT, Tetrick LE (2012) Perceived unfairness and employee health: A meta-analytic integration. *J. Appl. Psych.* 97(2):235–272.

Selden TM, Sing M (2008) Aligning the Medical Expenditure Panel Survey to aggregate U.S. benchmarks. Working paper, Agency for Healthcare Research and Quality, Rockville, MD.

Shadish WR, Haddock CK (2009) Combining estimates of effect size. *The Handbook of Research Synthesis*, 2nd ed. (Russell Sage Foundation, New York), 257–278.

Shields M (2006) Stress and depression in the employed population. *Health Rep.* 17(4):11–29.

Sing M, Banthin JS, Selden TM, Cowan CA, Keehan SP (2006) Reconciling medical expenditure estimates from the MEPS and NHEA, 2002. *Health Care Financing Rev.* 28(1):25–40.

Singh GK, Siahpush M (2006) Widening socioeconomic inequalities in US life expectancy, 1980–2000. *Internat. J. Epidemiology* 35(4):969–79.

Sparks K, Cooper C, Fried Y, Shirom A (1997) The effects of hours of work on health: A meta-analytic review. *J. Occup. Organ. Psych.* 70(4):391–408.

Strully KW (2009) Job loss and health in the US labor market. *Demography* 46(2):221–246.

Sudano JJ Jr, Baker DW (2003) Intermittent lack of health insurance coverage and use of preventive services. *Amer. J. Public Health* 93(1):130–137.

Sullivan D, Von Wachter T (2009) Job displacement and mortality: An analysis using administrative data. *Quart. J. Econom.* 124(3):1265–1306.

Sverke M, Hellgren J, Näswall K (2002) No security: A meta-analysis and review of job insecurity and its consequences. *J. Occup. Health Psych.* 7(3):242–264.

Tsutsumi A, Kayaba K, Kario K, Ishikawa S (2009) Prospective study on occupational stress and risk of stroke. *Arch. Internat. Med.* 169(1):56–61.

U.S. Census Bureau (2012) CPS table creator. Current Population Survey, Annual Social and Economic Supplement. Accessed January 2, 2013, http://www.census.gov/cps/data/cpstablecreator.html.

Vegso S, Cantley L, Slade M, Taiwo O, Sircar K, Rabinowitz P, Fiellin M, Russi MB, Cullen MR (2007) Extended work hours and risk of acute occupational injury: A case-crossover study of workers in manufacturing. *Amer. J. Industrial Medicine* 50(8):597–603.

Virtanen M, Nyberg ST, Batty GD, Jokela M, Heikkilä K, Fransson EI, Alfredsson L, Bjorner JB, Borritz M, Burr H, et al. (2013) Perceived job insecurity as a risk factor for incident coronary heart disease: Systematic review and meta-analysis. *British Medical J.* 347, http://dx.doi.org/10.1136/bmj.f4746.

von Känel R, Mills PJ, Fainman C, Dimsdale JE (2001) Effects of psychological stress and psychiatric disorders on blood coagulation and fibrinolysis: A biobehavioral pathway to coronary artery disease? *Psychosomatic Medicine* 63(4):531–544.

Wand GS, Mangold D, El Deiry S, McCaul ME, Hoover D (1998) Family history of alcoholism and hypothalamic opioidergic activity. *Arch. General Psychiatry* 55(12):1114–1119.

Wennberg JE, Fisher ES, Baker L, Sharp SM, Bronner KK (2005) Evaluating the efficiency of California providers in caring for patients with chronic illness. *Health Affairs* 24(Suppl. 3):526–543.

Wilper AP, Woolhandler S, Lasser KE, McCormick D, Bor DH, Himmelstein DU (2009) Health insurance and mortality in US adults. *Amer. J. Public Health* 99(12):2289–2295.

Woolhandler S, Himmelstein DU (1988) Reverse targeting of preventive care due to lack of health insurance. *J. Amer. Med. Assoc.* 259(19):2872–2874.

Woolhandler S, Himmelstein DU (1991) The deteriorating administrative efficiency of the US health care system. *New Engl. J. Med.* 324(18):1253–1258.

Woolhandler S, Campbell T, Himmelstein DU (2003) Costs of health care administration in the United States and Canada. *New Engl. J. Med.* 349(8):768–775.

World Bank (2012) World bank list of economies (July 2012). Accessed January 2, 2013, http://data.worldbank.org/about/country-classifications/country-and-lending-groups.

Wright DW, Beard MJ, Edington DW (2002) Association of health risks with the cost of time away from work. *J. Occup. Environ. Med.* 44(12):1126–1134.

Yang H, Schnall PL, Jauregui M, Su TC, Baker D (2006) Work hours and self-reported hypertension among working people in California. *Hypertension* 48(4):744–750.

Zhang J, Yu KF (1998) What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *J. Amer. Med. Assoc.* 280(19):1690–1691.

Zywicki TJ (2005) An economic analysis of the consumer bankruptcy crisis. *Northwestern University Law Rev.* 99(4):1463–1541.