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NO PAIN, NO GAIN:
WORK DEMAND, WORK EFFORT, AND WORKER HEALTH

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ABSTRACT

We combine Danish data on individuals' health with Danish matched worker-firm data, and find: One, within job spells, as firm sales increases, workers log longer hours and experience higher probabilities of stress and depression, and heart diseases and strokes; Two, the effects of firm sales on adverse health outcomes are more pronounced for high-risk groups: older workers, job-strained workers, and those with long initial work hours; Three, the worker cohorts who experience large sales increases develop higher risks of sickness in subsequent quarters. These novel results suggest that work demand increases individuals' workplace stress and elevates their sickness risk. We then compute the marginal disutility of our sickness variables, and show that the average worker's ex-ante welfare loss due to higher sickness rates accounts for nearly one quarter of her earnings gains from rising firm sales.

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1. Introduction

How do changes in demand for work affect workers' health? Higher demand likely raises income, and many studies show that higher income or wealth leads to better health. Reinforcing this view, the recent literature on "deaths of despair" suggest a connection between declining economic opportunities and worsening health, as do papers that implicate mass layoffs, plant closures and import competition.[1] Yet, the evidence is mixed. Increasing demand for labor that requires greater work load may increase work stress, and a medical literature has shown that self-reported work hours and stress are correlated with coronary heart diseases, strokes, and even mortality.[2] In addition, at the population health level, mortality and correlates of bad health (e.g. smoking, excess weight, physical inactivity) have been shown to be pro-cyclical, even though the exact channels remain an open question.[3]

In this paper, we examine how changes in firm sales affect the health of its employees. To be specific, an increase in firm sales tends to increase its labor demand, and so increase its employees' work load. We examine a hypothesis that an increase in work load wears down an individual worker's health capital and so makes her more likely to become sick. We base this hypothesis on the following premises. Survey data consistently identify work as a leading cause of self-reported stress.[4] In addition, a large medical literature has identified job strain as a major risk factor for stress and depression, heart diseases, and strokes, where job strain is measured as high demand and low control at work in survey data (e.g. Kivimaki et al. 2012). A key question for high work demand is whether one has enough time to do work (e.g. Fransson et al. 2012). Following this medical literature, we focus on the sickness variables that relate to stress, depression, heart diseases and strokes.

To carry out our inquiry, we draw on Danish administrative data that match the universe of private-sector

[1] See Case and Deaton (2015) on "deaths of despair", Sullivan and von Wachter (2009) for mass layoffs, Browning and Heinesen (2012) for plant closure and McManus and Schaur (2015) for import competition.

[2] E.g. Fransson et al. (2016), Virtanen et al. (2012), and O'Reilly and Rosato (2013).

[3] See Ruhm (2000, 2003, 2005) for pro-cylicality of US mortality and specific channels, Stevens, Miller, Page and Filipski (2015), argue that Ruhm (2000)'s result for mortality is driven by staffing changes at nursing homes. See also Lindo (2013), Tekin, McClellan and Minyard (2013), and Coile, Levine and McKnight (2014).

[4] Some place work as the leading cause (e.g. https://www.webmd.com/balance/guide/causes-of-stress#1) while others put work closely behind financial problems (e.g. https://www.cbsnews.com/news/the-biggest-cause-of-stress-in-america-today/).

Danish firms to the population of Danish workers. The matched worker-firm data provide rich information about Danish firms' sales and their input uses, such as capital, employment, and skilled labor, as well as detailed coverage of Danish workers' socio-economic characteristics, such as earnings, age, work hours, and occupation. We then merge in additional administrative datasets that cover every individual worker's prescription drug purchases, including cost and drug type, and every individual worker's hospitalization records, with the corresponding diagnostic codes. This rich data on individuals' health is available to us because Danish health care is free and universal, and every individual has access to health care, regardless of income and employment status. Our data thus distinguish our work from previous research on health and labor market using U.S. data, where workers' access to health care is correlated with income and employment status (e.g. Currie and Madrian 1999), and enables us to separate the direct effect of work demand on health from an indirect effect operating through access to health care.

The comprehensive nature and panel structure of our Danish data are critical for our identification. First, we consistently track each worker and each firm over time, and so we condition on job-spell fixed effects in our estimation; i.e. the source of our variation is the change over time within a given worker-firm relationship. This approach allows us to control for the idiosyncratic and time-invariant factors that affect individuals' health, such as pre-natal and early-life environment, birth weight, and genetic differences.[5] Second, the richness of our data allows us to control for firms' input uses in our estimation, and so tighten the connection between increases in firm sales and increases in work demand. Third, the detailed coverage of worker characteristics gives us useful proxies for work load: individual worker's earnings, individual worker's total hours with her employer, including over-time hours, and the ratio of materials to labor hours at the firm level. Fourth, our health data allow us to link sickness directly to work load because diagnostic codes identify the hospitalizations that are caused by high work load. Fifth, some of our variables, including firm sales and worker sickness, are available at quarterly frequency, and these quarterly data enable us to do event studies, to clarify the timing of workers' sickness and their employers' sales increases.

[5] See Maccini and Yang (2000) and Black, Devereux and Salvanes (2007).

We find the following results using within-job-spell variation. First, workers log longer hours and obtain higher earnings as their employer experiences sales increases. Second, increases in firm sales also lead to higher probabilities that workers purchase anti-depressants (elasticity 0.054) and use heart-disease drugs (elasticity 0.05). Third, workers suffer higher incidences of high-work-load-related hospitalizations as firm sales increases (elasticity 0.39-0.42), and they also experience higher rates of hospitalizations due to heart attacks and strokes, and severe liver diseases, an indicator of alcoholism (elasticity 0.09-0.13). It is intuitive that we obtain the largest elasticity estimates for high-work-load hospitalizations, because they are the most closely related to workplace stress.

We then corroborate these results by investigating whether workers' sickness events happen after sales increases at their employers. We conduct our event study by using our quarterly data to perform multi-period difference-in-difference estimates (e.g. Jacobson, LaLonde and Sullivan 1993) that follow workers across job-spells. We find that the worker cohorts who experience large sales increases have elevated risks of anti-depressant uses, heart-disease-drug uses, and high-work-load hospitalizations in subsequent quarters. In other words, even though our sample, specification, and identification strategy are all different from our within-job-spell estimation, our results are qualitatively similar. In addition, the findings of our event study suggest that firm-level economic shocks may have longer-term effects on individual workers' health, beyond that experienced during a given job spell.

We next probe the mechanisms of our results, by examining the heterogeneous effects of firm sales across workers. Keep in mind that severe sickness events occur with low frequency in our data – for example, the probability each year that a given worker is hospitalized for a heart attack or a stroke is 0.16%. Our idea is that, if higher work load wears down workers' health capital and increases the probability of sickness, we are more likely to capture these effects when examining ex-ante-high-risk individuals, whose health capital is presumably already low before sales increases occur.

Our first measure of ex-ante high risk is age. We show that age is a significant risk factor for heart diseases, but not a risk factor for high-work-load hospitalizations. Consistent with this pattern, we find that the effects of

sales increases are much larger for older workers for heart-disease drugs and stroke hospitalizations, but statistically indistinguishable for older vs. younger workers for high-work-load hospitalizations. Notably, these results hold in both within-job-spell and event-study analyses. We construct two additional measures of ex-ante high risk following the medical literature: job strain and long initial work hours. We find that, when firms experience sales increases, the workers who worked long hours before the sales increase experience an increased incidence of anti-depressants use and heart-disease drug use. We also find that the effects of sales increases on high-work-load hospitalizations are stronger for job-strained workers. This result is intuitive, because a key question in the job-strain measure is, "Do you have enough time to work".

In order to quantify the economic significance of our results, we develop a highly stylized model where the average worker derives expected utility from healthy and sick states, and optimally chooses treatment upon entering a sick state. Her marginal disutility for disease *g* (with treatment) is the monetary compensation she demands in exchange for heightened risk of *g*. We show that, relative to disease *l*, the marginal disutility for *g* is high if *g* happens with a low frequency but has a high expenditure share. Intuitively, expenditure shares reflect disease frequency and severity, which also affect the weights that the average worker attaches to diseases in the change of expected utility.[6] The fact that expenditure share is high despite low frequency indicates high severity, or high marginal disutility.[7,8] We find that marginal disutility has intuitive variation across diseases in our data; e.g. it is DKK 35,600 for the heart diseases that can be treated with prescription drugs, but becomes sharply higher, at DKK 3.48 million, for the hospitalization due to heart attacks and strokes (1 DKK = 0.18 USD in our sample). We then use these values of marginal disutility to show that, as firm sales increases, the average worker's ex-ante expected welfare loss from higher sickness rates amounts to 23.4% of her wage gains.

[6] A literature examines aggregate healthcare spending, focusing on the U.S. (e.g. Newhouse 1992, Hall and Jones 2007, Chandra and Skinner 2012). We focus on the expenditure shares of individual diseases within aggregate health spending, and propose the conditions under which expenditure shares relative to frequency provide useful approximations for marginal disutility.

[7] Because severity is post-treatment, our marginal disutility takes medical technology for treatment into account. In deriving this result, we also allow the private- and social-cost functions to differ, and so accommodate moral hazard, a very important feature of the healthcare system (e.g. Cutler and Zeckhauser 2000).

[8] A literature measures quality of life, or ex-post utility loss in sickness, using survey data. Examples include QALY, or Quality-Adjusted Life Years, (e.g. Torrance 1986, Cutler, Richardson, Keeler and Staiger 1997), and DALY, or Disability-Adjusted Life Years (e.g. Murray and Acharya 1997). Quality of life is related to, but distinct from, severity and marginal disutility of diseases in our framework (see Theory Appendix 5).

We now discuss how our work complements previous research. A line of work uses cross-sectional variation to estimate how economic shocks affect health (e.g. Marmot, Rose, Shipley and Hamilton 1978, Marmot et al. 1991, Case and Deaton 2009, Black, Devereux and Salvanes 2012) by drawing on health-diagnostics data (e.g. blood pressure). Our panel data allow us to control for unobserved worker characteristics that affect health, to assess a wide range of health outcomes and track them over time, and to employ the complementary identifications of shocks within job spells, and shocks in an event study.

We also contribute to a literature that assesses the pro-cyclicality of health and mortality, including Ruhm (2000, 2003, 2005), Lindo (2013), Tekin, McClellan and Minyard (2013), and Coile, Levine and McKnight (2014). As this literature uses population health data, an important question is whether the economic shocks affect individuals' health directly, or through other external economic activities (e.g. Miller, Page, Stevens and Filipski 2009, Stevens et al. 2015).[9] Our matched worker-firm data enable us to better explore the mechanism linking increased firm sales to adverse health outcomes related to work stress, by exploring the response of high-work-load hospitalizations and the heterogeneous effects of sales increases in both within-job-spell and event-study analyses.

A literature shows that negative economic shocks, including mass layoff (e.g. Sullivan and von Wachter 2009), plant closure (e.g. Browning and Heinesen 2012), and import competition (e.g. McManus and Schaur 2015), tend to have adverse effects on health.[10] Our findings do not contradict this literature, for two reasons. First, the average worker in our sample is qualitatively different from those who experienced mass-layoffs. Second, we examine a different link between economic shocks and health: that work demand increases individuals' workplace stress and elevates their sickness risk.

The paper is organized as follows. Section 2 describes our data and section 3 spells out our main

[9] For example, changes in nursing home staffing can lead to decreased quality of care and increased mortality. There can be more traffic-accident deaths during economic expansion because (1) time-pressed and stressed drivers pay less attention to safety; or (2) there are more vehicles on the road.

[10] See also Browning, Danø and Heinesen (2006), Eliason and Storie (2007, 2009), Colantone, Crinò and Ogliari (2015), Schaller and Stevens (2015), Pierce and Schott (2016), and Adda and Fawaz (2017). Some studies of import competition examine health-related outcome, such as marriage (e.g. Autor, Dorn and Hanson 2015) and crime (e.g. Dix-Carneiro, Soares and Ulyssea 2015). Goldman-Mellor, Saxton and Catalano (2010) survey the studies at the intersection of economics and epidemiology that focus on mental health.

hypotheses and identification strategy. Section 4 explores how firm sales affect work load. Section 5 presents our main results, based on the complementary identifications of within-job-spell variation, and event-study variation. Section 6 shows the differential impacts of firm sales on ex-ante-high-risk workers. Section 7 conducts robustness exercises. Section 8 outlines our framework for marginal disutility and implements the quantification. Section 9 concludes.

2. Data

In this section we briefly describe the main features of our data. We report more details of data construction in the Data Appendix.

We start with Danish administrative data that matches workers to firms and the domestic production of these firms. The main data set is annual, covers the period 1996-2012, and matches the population of Danish workers to the universe of private-sector Danish firms. The primary data sources are: 1) the Firm Statistics Register ("FIRM"), which holds information about firm-level employment and industry codes; 2) the Integrated Database for Labor Market Research ("IDA"), where we obtain information about workers' demographic and labor market characteristics, such as earnings and age; 3) the link between firms and workers ("FIDA"); 4) Account Statistics ("FIRE"), where we get firm-level capital stock; and 5) the Prodcom database ("VARS")[11], which breaks down each firm's sales of own goods (goods extracted, produced, processed or assembled by the reporting firm) by 8-digit product codes.

To construct our main sample, we start from the firms and workers in the Prodcom sales data/registry, which covers all firms in manufacturing (including mining and quarrying) with at least 10 employees. We keep the firms with positive imports and exports, since we use offshoring and the numbers of export products and export destination countries as controls. We also drop the observations with missing information about key firm variables (sales, capital-labor ratio and the share of high-skilled workers). We select 20-60 year old full-time

[11] The Prodcom database is a survey used by Eurostat to provide comparable production statistics for EU countries. Bernard et al. (2019) is a recent example using the Belgian version of the database.

workers, and we drop all observations where the employment relationship lasts a single year. The employment relationships, or job spells, are defined by all unique combinations of worker and firm id. As we explain below, we will use changes over time within job spells as the identifying variation. Panels 1 and 2 of Table 1 report the summary statistics of the firm- and worker-characteristics variables that we use in our analyses.

We then bring in additional administrative datasets that contain comprehensive information about individuals' health care utilization during 1996-2017. We observe the *universe* of *transactions* for every person within the Danish healthcare system, including prescription drug purchases and hospitalization. These datasets are organized by the same worker identifiers as our worker-firm data, allowing us to merge them. In the literature, a common concern for data on the utilization of health care is that access to care could be correlated with individuals' socio-economic conditions (e.g. income and employment status), and that this correlation could contaminate the care-utilization data (e.g. Currie and Madrian 1999). This concern is unlikely to be a main issue for us, because the Danish healthcare system is almost entirely funded by the government, available to all Danish residents regardless of employment status, and virtually free to all.[12]

Many studies of mental health use self-reported survey data and measure stress by the Center for Epidemiological Studies Depression Scale (CESD). While CESD measures self-reported stress that is not sufficiently severe for medical treatment, independent corroboration of the self-reported effects is not always available. By focusing on administrative data, we are capturing events sufficiently severe that medical treatment was sought and provided by trained professionals. We also include indicator variables of heart diseases and strokes, as well as hospitalizations due to liver diseases, a commonly used measure for alcoholism.[13] Panel 3 of Table 1 reports the summary statistics of our sickness variables in the annual data of 1996-2012 for the full sample, and also for the sub-samples of younger (below 50) and older (above 50) workers.

[12] There are two main exceptions. 1. Dental care is not covered. 2. Patients bear some co-payments for prescription-drug expenses. We do not consider dental visits in our study, and the prescription co-pays are small enough (roughly 0.13 percent of median income) that income constraints on access are unlikely to be binding.

[13] It is common for studies of mental health to examine alcoholism (e.g. Goldman-Mellor et al. 2010). We do not look at sleep disorder for two reasons. One, there is a U-shaped relationship between self-reported stress and hours of sleep in some survey data (e.g. De Quidt and Haushofer 2016). Two, Goldman-Mellor et al. (2010) survey a large number of studies on mental health, and none of them examines sleep disorder.

The mean rate of anti-depressant uses in our full sample is comparable to the medical literature (e.g. Olsen et al. 2007). The medical literature also shows that, while age significantly increases the risk for heart diseases and strokes, the connection between age and higher risks of severe stress and depression is not as strong. These patterns are consistent with our register data. For example, while older workers use heart-disease drugs 3 times more often than younger workers, their use of anti-depressant drugs is similar to younger workers and their risk of hospitalizations due to high work load is less than younger worker. We will revisit the differences in sickness rates between older and younger workers in section 6, as a way to evaluate population groups that face higher ex-ante risk of different kinds of sickness events. Panel 3 of Table 1 also reports the shares that our sickness variables account for in total Danish healthcare expenses; e.g. hospitalization due to heart attacks and strokes has the highest expenditure share, even though it happens with relatively low frequency. We will make use of the expenditure-shares data in section 8 below.

We obtain a rough proxy for individuals' work load from the "LON" register, which holds information reported by firms about annual hours worked for each employee. The hours reported are actual hours worked defined as contractual (paid) hours including overtime work but excluding time absent from work (e.g. due to sickness, child care leave etc.). While this register covers workers in all private-sector firms with at least 10 employees, in principle, reporting requirements are not strictly enforced in practice. As a consequence, the coverage varies by year, ranging from 60%-85% of our observations. One potential concern is that some occupations (e.g. managers) may be more subject to the reporting rules than others (e.g. assembly line workers). We show (in the Data Appendix) that the fractions of workers that are covered in the work-hour sub-sample are similar to the full sample across 1-digit occupations. The median worker logs 1615 hours and the full distribution of annual hours worked in the sample is displayed in Figure 1.

We complement our annual data with quarterly payroll records with information about worker-level hours as well as earnings and quarterly firm-level sales, and report the summary statistics of our quarterly variables in panel 4 of Table 1. Again, the hours variable measures actual hours worked including overtime hours and excluding time absent from work. The higher-quality hours data covers 2008-2017, and is based on the "eIncome"

register, which holds information reported by firms about quarterly hours and earnings for all employees. We merge this register with the firm-level data from the VAT register ("FIKS"), which has information on total sales and total purchase of materials for all firms liable for value added taxation on a quarterly basis. We then apply the same cuts as the annual data; e.g. all 20-60 year old fulltime workers employed by firms with non-missing Prodcom sales.

Much of the Danish labor market is covered by bargaining agreements, with 37 hours per standard work week. However, these agreements vary across bargaining segments, and have room for flexibility in weekly hours (e.g. by stipulating that weekly hours should average 37 per year). Such nuanced patterns of work hours are what we observe in our quarterly data. On the one hand, the standard 37 weekly hours are prevalent in the cross section: 41% of the worker-quarter observations are recorded with 481 hours, the quarterly sum of 37 hours per week, while 16% (43%) have longer (shorter) hours. On the other hand, workers experience considerable deviations from the standard hours during a job spell. For example, the 5^{th}, 10^{th}, 25^{th}, 75^{th}, 90^{th} and the 95^{th} percentiles in the distribution of deviations in log hours from the job spell mean are -0.32, -0.13, -0.01, 0.07, 0.17 and 0.27 respectively. This within job spell variation in hours accounts for a substantial portion of the total variation in hours: A simple regression of (log) hours worked on job spell fixed effects shows that 82% of the total variance are explained by these fixed effects, leaving 18% to within-job-spell variation. In addition, the within-job-spell variation of worker hours is correlated with the within-job-spell movement of firm sales. For example, in the quarters with above (below) average firm sales within job spells, worker hours are on average 1.3 percent higher (lower). We will re-visit the connection between worker hours and firm sales in section 4 below.

Our annual and quarterly data are complementary. On the one hand, the annual data covers a longer period, and this is useful because some of our sickness variables are rare events (see Table 1). In addition, our main firm- and worker-level control variables, such as capital, labor employment, and labor market experience, are available in annual frequency only. On the other hand, the quarterly data gives us observations at higher frequency, which enable us to better identify the timing of sales increases relative to sickness events. As a result, we use both annual and quarterly data in our analyses, as we explain below.

3. Main Hypotheses, Specification, and Identification

We start by developing a simple model of work load, and show how it relates to firm TFP, output demand, sales and input uses. We then connect changes in work load to changes in worker's health capital and the likelihood of an adverse health event. To ease exposition we will drop subscripts initially, and add them back when we transit to the empirical specifications.

Assume that firms have CES production functions and sell their outputs in competitive markets.[14] Let Y denote firm sales, A TFP, and p output price. Then we have

$$Y = \psi\left(K^{\frac{\sigma-1}{\sigma}} + M^{\frac{\sigma-1}{\sigma}} + H^{\frac{\sigma-1}{\sigma}}\right)^{\frac{\sigma}{\sigma-1}}, \psi = pA, H = La, \sigma > 0. \quad (1)$$

In equation (1), ψ encompasses both output demand and TFP, and σ is the substitution elasticity in production. The inputs include capital, K, materials, M, and total labor, H. Total labor has two components: L is employment, the number of workers hired at the firm, and a is the work load of a given worker. It may be that employment is slow to respond to a change in sales, but that workers can increase their workload, a. We assume that firms face an internal labor supply curve of the form $w = c_2 a^{\gamma}, \gamma > 0$,[15] where w is the wage for one unit of labor. We can then link changes in workload, a, to increases in firm sales as follows.

The CES bundle of inputs can be approximated as a log linear expression of input uses; i.e. $ln\left(K^{\frac{\sigma-1}{\sigma}} + M^{\frac{\sigma-1}{\sigma}} + H^{\frac{\sigma-1}{\sigma}}\right)^{\frac{\sigma}{\sigma-1}} \approx \theta_M \, lnM + \theta_K \, lnK + \theta_H \, ln(La) + constant$, where the θ's are positive constants and $\theta_H + \theta_K + \theta_M = 1$. We combine this approximation with the firm's labor demand and supply equations, to obtain an expression for workload as a function of other input uses and the parameter, ψ.

$$ln\, a = constant + \frac{\sigma ln\, \psi}{\sigma\gamma - (\theta_H - 1)} + \frac{\theta_K \, ln\, K}{\sigma\gamma - (\theta_H - 1)} + \frac{\theta_M \, ln\, M}{\sigma\gamma - (\theta_H - 1)} + \frac{(\theta_H - 1)\, ln\, L}{\sigma\gamma - (\theta_H - 1)}. \quad (2)$$

[14] We obtain similar results when the output market is monopolistic competition and consumers have CES preferences.

[15] The internal labor supply curve is a simple and transparent way to capture the main predictions of a more elaborate model, where workers and their employer bargain over work load (we are grateful to an anonymous referee for pointing this out). In our working paper, we spelled out the details of this multi-lateral bargaining model, and obtained similar predictions.

Equation (2) says that, fixing other inputs (including the number of workers hired), the elasticity of workload with respect to ψ is is $\sigma/(\sigma\gamma - (\theta_H - 1)) > 0$; i.e.

Hypothesis 1 Following an increase in output demand or TFP, sales increase and employees experience an increase in their work load.

Because the variable ψ in equation (2) is hard to measure in the data, we substitute it out using the expression for sales, equation (1), to obtain

$$ln\, a = constant + \frac{\sigma ln\, Y}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{\theta_K\,(\sigma-1) ln\, K}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{\theta_M\,(\sigma-1) ln\, M}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{((\sigma-1)\theta_H+1)\, ln\, L}{\sigma(\gamma+\theta_H)+1-\theta_H}. \quad (3)$$

Here, the elasticity of workload with respect to sales is $\sigma/(\sigma(\gamma_2 + \theta_H) + 1 - \theta_H) > 0$. Equation (3) relates changes in work load to changes in firm sales, conditioning on the use of other factors of production, and so allows us to take Hypothesis 1 to the data. Equation (3) also clarifies that, in our estimation, our main explanatory variable, firm sales, is closely related to work load, because we control for firms' input uses.

We next connect changes in sales and workload to worker health. A worker has health capital *E*, and she becomes sick when *E* falls below the threshold E_S.[16] Let E_0 denote the initial value, or endowment, of health capital. We assume that $E_0 > E_S$, because we focus on the initially healthy workers in our estimation. We also assume, following our discussions in the introduction, that an increase in a worker's load wears down her health capital and so makes her more likely to become sick.

Suppose $E = E_0\, \xi/(ln\ a)$, where *a* is work load, and ξ is a random variable that captures the stochastic nature of health. Then the worker's probability of getting sick is

$$S = \text{Prob}(E < E_S) = \text{Prob}(\xi < E_S(ln\ a)/E_0) = F(\frac{E_S\, ln\, a}{E_0}),$$

where *F*(.) is the cdf of the random variable ξ. This expression implies that

$$\frac{\partial S}{\partial ln\psi} = f(\frac{E_S\, ln\, a}{E_0}) \frac{\partial\, ln\, a}{\partial ln\psi} \frac{E_S}{E_0}, \quad (4)$$

where *f*(.) is the pdf of ξ. Since $\partial\ ln\ a\ /\partial ln\psi > 0$, by Hypothesis 1, equation (4) implies that $\partial S/\partial ln\psi > 0$; i.e.

Hypothesis 2 Following an increase in output demand or TFP, sales rise and employees become more likely to

[16] Nielson (2016) provides a graphical illustration of this point.

get sick.

While we have assumed away heterogeneity across workers to this point, equation (4) provides insight into two potential avenues to study the differential effects of sales changes on workers' health. First, $\partial S/\partial ln\psi$ is large if $\partial\ ln\ a\ /\partial ln\psi$ is large. That is, one could estimate the differential impacts of sales increases on individuals' work load, and then investigate whether those with large work-load increases are more likely to get sick. Second, $\partial S/\partial ln\psi$ is large if E_0 is close to E_S, so that E_S/E_0 is large (recall that $E_S < E_0$). That is, one could explore whether sales increases have larger impacts on the health of the workers whose health-capital endowment is close to the sickness threshold.

The first approach is a challenging path, for two reasons. One, in the data, an individual's work load combines observed (e.g. work hours) and unobserved (e.g. work intensity) components, and we provide evidence in the next section that the unobserved component is likely to be significant. Two, the response of an individual worker's hours (an observed component of work load) to firm-sales changes might be confounded by her underlying health capital. In the medical literature, Sokejima and Kagamimori (1998) show that there is a U-shaped relationship between the probability of heart attacks and (self-reported) work hours in the previous month.

As a result, we pursue the second avenue, where we lean on the medical literature to identify characteristics of workers who are, ex-ante, at high-risk, and examine whether sales increases result in worse health outcomes for these workers. In other words,

Hypothesis 3 Following an increase in output demand or TFP, sales rise and high-health-risk employees experience larger increases in the probability of sickness.

We examine evidence for Hypothesis 1, linking work load to sales, in the next section. Hypothesis 2 is the main focus of our study, and we take it to the data in section 5. Hypothesis 3 helps clarify the mechanism of Hypothesis 2, and we present evidence for it in section 6. We now transition to the empirical specifications for Hypothesis 1~3.

Let the subscript *i* index workers, *j* index firms, and *t* index years. We assume that a worker's health capital in a given year relative to their base year is $E_{S,ijt}/E_{0,ij} = R_i u_{ijt}$, where $R_i > 0$ is a binary variable that takes a

larger value for high-risk workers. Then for the sickness probability, we have $S_{ijt} = F[u_{ijt}(R_i lna_{ijt})]$, which can be expressed as an equation for u_{ijt} and $R_i lna_{ijt}$ through a first-order Taylor approximation. We assume that u_{ijt}, the residual component of individuals' health, depends on $\mathbf{x_{it}}$, the vector of time-varying worker characteristics, α_{ij}, job-spell fixed effects, as well as the error term, ε_{ijt}. Meanwhile, lna_{ijt} is given by equation (3), as a function of log firm sales and $\mathbf{z_{jt}}$, the vector of firms' input uses.

We then obtain two equations for S_{ijt}, one for the high-risk-worker sub-sample, and the other for everybody else. In both equations, S_{ijt} depends on log firm sales, firm controls $\mathbf{z_{jt}}$, worker controls $\mathbf{x_{it}}$, fixed effects α_{ij}, and error ε_{ijt}. In our estimation, we pool across the two sub-samples because the high-risk-worker sub-sample could be small in size.

We first examine Hypothesis 2, or the empirical relationship between sickness rate and firm sales. To be specific, we use our annual data to estimate

$$S^d_{ijt} = \phi_1 \, lnY_{jt} + \boldsymbol{x_{it}} b_1 + \boldsymbol{z_{jt}} b_2 + \alpha_{ij} + \alpha_t + \varepsilon_{ijt}. \quad (5)$$

In equation (5), the superscript "*d*" indicates the sickness variables we examine, and α_t represents year fixed effects. ϕ_1, the coefficient estimate of firm *j*'s log sales, lnY_{jt}, closely matches Hypothesis 2, because sales changes net of changes in input uses are likely due to changes in output demand or TFP, as spelled out in equation (3). With α_{ij}, job-spell fixed effects, the identification in equation (5) comes from within job spells, over time; i.e. we ask, conditioning on the match, whether individual workers suffer an increased risk of adverse health outcomes when their firms experience increased sales.

We supplement equation (5) with the following event study, to investigate whether individual workers' health declines after increases in firm sales,

$$S^d_{ijq} = \alpha_i + \alpha_q + \sum_{k=-8}^{T} \gamma_k I\{large\ Y_{ijq}\} + \varepsilon_{ijq}, \quad (6)$$

where *q* denotes quarters, α_q quarter fixed effects, and α_i worker fixed effects. Like other event studies, equation (6) is an extension of difference-in-difference to a dynamic setting, and so identification comes from both cross-cohort comparisons and over-time comparisons within the same cohort. In (6), we include 8 lead quarters before the main event, a large increase in firm sales, when the indicator variable, $I\{large\ Y_{ijq}\}$, turns on. We also

estimate the full set of dynamic effects, from the quarter of the main event, $k = 0$, to the last quarter in our data, $k = T$, following Borusyak and Jaravel (2018). We estimate (6) using our quarterly data.

Having provided Hypothesis 2 with the complementary identifications of equations (5) and (6), we move on to investigate whether sales increases have larger impacts on high-risk individuals' health, as proposed in Hypothesis 3, and estimate

$$S_{ijt}^{d} = \phi_1 \, lnY_{jt} + \phi_2 \, lnY_{jt} I\{R_i = high\ risk\} + \boldsymbol{x_{it}} b_1 + \boldsymbol{z_{jt}} b_2 + \alpha_{ij} + \alpha_t + \varepsilon_{ijt}. \quad (7)$$

Equation (7) is an extension of equation (5) with the additional interaction term between log firm sales and the indicator variable for high-risk workers, $I\{R_i = high\ risk\}$. Hypothesis 3 implies that $\phi_2 > 0$. The main ingredient of implementing equation (7) is to identify high-health-risk vs. low-health-risk workers. Finally, we examine Hypothesis 1, by using proxy measures of individuals' work load that are available in our data, lnh_{ijt}, and estimate

$$lnh_{ijt} = \phi_1 \, lnY_{jt} + \boldsymbol{x_{it}} b_1 + \boldsymbol{z_{jt}} b_2 + \alpha_{ij} + \alpha_t + \varepsilon_{ijt}. \quad (8)$$

The identification of equation (8) is the same as equation (5).

We now clarify our main identification assumption: firm sales are exogenous to the health of individual workers, conditional on the control variables in our estimation. In addition, we do not distinguish whether sales changes come from output demand or TFP. These identification assumptions are similar to the literature on the pro-cyclicality of health,[17] and that on the health effects of mass layoffs and plant closures.[18] Note that the studies in both literatures typically do not control for worker fixed effects.[19]

There are a series of possible threats to identification that we discuss, in detail, in sections 4-7 below.

[17] Examples include Ruhm (2000), Coile et al. (2014), and Stevens et al. (2015). This literature assumes that the main explanatory variable, state unemployment, is exogenous to workers' health, and does not distinguish whether state unemployment is caused by state productivity or aggregate demand.

[18] Quoting Sullivan and von Wachter (2009), "Firm-level employment declines should be exogenous to individual workers' health developments." This study does not distinguish whether employment declines are due to firm productivity or output demand. Other studies make similar identification assumptions (e.g. Eliason and Storie 2007, Browning and Heinesen 2012).

[19] There are some exceptions. The Framingham heart sample has panel data, and is used in cardiology to study the physiological and behavioral risk factors of heart diseases, such as obesity (e.g. Hubert et al. 1983). These studies do not focus on economic shocks. The Whitehall sample is also panel data, and is used in epidemiology to study the psychosocial and behavioral risk factors of health, such as job control (e.g. Bosma et al. 1997). The main explanatory variables in these studies are typically self-reported.

Here, we outline the main issues, and how we address them. The first is that there are differences across firms in technology that are correlated with sales and with risks to worker health, and that there are differences across individuals, or worker-firm matches, that are correlated with health.[20] These are accounted for by utilizing within job-spell variation, to sweep out potentially large cross-firm, cross-individual and cross-job-spell variation in these health risks, and by incorporating the time-varying use of other inputs within job-spells, such as capital, that may be correlated with both sales and health outcomes, following equation (5).

The second is reverse causality, that changes in workers' health capital and sickness affect firm sales. . Reverse causality may affect our results in two ways. One, a decline in individual workers' health for reasons exogenous to the firm is revealed through an increased incidence of seeking treatment, and that decline in health adversely affects firm productivity and sales. Reverse causality of this sort would create a bias against our hypothesized result that a rise in firm sales worsens health (and increases healthcare utilization). Two, already sick workers may seek treatment for exogenous reasons. Treatment makes them healthier, more productive, and increases sales. This would bias results our estimates upward. Notably, these two channels imply a fundamental difference in the link between worker health and healthcare utilization. The first (second) argument says that, within a job spell, a worker is sicker if she uses more (less) health care. While the second argument may be important where access to health care depends on worker income, it may be less so in Denmark, where access to health care is universal. Nevertheless, we address this concern by investigating carefully the timing of health and sales changes, as in equation (6), by testing the sensitivity of our results to cases where adverse health outcomes for individual workers are more likely to affect overall sales, and by using instruments for sales.

The third is simply that we are picking up spurious correlation between health outcomes and sales. We address this by examining whether ex-ante at-risk workers are more likely to suffer adverse health outcomes following a sale increase, as in equation (7), and by using placebo tests to investigate changes in health outcomes unrelated to work load.

[20] Examples of individual characteristics include pre-natal factors, birth weight, and genetic differences (e.g. Maccini and Yang 2009, Black et al. 2007). As an example of worker-firm characteristics, type-A-personality individuals may be more likely to work with firms with good growth potential. Helpman et al. (2010) examine worker-firm match specific productivity.

4. Preliminary Results: Sales Changes and Work Load

In this section, we take Hypothesis 1 to the data in two ways. First, we look within job-spells to link changes in firm sales to increased individual worker hours and earnings as observable components of workload. Second, we look at a given firm to see how changes in sales affect the use of measurable labor relative to material inputs to provide an indirect indication of changes in unobservable workload.

More formally, let us distinguish between observable and unobservable components of workload for an individual. We observe hours worked for individual i, in firm j at time t as h_{ijt}. The unobserved component of workload is v_{ijt} so that total work load $a_{ijt} = h_{ijt}v_{ijt}$. Under an assumption that the firm faces an upward sloping internal labor supply curve, an increase in workload should also show up in increased earnings within job-spell.

We examine the effects of firm sales on observed work load by implementing equation (8). We use the log of total hours by worker *i* for firm *j* in year *t* for lnh_{ijt}, and we examine the log of her earnings as well. For the vector of time-varying worker controls, $\boldsymbol{x_{it}}$, we use union membership, marital status, plus two dummy variables for experience. Meanwhile, the vector of firm characteristics, $\boldsymbol{z_{jt}}$, includes the share of high-skilled workers, and the logs of employment, capital-labor ratio, and offshoring (i.e. imported materials). We include job-spell and year fixed effects, and cluster our standard errors by firm. Column (1) of Table 2 shows that the elasticity of hours with respect to sales is 0.016, and it is statistically significant. Column (2) shows that the elasticity of earnings is 0.008, and it is significant as well.

While the results in columns (1) and (2) suggest that workers have increased work load as their employers experience sales increases, the number of observations in column (1) is smaller than in column (2), because the hours variable in our annual data does not cover the full sample. We thus also estimate equation (8) using our quarterly data, where the hours variable covers the full sample, and report the results in columns (3)-(8) of Table

2. In these columns, hours, earnings, sales and materials are at quarterly frequency, and all the other variables are annual. We include job-spell and quarter-year fixed effects,[21] and cluster by firm.

In columns (3) and (4), we control for the same set of worker and firm characteristics as in columns (1) and (2), and obtain an elasticity estimate of 0.023 for hours and 0.036 for earnings, both significant. In columns (5) and (6), we add the log of materials as control. Although our elasticity estimates are smaller in magnitude (0.018 for hours and 0.031 for earnings), they remain significant. As we discussed previously (section 2), many worker-quarter observations report exactly 37 hours per week, and one may be concerned that these workers are unable to adjust their hours. In column (7), we apply the specification of column (5) to the sub-sample of firms with below-the-median proportions of workers who report exactly 37 weekly hours. The hours elasticity is 0.038, larger than in column (5). In column (8), we examine the firms with above-the-median proportions of 37-weekly-hour workers. Although the hours elasticity becomes smaller, 0.009, it remains statistically significant.

Overall, Table 2 suggests that, within job-spells, workers increase hours and obtain higher earnings when their employers experience positive economic shocks. These results address the concern that firms may face a perfectly elastic supply curve for employment, in which case individual workers' earnings and hours would not rise in response to an increase in work demand.

While Table 2 is qualitatively consistent with our Hypothesis 1, one may be concerned that the hours elasticity estimates are quantitatively small. We believe that the response of total hours likely understates the response of work load, because hours do not capture such important aspects of work load as work intensity (e.g. a worker's output during a given hour) and pressure to perform. This is especially relevant if the magnitude of hours response is more constrained by labor-market institutions (e.g. regulations and contracts) than the response of work intensity. In other words, the unobserved work load, v_{ijt}, may also respond to sales increases.

One element of v_{ijt} is worker-level output. While we do not observe it in our data, several studies that observe worker-level output, from individual firms, report substantial responses in individuals' outputs. In the

[21] In the working-paper version of our study, we experimented with firm-year fixed effects and worker-year fixed effects, and obtained similar results.

randomized experiment studied in Bloom et al. (2014), Chinese call-center workers are not allowed to change their total daily hours, but they make more calls and take fewer breaks. In Hamilton et al. (2003), Californian garment workers put in slightly more weekly hours and sew garments substantially faster. Bandiera et al. (2005) show that, while U.K. farm workers pick more fruits per hour, the change in their daily hours is statistically indistinguishable from 0.[22]

While we cannot get at the unobserved work load, v_{ijt}, directly, we can make inferences about its response to sales changes in the following way. Our CES production function, equation (1), implies that

$$ln\frac{M_{jt}}{H_{jt}} = \sigma ln\frac{w_{jt}}{p_{M,jt}},$$

where $p_{M,jt}$ denotes the price of materials. Consider the benchmark scenario where a firm faces fixed prices for material and labor inputs. Then this expression says that a rise in sales results in no change in the relative use of inputs, $\frac{M_{jt}}{H_{jt}}$, in the benchmark scenario.

Now, recall from the previous section that total labor, H_{jt}, is comprised of the number of workers and total work load per worker. In our data, we directly observe employment, L_{jt}. We also observe average hours per worker, $\tilde{h}_{jt}$, the observed portion of work load. This means that the total measured hours, by firm, that we observe in our data, $L_{jt}\tilde{h}_{jt}$, differs from the total labor in our theory, $H_{jt} = L_{jt}\tilde{h}_{jt}\tilde{v}_{jt}$, by the unobserved work load, $\tilde{v}_{jt}$. We can then compare how total measured hours, $L_{jt}\tilde{h}_{jt}$, responds relative to materials, to infer the response of the unobserved work load, $\tilde{v}_{jt}$, as sales rise.[23]

To be specific, we regress log materials, M_{jt}, log total measured hours, $L_{jt}\tilde{h}_{jt}$, and the log of materials to total measured hours, $(M_{jt}/L_{jt}\tilde{h}_{jt})$, on log sales, using firm-quarter observations. We include firm fixed effects, to stay consistent with our identification for equation (5). Panel 1 of Table 3 reports the results for the

[22] In related studies, Bandiera et al. (2010), Lazear et al. (2016) and Mas and Moretti (2009) focus on individual workers' output, and do not have data on their total hours.

[23] This approach is conceptually related to studies that use firms' materials purchases as a proxy for the correlation between firm-specific productivity shocks and firms' labor and capital inputs in the productivity-estimation literature (e.g. Levinsohn and Petrin 2003 and De Loecker and Warzynski 2012).

full sample. Log materials are highly correlated with log sales, with elasticity of 0.65. Total measured hours also responds to sales, with an elasticity of 0.37. Combining these, we see that the use of materials relative to total measured hours rises in the face of a sales increase, with an elasticity of 0.28.

We now interpret these results using our simple model in section 3. What we observe in the data is

$$ln\frac{M_{jt}}{L_{jt}\tilde{h}_{jt}} = ln\frac{M_{jt}}{H_{jt}} + ln\tilde{v}_{jt} = \sigma ln\frac{w_{jt}}{p_{M,jt}} + ln\tilde{v}_{jt},$$

Why would this ratio be increasing in sales? One, firms face an upward sloping internal labor supply curve and wages increase in sales as we argued, in theory, in section 3, and showed, in our data, in Table 2. The rise in wage tends to induce a positive correlation between materials to total labor, M_{jt}/H_{jt},and sales. Two, the total measured hours in Table 3, $L_{jt}\tilde{h}_{jt}$, does not include the unobserved work load, $\tilde{v}_{jt}$, and so undercounts the total labor in the benchmark scenario, H_{jt}. The omission of $\tilde{v}_{jt}$ also implies a positive correlation between materials per measured hour, $\frac{M_{jt}}{L_{jt}\tilde{h}_{jt}}$, and sales, if $\tilde{v}_{jt}$ increases when firms increase their sales. This interpretation is consistent with the findings of the worker-level-output studies that we previously discussed, and provides additional evidence for Hypothesis 1.

In the rest of Table 3, we examine whether the response of unobserved work load is related to the proportion of 37-weekly-hour workers by firm. We split the sample by the median of the proportion of 37-weekly-hour workers, as in columns (7) and (8) of Table 2, and estimate the same regressions as in panel 1 of Table 3. The elasticity estimates, reported in panels 2 and 3 of Table 3, are similar to panel 1, suggesting that unobserved work load may have similar responses to sales for these two sub-samples, for which observed work load shows different responses to sales in Table 2.

5. Main Results: Sales Changes and Sickness

5.1 Within Job Spells

We now examine how changes in firm sales affect individual workers' adverse health events within job spells, by estimating equation (5). We use our annual data because they span a long horizon, which is important

for isolating relatively infrequent adverse health events within job spells. For sickness condition *d*, our dependent variable, S_{ijt}^{d}, takes the value of 1 if a worker has sickness *d* in year *t*, and 0 otherwise. Because sick individuals' physiology might be different from healthy ones', we follow the common practice in the medical literature and drop those who are initially sick with condition *d* when our dependent variable is S_{ijt}^{d}. This means that our sample size varies slightly across *d*.

Our main explanatory variable is the log of *j*'s sales in *t*, and we control for the following inputs by *j*: employment, capital, fraction of high-skilled labor, and offshoring (imported inputs). We also control for job-spell and year fixed effects,[24] and cluster our standard errors by firm. We also control for the same set of worker and firm characteristics as in columns (1) and (2) of Table 2. The time-varying worker characteristics control for potential confounding factors of individuals' health; e.g., experience is highly correlated with age, and we obtain very similar results when we use age instead of experience (Data Appendix). Meanwhile, the time-varying firm characteristics help separate the changes in sales that lead to changes in work demand from those related to changes in input uses, such as capital and materials. As we explained in section 3, firm sales net of input uses measure the work load for *j*'s workers, according to equation (3).

We first examine the use of anti-depressant drugs. We report the coefficient estimates normalized by the mean rate of anti-depressant use, to express the effect as the elasticity of stress with respect to sales, in column (1) of Table 4. We obtain an elasticity estimate of 0.054, which is marginally significant. In other words, as consistent with Hypothesis 2, workers are more likely to suffer from depression as their employers experience positive economic shocks.

Is the increase in anti-depressant uses related to workplace stress? As we explained in the introduction, survey data show that work is a leading cause of self-reported stress. These data also identify the following common causes of self-reported stress: financial problems, death or sickness of family members, and trauma from

[24] In the working-paper version of our study, we experimented with industry and region fixed effects, and obtained similar results. Note that our job-spell fixed effects pose a computational challenge for non-linear specifications, such as Probit or Logit, because the marginal effects there depend on the values of all the fixed-effects parameters (e.g. Wooldridge 2002), and we have over 700,000 of them in our estimation. We think about our results as a linear approximation around the sample means of the sickness variables.

violent crime. We can exclude financial problems because earnings increase (Table 2), and the other external factors are unlikely to correlate with sales increases in the worker's firm. This leaves us with work.

Still, we can take another step in pinning the increase in depression on work load by drawing on diagnostic information in our hospitalization data. Specifically, the Danish health system provides diagnostic codes for hospitalization associated with "burn-out", "lack of relaxation and leisure" and "stressful work schedule". We call this group narrow burnout. We then broaden our inquiry to include other work-related hospitalizations, such as "discord with boss and workmates" and "uncongenial work environment", while excluding those that are clearly related to job insecurity, such as "threat of job loss" and "change of job". We refer to this second group as broad burnout.

We report the results for narrow and broad burnout in columns (3)-(4) of Table 4. Although hospitalization due to burnout is rare, we obtain large elasticity estimates of 0.39 and 0.42, respectively, both statistically significant. These results establish a tight connection between work load and stress, because by construction, narrow and broad burnouts are likely related to work load.

We now further expand the scope of our inquiry, to examine whether changes in firm sales affect workers' rates of heart diseases, and hospitalization due to heart attacks, strokes, or alcoholism. Our first dependent variable is whether workers purchase prescription drugs for heart diseases and strokes in year *t*. We obtain a significant elasticity estimate, 0.050 in column (2) of Table 4. In column (5) of Table 4, our dependent variable is whether workers are hospitalized because of heart attacks or strokes in year *t*. Our elasticity estimate is again significant, 0.090. In column (6) of Table 4, our dependent variable is hospitalizations due to liver diseases, a commonly used measure for alcoholism. We obtain a significant elasticity estimate of 0.13.

The R^2 values in Table 4 are within-job-spell, and not comparable to the traditional R^2 that includes the explanatory power of the job-spell fixed effects. Their values are low because the dependent variables of sickness indicators exhibit no within-job-spell variation for the vast majority of our observations,[25] and it is very difficult

[25] To see this point, consider the use of heart-disease drugs. Most individuals never use them, and so their within-job-spell variation is zero.

to explain, *specifically*, who becomes sick and when. Meanwhile, our estimation results in Table 4 suggest that, when many firms in the economy experience sales increases, *some* of their employees are likely to develop depression, burnouts, heart diseases or strokes.

Among the sickness variables we have examined in Table 4, burnout hospitalizations have the largest elasticity estimates. This is intuitive, because burnout hospitalizations are the most closely-related to workplace stress. To see this point, compare burnout hospitalizations with anti-depressants. For both sickness conditions, the change in the probability of sickness in response to sales increases, which we estimate, is closely related to workplace stress. However, the mean rate of anti-depressant uses encompasses a much wider range of causes than burnout hospitalizations. This is consistent with a smaller *elasticity* estimate for anti-depressants than for burnout hospitalizations.

Summarizing, our results in Tables 4 show that, when a firm experiences a positive economic shock, the workers suffer an elevated chance of stress and depression, heart attacks and strokes, and alcoholism, as well as hospitalization due to work burnouts. These results are consistent with Hypothesis 2.

To be clear, our sickness variables come from administrative data on healthcare utilization, and we do not observe all stress, as measured by the CESD scale (see section 2). This implies that our results likely provide conservative estimates of the effects of work demand on general mental health, because an individual with no anti-depressant use may still feel lonely and sad. In addition, because seeking treatment represents a higher threshold than the CESD scale, the effects of work demand that we identify are economically significant (see section 8). On the other hand, some sick individuals might decline treatment. We don't believe that this is a major issue in our data, for two reasons. One, as we explained in section 2, Denmark has universal healthcare, and prescription-drug copays are very low relative to income. Two, many of our results are for severe sickness that leads to hospitalization. Here, decline of treatment is unlikely, given that hospital care is free in Denmark.

A related concern is that, in response to sales increases, workers may increase healthcare utilization because of higher income. While this is important in the U.S. data,[26] where access to healthcare is related to

[26] Examples include Ruhm (2003), Moran and Simon (2006), Sullivan and von Wachter (2009), and Coile et al. (2014).

employment and income, healthcare in Denmark offers universal coverage and very low out-of-pocket expenses, as we explained above. Krasnik et al. (1997) combine surveys and administrative data, and report that healthcare utilization in Denmark is strongly correlated with healthcare needs, and not with organizational and social factors. Still, high-income patients may choose branded drugs while low-income ones stay with generic drugs. This is why we have chosen to use indicator variables of prescription-drug purchases in our estimation, even though we observe individuals' prescription-drug expenses in our data.

We also believe that the income effect is unlikely to drive our result for liver-disease hospitalization, an indicator of alcoholism, for two reasons. First, while studies using population-level data tend to find a positive association between income and alcohol consumption (e.g. Ruhm 1995), those using individual-level data typically show a negative relationship between economic conditions and alcohol abuse (e.g. Goldman-Mellor et al. 2010). The latter studies are more relevant for our work, because alcohol consumption includes both normal and abusive drinking. Second, the small magnitude of the elasticity of income with respect to sales, 0.0084 (column 2, Table 2), limits the size of the income effect. To illustrate this point, we assume that the income elasticity of liver-disease hospitalization were as high as that of alcohol consumption, 0.174 (Ruhm 1995, Table 5). Even under this very strong assumption, the income effect accounts for 1.15% of our elasticity estimate in column (8) of Table 4 (1.15% = 0.174 x 0.0084/0.128).

Our identification so far examines changes within job spells, but leaves out the effects for the workers who separate. We address this issue of selection in our event study below, by constructing worker cohorts and tracking them over time. This event study approach also enables us to address concerns about reverse causality; i.e. that changes in worker health for reasons exogenous to the firm may affect firm sales. (To further address reverse causality, in robustness exercises (section 7 below), we instrument for firm sales, and experiment with excluding managers, who are more likely to influence the effectiveness of the firm as a whole.)

5.2. Sales Changes and Sickness: Event Study

In this sub-section, we conduct our event study by estimating equation (6). Because timing is the central focus of our inquiry, we use our quarterly data. While the quarterly data have a shorter span than the annual data,

we do not use within-job-spell changes for identification in our event study, as we explain below.

An "event" in our context is a "large" sales increase. We first compute the deviation of log firm sales, by quarter, from the firm-specific sample mean, tabulate the distribution of this deviation, and define a "large" sales increase as the top decile of this distribution. Event time, *k*, is the number of quarters relative to *q*; e.g. *k* = -1 is one quarter before the shock, *k* = 8 is 8 quarters after, etc. We then follow the event-study literature (e.g. Couch and Placzek 2010, Davis and von Wachter 2011), and impose the following requirements for the workers to be included in our sample. They are observed for at least 8 consecutive quarters before the shock, aged 25-55 at event time *k* = -1, and hit by a large sales shock at least once. These requirements leave us with an unbalanced panel of roughly 3.3 million observations, with no control group. Because we drop the initially sick individuals with sickness condition *d* when our dependent variable is S_{ijt}^{d}, our sample size varies slightly across *d*.

For each worker, we focus on the first shock event that happens in or after 2010Q1 (to ensure that we have 8 pre-shock quarters). When this event happens for worker *i* in quarter *q*, who is with firm *j*, our shock indicator $I\{large\ Y_{ijq}\}$ switches to 1, and remains 1 for the rest of our sample. All the workers employed with the firms for which $I\{large\ Y_{ijq}\} = 1$ at quarter *q* are in cohort *q*, and remain so for the rest of our sample, even if they switch employers. We condition on worker fixed effects in all regressions, and also control for quarter-year dummies. We also cluster by firm, as in our previous results in Table 4.

We rely on the timing of the shock for identification; i.e. our identification assumption is that the timing of firm-level sales shocks is exogenous to individual workers' health. This identification strategy is fairly common in the literature (e.g. Parker et al. 2013). In our estimation, the *k*-quarter lagged effect, γ_k in equation (6), is identified from the comparison between cohort *q*, in absolute time $q + k$, vs. all the workers who remain untreated by $q + k$, as well as the comparison between cohort *q* in time $q + k$ vs. cohort *q* itself in time $q - 8$ through $q - 1$. Borusyak and Jaravel (2018) show that in general, γ_k is a weighted average of these cross-cohort differences and over-time differences. In implementing our estimation, we follow Borusyak and Jaravel (2018) and set to 0 the coefficients of the two farthest-apart pre-shock quarters, *k* = -8 and *k* = -1. In addition, we do not impose a cap on the number of post-shock quarters we examine, but estimate all the 40 γ_k's that can be estimated using our data

(8 pre-shock quarters, the event quarter 0, plus 31 post-shock quarters).

Figure 2 provides a visualization of how workers are exposed to large sales changes and the persistence of large shocks. By construction all workers are hit by a sales shock at event quarter $k = 0$ after having been observed in the data for at least eight quarters. We allow workers to experience large sales changes during the required eight pre-shock quarters.[27] We also allow workers to experience large sales changes after event quarter $k = 0$, in order not to condition on future outcomes. Figure 2 shows that 60 percent of the workers experience a large sales shock at event quarter $k = 1$, around 40 percent also experience sales shocks (relative to the firm mean) the following quarters, and the large-sales-change exposure then reaches a stable level that is comparable to pre-shock quarters after eight quarters.

The top 4 graphs in Figure 3 show the coefficient estimates of γ_k and their 95th confidence intervals for the following sickness variables: anti-depressants, heart-disease drugs, and narrow and broad burnout hospitalizations. The top left graph shows that the effect of large sales increases on the probability of anti-depressant uses becomes positive and significant in event time $k = 8$, and remains so for the rest of our sample. The magnitudes of the coefficient estimates tend to increase over time, which is intuitive, because anti-depressant uses could be persistent; i.e. the coefficient estimate of γ_k likely captures the cumulative effects of large sales increases up to event time k. The top right graph shows that the effect of large sales increases on heart-and-stroke drugs becomes positive and significant at event time $k = 11$, and remains so for the rest of our sample. Since the use of heart-and-stroke drugs is also persistent, the magnitudes of the coefficient estimates tend to increase over time as well.

The middle left graph of Figure 3 shows that workers experience elevated risks of narrow burnouts 8 and 12 quarters after their employers register large sales increases, and the middle right graph shows that the results for broad burnouts are very similar to narrow burnouts. The magnitudes of the coefficient estimates for narrow

[27] Workers have exposure before $k = 0$ because we require the main shock event to happen in or after 2010 Q1, to ensure that we have at least 8 pre-shock quarters.

and broad burnouts do not show an upward trend, unlike in the top graphs of Figure 3. This result is intuitive, because narrow- and broad-burnout hospitalization events are not persistent.

In all the four graphs of Figure 3 that we have examined, the effect of large sales increases first becomes statistically significant 8 quarters or later after the main event. This pattern may be related to the time path of large sales increases, shown in Figure 2, that workers' exposure to them remains elevated for up to 8 quarters after the main event. We also see, from Figure 3, that the confidence intervals widen as we approach the end of our sample. This feature is consistent with Borusyak and Jaravel (2018), and suggests that we should interpret the coefficient estimates near the end of our sample with caution. Finally, the next two graphs of Figure 3 show the results for the hospitalizations due to heart attacks or strokes, and those for liver diseases. The coefficient estimates are not statistically significant.

To summarize, we have used a different sample, a different specification, and a different identification strategy, to study the effects of firm sales on worker sickness. Despite these differences, we obtain qualitatively similar results, and they provide additional evidence for Hypothesis 2. These new results also complement our previous within-job-spell results in several ways. First, the worker cohorts in the event study are fixed by construction, and include the workers who separate from their employers after large sales increases. This helps address the issue of selection for our job-spell-fixed-effects regressions. Second, Figure 3 shows that workers' sickness events happen after large sales increases, and so provide evidence against reverse causality, a potential issue for our previous results. Finally, our event-study suggests that firm-level economic shocks may have longer-term effects on individual workers' health, and this finding enriches our previous results.

5.3 Placebo Tests

We conduct placebo tests in this sub-section, to address the concern that we might be picking up spurious correlations between health outcomes and sales increases. Our placebo variable is inborn errors of metabolism (hospitalization), a genetic disease, and we report its summary statistics in Table 1. We have picked inborn errors

of metabolism because they are not connected with our main sickness variables, such as depression, heart diseases and strokes, or the risk factors of our sickness variables, such as high blood pressure and obesity.[28]

Column (7) of Table 4 shows the results of our placebo exercises for our job-spell-fixed-effects regressions. We obtain a negative and marginally significant coefficient for inborn errors of metabolism. The bottom graph of Figure 3 shows the results of our placebo exercises for our event study. The coefficient estimates for inborn errors of metabolism are statistically insignificant. These results suggest that sales increases have no effects on the health outcomes that are unrelated to work load and workplace stress, and provide evidence that the rest of our estimation results in section 5 are not due to spurious correlation. We will conduct additional placebo tests in our robustness exercises, section 8, below.

6. Mechanism: Differential Effects of Sales Changes

In this section, we follow up on our placebo tests, by showing that the effects of sales changes are more pronounced where we expect them to be, for high-health-risk workers, as spelled out in Hypothesis 3. We do so by following the medical literature to identify ex-ante (relative to firm sales changes) risk factors, such as old age. We first estimate equation (7), using within-job-spell variation, and then switch our identification strategy to event study, by estimating equation (6) for the sub-sample of older workers. These findings not only provide additional evidence against spurious correlation, but also clarify the mechanism of our results in the previous section.

6.1 Age

Our first measure of ex-ante high health risk is age. We have shown, in section 2, that age is a significant risk factor for heart diseases and strokes, but the connection between age and risks of severe stress and depression is not as strong. To recap, Table 1 shows that, as compared with the individuals under 50, those over 50 are much more likely to use heart-and-stroke drugs (mean rate = 7.2 per hundred, vs. 2.1), and to be hospitalized because

[28] We have thought about other placebo variables, but decided against them because they share common risk factors with our main sickness variables. E.g. glaucoma has high blood pressure as a risk factor, and stress may cause auto-immune diseases to flare up or to worsen their severity.

of heart attacks or strokes (4.6 per thousand, vs. 1.3). They are also more likely to use anti-depressants (mean rate = 3.7 per hundred, vs. 3.6), and to suffer from liver diseases (mean rate = 0.1 per hundred, vs. 0.09). Meanwhile, the individuals over 50 do not have elevated rates of hospitalizations due to narrow and broad burnouts, severe stress and depression.

We set the high-risk dummy, R_i, to 1 if worker *i* is over 50 years old at the beginning of a job spell. We then estimate equation (7) and report our results in Table 5. Again, we drop the initially sick individuals with condition *d* when our dependent variable is S_{ijt}^{d}, and cluster by firm.

Column (2) shows that the effect of sales changes on the use of heart-and-stroke drugs is more than one order of magnitude larger for older workers (0.00038 + 0.0087 = 0.0091) than for younger workers (0.00038). The difference becomes smaller, but remains very large, once we convert the coefficient estimates into elasticity estimates: the elasticity is 0.018 for those under 50, but 0.13 for those over 50. Column (6) shows that the effect on hospitalizations due to liver diseases is qualitatively similar to column (2), although the interaction between old age and log firm sales has marginally significant coefficient estimate. Meanwhile, column (1) shows that sales increases have larger effects on anti-depressant uses for older workers, and that the difference between older and younger workers is not as large as in column (2): the coefficient estimate is 0.032 (0.018 + 0.014) for older workers and 0.018 for younger workers, and the elasticity is 0.087 for older workers and 0.050 for younger ones. Finally, columns (3) through (5) show that the differential effects of sales increases for older workers are statistically insignificant for the other sickness variables.

Overall, the patterns of the differential effects of sales increases, reported in Table 5, match the patterns of sickness risk, reported in Table 1. In other words, for the sickness condition where old age is a significant risk factor (e.g. heart-and-stroke drugs), sales increases have very large additional effects on older workers. For anti-depressant uses, where old age is also a risk factor, sales increases have larger effects on older workers. For the sickness conditions where old age is not a risk factor (e.g. narrow burnouts), sales increases have similar effects for older and younger workers. These results are consistent with Hypothesis 3.

6.2 Long Initial Hours

Our next measure of ex-ante high health risk is motivated by the epidemiology studies that show that long initial hours, in the base year, have predictive power for future events of CHD, strokes and depression (e.g. Kivimaki et al. 2015).[29] This literature typically measures long hours as 55 hours/week or more of self-reported hours in survey data, and uses the individuals with normal hours as the comparison group (35-40 self-reported hours/week), given the findings in Sokejima and Kagamimori (1998).

In order to translate self-reported hours in survey data into the hours we observe in our register data, we tabulate self-reported hours in Americans' Changing Lives (ACL), which surveys a panel of individuals in 5 waves (1986, 1989, 1994, 2002 and 2011). We find that 55 self-reported hours/week is roughly the 90^{th} percentile of the distribution. Meanwhile, 35 and 40 self-reported hours/week are the 25^{th} and 50^{th} percentiles, respectively. We thus set our high health risk dummy, R_i, to 1 if, at the beginning of a job spell, worker i's annual hours are in the top decile of the annual-hours distribution. We keep the workers whose annual hours (again measured at the beginning of job spells) are in the 2^{nd} quartile of the distribution as the control group, and drop all the other workers, following the epidemiology literature on long hours.

We report the results of regression (7) in Table 6. Columns (1) and (2) show that, relative to normal-hours workers, those with long initial hours, at the beginnings of job spells, have higher risks for the uses of anti-depressants and heart-and-stroke drugs when their employers experience sales increases. We also obtain statistically significant results for hospitalizations due to liver diseases (column 6). Meanwhile, the interaction between long hours and sales changes has insignificant coefficient estimates for the other sickness variables. Overall, the results in Table 6 are consistent with Hypothesis 3. They also clarify that it is the level of workers' initial hours that place them at higher risks of adverse health events in the future, as sales increase for their employers.

6.3 Job Strain

For our last measure of ex-ante high health risk, we turn to a large body of epidemiology studies that have

[29] See also Virtanen et al. (2011), Virtanen et al. (2012), and Kivimaki and Kawachi (2015).

identified job strain as a risk factor for chronic heart diseases (CHD), strokes and depression.[30] This literature uses survey data to measure job strain, as high work demand and low work control. Examples of survey questions for work demand include, "Do you have enough time to work", and those for work control are, "Do you learn new things at work" (e.g. Fransson et al. 2012). We are not aware of any survey that covers as large a fraction of the population as our register data. We thus correlate the job-strain measure in survey data with detailed worker characteristics, and then use this correlation pattern to extrapolate job strain for our register data. We briefly outline the steps we have taken in the following paragraph, and relegate the details to the Data Appendix.

We use ACL, and classify an individual as job strained if her answer to the work-demand question, "Do you have enough time to work", in wave 1, is below the median, and her answer to the work-control question, "Are you bored with your work", is above the median.[31] We replicate the main findings of the above-mentioned epidemiology literature using ACL; i.e. the job-strain dummy has explanatory power for future events of heart diseases or strokes, conditional on age, gender and marital status. We then trim the ACL sample to resemble our register data (i.e. manufacturing workers aged 25-60 in wave 1), and map the ACL occupation and industry codes into those used in our register data.[32] We next correlate the job-strain dummy with age-by-gender and gender-by-occupation-by-industry dummies for the manufacturing sub-sample. This regression has $R^2 = 0.83$, and we gather the full set of coefficient estimates. Finally, we double check that the predicted-job-strain dummy still has explanatory power for future risks of heart diseases and strokes for the manufacturing sub-sample, if we use the 75^{th} percentile of the predicted values of job strain as the cut-off.

We now combine the regression coefficients from ACL with our register data on age-by-gender and gender-by-occupation-by-industry dummies, and compute the predicted values of job strain for our register data. Following our ACL results, we set the high-health-risk dummy, R_i, to 1 if worker i's predicted job strain is in the top quartile of the distribution at the beginning of a job spell. We estimate equation (7) and report the results in

[30] Examples include Kivimaki et al. (2012), Rosengren et al. (2014), Fransson et al. (2015), and Madsen et al. (2017).

[31] The answers range from 1 (strongly disagree) to 5 (strongly agree).

[32] We also have access to Danish Work Environment Cohort Study (DWECS), but the occupation codes in DWECS cannot be easily matched to the codes in our register data.

Table 7. Column (2) shows that the effects of sales increases on heart-and-stroke drugs are stronger for job-strained workers. This result is intuitive, given that job strain is a risk factor for CHD and strokes. Columns (3) and (4) show that the effects of sales increases on narrow and broad burnouts are stronger for job strained workers. These results are also intuitive, because the ACL work-demand question that we use to define job strain is, "Do you have enough time to work". On the other hand, the results for the other sickness variables are statistically insignificant. Overall, Table 7 provides additional evidence for Hypothesis 3.

6.4 Age: Event Study

In this sub-section, we complement our within-job-spell identification in sub-sections 6.1 through 6.3 with the following event study. We start from our event-study sample of sub-section 5.2, and consider the sub-sample of older workers, whose age is 50 or higher when they are first exposed to a large sales increase, at event quarter $k = 0$. This leaves us with 0.5 million observations. We then use the same procedure from sub-section 5.2 to estimate equation (6) for the older-worker sub-sample, and report the coefficient estimates for γ_k and their 95th confidence intervals (red dots and lines) in Figure 4. Figure 4 also shows the coefficient estimates for the full sample (blue dots and lines), from Figure 3, for comparison.

The top two graphs of Figure 4 are for heart-and-stroke drugs and heart-attack-and-stroke hospitalizations, for which age is a significant risk factor. In the left graph, we see that, for heart-and-stroke drugs, the coefficient estimates for older workers tend to have wider confidence bands than those for all workers, because of the smaller sample size. These estimates become statistically significant at event quarter $k = 14$, and show similar persistence and growth over time as compared with those for all workers. Notably, the magnitudes of the estimates are much larger for older workers than for all workers; e.g. at event quarter $k = 16$, the effect of large sales increases for older workers is roughly twice as large as that for all workers. Meanwhile, the top right graph shows that, for heart-attack-and-stroke hospitalizations, large sales increases also have much larger effects on older workers. For example, at event quarter $k = 13$, when the effect on older workers is statistically significant, it exceeds the effect for all workers by a factor of 10. The effects for older workers are also significant at event quarters 3 and 15.

The next two graphs of Figure 4 are for anti-depressants and alcohol-related hospitalizations, for which

age is a (mild) risk factor. From the left graph, we see that, the coefficient estimates for anti-depressants become statistically significant at $k = 9$ for older workers, and show similar over-time persistence and growth as compared with those for all workers. Meanwhile, the effects of large sales increases for older workers are broadly similar to those for all workers. On the other hand, the graph on the right shows that the coefficient estimates for alcohol-related hospitalizations are statistically insignificant for older workers.

The bottom two graphs of Figure 4 show the results for burnout hospitalizations. For these sickness conditions, old age is not a risk factor, and we see that large sales increases have similar effects, overall, for older workers and for all workers. The coefficient estimates are statistically significant for narrow and broad burnouts at event quarter $k = 7$ for older workers.

In summary, our results in Figure 4 corroborate our results in Table 5, despite the differences in sample, specification, and identification. We see, again, that large sales increases have more pronounced effects on older workers for the sickness conditions for which age is a significant risk factor. We also see that older workers' sickness events happen after sales increases. These results provide additional support for Hypothesis 3, and additional evidence against both reverse causality and spurious correlation.

7. Robustness Exercises

In this section, we first show that our main results hold for a number of robustness exercises, focusing on the concerns of reverse causality and endogeneity, which we outlined in section 3. We then explore how the effects of firm sales vary with gender, local-labor-market tightness, and worker tenure. We gather our results in Table 8. To save space, we only report sample size, and the coefficient estimates of firm sales and other explanatory variables of interest. The results for the other control variables are available upon request. We also limit the results we present in the following way, to keep our exposition concise. Among the proxy variables for work load, used in regression (8), we focus on hours. Among the estimations for the sickness variables, regressions (5) through (7), we focus on (5), the baseline job-spell-fixed-effects regression.

Panel 1 of Table 8 recaps our main results. Column (1) is for log hours, from column (1) of Table 2, and

columns (2)~(7) are for the sickness variables, from columns (1)~(6) Table 4. In Panel 2 of Table 8, we experiment with dropping managers. We do so because an individual worker's health is more likely to affect the sales of her employer if she holds a managerial position. We obtain similar results as compared with our main results in Panel 1. These findings provide additional evidence against reverse causality.

We next address the concern of endogeneity. Two examples include organizational changes (e.g. Dahl 2011) that can lead to worker stress, and the adoption of new technology in the firm. It is unclear how these would operate, as organizational change may be either positively or negatively correlated with firm performance, and new technology might increase or decrease worker stress depending on its nature and implementation.

In Panel 3 of Table 8, we control for organizational changes by including the following measures for the complexity of the firm as an organization: the numbers of management layers (Caliendo, Monte and Rossi-Hansberg 2015), imported and exported products, and import source countries and export destination countries, by firm by year. We normalize these complexity variables by firm sales, include the logs of the ratios in our estimation, and report 100 times the coefficient estimates in panel 3. The coefficient estimate for the number of management layers is mostly positive for the sickness variables, suggesting that changes in organizational complexity tend to increase worker stress, consistent with Dahl (2011). These coefficient estimates, however, lack statistical significance.[33] Meanwhile, the changes in firm sales still have significant impacts on anti-depressant uses and burnout hospitalizations.[34]

In Panel 4 of Table 8, we address the concern of endogeneity by constructing the following Bartik-style/shift-share instrument for firm sales.[35] Let Q_{kt} denote the aggregate output of product k in year t in Denmark, and s_{jk} denote product k's share in firm j's output in the pre-sample year of 1994. Then our instrument for j's sales, Y_{jt}, is $I_{jt} = \sum_k s_{jk} Q_{kt}$. The intuition of our instrument is as follows. To the extent that output demand, Q_{kt},

[33] The coefficients of the other complexity variables are mostly insignificant, too.

[34] Because the elasticity of sickness rate with respect to sales depends on the coefficients of log sales and all the complexity variables, we choose to report 100 times the raw coefficient estimates. Their magnitudes are not directly comparable to Panel 1, where we report elasticity estimates.

[35] This type of instrument is widely used in the literature (e.g. Blanchard and Katz 1992, Card 2009, Luttmer 2006, Hummels et al. 2014, and Nakamura and Steinsson 2014).

fluctuates over time for exogenous reasons, firm j's exposure to these fluctuations depends on the pre-sample output shares, s_{jk}. While some Danish firms may produce large shares of the aggregate national output, Q_{kt}, for some products, our identification requires that the shocks are exogenous to individual workers, and predicted firm sales are likely to be less correlated with omitted variables and more exogenous to individual workers than actual sales.

Our shift-share variable is positive and significant in the first stage estimation, and its F-statistics is 33.52 (see the Data Appendix for more details). We report the results of the second stage estimation in Panel 4 of Table 8. The coefficient estimates are positive and significant for most sickness variables, and also for log hours. They are also larger in magnitude than our benchmark estimates in Panel 1, where we use actual sales, although they are not statistically different from the benchmark numbers. These findings are consistent with our earlier discussions, in sub-section 5.1, that our benchmark estimates may be conservative, since reverse causality may bias these estimates towards 0.

We now explore whether firm sales have different effects for men vs. women. We do so for two reasons. One, men and women respond differently to stressful events (e.g. Altemus, Sarvaiya, and Epperson 2014). Two, they also have different rates of depression; e.g. in our data, while 3.04% of men use anti-depressants, 4.71% of women do so (Data Appendix). In Panels 5 and 6 of Table 8, we do our estimation separately for the sub-samples of men and women, and our findings are as follows. First, the effect of firm sales is significant for most sickness variables for both genders; i.e. increases in firm sales lead to adverse health outcomes for both men and women. Second, firm sales are significant for men's hours. Finally, the difference between men and women in the elasticity of sickness with respect to sales varies across sickness variables. Men have larger elasticity estimates for work-burnout-related hospitalizations, while women show stronger responses for anti-depressants, heart-disease drugs, and hospitalizations due to strokes and alcoholism.

We then investigate whether the effects of firm sales vary with the unemployment (UI) rate in the local labor market. A high UI rate may imply a large pool of potential workers, so that the firms find it easy to increase employment when sales increases. Alternatively, the high UI rate may decrease the workers' outside option and

induce them to provide more effort (e.g. Lazear, Shaw and Stanton 2016). As a result, how the effects of firm sales vary with labor-market tightness is ambiguous. We calculate UI rate by commuting zone by year (see Data Appendix for the details), augment our regressions with the interaction between UI rate and log firm sales, and report 100 times the coefficient estimates in Panel 7 of Table 8.[36] The coefficient estimate of the UI-rate interaction is sometimes negative and sometimes positive. Meanwhile, the coefficient estimate for log sales remains positive and significant.

We next explore how the effects of firm sales vary with worker tenure. While recent hires might have an easier time adjusting work load than high-tenured workers, they are also more likely to have experienced prior displacement and so more sensitive to changes in stress. Therefore, how the effects of firm sales vary with tenure is ambiguous. We classify workers as high-tenure if they have two or more years of tenure, and report 100 times the coefficient estimates in Panel 8 of Table 8. The coefficient estimates of the interaction between log sales and the high-tenure dummy do not show a clear pattern, while those of log sales are positive and significant.

Finally, we report the results for our placebo variable, in-born errors of metabolism, in column (8) of Table 8. Its coefficient estimates tend to be negative, and even when they are positive, they are statistically insignificant. These results provide further evidence that our main results, for the other sickness variables, are not driven by spurious correlation.

8. Marginal Disutility and Welfare Loss from Higher Sickness Rates

So far we have reported a rich set of results showing that increases in firm sales make individual workers less healthy, but they also enable those workers to increase their earnings. We now draw out the economic significance of these results, by quantifying the average worker's expected welfare loss from increased sickness rates, and then comparing this loss with the average worker's wage gains. The key idea in this exercise is that the share weight of a particular illness in workers' expected welfare loss is in proportion to the marginal disutility of

[36] Note that we report the raw coefficient estimates in Panel 7, and their magnitudes are not directly comparable to Panel 1, where we report elasticity estimates.

that illness. Under some restrictive assumptions, that weight, in turn, can be related to the health expenditure share of that illness. Using data on expenditures then enables us to provide a proxy measure of the compensating variation a worker would demand for higher risks of that illness; i.e. its marginal disutility. We first lay down the theoretical motivation for our approach, and then perform quantification.

8.1 Theoretical Motivation

We follow the standard framework in the health literature, and assume that the average worker may live in the healthy state, with income *I* and utility *u(.)*, or sick state $g = 1 \ldots S$, with utility $v_g(.)$ and income I_g. I_g includes both monetary income and disutility from sickness, and takes treatment into account. Assume that $I_g < I$ for all g and $v_g(I) \leq u(I)$ for all income level *I*; i.e. after treatment, utility remains lower when sick. Let the probability of sick state g be $p_g > 0$. Then the average worker's expected utility is $(1 - \sum_g p_g)u(I) + \sum_g p_g\, v_g(I_g)$, where $1 - \Sigma_g p_g > 0$ is the probability of being healthy. Consider compensation *M*, invariant across state, that equates this expected utility with the disease-free benchmark of *u(I)*:

$$u(I) = u(I + M) + \sum_g p_g\, [v_g(I_g + M) - u(I + M)]. \qquad (9)$$

In equation (9), *M* is the compensating variation relative to the disease-free benchmark, and provides the monetary value of the average worker's welfare loss from the presence of the sick states. It follows, then, that the average worker's expected welfare loss from higher sickness rates equals

$$\frac{\partial M}{\partial \psi} = \sum_g \frac{\partial M}{\partial p_g} \frac{\partial p_g}{\partial \psi},\ \frac{\partial M}{\partial p_g} > 0\,. \qquad (10)$$

In equation (10), ψ represents shocks to TFP or demand facing the firm, which corresponds to *j*'s sales conditional on input uses in our empirics. Since we have estimated how sickness rates respond to sales, $\partial p_g / \partial \psi$, in section 5, equation (10) says that we can calculate the average worker's expected welfare loss, $\partial M / \partial \psi$, if we know the values of the marginal disutility, $\partial M / \partial p_g$. The intuition of $\partial M / \partial p_g$ is similar to the value of a statistical life or injury, or VSLI; i.e. it shows the monetary compensation that the average worker demands in exchange for

heightened risk of disease g.[37]

Our quantification of $\partial M / \partial p_g$ starts with the utility share weight,[38]

$$\beta_g \equiv \frac{\partial \ln M / \partial \ln p_g}{\sum_g \partial \ln M / \partial \ln p_g}, \tag{11}$$

where β_g is positive and sum to 1 across diseases. Equation (11) says that β_g is high if an increase in the sick rate of g, p_g, has a large effect on the average worker's expected welfare loss. Assuming $v_g(.) = v_l(.)$, a common assumption in the state-dependence literature,[39] we show that (Theory Appendix 3)

$$\frac{\partial M / \partial p_g}{\partial M / \partial p_l} = \frac{\beta_g / p_g}{\beta_l / p_l} \text{ for all } g \neq l \tag{12}$$

Equation (12) says that relative to disease l, the marginal disutility of disease g is high if g has a low frequency, p_g, but a high utility share weight, β_g. This is intuitive, because a low frequency tends to reduce utility share weight, β_g. The fact that utility share weight is high despite low frequency indicates low post-treatment income, I_g, or high marginal disutility.

We now relate β_g to expenditure shares, by laying out a simple framework of optimal treatment choice. Let s_g denote the pre-treatment pain of disease g in monetary equivalent terms. s_g may capture the effect of disease symptoms and/or mortality risk. Treatment, t_g, ranges in effectiveness from 0 to 100%. The private cost of treatment is $c(t_g, s_g)$, which captures both monetary costs (e.g. co-pay) and non-monetary costs (e.g. disutility from treatment), and is affected by the state of medical technology and institutional features of the healthcare

[37] Theory Appendix 2 explores additional theoretical properties of $\partial M / \partial p_g$.

[38] An alternative approach is to identify the full set of structural parameters and then plug them into equations (9) and (10). However, the literature has not reached a consensus about how to identify these parameters. E.g. Viscusi and Evans (1990) and Finkelstein et al. (2013) report negative state dependence; i.e. $u'(.) > v_g'(.)$. Lillard and Weiss (1998), Edwards (2008) and Ameriks et al. (2016) report positive state dependence; i.e. $u'(.) < v_g'(.)$. Evans and Viscusi (1991) report zero state dependence; i.e. $u'(.) = v_g'(.)$.

[39] One exception is Evans and Viscusi (1990), who allow $v(.)$ to differ across two injury types but find results consistent with $u(.) = v_1(.) = v_2(.)$. Note that this assumption does not imply the same utility level for diseases g and l, since I_g and I_l can be different. In addition, it does not specify how $v_g(.)$ and $v_l(.)$ compare with $u(.)$; i.e. we still accommodate positive, negative or zero state dependence.

system.[40] If sick, the average worker chooses treatment to maximize $I_g = I - s_g(1 - t_g) - c(t_g, s_g)$.[41] The optimal treatment can be 100%, which is full recovery, or less than 100%, which is partial recovery.

Let $E_g = p_g C(t_g, s_g)$ denote expenditure on g, where $C(.)$ denotes the social monetary cost of treatment. $C(.)$ differs from the private cost, $c(.)$, because some diseases can be communicable, and also because healthcare expenses are typically paid for using public resources or through health insurance. Let $\mu_g = E_g/(\sum_g E_g)$ denote the expenditure share of g. Assume that $c(.)$ and $C(.)$ are increasing and convex with respect to treatment. We show (Theory Appendix 4) that under some technical assumptions, for all diseases $l \neq g$,

Proposition 1 Under partial recovery, μ_g has the same rank order as β_g if $p_g = p_l$, and $\mu_g/\mu_l = \beta_g/\beta_l$ if $s_g = s_l$. Under full recovery, $\mu_g/\mu_l = \beta_g/\beta_l$.

Proposition 1 says that expenditure shares vary across diseases in the same direction as utility share weights, β_g, and so they provide a useful measure for β_g. This is intuitive, because expenditures reflect disease frequency and severity, two important factors that also affect β_g. This result is also useful for our quantification below, because data on disease frequencies and expenditures are easy to obtain. Importantly, we do not need private and social costs to be identical for Proposition 1. This means that our results accommodate moral hazard, a very important feature of the healthcare system (e.g. Cutler and Zeckhauser 2000).[42] We make no assumptions about how the first-order derivatives, $u'(.)$ and $v_g'(.)$, compare with each other, either, and so we accommodate positive, negative or zero state dependence.

On the other hand, we have derived Proposition 1 under strong assumptions; e.g. we have subsumed the supply side of health care into the reduced-form private and social cost functions. As a result, Proposition 1 should be treated with caution, since it can be challenging to identify the exact health benefits of expenditure for a large

[40] A literature studies whether physicians' financial incentives lead them to prescribe suboptimal treatments (e.g. McGuire 2000), and most studies focus on the U.S. We assume, following this literature, that physicians have patients' best interests in mind, and abstract away from physicians' financial incentives, given the differences in the healthcare system between Denmark (and many other high-income countries) and the U.S. We discuss capacity constraints in Theory Appendix 4.

[41] We have implicitly assumed that the amount of resources, I, is not affected by sickness. This is a common assumption in the literature (e.g. Hall and Jones 2007, Finkelstein et al. 2013).

[42] In the U.S., low-health-risk individuals may choose not to buy health insurance. Adverse selection is likely to be far less important for Denmark, and many other high-income countries, where healthcare is universal.

number of diseases. A large literature has shown that many factors affect healthcare spending, and high expenditure may not always imply large health benefits.[43] We discuss the qualifications and caveats of our approach in the context of this literature in Theory Appendix 4.

8.2 Quantification

We now use the framework developed in the previous sub-section to guide our quantification. Because our framework builds on a highly stylized model, the goal of our quantification is not to deliver structural parameters. Rather, we aim to use readily available data to provide reasonable approximations to marginal disutility, $\partial M / \partial p_g$, and then use these values to gauge the average worker's welfare loss due to the higher sickness rates that result from rising firm sales.

We start with equation (12), where the disease frequency, p_g, comes from the mean values that we report in Table 1, and the utility share weight, β_g, comes from the expenditure shares in Table 1. For ease of reference, we gather these data in columns 1 and 2 of Table 9. Of the six sickness variables we examined previously, we leave out narrow burnouts because they are a subset of the hospitalizations included in broad burnouts.

Table 9 shows that the variation of sickness rates and expenditure shares across diseases is both intuitive and consistent with our theory. For example, the stress and heart diseases that can be treated with prescription drugs happen with high frequencies but have low expenditure shares, suggesting that these conditions are not very severe. In comparison, the hospitalizations due to heart attacks and alcoholism are severe. Consistent with this, they have high expenditure shares despite low frequencies.

Equation (12) delivers the ratios of marginal disutility, but not the levels. We follow the literature to estimate the marginal disutility of work injury, which is VSI, and then combine VSI with the *ratios* of marginal disutility, delivered by (12), to obtain the *levels* of marginal disutility. We have included the mean rate and expenditure share of injury in the last row of Table 9.

[43] E.g. Cutler (2005) argues that "where we have spent a lot, we have received a lot in return". Chandra and Skinner (2012) note that the literature has reported both positive and negative correlations between healthcare spending and outcome for the U.S., and provide evidence that expensive treatments with limited benefits are more widely used in the U.S. than in Europe. See also Newhouse (1992), Hall and Jones (2007), and Howard et al. (2015).

In order to obtain the marginal disutility of injury, we follow the VSI literature and estimate a Mincer regression augmented by occupational injury rate,[44] and use the log-wage-injury-rate gradient to compute VSI (see the Data Appendix for the details). Because the estimated gradient has its 95% confidence interval, we can compute the 95% confidence interval of VSI as well. We report our VSI estimate, 1.56 million DKK, in column 3 of Table 9, and report its 95% confidence interval in column 4. Our VSI estimate is larger than those obtained using U.S. data, because our data includes severe injuries only.

We then calculate the marginal disutility using equation (12), by combining our VSI estimate with the data for sickness rates and expenditure shares. We report our results in column 3 of Table 9. To see the intuition of our computation, consider the example of stroke hospitalization. As compared with injury, its expenditure share is slightly lower (2.02% vs. 2.26%), but its frequency is much lower (1.6 per thousand vs. 3.9 per thousand). As a result, equation (12) says that its marginal disutility is 2.23 times that of injury, or DKK 3.48 million (= 1.56 x 2.23). Since our VSI estimate has its 95% confidence interval, we use the upper and lower bounds of this interval to compute the corresponding bounds of our marginal-disutility values, and report them in Column 4 of Table 9.

Table 9 shows that the values of marginal disutility and their rankings across diseases are both intuitive. The two sickness conditions that can be treated by prescription drugs have low marginal disutility, 22,550 DKK and 35,600 DKK. Stress that can be treated with prescription drugs has a much lower marginal disutility (22,550 DKK) than broad burnouts, which require hospitalization (499,510 DKK). Both broad burnouts and strokes require hospitalization, but strokes are more severe, and so have a higher marginal disutility (3.48 million DKK). The marginal disutility of strokes hospitalization, in turn, is lower than that of mortality, which is $5-6.2 million (Viscusi and Aldy 2003), or DKK 27.78-34.44 million.

We now calculate the average worker's expected welfare loss using equation (10). We first translate our elasticity estimates in Table 4, $\partial \ln p_g / \partial \psi$, into estimates for level changes, $\partial p_g / \partial \psi$, by multiplying the elasticity estimates by the sickness rates, p_g. We report 100 times these estimates in column 5 of Table 9. Plugging

[44] The main idea of this regression is that workers take injury risks into account when making occupational choices, demanding high wages as compensation where occupational injury rates are high. This approach is compatible with positive, negative, or zero state dependence of utility, like our framework.

these estimates and the values of marginal disutility into (10), we obtain that, in response to a 10% increase in firm sales, the average worker suffers an expected welfare loss of 74.6 DKK.

In order to clarify the economic significance of this welfare loss, we compare it with the increases in earnings from rising sales. Using our results from column (2) of Table 2, we obtain that, following a 10% increase in firm sales, earnings increase by 318.7 DKK. As a result, the average worker's expected welfare loss amounts to 23.4% of her earnings gain. This result suggests that a substantial fraction of the monetary gains from rising sales can be attributed to compensating differential, for higher risks of sickness, in the spirit of Rosen (1986).

9. Conclusion

In this paper we merge matched worker-firm data from Denmark with administrative data on prescription drugs and hospitalization records, in order to study how changes in firm sales affect the health of its employees. Our rich data allow us to base our identification on changes within worker-firm matches, or job spells, examine sickness conditions that are explicitly tied to work burnouts, explore the differential effects of firm sales on ex-ante-high-risk workers, and check whether workers become sick after sales increases at their employers.

Our findings are as follows. First, within job spells, as firm sales increases, workers have longer hours and suffer higher probabilities of stress and depression, such as anti-depressant uses and burnout-related hospitalizations, as well as elevated risks for heart diseases and strokes, including stroke-related hospitalizations. Second, the effects of firm sales are more pronounced for the following high-risk groups: older workers, job-strained workers, and those with long initial work hours. Third, the effects of sales increases on worker sickness are robust to the use of quarterly-frequency data and multi-period difference-in-difference estimation; i.e. the worker cohorts who experience large sales increases develop higher risks of sickness in subsequent quarters, and the effects are larger for older workers for the sickness conditions where age is a significant risk factor. These novel results suggest that work demand increases individuals' workplace stress and elevates their sickness risk.

We then compute the marginal disutility of our sickness variables, and quantify the average worker's ex-ante welfare loss due to higher sickness rates. Although our approach builds on a highly stylized model, it uses

readily-available data and delivers sensible and intuitive marginal-disutility values across diseases. We find that the average worker's welfare loss accounts for nearly one quarter of her earnings gains from rising firm sales. More broadly, our approach extends VSLI to non-injury diseases, because the marginal disutility of diseases has the same intuition as VSLI. While the marginal-disutility values from our approach are clearly reduced-form, they may help gauge the economic benefits of government policies and regulations that reduce the risks of diseases, in the same way that VSLI gauges the economic benefits of reduced risks of mortality and injury.

Our results highlight the importance of workplace stress, which is emerging as an important public-health concern in the U.S.[45] For example, the U.S. CDC (Centers for Disease Control and Prevention) lists work-related stress as the leading workplace health problem, ahead of physical inactivity and obesity,[46] and Goh, Pfeffer and Zenios (2015) argue that workplace stress contributes $190 billion, annually, to U.S. healthcare costs. Unfortunately, the public provision of mental-health care lags far behind demand; e.g. in 44 U.S. states the biggest mental-health institution is a prison.[47] Fortunately, many employers are taking action. Large U.S. companies are offering training in cognitive behavioral skills, scented relaxation rooms, smart phone apps for mental-health issues, "living walls" decorated with plants, and outdoor cafes with wildflowers.[48] Perhaps these endeavors reflect a growing private sector recognition of the connection between work demand, work load and employee stress identified in our study.

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[45] Our results are also reminiscent of Sigmund Freud. In his classic, "Civilization and Its Discontents", he postulates that, as the civil society grows in terms of technology and profits, its citizens become neurotic and discontent. The recent hit song, Stressed Out, by the group Twenty One Pilots, echoes this theme.

[46] E.g. https://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2016/aag-workplace-health.pdf

[47] 'Mental Health: Out of the Shadows", Economist, April 25, 2015, 56-57.

[48] E.g. "To Cut Office Stress, Try Butterflies and Medication?", by Sue Shellenbarger, The Wall Street Journal, October 9, 2012, and "Management: Tackling Mental Health, One Text at a Time …", by Rachel Emma Silverman, the Wall Street Journal, July 20, 2016.

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Theory Appendix

1. Equations (2) and (3)

We first provide detailed derivations for equations (2) and (3), and then show that they generalize to monopolistically competitive output markets with CES preferences. Like in the text, we leave out subscripts, to ease exposition.

Let $V = \left(K^{\frac{\sigma-1}{\sigma}} + M^{\frac{\sigma-1}{\sigma}} + (La)^{\frac{\sigma-1}{\sigma}} \right)^{\frac{\sigma}{\sigma-1}}$. We first prove the log linear approximation of lnV; i.e.

$$lnV \approx \theta_M \, lnM + \theta_K \, lnK + \theta_H \, ln(La) + c_4 \qquad \text{(A1)}$$

where c_4 and the θ's are positive constants and $\theta_H + \theta_K + \theta_M = 1$.

Proof Let $y_1 = \ln(M/(La))$ and $y_2 = \ln(K/(La))$. Then $V = (La)(e^{y_1\frac{\sigma-1}{\sigma}} + e^{y_2\frac{\sigma-1}{\sigma}} + 1)^{\frac{\sigma}{\sigma-1}}$ and $\ln V = \ln(La) + g(.)$, where $g(.) = \frac{\sigma}{\sigma-1} ln(e^{y_1\frac{\sigma-1}{\sigma}} + e^{y_2\frac{\sigma-1}{\sigma}} + 1)$. The first-order Taylor approximation for $g(.)$ is $g(y_1, y_2) = g(y_{10}, y_{20}) + (\partial g(y_{10}, y_{20})/\partial y_1)\,(y_1 - y_{10}) + (\partial g(y_{10}, y_{20})/\partial y_2)\,(y_2 - y_{20})$, where y_{10} and y_{20} are constants. $\partial g(y_{10}, y_{20})/\partial y_1 = e^{y_1\frac{\sigma-1}{\sigma}}/(e^{y_1\frac{\sigma-1}{\sigma}} + e^{y_2\frac{\sigma-1}{\sigma}} + 1) = \theta_M$ and $\partial g(y_{10}, y_{20})/\partial y_2 = e^{y_2\frac{\sigma-1}{\sigma}}/(e^{y_1\frac{\sigma-1}{\sigma}} + e^{y_2\frac{\sigma-1}{\sigma}} + 1) = \theta_K$ lie between 0 and 1 for all values of y_{10} and y_{20}. Let $c_4 = g(y_{10}, y_{20}) - y_{10}\theta_M - y_{20}\,\theta_K$ and $\theta_H = 1 - \theta_K - \theta_M$, and we have equation (A1). **QED**.

It is easy to show that a firm's labor demand equation is

$w = \psi V^{\frac{1}{\sigma}}(La)^{-\frac{1}{\sigma}} = YV^{-\frac{\sigma-1}{\sigma}}(La)^{-\frac{1}{\sigma}}$.

We combine this expression with the supply equation for work load, $w = c_2 a^{\gamma}, \gamma > 0$, to obtain

$c_2 a^{\gamma} = \psi V^{\frac{1}{\sigma}}(La)^{-\frac{1}{\sigma}} = YV^{-\frac{\sigma-1}{\sigma}}(La)^{-\frac{1}{\sigma}}$.

We now take the log of both sides of this expression, and then substitute out lnV using the approximation (A1), to obtain equations (2) and (3) in the text.

If consumer preferences are CES with substitution elasticity σ_2, and the market for j's output is monopolistic competition, then $Y = (\frac{BA^{\sigma_2-1}}{P^{1-\sigma_2}})^{\frac{1}{\sigma_2}} V^{\frac{\sigma_2-1}{\sigma_2}}$, where B is consumer expenditure and P the CES price index. In this case, $\psi = (\frac{BA^{\sigma_2-1}}{P^{1-\sigma_2}})^{\frac{1}{\sigma_2}}$ and $Y = \psi V^{\frac{\sigma_2-1}{\sigma_2}}$. Firm j's labor demand equation is then

$w = \frac{\sigma_2-1}{\sigma_2}\psi V^{\delta}(La)^{-\frac{1}{\sigma}} = \frac{\sigma_2-1}{\sigma_2} YV^{-\frac{\sigma-1}{\sigma}}(La)^{-\frac{1}{\sigma}}, \delta = \frac{\sigma_2-1}{\sigma_2} - \frac{\sigma-1}{\sigma}$.

Note that σ_2 is the substitution elasticity for consumption, and σ is the substitution elasticity for production. We follow similar steps as above, and obtain the following generalizations of equations (2) and (3):

$$ln\, a = constant + \frac{ln\,\psi}{\gamma+1/\sigma-\delta\theta_H} + \frac{\delta\theta_K \ln K}{\gamma+1/\sigma-\delta\theta_H} + \frac{\delta\theta_M \ln M}{\gamma+1/\sigma-\delta\theta_H} + \frac{(\delta\theta_H-1/\sigma)\ln L}{\gamma+1/\sigma-\delta\theta_H},$$

$$ln\, a = constant + \frac{\sigma ln\, Y}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{\theta_K(\sigma-1)ln\, K}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{\theta_M(\sigma-1)ln\, M}{\sigma(\gamma+\theta_H)+1-\theta_H} - \frac{((\sigma-1)\theta_H+1)\ln L}{\sigma(\gamma+\theta_H)+1-\theta_H}.$$

2. First and Second Order Derivatives of M with respect to p_g

Let $p_H = 1 - \Sigma_g p_g > 0$ denote the probability of the healthy state. Differentiate both sides of equation (9)

$$\frac{\partial M}{\partial p_g}\sum\nolimits_g p_g[u'(I+M) - v_g'(I_g+M)] + u(I+M) - v_g(I_g+M) = \frac{\partial M}{\partial p_g}u'(I+M). \qquad \text{(A2)}$$

This implies

$$\frac{\partial M}{\partial p_g} = \frac{u(I+M) - v_g(I_g+M)}{u'(I+M) + \sum_l p_l[v_l'(I_l+M) - u'(I+M)]} = \frac{u(I+M) - v_g(I_g+M)}{p_H u'(I+M) + \sum_l p_l v_l'(I_l+M)} \quad \text{(A3)}$$

$\partial M/\partial p_g > 0$ because $v_g(I_g+M) \leq u(I_g+M) < u(I+M)$, and $u'(.) > 0$ and $v_g'(.) > 0$ for all g. This proves the claim that $\partial M/\partial p_g > 0$ in equation (10).

We now show properties of the second order derivatives; i.e. if $\partial M/\partial p_g$ is large, $\frac{\partial^2 M}{\partial (p_g)^2} > 0$ and $\frac{\partial^2 M}{\partial p_g \partial p_l} > 0$. To economize on notation, let $u'(.)$ denote $u'(I+M)$, and $v_g'(.)$ denote $v_g'(I_g+M)$, etc. We differentiate both sides of equation (A2) with respect to p_g, to obtain

$$\frac{\partial^2 M}{\partial (p_g)^2}\sum_g p_g[u'(.) - v_g'(.)] + (\frac{\partial M}{\partial p_g})^2 \sum_g p_g[u''(.) - v_g''(.)] + 2\frac{\partial M}{\partial p_g}[u'(.) - v_g'(.)]$$

$$= \frac{\partial^2 M}{\partial (p_g)^2} u'(.) + u''(.)(\frac{\partial M}{\partial p_g})^2$$

Re-arranging, we get

$$-\frac{\partial^2 M}{\partial (p_g)^2}\underbrace{[p_H u'(.) + \sum_g p_g v_g'(.)]}_{>0} = (\frac{\partial M}{\partial p_g})^2 \underbrace{[p_H u''(.) + \sum_g p_g v_g''(.)]}_{<0} - 2\frac{\partial M}{\partial p_g}\underbrace{[u'(.) - v_g'(.)]}_{??}.$$

In this expression, the sign of $u'(.) - v_g'(.)$ depends on the nature of state dependency. If $\partial M/\partial p_g$ is large, the first term on the right-hand side dominates, meaning that the right-hand side is negative. As a result, $\frac{\partial^2 M}{\partial (p_g)^2} > 0$.

We then differentiate both sides of equation (A2) with respect to p_l, to obtain

$$\frac{\partial^2 M}{\partial p_g \partial p_l}\sum_g p_g[u'(.) - v_g'(.)] + \frac{\partial M}{\partial p_g}\frac{\partial M}{\partial p_l}\sum_g p_g[u''(.) - v_g''(.)] + \frac{\partial M}{\partial p_g}[u'(.) - v_l'(.)] + \frac{\partial M}{\partial p_l}[u'(.) - v_g'(.)]$$

$$= \frac{\partial^2 M}{\partial p_g \partial p_l} u'(.) + u''(.)\frac{\partial M}{\partial p_g}\frac{\partial M}{\partial p_l}$$

Re-arranging, we get

$$-\frac{\partial^2 M}{\partial p_g \partial p_l}\underbrace{[p_H u'(.) + \sum_g p_g v_g'(.)]}_{>0} =$$

$$\frac{\partial M}{\partial p_g}\frac{\partial M}{\partial p_l}\underbrace{[p_H u''(.) + \sum_g p_g v_g''(.)]}_{<0} - \frac{\partial M}{\partial p_g}\underbrace{[u'(.) - v_l'(.)]}_{??} - \frac{\partial M}{\partial p_l}\underbrace{[u'(.) - v_g'(.)]}_{??}.$$

Again, the signs of $u'(.) - v_g'(.)$ and $u'(.) - v_l'(.)$ depend on the nature of state dependency. If $\partial M/\partial p_g$ is large, the first term on the right-hand side dominates, and so $\frac{\partial^2 M}{\partial p_g \partial p_l} > 0$.

The results in this section, about the properties of the first and second order derivatives of M, suggest that the relationship between M and p_g is reminiscent of a cost function.

3. Equation (12)

We start by using the definition of β_g, to derive

$$\frac{\beta_g}{\beta_l} = \frac{\partial \ln M / \partial \ln p_g}{\partial \ln M / \partial \ln p_l} = \frac{(\partial M / \partial p_g)(p_g / M)}{(\partial M / \partial p_l)(p_l / M)} = \frac{p_g(\partial M / \partial p_g)}{p_l(\partial M / \partial p_l)}.$$

Re-arrange this expression, and we get equation (12). In addition, we can combine this expression with equation (A3), to show that

$$\frac{\beta_g}{\beta_l} = \frac{p_g[u(I+M) - v_g(I_g+M)]}{p_l[u(I+M) - v_l(I_l+M)]}. \quad \text{(A4)}$$

This equation says that β_g is high if disease g happens with a high frequency (i.e. high p_g), or if it is severe post treatment (i.e. low I_g).

4. Proposition 1

The technical assumptions for Proposition 1 are

$c(t_g, s_g)$ and $C(t_g, s_g)$ are increasing in s_g, and have the same rank order, (A5)

or

$\partial c(.)/\partial s_g < 0$, $\partial C(.)/\partial s_g < 0$, and both are large in magnitude. (A6)

Assumption (A5) implies that

$C(t_g, s) < C(t_l, s) \Leftrightarrow c(t_g, s) < c(t_l, s)$, and $C(t, s_g) < C(t, s_l) \Leftrightarrow c(t, s_g) < c(t, s_l)$.

To see the intuition of Proposition 1 under assumption (A5), suppose disease g is painful; i.e. s_g is high. Then because g leads to a large welfare loss if untreated, the marginal benefit of treatment is high, and so treatment, t_g, is high. In addition, after treatment, severity, I_g, remains high, because treatment is less than 100% effective and also costly (severity, I_g, is post treatment, but pain, s_g, is pre-treatment). As a result, social expenditure on g is high, because social cost, $C(.)$, has the same rank order as private cost, $c(.)$.

To see the intuition of Proposition 1 under (A6), consider bacterial infection. It's painful if untreated (i.e. s_g is high), but antibiotics offers very effective treatment at low cost, as described by (A6). As a result, severity, which is after treatment, is low, and so is expenditure, implying that both the true and measured marginal disutility are low.

We start by showing Proposition 1 under strong additional assumptions, to illustrate the intuition of the proof. We then sequentially relax these additional assumptions, to show that Proposition 1 continues to hold.

4.1 Stylized Setting

Assume that both private and social cost functions depend on treatment, t_g, only, and that there is no mortality risk. We show Proposition 1 under assumption (A5).

In this setting, the average worker's optimization problem simplifies to

$$\max_{t_g} \{I_g = I - s_g(1 - t_g) - c(t_g)\}$$

The literature has considered two types of solutions: an interior solution, or partial recovery, with $t_g < 1$, and a corner solution, or full recovery, with $t_g = 1$. We examine partial recovery first, and then full recovery.

Under partial recovery, the solution to the worker's optimal treatment involves $v_g'(.)[s_g - c'(t_g)] = 0$, or $s_g = c'(t_g)$. We differentiate $s_g = c'(t_g)$ with respect to s_g, and then apply the Envelope Theorem, to get

$$\frac{\partial t_g}{\partial s_g} = \frac{1}{c''(t_g)} > 0, \frac{\partial I_g}{\partial s_g} = -(1 - t_g) < 0. \quad \text{(A7)}$$

We have explained the intuition of equation (A7) above, in our discussions for assumption (A5). Equation (A7) also says that treatment depends on pain, s_g, but not on frequency, p_g. This is intuitive, because people seek treatment after they get sick, not before.

We can now relate the utility share weight, β_g, to expenditure share, μ_g. First, compare two diseases, g and l, with the same frequency, $p_g = p_l$. Suppose g is more painful ($s_g > s_l$). Then by equation (A7), treatment for g is higher ($t_g > t_l$), and expenditure for g is also higher ($E_g > E_l$), by assumption (A5). In addition, by (A7), post-treatment net income is lower under g ($I_g < I_l$), and so by (A4), the utility share weight of g is higher ($\beta_g > \beta_l$). Clearly, $\mu_g = E_g/(\sum_g E_g) > \mu_l = E_l/(\sum_g E_g)$ if and only if $E_g > E_l$.

Second, suppose that $s_g = s_l$ but $p_g > p_l$ for diseases g and l. Then equation (A7) says that $t_g = t_l$, and assumption (11) says that $C(t_g) = C(t_l)$. Therefore, expenditures are proportional to frequency ($E_g/E_l = p_g/p_l$). Equation (A7) also says that net income, post treatment, is the same under g and l ($I_g = I_l$), and so by (A4), utility share weights are also proportional to frequency ($\beta_g/\beta_l = p_g/p_l$ since $v_g(.) = v_l(.)$). As a result, $E_g/E_l = \beta_g/\beta_l = \mu_g/\mu_l$.

Finally, suppose we have full recovery instead of partial recovery. This means that treatment is 100% effective; i.e. $t_g = t_l = 1$ for all diseases g and l. Clearly, $E_g/E_l = \beta_g/\beta_l = \mu_g/\mu_l$, as in the previous case. This completes the proof.

4.2 $c(s_g,t_g)$ and $C(s_g,t_g)$

We show that Proposition 1 continues to hold, under assumption (A5), when private and social costs depend on both treatment, t_g, and pain, s_g.

In this case, the worker's optimization problem is

$$\max_{t_g}\{I_g = I - s_g(1 - t_g) - c(t_g, s_g)\} \quad \text{(A8)}$$

Equation (A8) says that optimal treatment depends on pain, s_g, but not on frequency, p_g. This is intuitive, because people seek treatment after they get sick, not before.

First, suppose $p_g = p_l$ but $s_g > s_l$. We show that $E_g > E_l$ and $\beta_g > \beta_l$ if the cross-partial of the private-cost function is lower than the 1, which equals the cross-partial of the benefit to utility of treatment, $s_g t_g$; i.e. $\frac{\partial^2 c(.)}{\partial t_g \partial s_g} < 1$

Consider partial recovery first. Utility maximization implies that $\partial I_g/\partial t_g = 0$, or that $s_g = \partial c(.)/\partial t_g$. This implies that

$$\frac{\partial t_g}{\partial s_g} = \frac{1 - \frac{\partial^2 c(.)}{\partial t_g \partial s_g}}{\frac{\partial^2 c(.)}{\partial (t_g)^2}} > 0 . \quad \text{(A9)}$$

The numerator is positive by the assumption that $\frac{\partial^2 c(.)}{\partial t_g \partial s_g} < 1$, and the denominator is positive by convexity of the function $c(.)$. Meanwhile, using the envelope theorem on the expression $I_g = I - s_g(1 - t_g) - c(s_g,t_g)$, we get

$$\frac{\partial I_g}{\partial s_g} = -(1 - t_g) - \frac{\partial c(.)}{\partial s_g} < 0 . \quad \text{(A10)}$$

Equations (A9) and (A10) show that equation (A7) still holds. We can then follow similar steps as in the previous sub-section, to show Proposition 1.

Second, suppose we have full recovery. We immediately have $t_g = t_l = 1$, and so $I_g = I - c(s_g,1) < I_l = I - c(s_l, 1)$. This means $\beta_g > \beta_l$. On the other hand, $E_g = p_g C(s_g,1) > E_l = p_l C(s_l,1)$ since $s_g > s_l$. Note that we get weaker results than in the previous sub-section, because now pain itself affects expenditure directly.

Finally, suppose $p_g > p_l$ but $s_g = s_l$. Under partial recovery, $t_g = t_l$ and $I_g = I_l$ by equations (A9) and (A10). As a result, $E_g/E_l = \mu_g/\mu_l = p_g/p_l = \beta_g/\beta_l$. Under full recovery, again $t_g = t_l = 1$, and $I_g = I_l$ since $s_g = s_l$. As a result, $E_g/E_l = \mu_g/\mu_l = p_g/p_l = \beta_g/\beta_l$. This completes the proof.

4.3 Mortality Risk

We now show that Proposition 1 still holds, under assumption (A5), when pain, s_g, captures the total effect of disease symptoms and mortality risk.

Suppose that sickness g is associated with the mortality risk of p_{gD}, and p_{gD} is monotonically related to s_g with $\partial p_{gD} / \partial s_g > 0$. We also assume that treatment may reduce mortality risk; i.e. $\partial p_{gD} / \partial t_g \leq 0$.

Let v_D denote the utility level under mortality, and $v_{g1}(.)$ denote the utility function in sick state g, conditional on being alive. We normalize $v_D = 0$, as in Murphy and Topel (2003) and Hall and Jones (2007), and assume that the utility function $v_{g1}(.)$ has the same properties as the utility function we use in the text, $v_g(.)$.

Let I_{g1} denote the severity of g caused by the symptoms of g. We assume, as in the text, that I_{g1} depends on the pain, s_g, and treatment, t_g, with $\partial I_{g1} / \partial s_g < 0$. Then the expected utility in sick state g is equal to $(1 - p_{gD})v_{g1}(I_{g1}) + p_{gD}v_D = (1 - p_{gD})v_{g1}(I_{g1})$.

Given treatment, t_g, this expected utility is a monotonic function of s_g. To see this, suppose s_g increases. On the one hand, mortality risk, p_{gD}, increases, and this tends to decrease expected utility. On the other hand,

severity also increases (i.e. I_{g1} decreases), and this also tends to decrease expected utility. In other words, this setting formalizes the intuition that s_g captures the total effect of disease symptoms and mortality risk.

We now relate the expected utility to our framework in section 6, by noting that there exists I_g such that

$$v_g(I_g) = (1 - p_{gD})v_{g1}(I_{g1}). \quad \text{(A11)}$$

Equation (A11) defines I_g as an implicit function of t_g and s_g. This implicit function is the counterpart of equation (A8), and gives us a chance to work with a more general functional form than (A8). Below, we list the properties of this function that are important for our results (these properties all hold for equation (A8), with cost $c(s_g,t_g)$). 1. I_g is continuous and differentiable with respect to t_g and s_g. 2. If $t_g = t_l = 1$ and $s_g > s_l$, then $I_g < I_l$. 3. The choice of t_g to maximize I_g is a well-defined problem. 4. For the optimal treatment t_g, $\partial t_g / \partial s_g > 0$.

First, suppose $p_g = p_l$ but $s_g > s_l$. Under partial recovery, optimization sets $\partial I_g / \partial t_g = 0$. Apply the Envelop Theorem to equation (A11), and we have

$$\frac{\partial I_g}{\partial s_g} v_g^{'}(.) = -v_{g1}(.)\frac{\partial p_{gD}}{\partial s_g} + (1 - p_{gD})v_{g1}^{'}(.)\frac{\partial I_{g1}}{\partial s_g}. \quad \text{(A12)}$$

Equation (A12) says that $\partial I_g / \partial s_g < 0$, because $\partial p_{gD} / \partial s_g > 0$ and $\partial I_{g1} / \partial s_g < 0$. This result, together with the property that $\partial t_g / \partial s_g > 0$, implies that equation (A7) holds in this case. We can follow the same steps as in the previous sub-section, to show that $\mu_g > \mu_l$.

Second, under full recovery, we have $t_g = t_l = 1$, and so $I_g < I_l$, because $s_g > s_l$. We can follow the same steps as in the previous sub-section, to show that $\mu_g > \mu_l$.

Finally, suppose $p_g > p_l$ but $s_g = s_l$. We can follow the same steps as in the previous sub-section, to show that $E_g/E_l = \mu_g/\mu_l = p_g/p_l = \beta_g/\beta_l$.

4.4 Proposition 1 under Assumption (A6)

We now show that Proposition 1 also holds under assumption (A6). We focus on the case of $p_g = p_l$ but $s_g > s_l$ with partial recovery. The proof for the other cases is similar to the previous sub-section.

First, the treatment technology remains (A8). Because $c(t_g, s_g)$ and $C(t_g, s_g)$ are increasing and convex with respect to t_g, the average worker's optimal choice of treatment is a well-defined problem.

Under partial recovery, the Envelope Theorem implies that

$$\frac{\partial I_g}{\partial s_g} = -(1 - t_g) - \frac{\partial c(.)}{\partial s_g}$$

Under full recovery, we have

$$\frac{\partial I_g}{\partial s_g} = -\frac{\partial c(.)}{\partial s_g}$$

In both cases, $\partial I_g/\partial s_g > 0$ under Assumption (12) (i.e. $\partial c(.)/\partial s_g < 0$ and is large in magnitude).

On the other hand, the expenditure on g is $E_g = p_g C(.)$, and so

$$\frac{\partial E_g}{\partial s_g} = p_g\left(\frac{\partial C(.)}{\partial s_g} + \frac{\partial C(.)}{\partial t_g}\frac{\partial t_g}{\partial s_g}\right)$$

We have $\partial E_g/\partial s_g < 0$ under Assumption (A6) (i.e. $\partial C(.)/\partial s_g < 0$ and is large in magnitude).

Now consider the diseases $l \neq g$ with $p_g = p_l$ and $\beta_g > \beta_l$. Equation (A4) implies that $I_g < I_l$. We then have $s_g < s_l$, because $\partial I_g/\partial s_g > 0$. Thus $E_g > E_l$ and $\mu_g > \mu_l$, because $\partial E_g/\partial s_g < 0$.

4.5 Fixed Cost in Treatment

We first illustrate that Proposition 1 holds for some form of fixed cost in treatment. We then discuss the caveats of Proposition 1 for more general forms of fixed cost.

Suppose that the private cost function is linear in t_g, and has a fixed cost component; i.e. $c(t_g, s_g) = \lambda t_g + F(s_g)$, where λ is a positive constant. Assume that Assumption (A5) holds. This implies that $F(.)$ is an increasing function. Note that, in this case, $c(.)$ is no longer strictly convex in treatment, t_g.

Consider two diseases, g and l. Suppose l requires surgery, but g does not. Then $s_g < s_l$, and $F(s_g) = 0$, $F(s_l) > 0$. Suppose, also, that $p_g = p_l$. We show, below, that Proposition 1 holds for g and l.

If g and l both have partial recovery, or both have full recovery, then the analyses in sub-section 4.2 go through, and Proposition 1 holds.

Suppose, instead, that g has partial recovery, but l has full recovery. Because $s_g < s_l$ and $t_g < t_l$, $c(t_g, s_g) < c(t_l, s_l)$, and so $C(t_g, s_g) < C(t_l, s_l)$, by Assumption (A5). This implies that $E_g < E_l$ and $\mu_g < \mu_l$. On the other hand, let I_m denote the hypothetical post-treatment net income under the pain of s_g and full treatment. Because $t_g < 1$ is the optimal treatment under s_g, $I_m < I_g$. Because both I_m and I_l have full treatment and $s_g < s_l$, the analyses of sub-section 4.2 imply that $I_m > I_l$. As a result, $I_g > I_l$, and so $\beta_g < \beta_l$. This completes the proof.

A key element in our proof above is that diseases g and l have the same private cost function, $c(.)$. This implicit assumption limits the way that treatment technology differs between g and l. There could be scenarios where this implicit assumption might not hold. E.g. suppose that for disease l, surgery, and only surgery, delivers partial mitigation, but continued mitigation above this threshold is very low-cost. This means that $c_l(.) = 0$ if $t_l < \underline{t}$, and $c(.) = \lambda_l t_l + F(s_l)$ if $t_l \geq \underline{t}$, where $\lambda_l < \lambda$. In this case, the cost function of l, $c_l(.)$, differs from the cost function of g, outlined above. Whether Proposition 1 holds or not depends on the specific parameter values, such as $\underline{t}$, λ_l and λ.

4.6 Further Discussion and Comparison with Literature

First, Cutler et al. (1997) and Murray and Acharya (1997) use survey data to measure quality of life, or $v_g(I_g)/u(I)$. These surveys do not necessarily reflect individuals' actual choices. It is also necessary to take a stand on state dependence, in order to translate $v_g(I_g)/u(I)$ to our variables of interest, such as utility share weights and marginal disutility.

Second, in the framework of Hall and Jones (2007), there is no uncertainty about sickness, and healthcare spending reduces mortality risk. As a result, the marginal utility of consumption matters for the optimal healthcare spending. In our framework, however, this variable does not affect treatment and expenditure, because we assume, as in Ma and McGuire (1997), that the average worker takes the sickness rates as given, and seeks treatment after sickness, not before. We make these assumptions because our focus is the distinction among healthy and sick states.

Third, the marginal utility of consumption may also matter for the design of optimal health insurance (e.g. Cutler and Zeckhauser 2000). In our framework, we abstract away from the differences in health insurance across consumers, because Denmark, like many other high-income countries, has universal health care. We allow the private cost of treatment to differ from the social cost, and do not take a stand on the efficiency of the Danish healthcare system.

Fourth, suppose that the provision of treatment is capacity constrained in Denmark. Intuitively, what matters for our framework is how this constraint varies across diseases; i.e. whether assumptions (A5) - (A6) hold for the treatment, t_g, that the average worker chooses under capacity constraints.

Fifth, medical technology also matters for healthcare spending (e.g. Newhouse 1992, Cutler 2005). Proposition 1, under assumption (A6), shows that medical technology fits within our framework unless it has very different effects on c(.) and C(.). Howard et al. (2015) argue that anticancer drugs are expensive partly because of their producers' market power. To see how this affects our approach, suppose that the treatment providers of disease g charge a higher price than those of disease l, and this leads to higher expenditures on g than l. Our approach works in this case if g is more severe than l.

In summary, assumptions (A5)-(A6) clarify the conditions under which expenditure shares relative to frequency provide a useful approximation for marginal disutility. If we know, ex ante, that assumptions (A5)-(A6) do not hold for certain diseases, we can drop them. Their exclusion does not affect the marginal-disutility values of the remaining diseases, because these values are based on *ratios* of expenditure shares.

Table 1 Summary Statistics

	Obs	Mean	Std. Dev.	Below 50 years		Above 50 years		Expenditure share
				Mean	Std. Dev.	Mean	Std. Dev.	
Annual data (1996-2012)								
1. Firm characteristics								
Log sales	3,468,419	13.21	1.91					
Log employment	3,468,419	6.26	1.79					
Log capital labor ratio	3,468,419	19.71	0.97					
Share of high-skilled workers	3,468,419	0.21	0.16					
Log offshoring	3,468,419	17.65	2.74					
2.Worker characteristics								
Experience (years)	3,468,417	18.82	9.80	18.01	9.44	28.94	8.38	
Union membership (dummy)	3,468,406	0.85	0.35	0.85	0.36	0.87	0.34	
Married (dummy)	3,468,419	0.58	0.49	0.56	0.50	0.77	0.42	
Total hours	2,549,684	1529.95	398.84	1526.87	397.11	1570.21	418.70	
Log earnings	3,467,642	12.74	0.42	12.74	0.42	12.72	0.41	
3. Sickness (dummies)								
Anti-depressant (drug)	3,468,419	0.03582	0.1858	0.0357	0.1856	0.0369	0.1885	0.3012%
Heart disease (drug)	3,468,419	0.02439	0.1543	0.0206	0.1419	0.0722	0.2588	0.0005%
Narrow burnout (hosp.)	3,468,419	0.00005	0.0071	0.0001	0.0072	0.00004	0.0062	0.0006%
Broad burnout (hosp.)	3,468,419	0.00006	0.0077	0.0001	0.0078	0.00005	0.0068	0.0111%
Heart attack or stroke (hosp.)	3,468,419	0.00156	0.0394	0.0013	0.0362	0.0046	0.0677	2.0200%
Alcoholism (hosp.)	3,468,419	0.00090	0.0299	0.0009	0.0297	0.0010	0.0321	0.4653%
Metabolism (hosp.)	3,468,419	0.00001	0.0036	0.0000	0.0034	0.0000	0.0052	
4. Quarterly data (2008-2017)								
Log sales (firms)	11,628,136	11.99	2.06					
Log material use (firms)	11,626,041	11.57	2.10					
Log hours (workers)	11,628,136	6.06	0.34					
Log earnings (workers)	11,628,136	11.40	0.53					

Notes: Panels 1-3 have worker-firm-year observations, and Panel 4 has worker-firm-quarter observations. The expenditure shares are based on the health-expenses data in COFOG (Classification of the Functions of Government) by Statistics Denmark (see the Data Appendix for the details).

Table 2 Hours and Earnings

	Annual data		Quarterly data					
	Log hours	Log earnings	Log hours	Log earnings	Log hours	Log earnings	Log hours	Log hours
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log sales	0.016261**	0.008402***	0.0230***	0.0361***	0.0182***	0.0309***	0.0379***	0.0086***
	[2.10]	[6.44]	[4.20]	[6.41]	[4.20]	[5.58]	[15.43]	[3.04]
Experience 5-20 years	0.012691***	0.173958***	0.0241***	0.1115***	0.0242***	0.1116***	0.0149***	0.0327***
	[3.74]	[36.73]	[7.02]	[18.10]	[7.06]	[18.19]	[7.85]	[22.72]
Experience 20+ years	0.016211***	0.171409***	0.0226***	0.1036***	0.0227***	0.1037***	0.0148***	0.0305***
	[4.54]	[41.41]	[7.62]	[20.31]	[7.69]	[20.42]	[7.04]	[19.35]
Union membership	0.000586	0.040290***	0.0428***	0.0676***	0.0429***	0.0676***	0.0544***	0.0323***
	[0.24]	[8.53]	[13.08]	[13.11]	[13.13]	[13.11]	[41.24]	[22.38]
Married	-0.004470**	0.011562***	0.0085***	0.0229***	0.0085***	0.0229***	0.0088***	0.0084***
	[-2.21]	[9.56]	[7.89]	[8.11]	[7.90]	[8.12]	[10.55]	[13.02]
Log employment	-0.012659	0.061591***	0.0422***	0.0596***	0.0379***	0.0549***	0.0438***	0.0266***
	[-1.15]	[10.55]	[5.81]	[5.63]	[4.66]	[4.12]	[8.43]	[3.04]
Log capital labor ratio	-0.001005	-0.001236	-0.0032**	0.0018	-0.0032**	0.0018	-0.0050***	0.0003
	[-0.24]	[-0.76]	[-2.04]	[0.98]	[-1.97]	[0.92]	[-5.25]	[0.16]
Share high-skilled	0.150220*	0.149743***	0.0196	0.1378***	0.0168	0.1348***	-0.0045	0.0528**
	[1.74]	[5.76]	[0.73]	[3.20]	[0.57]	[2.96]	[-0.19]	[2.17]
Log offshoring	-0.000301	0.000467						
	[-0.21]	[0.99]						
Log materials					0.0102**	0.0112	0.0157***	0.0043
					[1.99]	[1.07]	[5.70]	[0.81]
Worker-firm FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes						
Quarter-year FE			Yes	Yes	Yes	Yes	Yes	Yes
Obs. No.	1,827,187	3,467,642	9,561,527	9,561,527	9,559,538	9,559,538	4,778,153	4,781,385
R^2 (within)	0.0029	0.0573	0.0189	0.1212	0.0190	0.1213	0.0298	0.0116
Job Spell No.	466,957	727,879	610,806	610,806	610,788	610,788	336,324	274,464

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1. The regression specification is equation (8). Annual data is 1996-2012, and quarterly data 2008-2017.

Table 3 Correlation between Inputs, Total Measured Hours, Input per Hour, and Sales

VARIABLES	Log Materials	Log hours	Log Materials per hour
1. Full Sample			
log Sales	0.6481***	0.3654***	0.2827***
	(0.0138)	(0.0149)	(0.0125)
Obs. No.	146,065	146,065	146,065
R^2 (within)	0.5236	0.1380	0.0961
2 low proportion of 37-hour workers			
log Sales	0.6772***	0.4001***	0.2772***
	(0.0198)	(0.0240)	(0.0214)
Obs. No.	68,987	68,987	68,987
R^2 (within)	0.5489	0.1635	0.1216
3 high proportion of 37-hour workers			
log Sales	0.6269***	0.3399***	0.2870***
	(0.0186)	(0.0190)	(0.0153)
Obs. No.	77,078	77,078	77,078
R^2 (within)	0.5100	0.1448	0.0973

Notes: Robust standard errors (clustered by firm) in brackets. *** p<0.01, ** p<0.05, * p<0.1. An observation is firm by quarter, and all specifications include firm fixed effects. Total hour by firm is the summation of all employees' work hours.

Table 4 Effects of Firm Sales on Worker Sickness

VARIABLES	(1) Anti-Depressants	(2) Heart-Disease Drugs	(3) Narrow Burnout	(4) Broad Burnout	(5) Stroke/Heart Attack	(6) Liver Diseases	(7) Metabolism
Log sales	0.054*	0.050**	0.392***	0.419***	0.090**	0.128**	-0.315*
	[1.94]	[2.19]	[2.79]	[3.09]	[2.53]	[2.01]	[-1.66]
Experience 5-20 years (x 100)	0.59***	-1.24***	-0.0041*	-0.0036	-0.065***	-0.0162*	0.0021
	[9.66]	[-27.64]	[-1.86]	[-1.60]	[-7.69]	[-1.73]	[1.35]
Experience 20+ years (x 100)	0.55***	-2.11***	-0.0028	-0.0023	-0.079***	-0.0126	0.0016
	[6.14]	[-26.14]	[-0.89]	[-0.71]	[-5.32]	[-1.04]	[0.83]
Unioned (x 100)	0.14**	0.32***	0.0004	0.0005	0.0128	-0.0001	0.0017
	[2.28]	[6.97]	[0.10]	[0.13]	[1.19]	[-0.01]	[1.12]
Married (x 100)	-0.0568	-0.58***	0.0059***	0.0066***	-0.026***	-0.024***	0.0005
	[-0.98]	[-14.72]	[3.13]	[3.39]	[-2.99]	[-3.12]	[0.48]
Log employment (x 100)	0.0687	-0.17**	-0.0017	-0.0024	-0.0092	0.0122	-0.0015
	[0.66]	[-2.21]	[-0.81]	[-1.14]	[-0.75]	[1.45]	[-1.46]
Log capital labor ratio (x 100)	-0.0164	-0.0515	-0.0018	-0.0020	-0.0011	-0.0057	0.0001
	[-0.41]	[-1.56]	[-1.42]	[-1.53]	[-0.17]	[-1.24]	[0.17]
Share high-skilled (x 100)	1.06**	-1.17**	0.0048	-0.0010	-0.0830	0.0059	0.0051
	[2.14]	[-2.50]	[0.35]	[-0.07]	[-1.02]	[0.12]	[1.05]
Log offshoring (x 100)	0.0030	-0.0052	0.0008	0.0008	0.0007	-0.0018	0.0000
	[0.30]	[-0.54]	[1.51]	[1.48]	[0.26]	[-0.98]	[0.16]
Obs. No.	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090	3,468,389
R^2 (within)	0.0084	0.0151	0.0000	0.0000	0.0003	0.0000	0.0000
Job Spell No.	713,133	721,511	727,916	727,908	727,404	727,088	727,939

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1. All specifications use annual data, and include job-spell fixed effects and year fixed effects. For each sickness condition, we drop the individuals who are initially sick with this condition, and so our sample size varies slightly across columns. The coefficients of log sales have been normalized by mean sickness rates, and so are elasticity estimates. Columns (1) and (2) are prescription drugs, and the other columns are hospitalizations.

Table 5 Differential Effects of Firm Sales: Old Age

VARIABLES	(1) Anti-Depressants	(2) Heart-Disease Drugs	(3) Narrow Burnout	(4) Broad Burnout	(5) Stroke/Heart Attack	(6) Liver Diseases
Log sales	0.00179*	0.00038	0.000020***	0.000024***	0.000088**	0.000094*
	[1.86]	[1.64]	[2.67]	[3.01]	[2.16]	[1.91]
Log sales x Age 50	0.00142***	0.00874***	-0.000001	0.000009	0.000519	0.000212*
	[2.82]	[2.70]	[-0.14]	[0.68]	[1.20]	[1.93]
Experience 5-20 years (x 100)	0.595***	-1.2336***	-0.0041*	-0.0036	-0.0643***	-0.0160*
	[13.55]	[-45.00]	[-1.83]	[-1.57]	[-9.44]	[-1.76]
Experience 20+ years (x 100)	0.557***	-2.0866***	-0.0028	-0.0023	-0.0777***	-0.0121
	[9.29]	[-44.12]	[-0.89]	[-0.69]	[-6.13]	[-1.04]
Unioned (x 100)	0.143**	0.318***	0.0004	0.0005	0.0128	-0.0001
	[3.21]	[9.69]	[0.14]	[0.18]	[1.23]	[-0.01]
Married (x 100)	-0.0562	-0.575***	0.0059***	0.0066***	-0.0262***	-0.0239***
	[-1.47]	[-22.75]	[3.46]	[3.69]	[-3.13]	[-3.49]
Log employment (x 100)	0.0745	-0.137*	-0.0017	-0.0024	-0.0070	0.0131
	[1.31]	[-3.46]	[-0.85]	[-1.17]	[-0.69]	[1.85]
Log capital labor ratio (x 100)	-0.0163	-0.0508	-0.0018	-0.0020	-0.0011	-0.0057
	[-0.70]	[-2.74]	[-1.72]	[-1.85]	[-0.20]	[-1.50]
Share high-skilled (x 100)	1.061**	-1.193**	0.0048	-0.0010	-0.0843	0.0053
	[3.73]	[-4.47]	[0.40]	[-0.08]	[-1.24]	[0.13]
Log offshoring (x 100)	0.0031	-0.0046	0.0008	0.0008	0.0007	-0.0018
	[0.33]	[-0.58]	[1.72]	[1.70]	[0.33]	[-1.14]
Obs. No.	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090
R^2 (within)	0.0084	0.0152	0.0000	0.0000	0.0003	0.0000
Job Spell No.	713,133	721,511	727,916	727,908	727,404	727,088

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1 All specifications use annual data, and include job-spell fixed effects and year fixed effects. For each sickness condition, we drop the individuals who are initially sick with this condition, and so our sample size varies slightly across columns. Columns (1) and (2) are prescription drugs, and the other columns are hospitalizations.

Table 6 Differential Effects of Firm Sales: Long Initial Hours

VARIABLES	(1) Anti-Depressants	(2) Heart-Disease Drugs	(3) Narrow Burnout	(4) Broad Burnout	(5) Stroke/Heart Attack	(6) Liver Diseases
Log sales	0.001363	0.000340	0.000015	0.000017	0.000181**	0.000066
	[1.52]	[1.01]	[1.07]	[1.37]	[2.08]	[0.65]
Log sales x Long Hours	0.003566***	0.002336***	0.000010	0.000014	0.000085	0.000202**
	[4.62]	[6.19]	[0.58]	[1.02]	[0.84]	[2.02]
Experience 5-20 years (x 100)	0.3685***	-1.3621***	-0.0029	-0.0032	-0.0405***	-0.0205
	[4.76]	[-28.72]	[-0.95]	[-1.03]	[-3.27]	[-1.14]
Experience 20+ years (x 100)	0.4204***	-2.4184***	-0.0030	-0.0029	-0.0630***	-0.0230
	[4.45]	[-34.17]	[-0.66]	[-0.63]	[-3.27]	[-1.14]
Unioned (x 100)	0.1401	0.4397***	-0.0009	-0.0006	0.0165	-0.0066
	[1.87]	[7.33]	[-0.16]	[-0.10]	[0.88]	[-0.70]
Married (x 100)	0.0517	-0.5692***	0.0047*	0.0056**	-0.0245*	-0.0230**
	[0.89]	[-14.91]	[1.76]	[1.99]	[-1.94]	[-2.30]
Log employment (x 100)	0.0271	-0.2182**	-0.0015	-0.0012	-0.0121	0.0159
	[0.37]	[-3.72]	[-0.65]	[-0.49]	[-0.83]	[1.54]
Log capital labor ratio (x 100)	0.0095	-0.0752*	-0.0006	-0.0007	-0.0130	0.0039
	[0.31]	[-2.68]	[-0.39]	[-0.47]	[-1.69]	[0.76]
Share high-skilled (x 100)	0.9237	-1.6008***	-0.0022	-0.0059	-0.1551	-0.0605
	[2.31]	[-3.83]	[-0.18]	[-0.44]	[-1.52]	[-1.02]
Log offshoring (x 100)	0.0166	-0.0145	0.0001	-0.0000	0.0004	-0.0026
	[1.35]	[-1.25]	[0.09]	[-0.03]	[0.11]	[-1.07]
Obs. No.	1,576,088	1,588,574	1,600,260	1,600,259	1,599,341	1,599,148
R^2 (within)	0.0076	0.0165	0.0000	0.0000	0.0002	0.0001
Job Spell No.	380,274	383,531	386,854	386,853	386,582	386,512

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1. All specifications use annual data, and include job-spell fixed effects and year fixed effects. For each sickness condition, we drop the individuals who are initially sick with this condition, and so our sample size varies slightly across columns. Columns (1) and (2) are prescription drugs, and the other columns are hospitalizations.

Table 7 Differential Effects of Firm Sales: Job Strain

VARIABLES	(1) Anti-Depressants	(2) Heart-Disease Drugs	(3) Narrow Burnout	(4) Broad Burnout	(5) Stroke/Heart Attack	(6) Liver Diseases
Log sales	0.001934*	0.001147**	0.000019***	0.000025***	0.000139**	0.000116**
	[1.94]	[2.08]	[2.72]	[3.04]	[2.52]	[2.01]
Log sales x Job Strained	-0.000010	0.000251***	0.000002***	0.000002**	0.000002	-0.000003
	[-0.57]	[15.91]	[2.59]	[2.30]	[0.56]	[-1.08]
Experience 5-20 years (x 100)	0.5934***	-1.2505***	-0.0041*	-0.0037	-0.0650***	-0.0161*
	[13.46]	[-45.19]	[-1.85]	[-1.59]	[-9.51]	[-1.78]
Experience 20+ years (x 100)	0.5537***	-2.1230***	-0.0030	-0.0024	-0.0791***	-0.0124
	[9.20]	[-45.12]	[-0.93]	[-0.73]	[-6.22]	[-1.07]
Unioned (x 100)	0.1428**	0.3154***	0.0004	0.0005	0.0128	-0.0000
	[3.22]	[9.60]	[0.13]	[0.17]	[1.23]	[-0.01]
Married (x 100)	-0.0568	-0.5813***	0.0059***	0.0066***	-0.0264***	-0.0240***
	[-1.48]	[-23.15]	[3.46]	[3.69]	[-3.16]	[-3.50]
Log employment (x 100)	0.0686	-0.1679**	-0.0016	-0.0024	-0.0091	0.0122
	[1.19]	[-4.08]	[-0.83]	[-1.17]	[-0.87]	[1.70]
Log capital labor ratio (x 100)	-0.0164	-0.0515	-0.0018	-0.0020	-0.0011	-0.0057
	[-0.70]	[-2.79]	[-1.72]	[-1.85]	[-0.21]	[-1.51]
Share high-skilled (x 100)	1.0637***	-1.1541***	0.0049	-0.0009	-0.0828	0.0056
	[3.73]	[-4.33]	[0.41]	[-0.07]	[-1.22]	[0.13]
Log offshoring (x 100)	0.0030	-1.2336***	0.0008	0.0008	0.0007	-0.0018
	[0.32]	[-0.62]	[1.73]	[1.70]	[0.31]	[-1.15]
Obs. No.	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090
R^2 (within)	0.0084	0.0153	0.0000	0.0000	0.0003	0.0000
Job Spell No.	713,133	721,511	727,916	727,908	727,404	727,088

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1. All specifications use annual data, and include job-spell fixed effects and year fixed effects. For each sickness condition, we drop the individuals who are initially sick with this condition, and so our sample size varies slightly across columns. Columns (1) and (2) are prescription drugs, and the other columns are hospitalizations.

Table 8 Robustness Exercises

	log(Hours) (1)	Anti-Depressants (2)	Heart-Disease Drugs (3)	Narrow Burnout (4)	Broad Burnout (5)	Stroke/Heart Attack (6)	Liver Diseases (7)	Metabolism (8)
1. Benchmark (elasticity)								
log(sales)	0.0163**	0.054*	0.050**	0.392***	0.419***	0.090**	0.128**	-0.315*
	[2.10]	[1.94]	[2.19]	[2.79]	[3.09]	[2.53]	[2.01]	[-1.66]
Obs. No.	1,827,187	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090	3,468,389
2. Drop Managers (elasticity)								
log(sales)	0.0165**	0.053*	0.049**	0.431***	0.436**	0.094***	0.134**	-0.315*
	[2.12]	[1.89]	[2.14]	[2.98]	[3.14]	[2.71]	[2.03]	[-1.66]
Obs. No.	1,772,735	3,317,353	3,350,637	3,376,158	3,376,125	3,374,188	3,373,071	3,376,282
3. Organizational Controls								
log(sales) x 100	3.6	0.425***	0.087	0.012***	0.013***	0.044	-0.018	0.0004
	[1.01]	[3.29]	[0.76]	[2.63]	[2.60]	[1.53]	[-0.90]	[0.17]
log(# layers/sales) x 100	1.03	0.004	0.041	0.0063*	0.006	-0.0016	-0.0186	0.0007
	[0.35]	[0.04]	[0.47]	[1.72]	[1.46]	[-0.07]	[-1.11]	[0.44]
Obs. No.	1,822,582	3,384,871	3,418,939	3,444,905	3,444,872	3,442,910	3,441,749	3,445,028
4. Predicted Firm Sales (elasticity)								
log(predicted sales)	0.0262***	0.083**	0.057*	0.608***	0.670**	0.092**	0.205***	0.079
	[3.32]	[2.54]	[1.76]	[2.58]	[2.35]	[2.10]	[2.91]	[0.32]
Obs. No.	1,701,466	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090	3,397,420
5. Men Only (elasticity)								
log(sales)	0.0167**	0.044**	0.045**	0.607**	0.657**	0.071*	0.090	-0.630**
	[2.42]	[2.08]	[2.06]	[2.07]	[2.45]	[1.94]	[1.48]	[-2.17]
Obs. No.	1,229,150	2,309,206	2,322,012	2,342,638	2,342,623	2,340,885	2,340,006	2,342,748
6. Women Only (elasticity)								
log(sales)	0.0151	0.066*	0.090**	0.109	0.105	0.231***	0.323***	0.236
	[1.29]	[1.94]	[2.34]	[0.48]	[0.64]	[3.36]	[3.10]	[0.78]
Obs. No.	597,982	1,098,389	1,119,921	1,125,426	1,125,408	1,125,176	1,124,885	1,125,442

Table 8 Robustness Exercises, Continued

	log(Hours) (1)	Anti-Depressants (2)	Heart-Disease Drugs (3)	Narrow Burnout (4)	Broad Burnout (5)	Stroke/Heart Attack (6)	Liver Diseases (7)	Metabolism (8)
1. Benchmark (elasticity)								
log(sales)	0.0163**	0.054*	0.050**	0.392***	0.419***	0.090**	0.128**	-0.315*
	[2.10]	[1.94]	[2.19]	[2.79]	[3.09]	[2.53]	[2.01]	[-1.66]
Obs. No.	1,827,187	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090	3,468,389
7. Local Labor Market Tightness								
log(sales) x 100	1.74**	0.19**	0.13**	0.002***	0.003***	0.011**	0.012**	-0.0007**
	[2.29]	[1.97]	[2.42]	[2.90]	[3.47]	[2.13]	[2.25]	[-2.09]
log(sales) x UI_rate x 100	-2.22	0.09	-0.23*	-0.008*	-0.0012**	0.06*	-0.012	0.0043
	[-1.00]	[0.52]	[-1.64]	[-1.54]	[-2.15]	[1.78]	[-0.49]	[1.58]
Obs. No.	1,827,132	3,392,447	3,426,588	3,452,710	3,452,677	3,450,709	3,449,595	3,452,837
8. Worker Tenure								
log(sales) x 100	1.37*	0.167*	0.127**	0.0019***	0.0025***	0.0131**	0.0104*	-0.0005*
	[1.78]	[1.66]	[2.30]	[2.75]	[3.03]	[2.35]	[1.76]	[-1.78]
log(sales) x high_tenure x 100	0.26***	0.031***	-0.0068***	0.0000	0.0001	0.0010**	0.0014***	0.0000
	[8.52]	[15.28]	[-3.89]	[0.53]	[0.54]	[2.49]	[3.83]	[0.97]
Obs. No.	1,827,192	3,407,793	3,442,131	3,468,263	3,468,230	3,466,260	3,465,090	3,468,389

Notes: t-statistics in brackets, with clustering by firm in all columns. *** p<0.01, ** p<0.05, * p<0.1. All specifications use annual data. Column (1) uses the same data and includes the same set of controls as column (1) of Table 2. The other columns use the same data and include the same set of controls as Table 4. Panel 3 includes the following additional control variables: the logs of the numbers of imported and exported products, and the numbers of import source countries and export destination countries, normalized by firm sales. Columns (2) and (3) are prescription drugs, and columns (4)-(8) are hospitalizations. Panels 3, 7 and 8 report 100 times the coefficient estimates.

Table 9 Marginal Disutility (With Treatment) of Sickness Variables

	Frequency	Expenditure Share	Marginal Dis-utility (DKK 000)	95% Confidence Interval for 3 (DKK 000)	ΔSick_Rate /ΔSales x 100
Sickness	1	2	3	4	5
Anti-depressants	0.0358	0.30%	22.55	[1.94, 43.17]	0.1931
Broad burnouts	0.00006	0.011%	499.51	[42.97, 956.04]	0.0025
Stroke (hospitalization)	0.0016	2.02%	3476.66	[299.10, 6654.22]	0.0140
Heart diseases (drugs)	0.0244	0.32%	35.60	[3.06, 68.14]	0.1213
Liver Diseases (Hosp.)	0.0009	0.47%	1392.86	[119.83, 2665.88]	0.0115
Injury	0.0039	2.26%	1556.84	[133.94, 2979.75]	N.A.

Notes: Columns 1 and 2 are, respectively, the mean sickness rates and expenditure shares in Panel 3 of Table 1. Column 5 is our elasticity estimates in Table 4 multiplied by the sample means in column 1 multiplied by 100. The value of injury in column 3 is the estimated VSI in our data, and its 95th confidence interval is reported in the last row of column 4. The rest of the marginal-disutility values and their confidence intervals in columns 3 and 4 are computed using equation (10) plus the estimated VSI and its confidence interval.

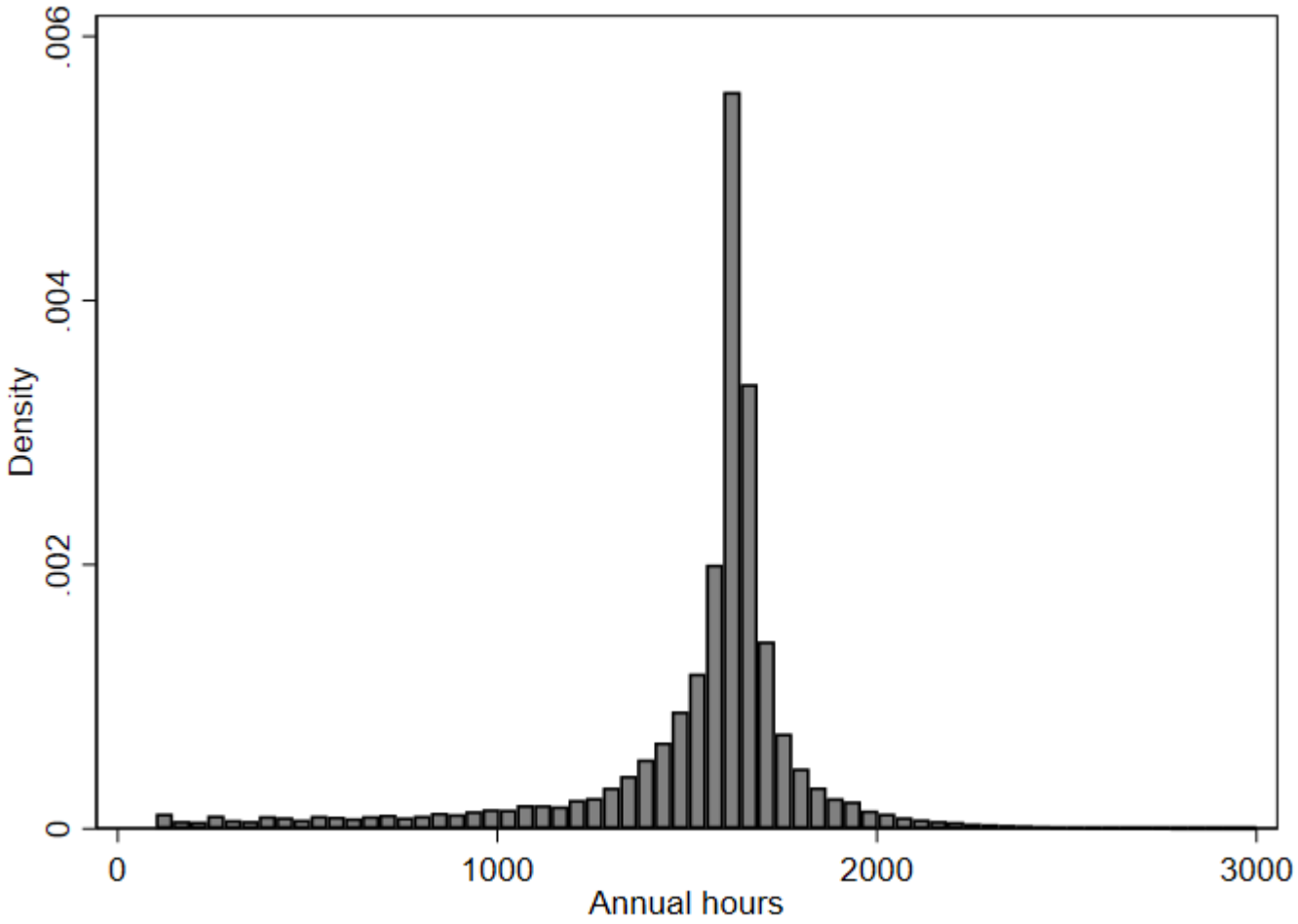

Figure 1 Distribution of Annual Hours

Notes: The results are based on annual data from the "LON" register, which holds information reported by firms about annual hours worked for each employee. The hours reported are actual hours worked defined as contractual (paid) hours including overtime work but excluding time absent from work (e.g. due to sickness, child care leave etc.).

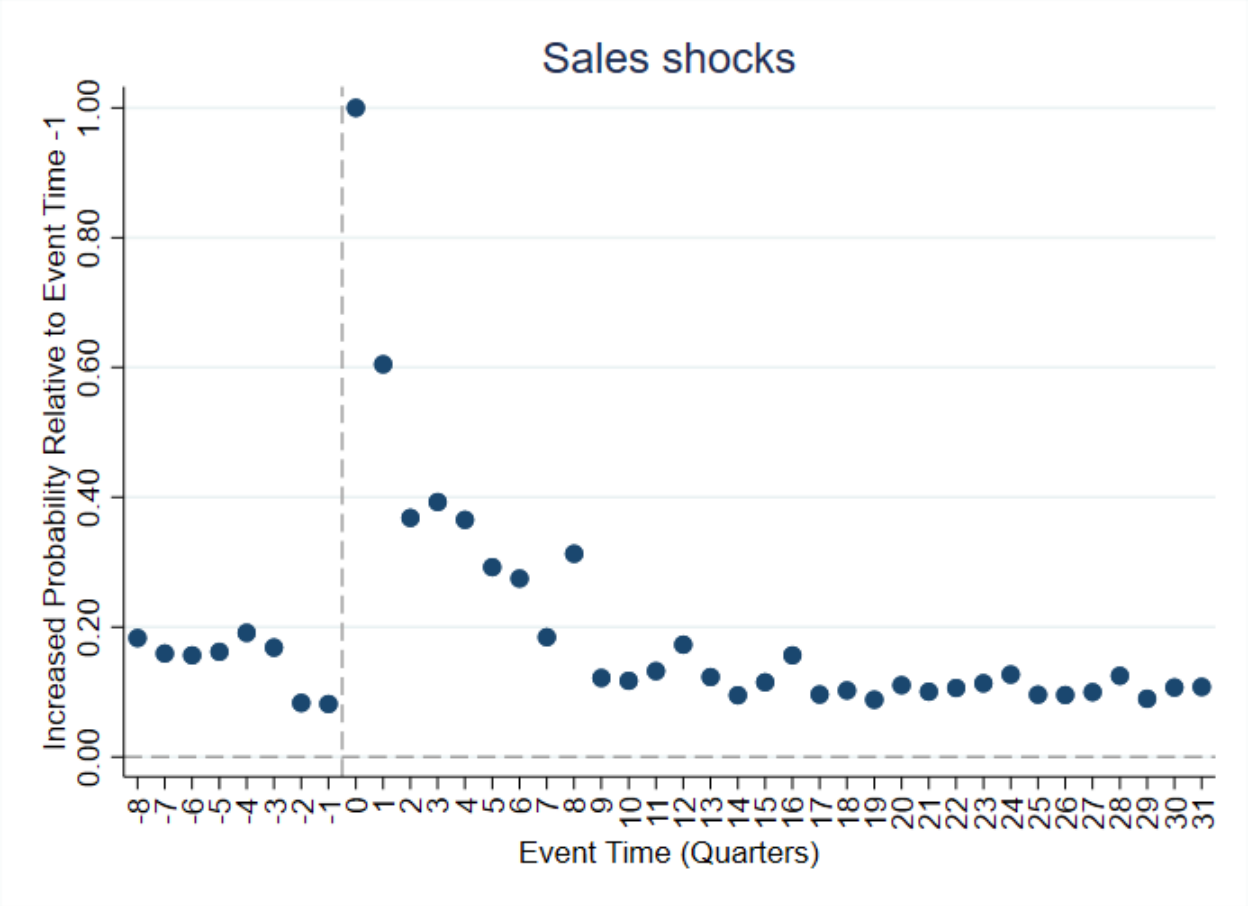

Figure 2 Workers' Exposure to Large Sales Increases

Notes: The results are based on quarterly data. Event quarter 0 is the quarter after 2010Q1 when the first large-sales increase happens. The other event quarters are relative to event quarter 0.

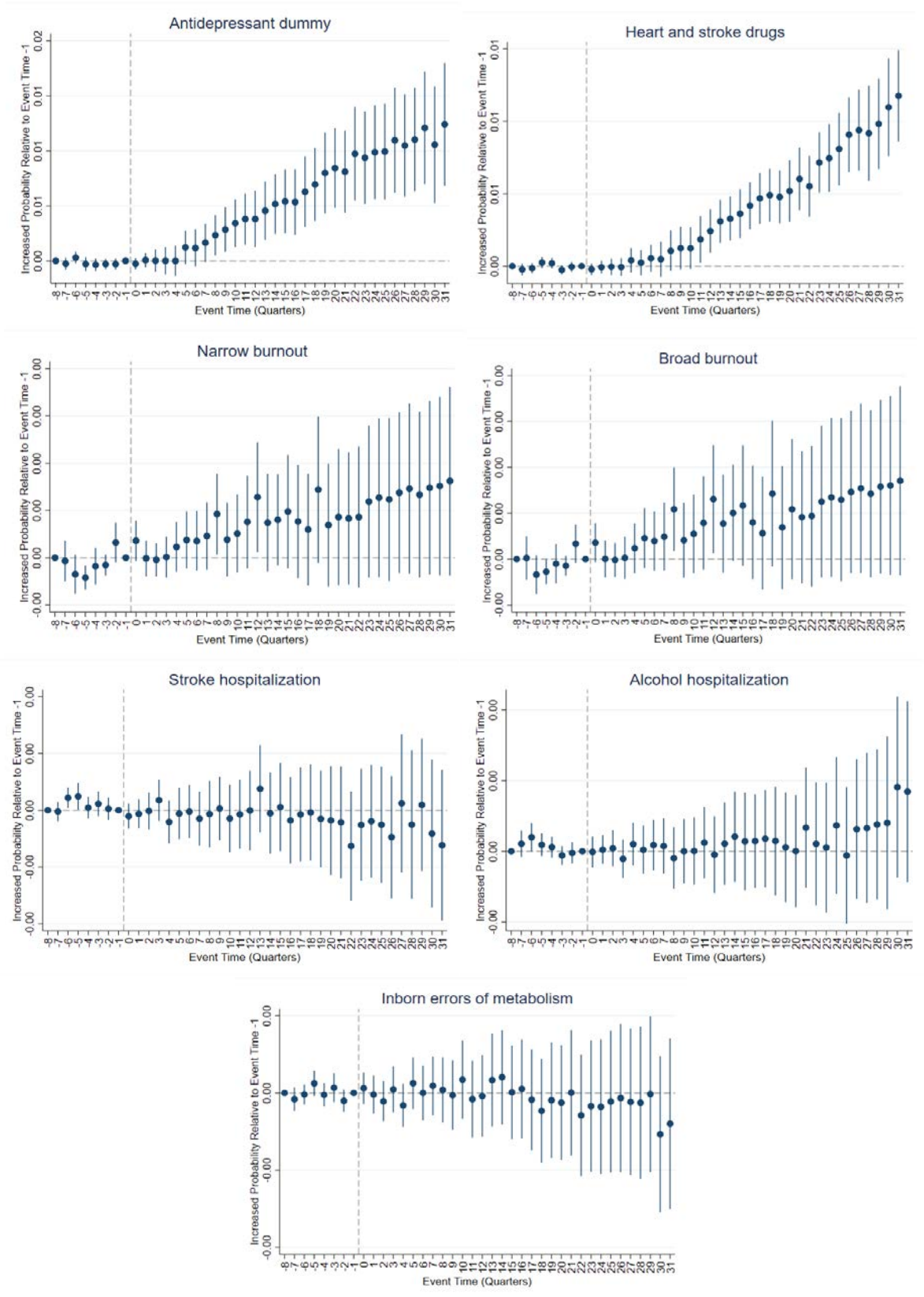

Figure 3 Effects of Large Sales Increases on Workers' Sickness

Notes: Event quarter 0 is the quarter after 2010Q1 when the first large-sales increase happens. The other event quarters are relative to event quarter 0. All specifications use quarterly data, and include worker fixed effects and quarter-year fixed effects. The solid dots show the coefficient estimates of the effects of large sales increases, and the solid lines show their 95^{th} confidence intervals.

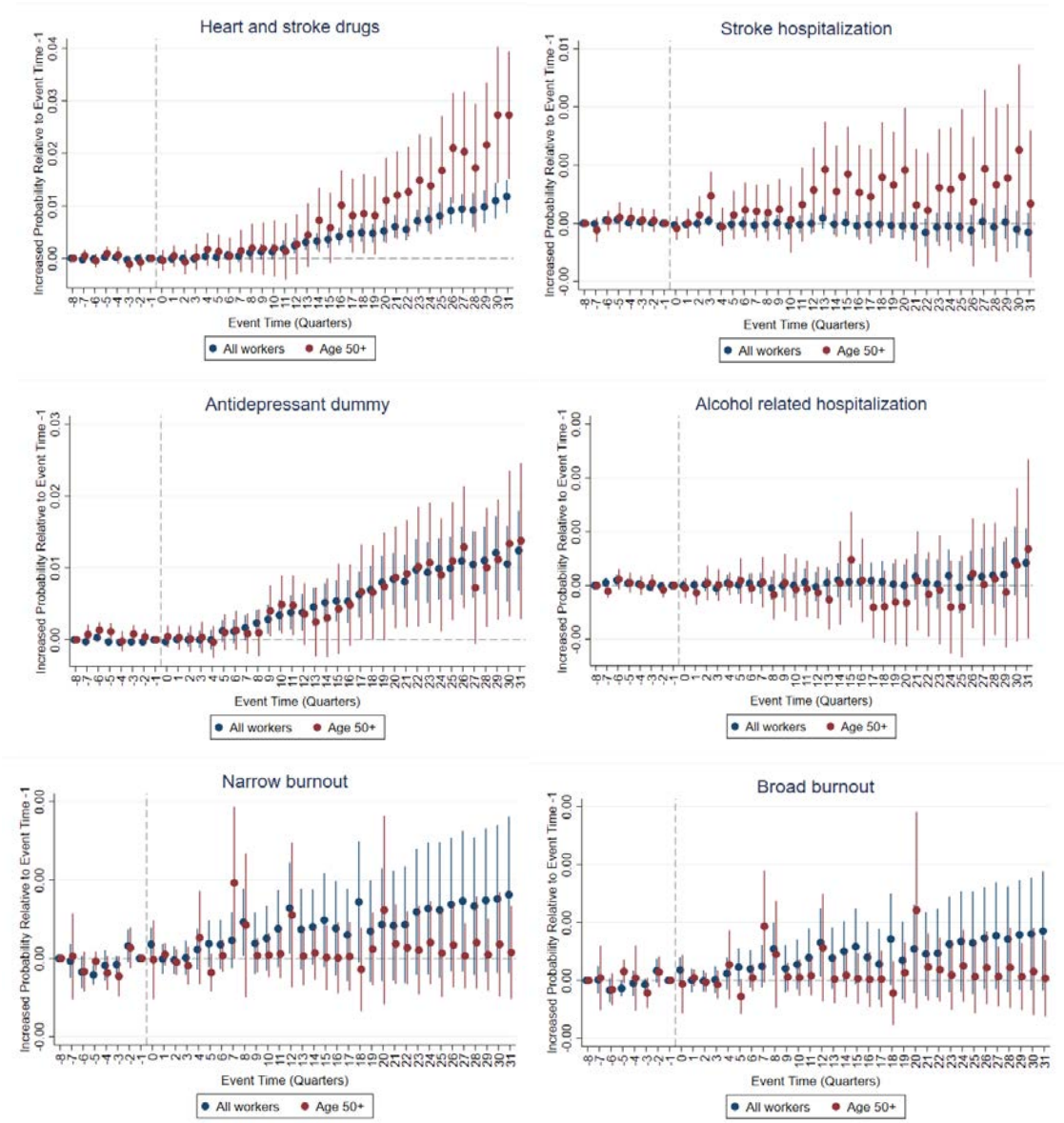

Figure 4 Effects of Large Sales Increases on Older Workers' Sickness

Notes: Event quarter 0 is the quarter after 2010Q1 when the first large-sales increase happens. The other event quarters are relative to event quarter 0. All specifications use quarterly data, and include worker fixed effects and quarter-year fixed effects. The red dots and lines show the effects of large sales increases for the older workers and their 95th confidence intervals, and the blue ones show the effects for all workers, from Figure 3.

1. More Details for Data Construction (Section 2)

1.1 Sample Cuts

Starting with the worker-year sample of firms and their workers in the Prodcom sales data/registry (manufacturing firms including mining and quarrying with at least 10 employees), we first drop observations with zero imports or exports. This leaves us with 94.8% the original sample. Next we drop observations with missing information about key firm variables (sales, capital-labor ratio and the share of high-skilled workers), reducing the coverage rate further to 93.7%. We then select 20-60 year old workers (88.3%) and require they work fulltime (84.4%). Finally we drop all observations where the employment relationship lasts a single year, which leaves us with a sample covering 78.2% of the original sample.

1.2 Administrative Data on Health

Prescription drugs data are drawn from the "Register of Medicinal Product Statistics" maintained by Statens Serum Institut (SSI). These data include each individual's drug classification following the 4-digit Anatomical Therapeutic Chemical (ATC) codes, copay (out-of-pocket expenses by patients) and total prescription drug cost for the Danish government. For all Danish full time workers aged 20-60 during 1995-2009, the median out-of-pocket expense for prescription-drug copay is 404 DKK while the median labor income is 296,379 DKK (1 DKK is about 0.18 USD in this time period). The ATC codes of our prescription-drug variables are as follows. Anti-depressants = N06AA, AF, AG and AX. Drugs for heart diseases = B01 and C01.

The data on hospitalization includes the diagnosis, by the International Classification of diseases (ICD10), and the total cost of in-patient care for the Danish government. The ICD 10 codes for our hospitalization variables are as follows. Narrow burnout = Z730, 731, 732, 733, Z563 and Z566. Broad burnout = narrow burnout plus Z564, 565, 568 and 569. Heart attacks or strokes = I21, 61 and 63. Alcoholism = F100, 102, 104, 105, I85, K70, 860, T500 and 510. Inborn errors of metabolism = D590, 591, 869, 835, I730, 789, K224, 509, 743, 744, 745, 754, 830, 900, L400, 401, 402, 405, 408, M009, 069, 080, 109, 119, 150, 160, 161, 179, 190, 199, 255, 321, 339, 340, 341, 342,, 348, 349, 350, 353, 359, 459, 478, 609, 791, 796, R760, 761, 768, 769.

1.3 Healthcare Expenses

Table A1 reports the share of healthcare expenses in GDP for Denmark by year, for the period 2010-2015. This share is stable over time. Total health care expenditure includes all private expenditure related to doctors and medicinal products (drugs plus medical equipment, such as hearing aid). It also includes all public expenditure, the category health in COFOG (Classification of the Functions of Government) by Statistics Denmark, which includes doctors, medicinal products, hospital service and R&D. Total health care expenditure excludes elderly care and income transfers, such as cash benefits for sick leave.

Table A2 breaks the healthcare expenses into the following categories for 2010. Hospital services include regular hospitals, plus costs to birthing clinics and rehabilitation centers. Within non-hospital services, medical products and equipment include all products used in the interest of health not related to hospitals; e.g. prescription drugs, experimental medicine, prosthesis equipment and glasses. Paramedic care includes all non-doctoral care that is not related to hospitals. Other public health care is mainly administrative costs, R&D and preventive services (such as vaccine).

We obtain the Diagnosis-Related Group (DRG) expenses for our hospitalization variables from the hospitalization registry. The DRG expenses have narrower coverage than the category of hospital services in Table A2. As a result, we compute the shares of our hospitalization variables within the total DRG expense, and assume that they are equal to the shares within the category of hospital services. For example, since heart attacks and strokes have a share of 3.23% within the total DRG expense, their total expense is 113.15 x 3.23% = 3.65 billion DKK.

1.4 Employment Shares for the Work-Hours Sub-sample, Annual Data

Table A3 tabulates the fractions of 1-digit occupations in employment for our main sample in the annual data, as well as for the work-hour subsample. The employment shares are similar.

2. More Details for Main Results (Sections 5 - 6)

2.1 Age vs. Experience as Control

Table A4 shows the results for anti-depressants and heart-disease drugs when we replace the control variables of experience with age. The coefficient estimates for log sales are very similar to Table 4.

2.2 Notes for Americans' Changing Lives (ACL)

ACL consists of five waves: 1986, 1989, 1994, 2002 and 2011. We organize our analyses in two steps. In Step 1, we replicate the main findings of the medical literature (discussed in sub-section 6.3 in the text); i.e. that job strain in the base year is a risk factor for future strokes. In Step 2, we map the ACL occupation and industry codes into those used in our register data, and correlate the job-strain dummy with occupation and industry dummies.

Step 1

We closely follow the medical literature in constructing our variables, specifying our regression, and conditioning our sample.

For our dependent variable, *FutureStroke*, we take the following survey questions about strokes from waves 3 through 5: did you have a stroke in the past 12 months; did you take medication or treatment for stroke in the past 12 months; have you been told by a doctor that you had a stroke. *FutureStroke* equals 1 if the answer to any of these questions is yes.

For our main explanatory variable, *JobStrain*, we take the following two groups of survey questions from wave 1. The work-demand questions include: does your work require working fast; do you have enough time to work; is your work free from conflicting demand. Meanwhile, the work-control questions include: are you bored with your work; do you decide how to work; do you have a lot to say about work. The answers to these survey questions are coded from 1 (strongly disagree) to 5 (strongly agree). *JobStrain* equals 1 if the answer to the enough-time-to-work question is strictly less than the median (which indicates high work demand), and the answer to the bored-with-work question is strictly larger than the median (which indicates low work control). We discuss alternative measures of *JobStrain* below.

For our control variables, we take the following demographics information from wave 1: age, sex and marital status. We estimate the following logit regression

$$FutureStroke_i = \alpha + \beta JobStrain_i + \gamma Demographics_i + \varepsilon_i, \quad \text{(D1)}$$

where *i* subscripts individuals. We drop from our sample the individuals who suffered a stroke in wave 1, and those whose health impeded their daily activities in wave 1.

We report our results in Table A5. Column (1) shows that the coefficient estimate for *JobStrain* is positive and significant, and this result replicates the main findings of the medical literature. In column (2), we have *JobStrain* = 1 if the answer to the enough-time-to-work question is less than or equal to the median, and the answer to the bored-with-work question is larger than or equal to the median. Our result is qualitatively similar to column (1), but not statistically significant. We also experimented with other measures of *JobStrain*, such as using the average of all three work-demand questions to measure high demand, and using the average of all three work-control questions to measure low control. These results (available upon request) are similar to column (2); in particular, the coefficient estimates of the alternative measures of *JobStrain* lack statistical significance.

Step 2

We take the individuals' occupation and industry affiliations from wave 1. The occupation affiliation follows the 1970 U.S. Census codes. We map them into 2000 U.S. census codes (using IPUMS' integrated crosswalk), and then into ISCO 88 codes (using the mappings from the U.S. National Crosswalk Center). Meanwhile, the industry affiliation in ACL wave 1 follows the 1970 U.S. Census codes. We map them into 1990 U.S. Census codes (using IPUMS' integrated crosswalk), and then into 2002 NAICS codes (using U.S. Census Bureau's crosswalk), and finally into NACE 1.1 codes (using U.S. Census Bureau's crosswalk).

We then correlate *JobStrain* with age-by-gender and gender-by-occupation-by-industry dummies for the manufacturing sub-sample. This regression has $R^2 = 0.83$, and the full list of coefficient estimates are available upon request. These estimates allow us to generate predicted values of job strain. We first use the 75^{th} percentile of the predicted values to construct *JobStrain* for the manufacturing sub-sample, and estimate the logit regression (D1). Column (1) of Table A6 shows that the coefficient estimate of *JobStrain* is positive and significant,

suggesting that the predicted values of job strain have explanatory power for future risks of heart diseases and strokes. When we use the 90th percentile as the cut-off for *JobStrain* instead, we obtain a positive, but insignificant, coefficient estimate (column (2) of Table A6).

3. Notes for Section 7

First Stage IV Results We present our first-stage results in Table A7. Our instrument is $\log(I_{jt}) = \log(\sum_k s_{jk} Q_{kt})$, where Q_{kt} is the aggregate output of product k in year t in Denmark, and s_{jk} is product k's share in firm j's output in the pre-sample year of 1994. We include the same set of fixed effects and the same set of worker- and firm-characteristics as controls as in Table 4, and cluster our standard errors by firm. The coefficient estimate of our instrument is 0.725, and the F-statistic for our instrument is 33.52.

Additional Summary Statistics: Table A8 shows the mean values of our sickness variables by gender. We use these mean values to calculate the elasticities in panels 5 and 6 of Table 8.

Commuting Zones: Commuting zones are based on geographically connected municipalities. 275 municipalities in Denmark are merged into 51 commuting zones such that the internal migration rate is 50% higher than the external migration rate. The commuting zone unemployment rate has substantial variation across workers and over time ranging from 1.4% to 16.8% with a mean of 5.3%.

4. Notes for Section 8

Injury Data: In order to estimate VSI, we draw on the following data for severe work injury in Denmark. When a worker is injured on the job, he/she may file a petition for compensation with the National Board of Industrial Injuries (NBII). If the job injuries are severe enough to cause permanent damages to the workers' earning and working abilities, then the workers are also eligible for a one-time, lump-sum monetary compensation. We observe all the petitions filed during 1995-2009, and the final decision by NBII for each petition. To measure injury we consider whether an individual receives positive monetary compensation from NBII. The mean injury rate is 3.9 per thousand in our sample, lower than in the U.S. data, probably because we only include severe injuries while the U.S. data includes all injuries. In addition, most workers stay employed with the same firm after injury in our data.

One potential concern with our injury dummy is that the standard used by NBII to award compensation may endogenously respond to economic fluctuations (e.g. tougher standards during recessions). This is not the case in our data. During 2007-2009, Denmark's Great Depression, NBII accepted around 51% of all petitions, while during the pre-recession years of 2004-2006, NBII accepted about 48% of all petitions.

Estimation of VSI: To carry out the estimation, we examine all full-time Danish workers in the private sector aged 18-65 in 2006. We follow the literature (e.g. Viscusi and Aldy 2003) and run a Mincer regression, augmented by the occupational injury rate. Our dependent variable is the log of annual wage. Our controls include age, experience, experience square, tenure, dummies for marriage, kids, white-collar occupations, vocational education, college education, and native-born Danish. We cluster our standard errors by occupation.

We report the results in Table A9. Our estimate for the log-wage-injury gradient is 5.24, with the 95% confidence interval [0.45, 10.03]. This is consistent with the literature, given that our sample mean is 0.0039 (e.g. Hersch 1998 obtains an estimate of 1.2~1.6 using U.S. data of all injuries, where the sample mean is 0.03, and Martinello and Meng 1992 obtain 3.2~4.1 using Canadian data of severe injuries, where the sample mean is 0.023). Because the average wage in this sample is 297,164 DKK, our estimate for the marginal disutility of injury is DKK 1.56 million (= 297,164 x 5.24), with the 95% confidence intervals of [0.134, 2.978] million DKK.

Expenditure Share for Injury: In Denmark, injury-related expenses consist of medical expenses, for treatment, and cash benefits, such as the monetary compensation we discussed earlier in this section. For our quantification in section 8, we need medical expenses, not cash benefits. However, we observe cash benefits through our NBII injury data, but do not observe medical expenses. On the other hand, in the U.S., the medical expenses for work injury are typically fully covered through insurance (e.g. Powell and Seabury 2018), like in Denmark. The categories of injury-related expenses in the U.S. are also well studied, with medical expenses

accounting for roughly 50% of all expenses (medical expenses plus cash benefits) (e.g. Baldwin and McLaren 2016). Assuming that the Danish share of medical expenses is the same as in the U.S. data, we obtain that, for 2010, total medical expenses for injury are equal to the total cash benefits of $4.1 billion DKK. The expenditure share for injury, therefore, is 2.26% (= 4.1/181.2).

References:

Hersch, Joni, 1998. “Compensating Differentials for Gender-Specific Job Injury Risks”, American Economic Review June 1998, 88(3), pp. 598-607.

Martinello, F. and R. Meng, 1992. “Workplace Risks and the Value of Hazard Avoidance”, Canadian Journal of Economics, 25(2), 333-345.

McLaren, Christopher F., and Marjorie L. Baldwin. "Workers’ Compensation: Benefits, Coverage, and Costs,(2015 data)." National Academy of Social Insurance: Washington, DC (2016).

Online Data Appendix, NOT for Publication

Table A1 Total Danish health care expenditure as percentage of GDP

2010	2011	2012	2013	2014	2015
10.00%	9.91%	10.11%	9.94%	10.00%	9.97%

Notes: Our sources are Statistics Denmark, http://medstat.dk/, Forsikring & Pension, and Arbejdsmarkedets Erhvervssikring.

Table A2 Danish Healthcare spending by category 2010 (billion DKK)

Hospital Expenses	**113.15**
Non-Hospital Expenses	**56.37**
Medical products and equipment	24.81
Doctor Visits	23.48
Paramedic Care	8.10
Other Expenses	**11.60**
Other Public Health Care Expenses	9.98
Expenses by None-Profit Institutions	1.62
Grand Total	**181.15**

Notes: Our sources are Statistics Denmark, http://medstat.dk/, Forsikring & Pension, and Arbejdsmarkedets erhvervssikring.

Table A3 Employment Shares by 1-digit Occupation, Annual Data

Occupation	Full Sample Occp. Share	Hours Subsample Occp. Share
1	.032245	.0370792
2	.0715409	.0779478
3	.1439805	.1619491
4	.0627748	.0556741
5	.0115262	.0052905
6	.0042052	.0028871
7	.1983044	.1716986
8	.3877012	.3975089
9	.082292	.0891845
Missing	.0054299	.0007804

Notes: The results are based on annual data, and the occupation codes are 1-digit ISCO-88 codes.

Table A4 Prescription Drugs and Doctoral Visits with Age as Control

VARIABLES	Anti-Depressants (1)	Heart-Disease Drugs (2)
log(sales)	0.0542*	0.0501***
	[1.93]	[2.18]
Age	0.000514	-0.001851*
	[0.49]	[-1.80]
Union	0.001424**	0.003216***
	[2.26]	[6.76]
Married	-0.000277	-0.006138***
	[-0.48]	[-15.65]
log(employment)	0.000771	-0.001940**
	[0.74]	[-2.44]
log(K/L)	-0.000150	-0.000546
	[-0.38]	[-1.64]
Skilled-labor share	0.011235**	-0.012928***
	[2.25]	[-2.70]
log(offshoring)	0.000029	-0.000057
	[0.29]	[-0.58]
worker-firm FE	Yes	Yes
year FE	Yes	Yes
Obs. No.	3,407,793	3,442,131
R2	0.0083	0.0145
Job-Spell No.	713,133	721,511

Notes: t-statistics in brackets, with clustering at firm level in all columns. *** p<0.01, ** p<0.05, * p<0.1.

Table A5 JobStrain and FutureStroke in ACL

	(1)	(2)
JobStrain1	0.815*	
	(2.34)	
JobStrain2		0.168
		(0.62)
age	0.0325***	0.0310**
	(3.38)	(3.22)
Female	0.0879	0.0714
	(0.36)	(0.29)
Separate	0.546	0.546
	(1.10)	(1.10)
Divorced	0.255	0.266
	(0.79)	(0.83)
Widowed	0.0472	0.0407
	(0.10)	(0.09)
Never Married	-0.0686	-0.0424
	(-0.17)	(-0.10)
Constant	-4.578***	-4.462***
	(-9.05)	(-8.70)
N	1513	1513

Notes: The omitted category of sex is male, and that of marriage is “married”. JobStrain1 excludes median. JobStrain2 includes median.

Table A6 Predicted JobStrain and FutureStroke in ACL Manufacturing Sub-sample

	(1)	(2)
Prdt. JobStrain (75th pct)	3.547***	
	(4.43)	
Prdt. JobStrain (90th pct)		1.060
		(1.64)
age	0.0723*	0.0640*
	(2.39)	(2.46)
Female	-0.366	-0.678
	(-0.55)	(-1.12)
Divorced	0.788	0.382
	(0.93)	(0.54)
Widowed	0.503	1.081
	(0.35)	(0.91)
Never Married	0.250	0.0925
	(0.27)	(0.11)
Constant	-7.726***	-5.411***
	(-4.82)	(-4.49)
N	255	255

Notes: The omitted category of sex is male, and those of marriage is “married” and "separate". The sample consists of manufacturing workers in ACL.

Table A7 First Stage Estimation for Predicted Sales

VARIABLES	log(sales)
log(instrument)	0.725***
	[5.79]
log(employment)	0.452***
	[7.14]
log(K/L)	0.0312
	[1.60]
Skilled-labor share	-0.856***
	[-2.97]
log(offshoring)	0.0287***
	[3.87]
Exp. 5-20 years	-0.0080
	[-1.37]
Exp. 20+ years	-0.0099
	[-1.61]
Union	0.0022
	[0.31]
Married	-0.0059**
	[-2.49]
Obs. No.	3,338,233
R2	0.4533
Job-Spell No.	727,945
F-stat	33.52

Notes: t-statistics in brackets, with clustering at firm level in all columns. *** p<0.01, ** p<0.05, * p<0.1. The sample is the one we have used to estimate the effects on anti-depressants, and the results based on the samples of the other sickness variables are very similar. All specifications include job-spell fixed effects, and year fixed effects.

Table A8 Mean Sickness Rates by Gender

	Men	Women
Anti-Depressant (Drug)	0.0304	0.0471
Narrow Burnout (Hosp.)	0.00004	0.00007
Broad Burnout (Hosp.)	0.00005	0.00009
Heart Disease (Drug)	0.0289	0.0152
Heart Attack or Stroke (Hosp.)	0.0020	0.0007
Liver Disease (Hosp.)	0.0011	0.0005

Table A9 Estimation of VSI

Dep. Var. = log(annual wage)	
Occp. Injury Rate	5.239**
	(2.443)
female	-0.209***
	(0.0090)
kids	-0.0341***
	(0.0076)
married	0.0383***
	(0.0031)
native	-0.0907***
	(0.0098)
vocational school	0.0923***
	(0.011)
college	0.233***
	(0.013)
age	-0.0056***
	(0.0007)
experience	0.0569***
	(0.0019)
experience2	-0.0011***
	(3.98e-05)
tenure	0.0047***
	(0.0004)
Obs. No.	890,650
R^2	0.429

Notes: standard errors (clustered by occupation) in brackets. *** p<0.01, ** p<0.05, * p<0.1.