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The Wear and Tear on Health: What is the Role of Occupation?

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The Wear and Tear on Health: What Is the Role of Occupation?

Bastian Ravesteijn, Hans van Kippersluis, Eddy van Doorslaer*

September 16, 2013

Abstract

While it seems evident that occupations affect health, effect estimates are scarce. We use a job characteristics matrix in order to characterize occupations by their physical and psychosocial burden in German panel data spanning 26 years. Employing a dynamic model to control for factors that simultaneously affect health and selection into occupation, we find that manual work and low job control both have a substantial negative effect on health that increases with age. The effects of late career exposure to high physical demands and low control at work are comparable to health deterioration due to aging by 16 and 23 months respectively.

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Average health and life expectancy differ substantially across occupational groups (Marmot et al., 1991; Case and Deaton, 2005). For example, manual workers in the US are 50 percent more likely to die within a given year than workers in managerial, professional and executive occupations (Cutler et al., 2008). In eight European countries, the mortality rate for manual workers is higher than for nonmanual workers throughout the age distribution (Mackenbach et al., 1997; Kunst et al., 1998), and this gap has widened over time (Mackenbach et al., 2003). For the Netherlands, Ravesteijn et al. (2013) find a strong gradient in self-assessed health by occupational class, especially at an older age, and note that at the age of 60, 20 percent of elementary workers have exited the workforce into disability, as opposed to 8 percent among workers in occupations that require academic training.

Apart from occupation exerting a causal effect on health, this strong correlation between occupation and health could also stem from reverse causality, with health constraining occupational choice. Moreover, individuals in different occupational groups can differ in other observed and unobserved “third factors” that potentially influence health. For example, manual workers may have lower education, or they may have a different genetic predisposition. Both reverse causality and third factors may lead to selection effects: people with good health prospects are selected into certain types of occupation, and as a result the size of the association may be very different from the size of the causal effect of occupation on health.

In this paper we aim to assess the extent to which the observed association is due to causation from occupation to health. This is important from a fairness perspective, as well as from a productivity perspective. From a fairness perspective, health disparities that result from occupational stressors may be socially undesirable. Policymakers may want to distinguish between health disparities resulting from free choice behavior, such as smoking or drinking, and from occupational stressors that can only be chosen from a heavily constrained choice set. From a productivity perspective, policymakers and employers are interested to know which specific occupational characteristics are most harmful to health. For example, occupations with harmful ergonomic workplace conditions may simultaneously be characterized by low control possibilities at work, which may exert
an independent effect on health. Consequently, improved knowledge about these separated effects will allow for better-targeted efforts in order to reduce sickness absenteeism and disability through adjustment of specific labor conditions.

Many studies have documented strong associations between type of occupation and health (see e.g. Kunst et al., 1999; Goodman, 1999). Only very few attempt to obtain estimates of a causal effect and those that do often focus on very specific occupations or specific exposure to health-harming circumstances (e.g. Bongers et al., 1990, who study back pain among helicopter pilots). In the economic literature the relationship between occupation and health has received surprisingly little attention, yet interest has been growing in recent years. Case and Deaton (2005) show that the self-reported health of manual workers is lower and declines more rapidly with age than of nonmanual workers. Choo and Denny (2006) report similar patterns for Canadian workers while controlling for a more extensive set of lifestyle factors, and suggest that manual work has an independent effect on health over and above any differences in lifestyle across occupations. Using the longitudinal Panel Study of Income Dynamics (PSID), Morefield et al. (2012) estimate that five years of blue collar employment predicts a four to five percent increase in the probability of moving from good to bad health. Yet, as the authors acknowledge, these studies are limited in their ability to investigate the role of factors that may affect both selection into certain types of occupation and health itself.1

Using a three-digit occupational classification, Fletcher et al. (2011) combine the information on physical requirements of work and environmental conditions taken from the Dictionary of Occupational Titles (DOT) with occupational information in the US Panel Study of Income Dynamics (PSID). Their aim is to estimate the health impact of five year exposure to these physical and environmen-

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1Apart from current occupation, workers’ entire occupational history is likely to have an impact on current health. Fletcher and Sindelar (2009) use father’s occupation during childhood and the proportion of blue collar workers in the state as instrumental variables for first occupation, and find that first occupation in a blue collar occupation has a negative effect on self-assessed health. Kelly et al. (2011) question the statistical relevance of the two instrumental variables used in Fletcher and Sindelar (2009), and instead propose methods developed by Lewbel (2012) and Altonji et al. (2005) in order to investigate the causal effect of first occupation on health. They find that entering the labor market as a blue collar worker raises the probabilities of obesity and smoking, by 4 and 3 percent respectively. This indicates that the effect of occupation on health may – at least partly – be transmitted through lifestyles.
tal conditions. They include first-observed health and five-period lagged health in their model and they find negative effects of physical demands and environmental conditions on health for women and older workers but not for men and nonwhite women, and strong negative effects of environmental conditions for young men but not for young women. Fletcher et al. (2011) acknowledge that the potential endogeneity of occupation and occupational change does not allow their random effects estimates to have a causal interpretation. Reverse causality and unobserved third factors may lead to biased estimators, and in their approach it is impossible to disentangle the contributions of physical and psychosocial occupational stressors.

In this study, we aim to overcome these limitations in two ways. First, we estimate a fixed effects model that controls for lagged health in order to estimate the effect of exposure to occupational stressors in the previous year on current health. From a theoretical model of occupation and health over the life cycle, we derive health-related selection mechanisms into occupation, show how our econometric estimators relate to the structural parameters, and explicitly formulate conditions under which our estimates have a causal interpretation. We argue that, in the absence of exogenous variation in the regressor(s) of interest, but with panel data spanning 26 years, our model offers the most trustworthy causal estimates. Our alternative formulation of the Grossman (1972) model illustrates its implicit assumptions on the decaying effect of past shocks and health investment on current health. These insights are informative for anyone trying to provide theoretical foundations for an econometric dynamic panel data model.

Second, we argue that manual occupations are not only more physically demanding, but are simultaneously often characterized by low psychosocial workload. In previous research, most existing studies have characterized occupation with a binary indicator of manual versus nonmanual occupation, or have focused only on the manual aspects of occupation. This made it impossible to disentangle the contributions of the different ergonomic and psychosocial stressors, which meant that the resulting policy implications were unclear. In the present study, we link detailed Finnish data on occupational stressors to individual-level German longitudinal data. This provides a level of detail that was not available in earlier studies. The US DOT, for example, lacks information on psychosocial workplace conditions and exclusively includes information on physical requirements and en-
environmental conditions.

Our findings suggest that about 50 percent of the association between physical demands at work and self-reported health is due to the causal effect of physical demands. Selection accounts for the remaining 50 percent. The average immediate effect of a one standard deviation increase in the degree of manually handling heavy burdens (e.g. from a wholesale worker to a plumber or from a mail sorter to a bricklayer) is comparable to the effect of aging five months and the effect increases with age. A lower degree of control over daily activities at work (e.g. kitchen assistant instead of cook or nurse instead of physiotherapist) is harmful to health at older, but not at younger ages. Under the assumption that the coefficient of lagged health captures the decay rate of past choices and shocks, we estimate that exposure to a one standard deviation increase in handling heavy burdens between age 60 to 64 leads to a health deterioration that is comparable to aging 16 months. The estimated effect of exposure to low job control between age 60 to 64 is comparable to aging 23 months.

The paper is organized as follows: Section 1 discusses the theoretical relationship between occupation and health. Section 2 introduces the German Socioeconomic Panel. Section 3 outlines our empirical approach to estimating the effect of manual work on health. Section 4 presents the results. Section 5 discusses how our results relate to the literature and concludes.

1 Occupation and health over the life cycle

In the economics literature, health is treated as a durable capital stock which depreciates with age and can be increased by investment (Grossman, 1972). The age-related health depreciation rate is exogenous, but an individual can invest in his health through the purchase of (preventive or curative) medical care. The effect of behavior on health can be positive or negative. Occupational choice can be seen as a form of health disinvestment/erosion: an individual chooses an occupation that is characterized by a set of potentially harmful occupational stressors (Case and Deaton, 2005; Galama and van Kippersluis, 2010). Occupations with more harmful characteristics may yield higher earnings than other, less harmful occupations in the choice set of the individual. This difference in earnings is
known as the compensating wage differential (Smith, 1974; Viscusi, 1978). The additional earnings may be used in order to partially offset the detrimental effect of work on health by investing in health, but this is not necessarily the case, for example when the individual spends the additional income on consumption. This economic paradigm proves useful in detecting sources of health-related selection into occupation.

These insights are embedded in a theoretical model of an individual maximizing the expected present value of lifetime utility, which is derived from consumption \( c \) and health \( h \), by choosing levels of consumption \( c \), occupational stressors in vector \( o \), and health investment \( m \). Each occupation is characterized by physical and psychosocial occupational stressors which tend to be clustered, i.e. occupations with low psychosocial workload are often simultaneously characterized by high physical demands. Future utility is discounted at discount rate \( \beta \). The information set \( I \) includes endowments \( e \) and permanent health \( h_p \), all state and choice variables up to time \( t \), all future values of the aging rate, but not future unforeseen health shocks \( \eta \).

\[
max_{\{c_{t+j}, o_{t+j}, m_{t+j}\}} J_T \sum_{j=0}^{T-t} \beta^j u(c_{t+j}, h_{t+j}) \vert I_t \]

(1)

The health production function depends on (i) characteristics and circumstances that remain constant over time, embodied by permanent health \( h_p = f(e) \), which is a function of endowments and reflects all circumstances and personal characteristics that remain constant over the life cycle, (ii) foreseen health deterioration because of aging \( a \), (iii) a vector of occupational characteristics \( o \), (iv) medical investment \( m \), and (v) exogenous health shocks \( \eta \). The effect of occupational characteristics on health, \( \gamma_o \), is nonpositive, and \( 0 \leq \theta \leq 1 \), reflects diminishing marginal benefits to health investment. Total lifetime \( T \) is exogenous and known to the individual and the effects of occupational stressors, health investments and shocks are assumed to decay at the same rate \( \phi \), which lies between 0 and 1.

\[
h_{t+j} = h_p + \sum_{k=2}^{t+j} \left( a_k + \phi^{t+j-k} (\gamma_o o_k + \gamma_m m_k + \eta_k) \right)
\]

(2)

Expenditures on consumption and health investment, at prices \( p_c \) and \( p_m \) respectively, should not exceed the net value of wage earnings. The individual can lend
and borrow at real interest rate $r$, but he has to repay any remaining debt at the end of his life. Wage $w$ is a function of (i) current occupational choice $o$, (ii) current health $h$, and (iii) endowments $e$. 

$$s.t. \sum_{k=1}^{T} (p_c c_k + p_m m_k) \leq \sum_{k=1}^{T} (1 + r)^{k-1} w(o_k, h_k; e)$$ (3)

Consumption, health investment and occupational choice are chosen by equating marginal benefit to marginal cost. The marginal utility of consumption is equal to the shadow price of income $\lambda$ times the price of consumption.

$$\frac{\partial u_t}{\partial c_t} = \lambda p_c$$ (4)

For each occupational attribute $o_l$ in vector $o$, the marginal benefit of occupational stress is represented by the product of $\lambda$ and the instantaneous wage premium. The marginal cost includes the marginal deterioration of health in all future periods multiplied by (i) the discounted marginal utility of future health, and (ii) the product of $\lambda$ and the present value of the marginal wage returns to future health.

$$\lambda \frac{\partial w_t}{\partial o_{t,l}} = -\sum_{j=1}^{T-t-1} \left[ \beta^j \frac{\partial u_{t+j}}{\partial h_{t+j}} + \lambda \left( \frac{1}{1 + r} \right)^j \frac{\partial w_{t+j}}{\partial h_{t+j}} \right] \forall l$$ (5)

Health investment is the ‘mirror image’ of occupational choice. The marginal benefit (the product of the marginal effect of health investment on health and both the discounted marginal utility of health and the marginal wage returns to health in all future periods) is equated to marginal cost (the product of the shadow price of income and the price of medical care).

$$\sum_{j=1}^{T-t-1} \frac{\partial h_{t+j}}{\partial m_t} \left[ \beta^j \frac{\partial u_{t+j}}{\partial h_{t+j}} + \lambda \left( \frac{1}{1 + r} \right)^j \frac{\partial w_{t+j}}{\partial h_{t+j}} \right] = \lambda p_m$$ (6)

The theoretical framework shows how an individual takes into account the future consequences of his decisions while deciding on the optimal levels of harmful occupational stressors. Three insights from the theory are particularly noteworthy. First, both time-invariant initial endowments $e$, in the form of e.g. physical ability, intelligence or taste for adventure, and time-varying factors such as health shocks
\(\eta\) (e.g. a car accident or a disease onset) may influence both occupational choice and health status through (i) the marginal utility of health, (ii) the marginal wage returns to health, and (iii) the shadow price of income \(\lambda\). This means that workers may select themselves into certain types of occupations depending on exogenous factors that also influence health directly. The observed health differences across occupational class should therefore not be interpreted as evidence of a causal effect of occupation on health.

Second, in contrast to exogenous sources of health-related selection into occupation, such as endowments and shocks, individuals endogenously choose their level of health investment. Health investment may be correlated with occupational choice (i) because exogenous factors influence both, and (ii) because workers may choose to offset occupation-related health damage by investing in health: e.g. a bricklayer may seek physiotherapeutic treatment for his back pain or a manager may take yoga classes in order to improve his mental well-being.

Third, the relationship between work and health may change over the life cycle. This could occur for three reasons. First, as equation 6 illustrates, the expected wage returns on health investment decrease as the individual approaches the retirement age, which implies that individuals have fewer incentives to offset occupational damage to health by medical investment.\(^2\) Second, possibly \(\gamma_o\) changes over the lifetime, e.g. if health at older ages is more vulnerable to wear and tear at the workplace. Third, even if the effect of hard work is equal at all ages, the marginal effect of health repair may decrease with age such that full repair of health is no longer feasible at older ages.\(^3\)

In sum, our empirical identification strategy should (i) account for factors that may influence selection into type of occupation and may also be related to health, (ii) address how behavioral adjustments that affect health may coincide with occupational choice, and (iii) accommodate the changing relationship between occupation and health over the life cycle.

\(^2\)Although a model which endogenizes length of life as a function of health could explain an increase in medical investment at older ages.

\(^3\)Our model does not incorporate real-world labor market rigidities, yet these may also prevent individuals from switching occupations at older ages to optimize their exposure to occupational stressors.
2 Occupational stressors and the German Socioeconomic Panel

The German Socioeconomic Panel (GSOEP) started in 1984 and we use data from the 26 subsequent annual waves. The full sample consists of 358,281 person-wave observations. Sample sizes per wave range from 8,681 in the year 1989 to 20,912 in 2000. Respondents are followed over multiple waves but the panel is unbalanced since many respondents enter the sample after the year 1984 or leave the sample before 2009. We limit our sample to respondents who are employed or self-employed and of working age, i.e. between 16 and 65 years old. 35,258 individuals are recorded to have been working in at least one wave, 15,819 individuals have been working in at least six waves and 6,813 in at least eleven waves.

Respondents were asked to rate satisfaction with their own health on an integer scale from 0 to 10 which we will refer to as self-assessed health (SAH). Occupational titles were coded into the International Standard Classification of Occupations of the OECD (ISCO-88). These are 311 occupational classes that were grouped into ten ranked major occupational groups by the OECD. On the basis of the OECD classifications, we define white collar workers as legislators, senior officials, managers, professionals, technicians, associate professionals, clerks. We define blue collar workers as service workers and shop and market sales workers, skilled agricultural and fishery workers, craft and related trades workers, plant and machine operators, assemblers and workers in elementary occupations. This is in line with the distinction between manual and nonmanual work of Case and Deaton (2005). According to these definitions, we have a total of 122,419 person-wave observations for white collar occupation and 110,783 observations for blue collar occupation.

Figure 1 graphs age-predicted SAH for blue collar and white collar workers. Blue collar workers on average report better health at younger ages, whereas the opposite is true after the age of 28. Self-assessed health decreases for both blue collar and white collar workers over most of the age range, but increases after the age of 57. One should keep in mind, however, that these patterns only reflect the SAH ratings of those who are employed.\footnote{At older ages, unhealthy workers exit out of employment while healthy workers remain.} In line with Case and Deaton (2005),
in the pooled sample we find the health decline associated with age to be much steeper among blue collar than white collar workers. This is the observation that begs the question why blue collar workers run down their health at a much faster rate than white collar workers. Table 1 shows that the average SAH score for

![Figure 1: Health for blue and white collar workers.](image)

Predicted satisfaction with health for blue and white collar workers over the life cycle.

those who work is 7.05, and lower for blue collar workers (6.96) than for white collar workers (7.10). Blue collar workers on average have less education and earnings, are slightly younger and less likely to be female. Average education among blue and white collar workers is 10.47 and 12.89 years, respectively. If we disregard censoring, average net monthly labor earnings are 1,463 Euro for white collar workers and 1,098 Euro for blue collar workers. While the dichotomization into blue and white collar occupation represents a useful and convenient way to study health differences across broad occupational groups, it does not allow us to identify which aspects of occupational stressors associated with blue collar occupations matter most. In Finland, a Job Exposure Matrix (FINJEM) has been

\footnote{Health worsens from the top to the bottom of the OECD occupational ladder. 23 percent of legislators, senior officials and managers rate their health with a five or less, as opposed to 31 percent of elementary workers. 49 percent of legislators, senior officials and managers rate their health with at least an eight, while for elementary workers this is only 42 percent. This pattern is monotonic across the nine ranked major OECD occupational groups.}
constructed from a detailed survey on occupational stressors that maps occupational titles into three measures of occupational stressors (Kauppinen et al., 1998): (i) the manual handling of burdens, (ii) control possibilities at work and (iii) psychosocial workload. Even though such information on occupational stressors in Germany is unavailable,\textsuperscript{6} we use the FINJEM in order to map about 360 different occupational titles in the GSOEP into (ordinal) measures of occupational stressors, thereby assuming that the relationship between OECD classified occupations and occupational stressors in Finland and Germany is similar.

Table 2 shows the exposure of the nine major OECD occupational groups in the GSOEP to the three occupational stressors in the FINJEM. Two important observations can be made. First, blue collar occupations are not only characterized by more frequent manual handling of heavy burdens than white collar occupations, but also by lower psychosocial workload, which is often ignored in the previous literature. Control possibilities vary across occupations, but there is no clear pattern if we move up the occupational ladder. Second, there is ample variation in occupational characteristics even within the major occupational groups. Even though blue collar workers are generally more likely to have more demanding ergonomic conditions and lower psychosocial workload when compared to white collar workers, for many specific occupations this is not the case. A simple dichotomization of occupations into either blue or white collar would therefore neglect the considerable heterogeneity within these groups and the clustering of

\textsuperscript{6}The German Qualification and Career Survey only includes information on analytical tasks, manual tasks and interactive tasks, while we are interested in physical and psychosocial stressors.
Table 2: Occupational stressors across the major ISCO occupational groups.

<table>
<thead>
<tr>
<th>Manual handling of burdens, percentage above mean</th>
<th>Percentage with high job control</th>
<th>Percentage with high psychosocial workload</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Legislators, senior officials and managers</td>
<td>23</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Professionals</td>
<td>6</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td>Technicians and associate professionals</td>
<td>18</td>
<td>18</td>
<td>75</td>
</tr>
<tr>
<td>Clerks</td>
<td>9</td>
<td>4</td>
<td>54</td>
</tr>
<tr>
<td>Service workers and shop/market sales workers</td>
<td>90</td>
<td>10</td>
<td>68</td>
</tr>
<tr>
<td>Skilled agricultural and fishery workers</td>
<td>100</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Craft and related workers</td>
<td>76</td>
<td>9</td>
<td>22</td>
</tr>
<tr>
<td>Plant and machine operators and assemblers</td>
<td>87</td>
<td>37</td>
<td>15</td>
</tr>
<tr>
<td>Elementary occupations</td>
<td>100</td>
<td>4</td>
<td>12</td>
</tr>
</tbody>
</table>

Percentages reflect proportion above mean for manual handling of burdens, proportion exposed to low job control and high psychosocial workload. Manual handling of burdens is an index, job control and psychosocial workload are binary variables. White collar occupations above the dashed line, blue collar occupations below.

occupational stressors. In the remainder of this paper we will use the blue/white collar distinction as well as the variation on the basis of the three occupational stressors across the 360 occupational titles in order to estimate the effect of occupation on health.

3 Estimation of the effect of occupational stressors on health

We want to estimate the structural parameter $\gamma_o$ in equation 2 which refers to the health effects of exposure to occupational stressors $o$ in the previous year. Towards this aim, note that the one-period lag of the health production function (equation 2), which includes permanent health $h_p$, the health effects of aging $a$,
health investment \( m \) and shocks \( \eta \), is:

\[
h_{t+j-1} = h_p + \sum_{k=2}^{t+j-1} (a_k + \phi^{t+j-1-k}(\gamma'_o o_{k-1} + \gamma_m m_{k-1} + \eta_k))
\] (7)

Substituting equation 7 into equation 2 we obtain:

\[
h_{t+j} = (1 - \phi) \left( h_p + \sum_{k=1}^{t+j-1} (a_k) \right) + a_{t+j} + \gamma'_o o_{t+j-1} + \gamma_m m_{t+j-1} + \phi h_{t+j-1} + \eta_{t+j}
\] (8)

Switching to individual notation and demeaning the covariates to eliminate the time-invariant factors, we obtain a fixed effects within estimator:

\[
h_{i,t+j} - \bar{h_i} = \phi (h_{i,t+j-1} - \bar{h_i}) + \gamma'_o (o_{i,t+j-1} - \bar{o_i}) + \delta'(x_{i,t+j} - \bar{x_i}) + \varepsilon_{i,t+j}
\] (9)

where any unobserved heterogeneity that is constant over time and may be correlated with occupation (such as permanent health \( h_p \) in equation 2) is eliminated:

\[(1 - \phi)h_p - (1 - \phi)\bar{h_p} = 0\]. Coefficient \( \phi \) of the demeaned one period lag of health can be interpreted as the decay parameter through which occupational choice \( o \), health investment \( m \), and unforeseen shocks \( \eta \) in period t-2 and earlier periods affect current health.

\( x \) is a vector of control variables consisting of age, age squared, age to the third power, and wave dummies in order to control for common time trends. We assume that the effect of age is smooth and can be approximated by an age polynomial of the third degree. A less flexible approximation of the age effect, such as only controlling for a linear term, would bias our estimates of \( \gamma_o \) if health deteriorates more rapidly at older ages, and workers at older ages would be more or less likely to be exposed to certain occupational stressors.

The error term is \( \varepsilon_{i,t+j} = \gamma_m (m_{i,t+j-1} - \bar{m_i}) + \eta_{i,t+j} - \bar{\eta}_i \), which implies two things. First, the ordinary least squares estimator of \( \phi \) is biased since \( h_{i,t+j-1} \) is correlated with \( \bar{\eta}_i \), and \( \bar{h}_i \) is correlated with \( \eta_{i,t+j} \). Yet, importantly the estimator is consistent for large \( T \) (Nickell, 1981; Bond, 2002). Second, the estimator of \( \gamma_o \) is biased if occupation and health investment are correlated. The theory suggests individuals simultaneously choose occupation and health investment, such that the estimates should be interpreted as the sum of the structural effect of occupation.
and health investment decisions related to occupation.

Self-reported health, on a five-point ordinal scale from poor to excellent, has been shown to be a reliable predictor of mortality and morbidity (e.g. Idler and Benyamini, 1997; Mackenbach et al. 2002). We use satisfaction with health (on a 0-10 integer scale) as a proxy for health, which exhibits more variation than the five-point measure. Ferrer-i-Carbonell and Frijters (2004) show that for the variable that measures satisfaction with life on a ten point scale, assuming ordinality or cardinality makes little difference, such that a linear specification is acceptable. Reporting heterogeneity due to the fact that different subgroups may report the same objective health status differently (Lindeboom and Van Doorslaer 2004), is eliminated by the individual fixed effect to the extent that it is time-invariant.

Our estimates are based on variation in occupational stressors and health over time within individuals for whom we observe occupation in the previous year and health in the current year. Nonworking individuals generally remain in the sample, except in the case of attrition due to mortality or nonresponse. Health-related attrition leads to a downward bias – in absolute value – of our estimators if individuals with the largest occupation-related health deterioration are more likely to attrite. Our estimates should therefore be interpreted as a lower bound on the true effect of occupational stressors.

4 Results

4.1 Main results

Table 3 shows the main results compared for five different models, where we first present results for a dichotomous indicator for blue/white collar (columns 1 and 2) and then present results where we break down occupation into three occupational stressors (columns 3 to 5). To get an idea about the order of magnitude of the coefficients, note that the average health deterioration of getting one year older (obtained from an individual fixed effects regression of satisfaction with health on age) is \(-0.0666 (0.0006)\) in our sample.

The bivariate association in column 1 between satisfaction with health and blue
or white collar occupation in the previous year tells us that blue collar workers are in worse health, and that the size this health gap is similar to the average effect of aging 25 months, which is a sizable and economically meaningful difference. Column 2 shows the results for the model described by equation 9. Much of the association appears to be driven by selection effects, as the estimate of the causal effect is now -.0343 (.0171) compared to -.1418 (.0091) in column 1. We conclude that the health effect of exposure to a blue collar instead of a white collar occupation in the previous year is comparable to the average health effect of aging six months. Column 3 breaks down occupation into three dimensions of occupational stressors: manual handling of heavy burdens, job control, and psychosocial workload in the preceding year. Manual handling of burdens and low job control are associated with worse health, while workload is positively associated with health. Given our theoretical model, we expect strong health-related selection into occupation that could drive these associations. Column 4 shows estimates of the effects of these three occupational stressors according to the specification in equation 9. We conclude that approximately fifty percent of the negative association between manual handling of heavy burdens and health can be explained by selection, and our point estimate (-.0275) of the causal effect of a one standard deviation increase in manually handling heavy burdens compares to a health effect of aging five months.

The estimates of the causal effects of psychosocial stressors (control possibilities at work and workload) in column 4 are not significantly different from zero. As we have seen in table 2 psychosocial workload was higher among white collar workers and lower among blue collar workers with the exception of service workers and shop and market sales workers. Socioeconomic factors, such as education, influence both occupational rank – and therefore workload – and health status, which leads to selection bias of the naïve estimator in column 3. A comparison of the point estimates for workload in columns 3 and 4 confirms that selection effects are important for psychosocial workload: the 95 percent confidence interval of the causal estimate [-.0218, .0332] lies well below the confidence interval of the association [.4593, .8430], plausibly because of the elimination of much of the omitted variable bias that plagues the results in column 3.

We add a vector of occupational stressors interacted with age to the set of inde-
Table 3: Results.

<table>
<thead>
<tr>
<th>Associations for blue and white collar</th>
<th>FE &amp; LDV for blue and white collar</th>
<th>Associations for stressors</th>
<th>FE &amp; LDV for stressors</th>
<th>FE &amp; LDV for age interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue collar occupation at t-1</td>
<td>-.1418** (.0092)</td>
<td>-.0343* (.0171)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manual handling of burdens at t-1</td>
<td>-0.567** (.0050)</td>
<td>-0.0275** (.0090)</td>
<td>0.0436 (.0260)</td>
<td></td>
</tr>
<tr>
<td>Control possibilities at t-1</td>
<td>0.0582** (.0148)</td>
<td>-0.0120 (.0217)</td>
<td>0.1513* (.0687)</td>
<td></td>
</tr>
<tr>
<td>Workload at t-1</td>
<td>0.0651** (.0098)</td>
<td>0.0057 (.0140)</td>
<td>-0.0358 (.0451)</td>
<td></td>
</tr>
<tr>
<td>Age * manual handling of burdens at t-1</td>
<td></td>
<td></td>
<td></td>
<td>-0.0019** (.0006)</td>
</tr>
<tr>
<td>Age * control possibilities at t-1</td>
<td></td>
<td></td>
<td></td>
<td>-0.0042* (.0017)</td>
</tr>
<tr>
<td>Age * workload at t-1</td>
<td></td>
<td></td>
<td></td>
<td>0.0011 (.0011)</td>
</tr>
<tr>
<td>Health at t-1</td>
<td>0.0985** (.0032)</td>
<td>0.0985** (.0032)</td>
<td>0.0983** (.0032)</td>
<td></td>
</tr>
<tr>
<td>Individual fixed effects</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>estimator controlling for a third order age polynomial and wave dummies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>202,109</td>
<td>201,750</td>
<td>200,435</td>
<td>200,080</td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.0012</td>
<td>0.5645</td>
<td>0.0012</td>
<td>0.5646</td>
</tr>
</tbody>
</table>

Main results for satisfaction with health. Panel-robust standard errors in parentheses. * indicates significance at 5 percent level, and ** at 1 percent level. Fixed effects specifications are computed by subtracting individual averages for each regressor. Reference category for columns 1 and 2 is working in a white collar occupation. Intercept not shown.
dependent variables in column 5 of table 3 in order to investigate whether the causal effect of occupational stressors differs by age. The model nests age-dependent and age-independent effects: the coefficients of the interaction terms would be equal to zero if the effects of the occupational stressors do not vary with age. The coefficients of the noninteracted occupational stressors in rows three to five of column 5 cannot be interpreted directly, as they refer to the hypothetical effects of occupational stressors at the age of zero. The coefficients of the interaction terms in rows six to eight indicate how the effects of the occupational stressors change as workers grow older.

Strikingly, predicted health deterioration due to a one standard deviation increase in handling heavy burdens is equal to zero at the age of 23, but at the age of 63, the point estimate of the effect is comparable to aging almost 14 months (.0436-.0019*63=-.077). Low job control has a negative effect after the age of 36: being in a job with low instead of high job control at the age of 63 leads to a predicted health deterioration comparable to the effect of aging 20 months (.1513-.0042*63=-.1133). We conclude that the effect of manual work and job control is age-dependent. The effect of the interaction of workload and age is not significantly different from zero, possibly due to our crude, dichotomous measure of workload or because workload is only important for a subset of occupations.

The health effects of past exposure to occupational stressors cannot simply be added to obtain cumulative effects. Under quite demanding assumptions, we can compute cumulative health effects by using the estimated coefficient of the lagged dependent variable, $\phi$ in equation 2, which by assumption is the uniform exponential decay rate at which past health investment, occupational stressors, and shocks affect current health. The point estimates of $\phi$ in table 3 suggest that roughly ten percent of the occupation-related health damage in period t-2 persists in period t. Using the point estimates in column 5, the point estimate of health damage at the age of 65 due to a one standard deviation increase in manual handling of heavy burdens between ages 60 to 64 is $\sum_{k=60}^{64} \cdot .0983^{64-k}(.0436-.0019* k) = -.0865$, which is comparable to the average health effect of aging almost 16 months. Likewise, the point estimate of the effect of working in low control occupations between ages 60 to 64 is -.1398, which is comparable to the effect of aging 23 months.
4.2 Robustness checks

Individuals in different occupations may have different biological aging rates, while we have assumed uniform aging effects in the preceding analyses. If the health of individuals in manual occupations would decline more strongly, our results would overestimate the harmful effects of physical stressors. In column 1 of table 4 we allow for different aging rates by interacting an education dummy with a third degree age polynomial. Our estimates are similar to our findings in table 3, and we find no significant differences in the biological aging rate between individuals with high and low educational attainment. The estimator of the coefficient of the lagged dependent variable is consistent if the number of time periods in the sample goes to infinity. Our sample spans 26 years and is unbalanced: it includes individuals who are observed in a lower number of waves. We repeat our analysis for a subsample of 10,373 individuals who are employed in at least nine years in order to counter the downward bias of the estimator of the lagged dependent variable that plagues short panels (Bond, 2002). The number of person-wave observations drops from 200,080 in our baseline specification in column 5 of table 3 to 136,470 in column 2 of table 4. The coefficients of the (age interacted) occupational stressors are similar to those in our baseline specification. However, the coefficient of lagged health is now larger than before, suggesting that past health investment, occupational stress, and health shocks are more persistent than appeared in our analysis of the full sample. We conclude that our estimates of the effects of occupational stressors are robust across specifications, but that an analysis on the full sample leads to underestimation of the coefficient of lagged health. We may have underestimated the cumulative effects of occupational history by underestimating $\phi$, and the predictions in the previous paragraph provide – in absolute terms – the lower bounds on the effects, which means that the true health effects may be even larger.

Angrist and Pischke (2009) voice worries about the violation of strict exogeneity in fixed effects dynamic models, particularly when using short panels. They propose to check robustness by separately estimating both a fixed effects and a lagged dependent variable model. Column 3 of table 4 presents results from a fixed effects model without a lagged dependent variable. With respect to equation 9, the error term would now include the deviations of the effects of health
Table 4: Robustness.

<table>
<thead>
<tr>
<th></th>
<th>Control for individuals</th>
<th>Only individuals</th>
<th>FE</th>
<th>LDV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
</tr>
<tr>
<td>Manual handling of burdens at t-1</td>
<td>.0317 (.0268)</td>
<td>.0447 (.0276)</td>
<td>.0534* (.0262)</td>
<td>.0435** (.0149)</td>
</tr>
<tr>
<td>Control possibilities at t-1</td>
<td>-.1350 (.0696)</td>
<td>.1911** (.0728)</td>
<td>-.1562* (.0691)</td>
<td>-.0707 (.0447)</td>
</tr>
<tr>
<td>Workload at t-1</td>
<td>-.0370 (.0463)</td>
<td>-.0445 (.0480)</td>
<td>-.0404 (.0455)</td>
<td>-.0645* (.0288)</td>
</tr>
<tr>
<td>Age * manual handling of burdens at t-1</td>
<td>-.0016** (.0007)</td>
<td>-.0019** (.0007)</td>
<td>-.0022** (.0007)</td>
<td>-.0023** (.0004)</td>
</tr>
<tr>
<td>Age * control possibilities at t-1</td>
<td>.0039* (.0017)</td>
<td>.0049** (.0018)</td>
<td>.0044* (.0017)</td>
<td>.0008 (.0011)</td>
</tr>
<tr>
<td>Age * workload at t-1</td>
<td>.0011 (.0011)</td>
<td>.0014 (.0012)</td>
<td>.0012 (.0011)</td>
<td>.0029** (.0007)</td>
</tr>
<tr>
<td>Health at t-1</td>
<td>.0976** (.0032)</td>
<td>.1378** (.0034)</td>
<td>.5420** (.0023)</td>
<td></td>
</tr>
<tr>
<td>Third order age polynomial and wave dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Third order age polynomial interacted with age</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Individual fixed effects</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Education and gender</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Observations</td>
<td>196,935</td>
<td>152,244</td>
<td>200,435</td>
<td>196,935</td>
</tr>
<tr>
<td>$R^2$</td>
<td>.5647</td>
<td>.5647</td>
<td>.5604</td>
<td>.3351</td>
</tr>
</tbody>
</table>

Robustness checks for satisfaction with health. Panel-robust standard errors in parentheses. * indicates significance at 5 percent level, and ** at 1 percent level. Fixed effects specifications are computed by subtracting individual averages for each regressor. Second column on sample of individuals who are observed in at least 10 waves. Intercept not shown.
investment, occupational stressors, and health shocks before period t-1 from their individual averages. If a past health shock would have a negative effect on current health and lead to higher occupational stress in the previous period, we would overestimate the effect of occupational stressors since this leads to additional correlation between $o$ and the error term. The point estimates in column 3 suggest a somewhat stronger effect of manual handling of burdens and job control at older ages than the baseline specification, but these may result from the bias caused by past events that affected health. The lower $R^2$ compared to the baseline specification in column 5 of table 3 indicates that this model explains less variation in the outcome variable, possibly because the health effects of omitted variables are no longer proxied by lagged health.

In a model in which we control for a lagged dependent variable, but not for individual-specific fixed effects, the estimator of the decay parameter $\phi$ in equation 9 is biased towards one: all past deviations from the steady state of health “die out” while the time-invariant individual fixed effects are constant over time. In this specification we therefore overestimate the impact of past events on current health, and we only partly control for unobserved time-invariant heterogeneity. By not subtracting averages in equation 9, the error term now includes $(1 - \phi)h_p$, which may be correlated with lagged health and the occupational characteristics. In order to proxy for time-invariant unobserved factors otherwise picked up by the fixed effect, we control for years of schooling and gender. Our estimates are now mostly driven by variation between individuals a moment in time. The relatively low $R^2$ in column 4 of table 4 reflects how this only partially enables us to control for unobserved heterogeneity. The coefficient of the interaction between age and manual handling of burdens is similar to our earlier results, but the coefficient of the interaction between age and job control is no longer significant. Workload now seems to have a positive effect, but this may be due to the fact that, by not controlling for individual-specific fixed effects, we insufficiently control for the selection of healthy individuals into occupations characterized by high workload.

Other methods have been proposed to consistently estimate $\gamma_o$ in equation 9 in short panels, of which the so-called Arellano-Bond estimator (Arellano and Bover, 1995; Blundell and Bond, 1998) is the most prominent one. The Arellano-Bond estimator is based on the first-difference estimator. Most important assumption
is that the second and further lags of health, are uncorrelated with the first differences of the error term and can be used as instrumental variables for $h_{t-1} - h_{t-2}$. Unfortunately, in our case the Arellano-Bond test for autocorrelation rejects this assumption, which is perhaps not so surprising since in the case of health, using lagged values as instruments seems hard to justify. Chronic illnesses or the introduction of a new medical drug may progressively affect health over time. This leads to second or higher order serial correlation in the differenced error term and violation of the exogeneity assumption. In an attempt to overcome this problem one could include more lags of the regressors in the model and use further lags of the instruments in order to attempt to get rid of the autocorrelation in the error term, but we still find higher order autocorrelation in these models, rejecting validity of the instruments.\footnote{Limiting the number of waves could give us the false illusion that serial correlation of the error term is not a problem, simply because of the low power of the test. Blundell and Bond and Michaud and Van Soest (2008) used short panels of six waves, and they “use up” even more waves because of the inclusion of lagged values of the dependent variable. The autocorrelation tests in these studies do not reject the assumption of no autocorrelation in the error term but this could be due to limited test power with the small number of waves. If we include one and two period lags of the dependent variable (Michaud and Van Soest (2008)), we find no second order autocorrelation, but we do find autocorrelation of the third order which would still violate the Arellano Bond assumptions. Including third or fourth lags seems to shift downwards the order of autocorrelation, instead of solving the problem. Performing the Sargan test may not be very informative since this test assumes that at least one instrument is exogenous, which is an assumption we are not willing to make.}

5 Conclusion

We find that exposure to both high physical occupational stress and low job control has a negative effect on health. The immediate effect of exposure to a one standard deviation increase in the degree of handling heavy burdens (e.g. from mail sorter to a bricklayer) during one year is comparable to aging five months. This effect becomes stronger with age: just before reaching the retirement age, a similar increase in handling heavy burdens is comparable to aging fourteen months. Low job control is equally harmful to health, but only after age 36. After age 60, the immediate effect of low job control (e.g. nurse instead of physiotherapist) is equivalent to aging 20 months. The estimated causal effect of
carrying heavy burdens accounts for roughly 50 percent of the bivariate association between occupation and health. This implies that selection into occupation by prior health and/or other factors such as education accounts for the other half of the observed association.

We derive the empirical specification from a theoretical model of occupation and health over the life cycle and outline the conditions under which we can obtain causal estimates using a detailed longitudinal dataset over many time periods (26 years). We show how a fixed effects lagged dependent variable model neutralizes several time-invariant and time-varying sources of selection bias and argue that the resulting estimators are preferable in the absence of exogenous variation in occupational stressors. Moreover, our study generalizes across the labor force, in contrast to local effect estimates based on a particular reform that affected only part of the employed population. We show that the coefficient of the lagged dependent variable should be interpreted as a decay parameter that captures the effects of past unobserved factors – which affected health in the previous period but could also have affected occupational choice – on current health.

We were able to separate the health effects of physical and psychosocial stressors by linking German longitudinal data on occupational titles to Finnish data on occupational stressors on the level of detailed occupational titles. However, as we did not observe individual levels of health investment, we were unable to disentangle the effects of the occupational stressors and any health investment that was made in response to occupational choice. Our estimates should therefore be interpreted as the sum of the direct effect of occupation and the health effect of any behavioral response to occupational choice.

Occupational health and safety policies, career development programs, and retirement policies should be based on the insight that exposure to physically demanding manual handling of burdens and low job-control is harmful to health at older ages. Shielding older workers from these conditions prevents health deterioration among vulnerable groups of workers and is likely to have a protective effect against illness-related absenteeism and labor force exit due to disability.
References


