ANNALS OF THE NEW YORK ACADEMY OF SCIENCES

Issue: The Biology of Disadvantage

Work and its role in shaping the social gradient in health

Jane E. Clougherty,¹ Kerry Souza,² and Mark R. Cullen³

¹Harvard School of Public Health, Boston, MA, USA. ²Department of Epidemiology, Harvard School of Public Health, Boston, MA, USA. ³Division of General Internal Medicine, School of Medicine, Stanford University, Stanford, CA, USA Address for correspondence: Mark R. Cullen, Division of General Internal Medicine Stanford University School of Medicine, 251 Campus Dr. MSOB Room 338, Stanford, CA 94305, USA. mrcullen@stanford.edu

Adults with better jobs enjoy better health: job title was, in fact, the social gradient metric first used to study the relationship between social class and chronic disease etiology, a core finding now replicated in most developed countries. What has been less well proved is whether this correlation is *causal*, and if so, through what mechanisms. During the past decade, much research has been directed at these issues. Best evidence in 2009 suggests that occupation *does* affect health. Most recent research on the relationship has been directed at disentangling the pathways through which lower-status work leads to adverse health outcomes. This review focuses on six areas of recent progress: (1) the role of *status* in a hierarchical occupational system; (2) the roles of *psychosocial job stressors*; (3) effects of workplace *physical and chemical hazard exposures*; (4) evidence that *work organization* matters as a contextual factor; (5) implications for the gradient of new forms of nonstandard or "precarious" employment such as contract and shift work; and (6) emerging evidence that women may be impacted differently by adverse working conditions, and possibly more strongly, than men.

Keywords: occupational health; psycho-social stressors; work organization; women in the workplace; nonstandard work

Introduction

It has long been recognized adults with better jobs enjoy better health than those with less prestigious, less remunerating employment. Indeed job, rather than other measures of wealth and education, was the social gradient metric first used to understand the relationship between occupational position and chronic disease etiology in the landmark Whitehall studies of the 1970s (Fig. 1).¹

The core finding, an inverse association between job status and measures of chronic disease incidence, prevalence, and mortality, has been replicated in most developed countries, although in Europe job appears to show a less pronounced association than income or education.² The United States is no exception; Figure 2 depicts incidence rates by job grade for six chronic illnesses in a large, diverse, and geographically dispersed employee population of a single U.S. corporation, for 1996–2003.³

These relationships hold even after adjustment for other measures of socioeconomic status such as education and income, although these are highly correlated in most populations in developed countries.²

What has been less clear is whether this correlation is *causal*, and if so, through what mechanisms. During the past decade, much research has been directed at these issues, although progress has been slow, especially in the important area of cardiovascular disease, for both technical and practical reasons.^{4.5} The most serious technical problems relate to selection—workers with cardiovascular risk or symptoms often leave exposed jobs early, confounding associations; the practical problem is the difficulty of obtaining detailed occupational exposure data for large samples of subjects. These limitations notwithstanding, we will review the general evidence for causality here, and discuss possible pathways in the several sections that follow.

Two noncausal links between occupational status and health have been hypothesized. First, *reverse causation* would suggest that poorer health status, or adverse health risk profiles, leads to lower job

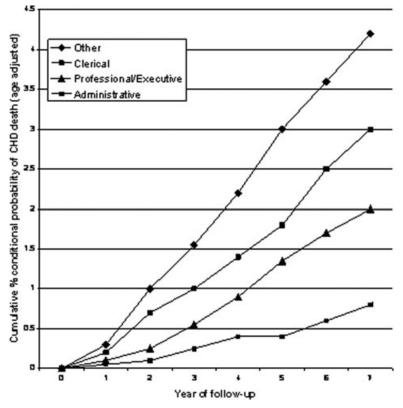


Figure 1. Whitehall Study results depicting the relationship between job category and mortality from coronary heart disease.

status, through a compromised ability to work as effectively or consistently as healthier workers.⁶ There is some longitudinal evidence that poor childhood health leads to lower professional achievement in

adulthood,⁷ and some evidence of preexisting health gradients by profession at the time of employment.⁸ Nevertheless, there is little evidence of measurable health differences among young adults strong

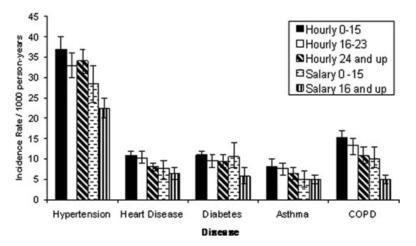


Figure 2. Male incidence rates for major chronic diseases of working-age people for each of five job grades, ranging from low-grade blue-collar (*left bars*) to highest professional/executive grade at Alcoa.

enough to explain job attainment differences after adjustment for education. In fact, most lower-grade jobs in our society (e.g., mining, construction, farming, manufacturing) require demonstrable physical capabilities, especially in comparison to high-grade jobs, and employees in physically demanding jobs are more thoroughly screened for fitness at hire. These observations suggest that the reverse may actually be true-that significant physical incapacity can serve to exclude individuals from physical employment, and is the basis for well-described healthy worker selection effects among employees performing physical labor.9 Some studies have attempted to apportion the job status-health association into causal and selective components using longitudinal data,^{10–12} but with limited success.

Causal and selective components of job status are difficult to disentangle, in part, due to persistent confounding by social class and its associated (unmeasured) health risks. Blue-collar workers in competitive economies tend to derive from lower socioeconomic status (SES) backgrounds, with lesser education and fewer job options. In the United States, blue-collar and low-grade service jobs are disproportionately performed by males and minorities, and generally include, for example, a higher prevalence of smokers.¹³ In other words, workers from more privileged backgrounds, already on a better health trajectory, are more likely to seek and achieve better jobs. As such, occupation may serve only as a surrogate measure for such selection effects. Because experimental data will never be available (comparable to, say, lottery winners for the study of income effects on health, or housing lotteries such as Moving to Opportunity¹⁴), the evidence for and against this argument must be strictly observational. Studies of British birth cohorts have demonstrated that pre-hire factors explain some of the subsequent pattern of adult health,⁷ but may act additively with subsequent occupation and income.15 Studies of the U.S. Alcoa aluminum manufacturing population, stratified by propensity scores of risk factors at hire, demonstrate added effects associated with duration of lower-status, but not higher-status work, suggesting that endogenous differences at onset do not explain the outcomes entirely, or may indicate susceptibility to subsequent exposures.16,17

Thus, while the causal role of job as a partial explanation for the SES gradient remains far from

fully established, and its interplay with other SES indicators such as education and wealth remains underexplored, the best evidence in 2009 suggests that occupation matters (causally) for health. As such, much recent research on work and health has been directed at disentangling the multiple pathways through which lower-status work may lead to adverse health outcomes. Figure 3 depicts the conceptual framework from which we have examined the evidence linking work and adult health. Childhood SES may contribute to adult work roles via educational and/or direct (early adult) health pathways, as well as some "opportunity" pathways, less easily measured. In adulthood, additional benefits or hazards to health may be mediated in turn through multiple pathways including social onessocietal status, income, benefits, etc.--and pathways linked more closely to the context and content of work itself. To capture job content, we include separate physical aspects (e.g., work load, chemical and physical environmental factors) from psycho-social aspects, such as job strain or a need for constant vigilance. The social context in which these exposures occur, especially psychosocial stressors, may modify the impact of physical exposures, comparable to emerging evidence that poorer children tolerate air pollution less well than middle-class children comparably exposed,¹⁸ Not depicted in the schema, but noteworthy, is the likelihood that each of the pathways may operate differently in women than in men.

We devote the rest of this review to the evidence for several salient work-related mediating pathways, with a focus on five areas of recent progress. First, we discuss the role of status in a hierarchical occupational system, and, relatedly, the roles of psychosocial job stressors, and workplace physical and chemical hazard exposures. We then review recent evidence that work organization as a contextual factor (as opposed to job-specific stressors within a workplace) may significantly influence employee health. One specific aspect of that context relates to the changing nature of the work-employee contract. As nonstandard work arrangements become increasingly common, this will raise new questions about the health impact of job insecurity, contract work, self-employment, and, increasingly, underemployment, which many working adults will experience at some time in their working life if current patterns

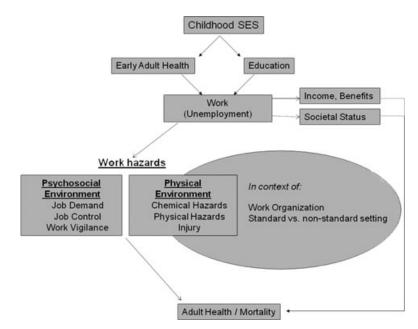


Figure 3. Conceptual framework showing the links between work and adult health.

persist. Finally, as women increasingly perform work roles once almost exclusively male, new questions have arisen about persistent sexstratification in the workforce, and possible modification in the health effects of work conditions on health by sex and gender.

(1) The role of status: does position in an occupational hierarchy in and of itself confer health risks or benefits?

Occupational status, frequently used as one of the core components of the SES construct, includes a highly complex bundle of factors whose impact on health may be mediated through many alternate pathways. At the most obvious level, occupational status confers more or less societal prestige, in addition to defining day-to-day working relationships. Professionals, for example, are generally more highly esteemed in our culture than laborers; executives more than clerical workers. There is anthropologic and biologic evidence to suggest humans, like other primates, are naturally hierarchical, and that relative position, in and of itself, confers health effects. Within the work setting, job status often translates into tangible benefits and hazards as well, including income level and fringe benefits, degree of control over work, level of workplace support to get work accomplished and, typically, lower frequency and degree of exposure to noxious physical environments. Teasing apart these components has been and remains a scientific challenge, especially given the implausibility of experimental or even quasiexperimental data sources: job status is inherently nonrandomly assigned.

Most research on job status has relied either on relatively coarse job classifications, or focused almost exclusively on administrative (i.e., "whitecollar") populations.^{19–25} Across more diverse cohorts, job grade may reflect a host of social, economic, and psychosocial risk factors varying with social class,^{26–28} and the effect of job grade itself may vary within broader occupational classes and settings.²⁹ Among white-collar workers, job grade may be mediated through workplace decisionmaking authority; among blue-collar workers, job grade may capture differences in chemical exposures, physical demands, or work pace. Some factors, such as income and job security, can vary within both groups.

The use of broad occupational classifications

Occupational classifications used in many epidemiological studies (i.e., manual/nonmanual distinctions,³⁰ professional grade,^{20,31} and census job classifications³²) have proven too coarse to capture fine-scale status differences most relevant to employee experiences within occupational settings. More recent work has attempted to understand health effects of status differentials within working classes, and to examine job grade effects among manufacturing and industrial workers, where job grade itself is confounded by (and may also, to some extent, consist of) a range of physical, chemical, and psychosocial stressors, including noise, heat, chemical exposures, a rapid pace of work, job strain, and socioeconomic risk factors outside the occupational setting.

The complexity of "job grade" in the blue-collar setting

The association between job status and health has been difficult to interpret, as job status encompasses many physical and psychosocial aspects of work, and is itself associated with socioeconomic risk factors and health behaviors.²⁶⁻²⁸ With improved methods for measuring work-related stressors (e.g., noise, ergonomic demands), we can now achieve a more refined understanding of job grade effects independent of the physical demands and exposures of work, although the task remains challenging as such exposures are persistently correlated. Recent studies have attempted to disaggregate job grade into its various components³³ including income,³⁴ effortreward imbalance,35 perceived fairness,36 knowledge of job-related risks and behaviors^{37,38} and job strain (decision latitude vs. demands).³⁹⁻⁴¹ Because of this complex exposure mix, most studies have focused on few industries or companies, in which these distinct aspects can be measured and isolated.42,43

In our data set of 15,000 employees across eight U.S. states, we compared incident hypertension among full-time hourly (mainly production, or "blue-collar") and salaried (production supervisors and administrative, largely "white-collar") personnel in a large aluminum manufacturing company. We used propensity stratification to account for strong SES bias in job placement, and to distinguish selection from exposure effects. We found elevated hypertension risks among blue-collar employees, relative to white-collar, which persisted after adjustment for income, education, and age, particularly relevant in this setting where the correlation between job grade and income is weak because total income is largely driven by how much overtime employees opt to work. The blue-collar

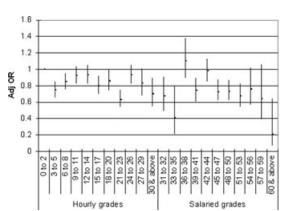


Figure 4. Odds of getting hypertension among full-time hourly and salaried workers by job grade; reference is the lowest category of hourly workers.

effect increased with more stringent case criteria, suggesting greater illness severity or chronicity among blue-collar workers. Higher job grades were significantly protective in a full model, particularly among hourly employees. This is depicted in Figure 4:

Tenure (years on the job) increased hypertension risks among blue-collar workers after adjustment for age, which may indicate the impact of cumulative exposure to physical, chemical, or psychosocial exposures, including heat, noise, and job strain above and beyond a status effect. Differences in chemical or physical exposures (e.g., closer proximity to smelting fumes among laborers than supervisors) may also account, in part, for some job grade effects within blue-collar groups. Lower-grade employees appear to benefit more per increment than workers in higher grades, possibly explaining apparently steeper job grade effects in women, who make up a larger portion of low-grade workers.⁴⁴ This parallels evidence for diminishing marginal health returns for income,45,46 lending some content validation to this singular observation.

Piecing the evidence together, the role of "status" as an explanatory factor in the SES gradient remains consistent with the data, even as behavioral, material, and other explanatory factors are considered. This appears to be true within blue-collar as well as white-collar workforces, and when smaller, rather than coarser gradations are considered. However, neither the strength of the association nor the causal pathway(s) are well established.

(2) Psychosocial job stressors (demand, control, reward): how do we understand and parameterize these constructs, and what do they contribute to explaining the gradient? Working life entails many types of demands—some physical, some mental—and many variants of "control," or means of meeting those demands. As such, one of the greatest challenges in job strain research lies in accurately characterizing the nature of "job strain," or work-related stress, and distinguishing its effects from those of concomitant physical, chemical, and financial stressors.

There is broad evidence linking psychosocial work characteristics to cardiovascular risk.^{25,47,48} Increased risks of hypertension and elevated ambulatory blood pressure have been associated with chronic job strain,^{49,50} low job control,⁴¹ and stressful work conditions including low promotion potential, little participation in decision-making, communication difficulties, unsupportive co-workers and foremen, and overall job dissatisfaction.⁴² Several biological and psychological pathways have been proposed to link psychosocial work conditions to hypertension,⁵¹ including chronic stress, irritation,⁵² suppression of anger and problems,⁵³ and selection by personality type.⁵⁴ To the extent that work is experienced as stressful, it may produce cumulative wear and tear (allostatic load), potentially suppressing immune function over time, increasing general susceptibility, reducing systemic regulation, and increasing broad disease risk⁵⁵ (Seeman et al., in this volume).

In previous decades, most of what was known about "job strain" stemmed from the development and application of the Karasek model of demand-control,56-58 which defined job strain as the bi-dimensional interaction between (1) job demands (usually psychological, rather than physical) and (2) decision latitude allowing the individual to meet those demands, often referred to as control. As was the case for job grade, most early work using the Karasek model included only white-collar populations, and generally found that greater control conferred better health outcomes. Using the demand-control model for their administrative cohort, Whitehall investigators concluded that much of the differential in IHD morbidity and mortality observed between job grades could be explained by control.48 Some more recent crosssectional studies have demonstrated associations between job strain and blood pressure among men in various occupations, effects which appear strongest among lower-SES workers⁵⁹ and most salient when observed during, rather than after, working hours.⁶⁰

Alternatively, the effort-reward imbalance model quantifies the lack of correspondence between jobrelated efforts (both physical and, more importantly, psychological) and rewards received for those efforts (including monetary remuneration, prestige, and career opportunities).⁶¹ A number of investigations, including cross-sectional and longitudinal results, although largely among men, support a link between effort-reward imbalance and cardiovascular outcomes.⁶² There is additional evidence that the effort-reward imbalance model may be most informative in combination with results of the job strain model.⁶³ The models overlap significantly along the control dimension; according to some evidence, persons with lower job control report more effort-reward imbalance.⁶⁴ It is important to note, however, that the models emphasize very different types of control; job strain emphasizes control over task performance, while effort-reward imbalance emphasizes control over "macro-level" issues such as salary and career advancement.65

The nature of job stressors, and, accordingly the critical aspects of control, may differ dramatically by setting. In the manufacturing setting, where job strain has been less explored, high decision latitude for a worker in a rapid-paced manual process can be highly stressful, possibly due to the need to maintain high levels of vigilance at all times. Furthermore, the forms of control valued by the individual workers performing physical (rather than sedentary) labor may be very different; some workers may value having input on how their work is designed or performed, while others (particularly those performing repetitive physically demanding tasks) may more greatly value the option to take a work break when needed. Perhaps not surprisingly, then, recent studies of the job strain model in the aluminum manufacturing sector have produced results that differ dramatically from the Whitehall results. While both demand and control measures predict injury risk,^{66,67} only demand appears to be associated with incident depression,68 and neither demand nor control produces the expected relationship with cardiovascular outcomes, as demonstrated in the Table 1; if anything lower control

Job characteristic	IHD	No IHD	Odds ratio
Demand			
High	91 (3.9)	2264 (96.1)	0.92 (0.69–1.23)
Moderate	147 (5.7)	2412 (94.3)	1.40 (1.08–1.80)
Low	107 (4.2)	2455 (95.8)	1 (ref.)
Control			
Low	107 (3.9)	2639 (96.1)	0.65 (0.50-0.84)
Moderate	116 (4.4)	2547 (95.6)	0.73 (0.56-0.94)
High	122 (5.9)	1947 (94.1)	1 (ref)

 Table 1. Numbers of cases and percent (in parentheses) with and without ischemic heart disease by levels of job demand and control in the Alcoa study

appears protective from ischemic heart disease in these workers.⁶⁸

Part of the discrepancy between these results may be derived from differences in the job strain measurement. Most studies over the past three decades have relied on subjective report of the major elements of strain.48,56,69 Because of concerns about conflation of predictors with outcomes, especially with mental health or other outcomes also measured by self-report,⁷⁰ many have proposed and utilized measures of strain associated with particular jobs or tasks, or external measures, rather than individual reports.⁷¹ These latter job-level strain assessments are often performed by external, presumably more objective, raters, although Kasl and others have argued that this approach introduces other measurement issues and biases.⁴³ Importantly, in the few cases in which subjective and external ratings have both been obtained, as in Whitehall, the two measures correlate poorly (r < 0.2).⁷² Recent analyses of absenteeism suggest that subjective measures add little to external ratings, and the latter has the advantage of representing "environmental" factors more amenable to intervention or modification.⁷² For this reason, external ratings have been relied upon in the previously cited Alcoa studies of job strain.

Finally, specific stressful characteristics of work have merited more involved study. Night shift work and threat avoidance vigilance have been associated with cardiovascular outcomes including hypertension and myocardial infarction.^{73–75} Some physical stressors associated with job tasks (e.g., exposures to noise, vibration, heavy lifting, or chemicals⁷⁶) have shown similar results, although, importantly, distributions of these exposures are tightly correlated, such that their health effects can be difficult to disentangle. One such chronic stress condition, threat avoidance vigilance, may help to explain elevated risks of hypertension co-occurring with injury risk in some manufacturing settings.

In summary, no more consistent picture of the long-debated job strain construct has emerged in the past decade to explain the SES gradient. Although survey-based exposure assessments continue to support the role of perceived strain especially low control—as a risk factor for cardiovascular and psychiatric disease in office workers, the same has not been consistently found among workers doing more physical jobs, in which workdemand may be a more salient risk. It appears likely these constructs themselves will undergo further scrutiny and change as the process of disentangling the relationship between work and the gradient evolves.

(3) Physical and chemical hazards: what do they contribute to the occupational gradient?

The study of occupational hazards and health dates back to the early 18th century, and remains a substantive discipline of research and practice in both clinical and public health.^{77,78} In addition to widespread risks of work-related physical injury, hundreds of acute and chronic work-related disorders have been characterized, and risk profiles associated with exposure to thousands of common toxic materials have been established, many supported through toxicologic experiments in laboratory animals. Despite this body of knowledge, and the broad investment in the United States and other developed countries to control and manage occupational risks, many social epidemiologists have argued that physical hazards contribute minimally to SES-health gradients at the societal level. This perspective stems from the exemplary demonstration of the health and mortality gradients among the administrative white-collar Whitehall I and II populations, wherein physical exposures such as heat, noise, and chemical hazards were negligible, and evidence that social gradients in health begin early in life, precluding any effect of work beyond that of parental occupation (again, status) or residual exposures to chemical exposures carried home on work clothes.

This evidence indicates that at-work exposures to physical agents are not a necessary component to social health gradients-indeed, SES-health gradients clearly can exist in the absence of physical hazardsalthough this is not proof that work-related physical hazards are noncontributory. Moreover, it is widely believed that while workers in an earlier period of industrial development endured tremendous physical hazards, the modern postdevelopment economy has all but eliminated "dangerous" sectors and nearly eliminated residual risks in the industrial sector through modern regulation. In this section, we review the evidence that both presuppositions are false, while we acknowledge that robust quantitative estimates for the contribution of physical work hazards to the social gradient in health are not available.

Exposures to physical environmental conditions at work, including trauma, chemicals, biologic agents, and physical hazards (e.g., heat, noise, radiation), are still widely prevalent in the United States and other OECD countries, and thus may contribute substantively to the social gradient in health. Using the most recent U.S. data available, work in the formal manufacturing, construction, mining, and agriculture sector continue to comprise roughly 25% of the total workforce, or 37.5 million men and women; the majority of them work in non-office capacities that entail some degree of chemical and/or physical hazard, including many lower-level salaried jobs.79 Another 15% of jobs, mostly in health, transportation, and food services, have substantial environmental exposures and among the highest injury rates of any sector. These BLS statistics do not include the far larger fraction of older adults who have moved over from the shrinking sectors such as manufacturing into service jobs or retirement, nor those working full or part time in the informal sector including very dangerous trades such as sex work, drug trafficking, personal, and domestic services, nor the very dangerous exposures incurred during military service. Put altogether, it is reasonable to estimate that upwards of half or more living U.S. adults have been in the past or are currently exposed to one or more potentially injurious physical, chemical, or biologic hazards; for many this is the most salient characteristic of their work.

A related issue is whether the distribution of hazardous exposures sufficiently parallel to the social gradient in health that a significant contribution is plausible. The answer is almost certainly yes. Putting aside for the moment the varying efficacy of presentday controls and regulations to mitigate workplace physical exposures, this proposition of a social gradient in workplace exposures has been assumed so self-evident that empiric proof has not been broadly presented across industries, although indirect evidence and within-industry associations abound. Most jobs in the formal industrial sectors described earlier require a high school education or less, and many (with the possible exception of the relatively small trade-union middle class) are marginally compensated relative to higher-status professions. Although labor market studies have documented that workers in overtly hazardous industries earn a hazard "premium," or proportionately higher wages in exchange for accepting greater risks, although recent reevaluation suggests this may not be as pervasive as generally believed.⁸⁰ To the extent they might impact (inversely) the gradient, such premiums have been largely linked to conspicuous on-the-job death risks and other immediate threats, not long-term health. Moreover, other evidence indicates that stratifying by job type, employees of larger, often multisite firms are better protected and better paid compared to those in more marginal workplaces, so the possibly perverse impact of such premiums on the SEShealth gradient is likely modest at best.^{81,82} Within workplaces, there is also evidence that higher-status workers enjoy better physical environmental conditions, as well as lower physical demands. For example, Figure 5 depicts the relationship between physical demand (1 = sedentary, up to 5 = extreme physical exertion) and job grade (here on a 12-point collapsed scale) within the hourly (bluecollar) workforce at a large aluminum smelting facility (unpublished data).

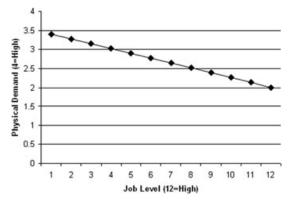


Figure 5. Modeled relationship between physical demand and job grade among male hourly workers at Alcoa.

Similarly, in this data set, dust exposures are also higher among lower-grade hourly employees. Noise exposures, however, distribute differently: higher-grade skilled tradesman (here, grades 22 and above on the full 36-point scale) experience higher noise exposures, on average, than those in lowerstatus jobs (Fig. 6).

As noted, exposures to all of these agents are negligible among the better-educated salaried workforce, which comprises about one third of all company employees.

By far the most salient hazard in the above workplaces is trauma and bodily injury. In 2007, almost 5500 U.S. men and women died from injuries sustained on the job. Another two million (122 per 10,000 workers) suffered injuries serious enough to require medical treatment and restricted or lost work time. These injuries are concentrated in workers performing heavier, more menial lower SES jobs by up to 10-fold in our study population. Previous estimates suggest these injuries cost the economy

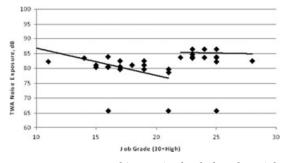


Figure 6. Average ambient noise levels based on job grade among hourly workers at Alcoa.

1-2% of GDP annually.83 While injuries by themselves cannot substantively account for health and mortality gradients, they do contribute significantly to differentials in an individual's ability to work productively into late middle-age and beyond. Indeed, musculo-skeletal disorders are a major cause of permanent work loss, constituting over 25% of the social security disability claims, compared with mental disorders around 30%, circulatory 10%, and nervous system also about 10%.84 More importantly, and inadequately studied, is the potential of these physical injuries and disability to impair health, particularly susceptibility to cardiovascular disease (CVD). One pathway suggested by the literature as a link between injury risk and CVD is hyper-vigilance, which may be highly prevalent among those in dangerous jobs or jobs with potential for catastrophic mistakes, as shown among commercial drivers and air-traffic controllers.85,86

The contribution of workplace chemicals to chronic respiratory disease has been well examined, with studies following two key approaches. First, classical occupational epidemiology prospective cohort studies have followed groups of workers exposed to various well-measured agents (e.g., coal dust, welding fumes) to estimate risks of "excess" chronic lung disease compared to less-exposed, but otherwise comparable, control populations. Extrapolation of risks to the larger population is then derived from the fraction of the population that is exposed, coupled with estimates of exposure intensity. In this manner, for example, risks associated with occupational exposures to hundreds of organic and inorganic chemicals have been established as major causes of asthma.87,88 Likewise, longitudinal studies of lung function in workers exposed to various dusts and fumes have demonstrated excess reductions in airflow and risk of chronic obstructive pulmonary disease (COPD), outcomes normally associated with smoking.⁸⁹⁻⁹¹ Second, the contribution of workplace airborne irritants and toxicants to chronic lung disease has been estimated by parsing risk factors observed in large heterogeneous populations to assess whether occupational exposures-generically defined from medical records, self-report, or questionnairescan explain differences in lung function or disease rates.^{92–94} Gratifyingly, this latter approach, which applies relatively exposure assessment methods in the occupational setting, has been corroborated by prospective results, yielding relatively consistent estimates of approximately 15% of adult asthma cases and 15–20% of the COPD burden due to dusts, fumes, and other physical exposures at work.⁹⁵ The contribution to lung fibrosis has historically been higher but is dropping with control of the most prevalent causal agents, asbestosis, and silica dust.

The contribution of workplace chemicals to the total cancer burden has been estimated by similar study designs. Malignant mesothelioma, an otherwise uncommon cancer, is largely attributed to asbestos exposure among workers in western countries.96 Lung and bladder cancers, much more common, are also closely associated with workplace chemicals, with upward of 20% of cases attributable to established causal agents in the workplace, including asbestos, petroleum combustion products, and arsenic.97 Some leukemias, liver and skin cancers (other than highly lethal melanoma) are less common, but occupational causes are well established.⁷⁸ For many other cancers and tumors, associations with workplace hazards are hypothesized but not yet proved. Overall estimates, using forward and backward approaches-studying cancer outcomes in cohorts of workers exposed to varying doses, or using broad patterns in the larger population to estimate attribution-range from 4% to 5% of the total cancer burden, with little change over the past three decades.^{98,99} The implication of these observations toward explaining the social gradient in health, however, is that most of the attributable cancer burden is heavily concentrated in cancer sites for which low SES figures most prominently as a risk factor, for example, lung, bladder, and liver.

Evidence of a causal association between workplace physical exposures and cardiovascular disease remains less well understood, but, due to the high prevalence of cardiovascular illness in developed countries, may be extremely important, even if the attributable fraction of cases is relatively small.^{4,5} Three nearly ubiquitous physical hazards in the industrial setting-noise, heat, and exertion-have each been linked to ischemic heart disease (IHD). For noise, the presumptive pathway is via elevated blood pressure, although evidence of a direct link remains limited¹⁰⁰ despite elegant physiologic models of adreno-medullary responses to noise in laboratory and animal experiments.¹⁰¹ Heat stress, a potent stimulus for increased cardiovascular demand, has been long recognized as a risk for myocardial infarction (MI) or sudden death in workers with existing heart disease.^{102,103} For this reason, most workers with heart disease and those taking vasoactive drugs for hypertension are excluded from working in hot environments. Because of such screening and self-selection away from such jobs, the impact of hot work on IHD morbidity and mortality has been difficult to assess. For high-exertion activity, the data are mixed; Paffenbarger famously showed that physical activity among longshoreman *reducess* IHD risk,¹⁰⁴ but the role of selection and the "healthy worker effect" in these estimates remains problematic, and heavy physical exertion may, with or without other environmental risks, be harmful.¹⁰⁵

As globalization and other economic transformations increase the proportion of work performed at nontraditional hours, concern for the health consequences of rotating or variable work shifts have intensified, with cardiovascular diseases and its antecedents the area of greatest concern.^{106,107} Methodological problems, most notably endogenous differences between those who do and do not end up working non-day shifts, have complicated study of this issue, but cohort studies in Denmark¹⁰⁸ and Finland¹⁰⁹ have found excess CVD outcomes in men who worked nights, while Kawachi et al. found a risk in female nurses proportional to the number of years the women worked alternative shifts.¹¹⁰ Others have failed to find such excesses, although there is also data linking increased BMI and insulin resistance to shift work,107 rendering this concern highly plausible given present knowledge.

Perhaps more concerning still are certain ubiquitous forms of dust. Evidence from environmental studies of *outdoor* air pollution consistently show that inhaled fine particulates (aerodynamic diameter less than 2.5 µm, or PM_{2.5}) increases risk of respiratory and cardiovascular mortality, with moderately elevated relative risks on the order of 1.2, and attributed annual excess mortality of 60,000 U.S. deaths/year.^{111,112} The data supports the attribution of a significant fraction of cardiovascular deaths to ambient air pollution, and an extensive literature has emerged exploring possible mechanistic pathways, including inflammatory effects in the lung, affecting vascular endothelium via circulating cytokines.¹¹³ These observations raise serious implications about the role of workplace particulates, often present in concentrations orders of magnitude above ambient levels. As such, occupational PM exposures could contribute to increased IHD mortality among workers in heavy industry, transportation, construction, and related fields, masked perhaps by salient healthy worker selection effects. Dusts in these industries have historically been considered a nuisance rather than a risk factor, and cardiovascular effects have been largely ignored among young, relatively healthy working populations. As such, evidence to inform this proposition remains limited in part because workplace measures of dust have historically differed from those used to study ambient air pollution. In summary, while physical hazards in the work environment may prove a major contributor to high rates of cardiovascular illness among lower social class workers, evidence at this time remains more limited than that for respiratory diseases and cancer.

Putting all the information together, poorer workers appear in general to get injured more and experience a wide range of hazardous chemical and other physical exposures. While a secure estimate of the contribution of these factors to the SES gradient is lacking, some disease specific estimates of this effect are emerging, although the contribution to the biggest killer—atherosclerotic cardiovascular disease—remains in need of much further research.

(4) Healthy work organization: is there a "neighborhood" effect in the workplace? And if there is, how might it impact the socioeconomic status gradient?

In recent years, social epidemiologists have reinvigorated investigation into the socioeconomic causes of poor health, and have demonstrated the necessity of disentangling proximal individual risk factors from more distal or "structural" risk factors. Occupational health researchers, in turn, have attempted to estimate the influence of the workplace environment and some of its structural characteristicsvariously conceived of and named "workplace culture," "work organization," and "workplace social capital," for example. Several workplace characteristics have been extensively measured and studied, toward understanding how workplace context, as distinct from workforce composition, contributes to ill health. Results of research point to the importance of work organizational factors to workers' health, and saliently, to the need for appropriate methods for examining and quantifying these separate contributions.

Only a few contructs have thus far been studied in the work context. "Safety culture," a commitment to safety that is expressed in appropriate policies and practices,¹¹⁴ has long been championed by human resources and safety professionals as integral to safe working environments. The construct of "safety culture," however, has not been easily or adequately measured, and in our own preliminary work in the manufacturing sector may prove unrelated to actual safety experience. This aspect of work organization has been more completely studied than in the pressure- and hazard-filled health care setting; in that arena the culture of the organization has been shown to be relevant both for the health/safety of patients and that of its workers. Abundant patient safety research demonstrates that patients well-being depends upon the success of "system," more so than individual-oriented measures.¹¹⁵ Analogously, research in the health care setting, particularly among nurses, points to the contribution of job design and organizational interventions to worker injury and illness outcomes, including needle-stick injuries.116,117

Workplace change situations have also provided a context in which to study the impacts on health and safety on work organization. For example, studies of the changes in culture that occur in anticipation of downsizing provide a window into the effect of broad psychosocial environment on worker health. A 2001 review of the evidence for the health impacts of work organization reported that 90% of 25 downsizing studies reviewed found a negative association with workers' health and safety outcomes.¹¹⁸ Job strain, while usually measured at the individual job level, may also be viewed as a social construct, reflecting qualities of the broader work environment. The manner in which work is organized dictates the demand and control associated with the performance of individual jobs, thus researchers have recently identified the value of measuring demand/control at the aggregate level. By modeling individual's responses to questions about job demand, control, and other exposures in this way, it appears that variation in health outcomes derives from both individual exposure and work unit aggregate measures.^{119–121}

As with job control, demand, and job strain, the effects of most working conditions have been examined at the individual level by comparing predictors such as individuals' working hours or perceived safety climate against individual health outcomes. These constructs can vary importantly across job settings, however; for example, Amick¹²² showed higher strain indices and poorer self-reported health across women employed in health care settings. Many workplace psychological processes likely operate on multiple levels: both the individual-level, and that of the work unit(s), departments, or plants, where presumably at least some fraction of the exposure originates. Newer modeling techniques allow for the estimation of effects at multiple levels (e.g., the individual and the plant).

Each of these studies provides some early clues, but the biggest strides in the past decade have come on the methodologic side. The tools available studying workplace-level factors have been honed by social epidemiologists, under the rubric of multilevel (or hierarchical) analysis and modeling. In occupational health, such models are increasingly being used to distinguish and quantify variability in effects of individual-level predictors from those of the workplace, department, or other organizational unit, with specific attention to potential "crosslevel" interactions between individual factors (e.g., age, sex) and aggregate characteristics (e.g., workplace size, type, region).¹²¹ Multilevel analysis, applied to occupational cohorts, accounts for clustering among workers in the same unit, rather than treating each subject (worker) as an independent observation, thus better accounting for autocorrelation in occupational data sets. Importantly, most occupational data sets are inherently hierarchical, with workers nested within organizational units; as such, multilevel modeling offers advantages for improved validity and efficiency over previous strategies, such as including binary indicators for each organizational unit into a single-level multivariate regression model, which accounts for a mean effect by plant, but does not allow for worker "fit" although interactions between individual and workplace characteristics.

Soderfeldt *et al.*¹²¹ first applied multilevel modeling to study the demand–control model in an occupational cohort, and concluded that the job variables in the demand–control model drew a significant portion of their variation from organizational level factors as opposed to specific features of the jobs or the people doing them. Later, results from the prospective Finnish Public Sector Study of over 25,000 public sector employees found, using intra-class correlations, that 21% and 15% of the variability in job strain and job demand, respectively, derived from the work unit level. The study reported that higher-strain jobs (assessed by selfreport or aggregate measure) conferred increased risk of disability, relative to low-strain jobs.¹²³ In a separate Finnish cohort of 2969 hospital personnel, both individual- and unit-level decision latitude (i.e., control) and "organizational justice," as measured by a survey instrument probing fairness, predicted certified sickness absence.¹¹⁹ Multilevel methods should, where feasible-these methods require minimum numbers of "clusters" or work units-be applied to studies of occupational exposures.

Putting all this together, there is already ample reason to suspect that (1) work organization matters for health and (2) tools are available to unravel the connections. What remains, however, is to demonstrate that lower SES workers are systematically exposed to "worse" cultures, and/or disproportionately impacted by them, although on the face of it, it seems reasonable to assume that work organization may prove an important contributor to the SES gradient.

(5) The new employment "contract" and worker health: what are the implications for the gradient?

Despite the work-related health and safety risks elucidated earlier, there is compelling evidence that work may confer important health advantages as well. Many studies have documented that stably employed adults experience better health and survival than those who are less stably or never employed. Recent evidence of the health benefits of work are documented in Finnish studies and strong evidence is provided by many occupational cohorts, in which those who retire early-even from hazardous occupations -have consistently worse outcomes than those who continue working, with large increases in adverse lifestyle, accidental death, and homicide.^{124,125} For this reason, short-term workers are generally excluded from cohort studies, as their inclusion may mask detrimental effects of work exposures. Much of this disparity has been attributed to selection effects, that is, that men and women with stable habits and lifestyle are more likely to be stably employed, and also to enjoy better health. Alternatively, there is some suggestion, including studies of nonvoluntary changes in workforce participation (e.g., eastern European males after the fall of communism)¹²⁶ that work itself may confer positive health benefits, though it has been difficult to establish what aspects of stable work (e.g., regular activity, sense of responsibility, self-efficacy, social support) may confer benefits beyond those attributable to income and material well-being.

Better understanding of these non-economic health aspects of work becomes even more salient as the older order of work is rapidly disappearing. Historically, most workers spent long stretches of their careers working for a single employer, under a covenant in which employers were generally expected to provide continued employment and security, in return for allegiance and continued efforts, for the common benefit of both parties-with or without the intercession of a union. This venerated arrangement has begun to erode as a newer one is developing, wherein workers sell portions of their labor effort in the marketplace. While the erosion of the old "contract" may confer benefits to some employees (e.g., flexibility, mobility, competitive pricing for relatively scarce skills), it likely also confers new hazards (e.g., economic insecurity, loss of social support networks, transfer of responsibility for retirement planning from employers to individuals). Unemployment, once only a risk to employees with less marketable or over-supplied skills, or workers in economically marginal sectors, has increasingly become a reality for many, if not most employees; many will experience periods of relative underemployment as market conditions fluctuate. While for many workers, especially in lower-status occupations, chronic risk of unemployment or underemployment is not new, the diffusion of un- and under-employment into the more highly educated workers has increased the overall prevalence of related risks, and created a compelling urgency to understand the health consequences of these economic and societal transformations.

While the health risks associated with unemployment per se have been studied (reviewed later), the impact of these other aspects have only recently been seriously examined as a contribution to social gradients in health. Later we review what has been learned so far, recognizing that the importance of these issues are likely to grow as the economy continues to transform.

Unemployment

Macroeconomic studies of the societal effect of increases and decreases in employment rates as they occur through business cycles have yielded, perhaps surprisingly, little clear indication that growth, with its attendant increases in the rate of employment, results in short-term gains in mortality or self-rated health, except psychologically; more surprisingly, modern studies of recession have shown either minimal or positive impacts on overall mortality, cardiovascular and accidental death rates, infant mortality, and self-rated health; only mental health and suicide appear "procyclical."127-129 Stated reasons include demonstrated decreases in adverse health behaviors during recessions, as well as less overall driving, risky-behaviors and the like. What is less clear from these reports is upon which segments of the population this "benefit" of unemployment is conferred, all the more vexing in light of multiple studies showing that unemployed individuals do poorly. Because of the important methodological issues raised by persistent selection effects and SES confounding associated with employment insecurity, the best studies of the health effects caused by unemployment are assessments of larger plant closings, wherein individual selection is minimized. Kasl¹³⁰ found worse self-reported health and increased adverse health behaviors after job loss, and similar observations were reported in more recent studies^{131,132} although longer-term consequences have been less well explored. There are many longitudinal studies of men and women who have become unemployed for various reasons, although these studies are often confounded by other factors, including income and education, which may themselves augur poor health outcomes. Jin et al.133 reviewed the health impacts of unemployment, and documented excess mortality among individuals who had ever been unemployed, compared with those who had not. Swedish twin registry studies, comparing mortality 10 and 24 years later among young adults unemployed in 1973, found broad mortality excess compared both to siblings and to the general population; these differences were not explained by prior smoking, drinking, or chronic disease status.^{134,135} The U.S. Health and Retirement Study found shortand long-term deleterious effects of involuntary job loss on physical function and cardiovascular disease among older workers, after accounting for smoking and income.^{136,137} Census studies in the United Kingdom and Denmark have also documented that cancer rates and mortality were higher among those previously unemployed, after controlling for tobacco use.¹³⁸ Mathers and Schofield,¹³⁹ likewise, showed the effects of unemployment to be greater than those of economic loss alone, suggesting that stable work may confer benefits beyond income and material well-being. Efforts to explain these negative health effects have focused on loss of social support¹⁴⁰ and psychological stressors including lowered self-esteem and depression.141,142 Some physical effects may be mediated through inflammatory pathways, as suggested by recent data from the CARDIA study, which showed elevated C-reactive protein (CRP) levels 15 years after baseline among men who had been unemployed 5 years previously, after adjustment for health behaviors and SES.143

Work insecurity

Several recent studies of perceived stress have suggested that prolonged fear of losing a job may be more damaging than job loss itself,^{144,145} particularly for mental health and self-reported health status.146,147 More worrisomely, Mattiasson148 showed direct effects of job-loss threat on cardiovascular risk factors, including rises in blood pressure and serum cholesterol, although the associated longerterm health outcomes and mortality gradients remain unestablished. Likewise, the elevated cardiovascular risk factors reported¹⁴⁹ among Swedish factory workers, and British civil servants in Whitehall II,¹⁴⁷ whose units were threatened with closure do not establish longer-term health outcomes. These studies focused primarily on the impact of fear of job loss occurring in the context of feared lay-offs and firm closings. Whether similar impacts will develop in workers for whom the old notion of job security itself no longer exists in the newer global economy remains an open question, but an urgent one.

Nontraditional, nonstandard, and contingent work

In economic recessions, these forms of employment obviously become more widespread, although as noted earlier the health consequences of recession itself remain disputed. Deeper structural changes in the nature of work, brought about by globalization, technological change, and economic restructuring may prove more intractable and, potentially, more consequential over the long term. Since the end of World War II most working adults in developed countries have been working under a "contract"-formal or otherwise-conferring relatively predictable and generally rising, wages and benefits in exchange for continued work and corporate fidelity. Work has traditionally been conducted at the employer's place of business, during regular business hours. These traditional characteristics of work have shifted in recent decades, with the rise of telecommuting, enabled by modern technologies, and changing family structures demanding greater flexibility in work hours and location. In 1995, the BLS estimated that 30% of all U.S. workers are on-call or day laborers, involuntarily part-time, self-employed, work for a contract labor firm, or work from home. A follow-up survey is not available, although evidence points to an increase in these nontraditional, nonstandard, contingent, or precarious forms of work, the health effects of which are now under scrutiny. Two thorough reviews^{118,150} emphasize potential health risks from these forms of work, which include material and income loss, greater work stress, enhanced work-family conflict due to irregular scheduling and high work demands; poorer regulation of the physical work environment, and adverse social effects including isolation.

The work categories described earlier, while sharing some common features, are very different, and it is probably inappropriate to lump self-employment by choice with involuntary part-time work, or combine forms of full-time employment with on-call or temporary work. Indeed, some efforts have been made to distinguish the health effects of these varying work structures,¹⁵⁰ particularly as many nontraditional job structures exist by choice, by men and women needing flexible hours to meet familycare demands, because these structures may confer opportunities for economic and personal growth not available in the traditional labor market. As such, health studies of "precarious employment" suffer from heterogeneity in the populations included, and the applicability of the results to particular industries past or future must be interpreted with caution. In addition, data on the SES distribution of precariously employed workers is limited, although Hipple¹⁵¹ has shown nontraditional work structures to be more prevalent among women, minorities, and poorer groups. It has also been assumed that such jobs within each broad

social class may be less desirable in the aggregate, and thus more likely held by *relatively* disadvantaged workers,¹¹⁸ which in turn makes distinguishing employment *consequences* from unmeasured selection effects problematic.^{152,153}

Many studies attempt to relate irregular employment conditions with various health outcomes and most, but not all, report adverse effects on injury rates, self-reported mental and physical health, or poorer health and safety performance.¹¹⁸ One consistent finding is that workers in nontraditional settings experience more injuries, and more severe injury (e.g., fatality).^{154–158} While the contribution of precarious employment to the health gradient is not immediately transparent, it underscores the likelihood that these job conditions may be consistently worse than those in traditional work settings, even after adjusting for the nature of the work. A few of these studies substantiate these claims using self-reported depression and fatigue,¹⁵⁹ musculoskeletal symptoms,160 and physical and psychological health,^{161–163} although objective and long-term measures are lacking. Finally, while the demonstration of adverse health and safety conditions and work culture associated with these jobs must be viewed more as process than outcome measure,^{159,164} the importance of these factors to deteriorating health and mortality suggests that such links will become manifest, perhaps justifying the use of the term "precarious," preferred by many investigators, to describe these job-types.¹¹⁸

(6) Sex, gender, and occupation: how have changes in the roles of women in workplaces affected the health of women?

The composition of the workforce is rapidly changing. However, despite greater numbers of women in traditionally "male" roles, gender stratification in job assignment persists overall.^{165,166} Accordingly, men and women continue to differ—on average at least—in job-related chemical exposures,¹⁶⁷ ergonomic demands,¹⁶⁸ accidents,¹⁶⁹ and psychosocial stressors.¹⁷⁰

The resulting workforce composition poses new challenges and offers new opportunities for occupational health research and control, as a wider range of individuals performing any specific job offers better opportunities to separate job-related effects from individual health risk characteristics. There remains, however, relatively little research on the effect of "male" work roles on women's health, and some studies show greater health differentials associated with blue-collar (relative to white-collar) work for women than men.^{16,171–173} These results have been surprising as, in most prior adult cohorts, women's risk of chronic illness, including cardiovascular illness, diabetes, and COPD, has been notably lower than men's until relatively late in life. Thus, the observation that illness should actually be higher for blue-collar women than men suggests either very strong gender-specific selection effects, or potent workplace health risk factors to which women may be particularly susceptible: chemical, physical, or social.

Even as sanctioned sex-stratification in work is diminishing, women and men continue to live and work within a gendered society; evidence indicates that women and men "choose" blue-collar work for very different reasons. In our data set of 15,000 aluminum manufacturing employees across eight U.S. states, we found a greater likelihood of blue-collar work among lower-educated, African-American, and single parent women than men.¹⁶ Women in hourly jobs tended to be from lower SES background, have greater financial need (e.g., single mothers), and were more likely to hold lower-grade (e.g., lower-skilled) hourly jobs, than were hourly men. These observations suggest that most of the hourly women had not planned to be so, but rather were drawn towards manufacturing jobs out of financial necessity. More of the hourly men in our data set, comparatively, were skilled individuals in higher-rank hourly jobs, or were hired by the company later in their careers.

To control as best possible for this differential selection, we used propensity score models to examine the effect of pre-hire sociodemographic characteristics and personal life factors (e.g., marital status, parity) on men's and women's employment decisions. After adjustment using these propensity scores, hourly work conferred greater risks of hypertension, a broad marker of cardiovascular risk, among women than men. As hinted earlier, the hourly women not only fared worse compared to their salaried counterparts but actually had *higher* rates of hypertension by age 40 than the hourly men! This observation proved stronger when we used more stringent case criteria. Tenure, a surrogate for cumulative exposure, conferred greater risks among women likely to be hourly, suggesting greater susceptibility to workplace risk factors with lower SES. In addition, higher job grades reduced risks more consistently among men than women. Finally, men in blue-collar jobs with higher physical demands displayed the protective effect of lower BMI, which was not observed in women, while women showed stronger negative health effects with physical strain.

Beyond this elevated chronic disease risk, hourly work appears to pose additional risks to women's health. Controlling for work tasks themselves, women get injured 40-60% more often, with greater injury severity.¹⁷⁴ Likewise, rates of absenteeism, and time to return to work after illness, are consistently worse in blue-collar women than their male counterparts.^{175,176} It remains unknown what portion of these elevated risks among women may be attributable to differences in sex-linked biological susceptibility, or to culturally derived gender differences, such as gendered selection into job roles, workplace harassment, or personal life circumstances. This critical distinction between sex (i.e., biological differences by chromosomal complement, including reproductive organs and hormonal composition) and gender (i.e., self-representation, socially derived behaviors and roles, and response by social institutions) is needed to accurately disaggregate work-related health risks for both sexes, and for designing effective workplace interventions.^{177–179}

Possible explanations for this array of adverse outcomes in the women are sex-linked differential susceptibility to workplace contaminants, or gender differences in the manufacturing environment experience. The same worksite and task may be experienced differently by women and men; for example, differences in anthropomorphic measurements and work equipment designed for male bodies can increase women's task burden.^{180–182} Women are still a tiny fraction of the blue-collar working population, and are more likely to experience harassment, discrimination,^{183,184} and low job control.170,185 Finally, non-workplace factors, including family roles and domestic responsibilities, may influence susceptibility to employment-related health effects; women's greater average time on household tasks¹⁸⁶ may affect fatigue, nonoccupational stress,^{187,188} response to workplace stressors, and return to work following illness or injury.^{189–191} One intriguing issue is the impact of status on women's health in this new frontier. Women still comprise

a small proportion of blue-collar workers, but are overly represented in low-grade jobs,¹⁹² and earn less than men in the same job category.¹⁶⁶ This gendered stratification is important because low job grade has been linked to heart disease,²¹ hypertension,²⁴ and injury.^{193,194} As much job grade research has focused on largely male, "white-collar" populations (e.g., Ref. 20), it remains unclear whether job status operates similarly among women in bluecollar jobs. Finally, "job grade" is associated with many chemical, physical, and psychosocial stressors, thus its "causal components" for health remain unknown, and may vary by age, gender, and setting.²⁹

Together, these observations suggest that, currently, blue-collar work settings may be disproportionately unhealthy for women. It is less clear, however, why this is so. The negative effect of blue-collar work on women's health, combined with higher risks associated with job tenure among women predicted to be hourly, together raise important questions about the nature of manufacturing work which may differently affect men and women. Why employment duration increases risks more among women than men is uncertain, but likely indicates different responses to cumulative physical, chemical, or psychosocial exposures at work. This effect appears particularly strong among those with greater social disadvantage at the time of employment, consistent with the life-course appreciation of the origins of the SES gradient now emerging.

Summary

Weighing the current evidence, it would appear that occupation-or at least some of its component elements-contribute causally to the health gradients evident in all developed societies. The evidence that hypertension risk drops with even small incremental rises in job grade, defined administratively among employees of a single large corporation in which socio-demographic and other health risks are controlled for, adds weight to the theory that status is, all by itself, a contributor across a broad array of occupations. The role of job stress, on the other hand, may be more nuanced; while job strain has been shown to contribute to cardiovascular disease and mortality in white-collar occupations, the contribution to those in more physical jobs is less obvious, although strain does appear to increase risk for injury and depression. For physical risk

factors—more prevalent in the modern workplace than many might suppose—the scorecard is getting clearer: chemicals contribute measurably to the gradients of chronic lung disease and cancer, and physical work is strongly associated with injury and musculo-skeletal disability. The role each of these factors plays in the cardiovascular disease gradient remains poorly understood, although there is more than a little reason for concern.

Concern about these and other health risks is especially great for women as they migrate into traditional male occupations: the delayed onset of cardiovascular disease in women, long attributed to hormonal influences, appears to have vanished among these women, who also get injured more often, miss more work, and recover less quickly from major illness and injury than their male counterparts. Unknown is whether these adverse effects owe to sex, that is biology, or gender, that is, social roles, an area worthy of urgent inquiry if preventive strategies are to be devised. Also desperate for further research is the role of work organization or context, which appears based on early evidence to modify, for good or ill, other work factors. Understanding these relationships may be particularly important as the prevailing culture of work itself changes in our society, from one in which a typical job occurred by daylight, at a defined place of employment, with a written or unwritten expectation that the relationship would, in general, continue over the long term, perhaps a lifetime. As the global economy shifts, the health implications of nonstandard work, changing shifts and work insecurity to SES gradients in health may come to become the most important questions of all, yet another area where our interest at present greatly exceeds our knowledge.

Conflict of interest

The authors declare no conflicts of interest.

References

- Reid, D.D. *et al.* 1974. Cardiorespiratory disease and diabetes among middle aged civil servants *Lancet.* 7856: 469–473.
- Mackenbach, J.P. *et al.* 2008. Socioeconomic Inequalities in Health in 22 European Countries. *N. Engl. J. Med.* 358: 2468–2483.
- Cullen, M. *et al.* 2006. Use of Medical Insurance Claims Data for Occupational Health Research. *J. Occup. Environ. Med.* 48: 1054–1061.

- MacDonald, L.A. *et al.* 2009. Occupation as socioeconomic status or environmental exposures: a survey of practice among population-based cardiovascular studies in the United States. *Am. J. Epidemiol.* DOI: 10.1093/aje/kwp082.
- Cullen, M.R. 2009. The search for preventable causes of cardiovascular disease: whither work? *Am. J. Epidemiol.* DOI: 10.1093/aje/kwp078.
- Bartley, M. 1988. Unemployment and health: selection or causation – a false antithesis? *Sociol. Health Illness* 10: 41–67.
- Mensah, F.K. & J. Hobcraft. 2008. Childhood deprivation, health and development: associations with adult health in the 1958 and 1970 British prospective birth cohort studies. *J. Epidemiol. Community Health* 62: 599– 606.
- 8. Rabinowitz, P.M. *et al.* 2006. Trends in the prevalence of hearing loss among young adults entering an industrial workforce 1985–2004. *Ear Hear.* **27:** 369–375.
- Checkoway, H., N. Pearce & D. Kriebel. 2004. Research methods in occupational epidemiology. *Monographs in Epidemiology and Statistics*. Oxford University Press. New York.
- 10. Chandola, T. *et al.* 2003. Health selection in the Whitehall II study, UK. *Soc. Sci. Med.* **56:** 2059–2072.
- 11. Claussen, B. *et al.* 2005. Intragenerational mobility and mortality in Oslo: social selection versus social causation. *Soc. Sci. Med.* **61**: 2513–2520.
- Heponiemi, T. *et al.* 2007. Relationship between unemployment and health among health care professionals: Health selection of health effect? *J. Psychosom. Res.* 63: 425–431.
- Kivimaki, M. *et al.* 2006. Work stress, weight gain and weight loss: evidence for bidirectional effects of job strain on body mass index in the Whitehall II study. *Int. J. Obesity* 30: 982–987.
- Leventhal, T. & J. Brooks-Gunn. 2003. Moving to opportunity: an experimental study of neighborhood effects on mental health. *Am. J. Public Health.* 93: 1576– 1582.
- Lidfeldt, J. *et al.* 2007. A prospective study of childhood and adult socioeconomic status and incidence of type 2 diabetes in women. *Am. J. Epidemiol.* 165: 882–888.
- 16. Clougherty, J.E. et al. Unpublished analysis.
- Clougherty, J.E. *et al.* 2008. Workplace status and risk of hypertension among hourly and salaried aluminum manufacturing employees. *Soc. Sci. Med.* 68: 304– 313.
- 18. Clougherty, J.E. *et al.* 2007. Synergistic effects of trafficrelated air pollution and exposure to violence on urban

asthma etiology. *Environ. Health Perspect.* **115:** 1140–1146.

- Aboa-Eboule, C. *et al.* 2007. Job strain and risk of acute recurrent coronary heart disease events. *JAMA* 298: 1652–1660.
- Marmot, M. & G. Smith. 1991. Health inequalities among British civil servants: the Whitehall II study. *Lancet* 337: 1387–1394.
- Marmot, M. *et al.* 1997. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 350: 235– 239.
- 22. Moller, J. *et al.* 2005. Work related stressful life events and the risk of myocardial infarction. Case-control and case-crossover analyses within the Stockholm heart epidemiology program (SHEEP). *J. Environ. Community Health* **59:** 23–30.
- Kivimaki, M. *et al.* 2007. Hypertension is not the link between job strain and coronary heart disease in the Whitehall II study. *Am. J. Hypertens.* 20: 1146–1153.
- Calhoun, H., H. Hemingway & N. Poulter. 1998. Socioeconomic status and blood pressure: an overview analysis. J. Human Hypertens. 12: 91–110.
- 25. Barbini, N. *et al.* 2005. Analysis of arterial hypertension and work in the epidemiologic study "Aging, Health, and Work." *Epidemiol. Prev.* **29:** 160–165.
- Erikson, R. 2006. Social class assignment and mortality in Sweden. Soc. Sci. Med. 62: 2151–2160.
- Kaplan, G. & J. Keil. 1993. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation.* 88: 1973–1998.
- Warren, J. *et al.* 2004. Job characteristics as mediators in SES-health relationships. *Soc. Sci. Med.* 59: 1367–1378.
- Sparacino, J. et al. 1982. Blood pressure of municipal employees: effects of job status and worksite. Percept. Motor Skills. 55: 563–578.
- Macleod, J. *et al.* 2005. Is subjective social status a more important determinant of health than objective social status? Evidence from a prospective observational study of Scottish men. *Soc. Sci. Med.* 61: 1916–1929.
- Schaufelberger, M. & A. Rosengren. 2007. Heart failure in different occupational classes in Sweden. *Eur. Heart J.* 28: 212–218.
- 32. Stevens, G. & J.H. Cho. 1985. Socioeconomic indexes and the new 1980 census occupational classification scheme. *Soc. Sci. Res.* **14:** 142–168.
- MacDonald, L. *et al.* 2001. Covariation between workplace physical and psychosocial stressors: evidence and implications for occupational health research and prevention. *Ergonomics* 44: 696–718.

- Lecluyse, A. 2007. Income-related health inequality in Belgium: a longitudinal perspective. *Eur. J. Health Econ.* 8: 237–243.
- Merritt, M. *et al.* 2004. Low educational attainment, John Henryism, and cardiovascular reactivity to and recovery from personally relevant stress. *Psychosom. Med.* 66: 49–55.
- DeVogli, R. *et al.* 2007. Unfairness and health: evidence from the Whitehall II study. *J. Epidemiol. Community Health* 61: 513–518.
- Nourjah, P. *et al.* 1994. Knowledge of risk factors and risk behaviors related to coronary heart disease among blue and white collar males. *J. Public Health Policy* 15: 443–459.
- Niknian, M. *et al.* 1991. Use of population-based data to assess risk factor profiles of blue and white collar workers. *J. Occup. Med.* 33: 29–36.
- Blumenthal, J., E. Thyrum & W. Siegel. 1995. Contribution of job strain, job status, and marital status to laboratory and ambulatory blood pressure in patients with mild hypertension. *J. Psychosom. Res.* 39: 133–144.
- 40. Kivimaki, M. *et al.* 2006. Why is evidence on job strain and coronary heart disease mixed? An illustration of measurement challenges in the Whitehall II Study. *Psychosom. Med.* **68**: 398–401.
- Steptoe, A. & G. Willemsen. 2004. The influence of low job control on ambulatory blood pressure and perceived stress over the working day in man and women from the Whitehall II cohort. *J. Hypertens.* 22: 915–920.
- 42. Matthews, K. *et al.* 1987. Stressful work conditions and diastolic blood pressure among blue collar factory workers. *Am. J. Epidemiol.* **126**: 280–291.
- 43. Kasl, S.V. 1981. The challenge of studying the disease effects of stressful work conditions. *Am. J. Public Health* **71:** 682–684.
- 44. Quinn, M.M. *et al.* 2007. Social disparities in the burden of occupational exposures: results of a cross-sectional study. *AJIM.* **50**: 861–875.
- 45. Mackenbach, J. *et al.* 2004. The shape of the relationship between income and self-assessed health: an international study. *Int. J. Epidemiol.* DOI: 10.1093/ije/dyh338.
- Kawachi, I. 2000. Income inequality and health. In Social Epidemiology. Berkman, L. & I. Kawachi, Eds. Oxford University Press. New York.
- Chandola, T., E. Brunner & M. Marmot. 2006. Chronic stress at work and the metabolic syndrome: prospective study. *BMJ (Clin. Res.)* DOI: 10.1136/bmj.38693.435301.80.
- 48. Kuper, H. & M. Marmot. 2003. Job strain, job demands, decision latitude, and risk of coronary heart disease

within the Whitehall II study. *J. Environ. Community Health* **57:** 147–153.

- Markovitz, J. *et al.* 2004. Increases in job strain are associated with incident hypertension in the CARDIA study. *Ann. Behav. Med.* 28: 4–9.
- Landsbergis, P.A. *et al.* 2003. Life-Course exposure to job strain and ambulatory blood pressure in Men. *Am. J. Epidemiol.* 157: 998–1006.
- Chandola, T. *et al.* 2008. Work and coronary heart disease: what are the mechanisms? *Eur. Heart J.* DOI:10.1093/eurheart/ehm584.
- 52. Folkow, B. 1981. Physiological aspects of primary hypertension. *Physiol. Rev.* **62:** 347–504.
- Cottington, E.M. *et al.* 1986. Occupational stress, suppressed anger, and hypertension. *Psychosom. Med.* 48: 249–260.
- Esler, M., S. Julius & A. Zweifler. 1977. Mild high-renin essential hypertension: neurogenic human hypertension? *N. Engl. J. Med.* 296: 405–411.
- McEwen, B.S. & T.E. Seeman. 1999. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann. N.Y. Acad. Sci.* 896: 30–47.
- Karasek, R. *et al.* 1981. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *Am. J. Public Health* 71: 694– 705.
- Karasek, R. & T. Theorell. 2000. The demand-controlsupport model and CVD. In *The Workplace and Cardio*vascular Disease. Vol. 15. Schnall, P.L. et al. Eds.: 78–83. Hanley & Belfus, Inc. Philadelphia.
- Karasek, R. *et al.* 1998. The Job Content Questionnaire (JCQ): an instrument for internationally comparative assessments of psycho-social job characteristics. *J. Occup. Health Psychol.* 3: 322–355.
- Landsbergis, P.A. *et al.* 2003. Lower socioeconomic status among men in relation to the association between job strain and blood pressure. *Scand. J. Work Environ. Health* 29: 206–215.
- 60. Landsbergis, P.A. *et al.* 2008. Work conditions and masked (hidden) hypertension – insights into the global epidemic of hypertension. *Scand. J. Work Environ. Health* (Suppl): 41–51.
- Siegrist, J. 1996. Adverse health effects of higheffort/low-reward conditions. J. Occup. Health Psychol. 1: 27–41.
- Belkic, K. *et al.* 2000. Psychosocial factors: review of the empirical data among men. In *The Workplace and Cardiovascular Disease*, Vol. 15. Schnall, P.L. *et al.* Eds.: 24–46. Hanley & Belfus, Inc. Philadelphia.

- 63. Peter, R. *et al.* 2002. Psychosocial work environment and myocardial infarction: improving risk estimation by combining two complementary job stress models in the SHEEP study. *J. Epidemiol. Community Health* **56**: 294–300.
- 64. Bosma, H. *et al.* 1998. Two alternative job stress models and the risk of coronary heart disease. *Am. J. Public Health* **88**: 68–74.
- 65. Belkic, K.L. *et al.* 2004. Is job strain a major source of cardiovascular disease risk? *Scan. J. Work Environ. Health* **30**: 85–128.
- Kim, C. *et al.* 2009. Job strain and the risk for occupational injury in small-to-medium-sized manufacturing enterprises: a prospective study of 1,209 Korean employees. *AJIM* 52: 322–330.
- 67. Cantley, L. *et al.* submitted for publication. An externally rated job demand survey to assess physical and psychological job stress in relation to injury risk in aluminum manufacturing jobs. *Occup. Environ. Med.*
- 68. Iennaco, J. *et al.* submitted for publication. Effects of externally rated job demand and control on depression diagnosis claims in an industrial cohort. *AJE*.
- Reed, D.M. *et al.* 1989. Occupational strain and the incidence of coronary heart disease. *Am. J. Epidemiol.* 129: 495–502.
- Landsbergis, P.A. & T. Theorell. 2000. Self-report questionnaires. In *The Workplace and Cardiovascular Disease*, Vol. 15. Schnall, P.L. *et al.* Eds.: 163–171. Hanley & Belfus Inc. Philadelphia.
- Greiner, B.A. & N. Krause. 2000. Expert-observer assessment of job characteristics. In *The Workplace and Cardiovascular Disease*. Vol. 15. Schnall, P.L. *et al.* Eds.: 175–183. Hanley & Belfus Inc. Philadelphia.
- 72. Rehkopf, D.H., H. Kuper & M.G. Marmot. under review. Discrepancy between objective and subjective measures of job stress and sickness absence.
- 73. Winkleby, M.A. *et al.* 1988. Excess risk of sickness and disease in bus drivers: a review and synthesis of epidemiological studies. *Int. J. Epidemiol.* **17:** 255–262.
- Boggild, H. & A. Knutsson. 1999. Shift work, risk factors and cardiovascular disease [review]. Scand. J. Work Environ. Health 25: 85–99.
- Belkic, K., R. Emdad & T. Theorell. 1998. Occupational profile and cardiac risk: possible mechanisms and implications for professional drivers. *Int. J. Occup. Environ. Health* 11: 37–57.
- Fine, L.J. 2000. Chemical and physical factors. In *The* Workplace and Cardiovascular Disease, Vol. 15. Schnall, P.L. et al. Eds.: 18–24. Hanley & Belfus Inc. Philadelphia.
- 77. Ramazzini, B. 1700. De Morbis Artificum.

- Rosenstock, L. et al. 2005. Textbook of Clinical Occupational and Environmental Medicine. Elsevier Ltd., London/W.B. Saunders. Philadelphia.
- 79. Bureau of Labor Statistics. 2006.
- Leigh, J.P. 1991. No evidence of compensating wages for occupational fatalities. *Indust. Relat.* 30: 382–395.
- Suruda, A. & D.F. Wallace. 1996. Fatal work-related injuries in the US chemical industry 1984–89. *Int. Arch. Occup. Environ. Health* 68: 425–428.
- Haas, J. & R. Buchan. 1995. Occupational health and safety hazards in Colorado small industry. *Appl. Occup. Environ. Hyg.* 10: 151–155.
- Institute of Medicine. 2001. Musculoskeletal Disorders and the Workplace: Low Back and Upper Extremities. National Academy Press. Washington, DC.
- 84. Social Security Administration. 2008.
- Belkic, K. *et al.* 1994. Mechanisms of cardiac risk among professional drivers. *Scand J. Work Environ. Health* 20: 73–86.
- Cobb, S. & R.M. Rose. 1973. Hypertension, peptic ulcer, and diabetes in air traffic controllers. *JAMA* 224: 489– 492.
- Chan-Yeung, M. & J.-L. Malo. 1999. Tables of major inducers of occupational airways disease. In *Asthma in the Workplace*. Bernstein, I.L. *et al.* Eds. Marcel Dekker. New York.
- Van Kampen, V., R. Merget & X. Baur. 2000. Occupational airway sensitizers: an overview of the respective literature. *Am. J. Indust. Med.* 38: 164–218.
- Seixas, N.S. *et al.* 1993. Longitudinal and cross-sectional analyses of coal mine dust and pulmonary function in new miners. *Br. J. Indust. Med.* 50: 929–937.
- Nakadate, T. *et al.* 1998. Change in obstructive pulmonary function as a result of cumulative exposure to welding fumes as determined by magnetopneumonography in Japanese arc welders. *Occup. Environ. Med.* 55: 673–677.
- 91. Coggon, D. & A.N. Taylor. 1998. Coal mining and chronic obstructive pulmonary disease: a review of the evidence. *Thorax* **53**: 398–407.
- Kauffmann, F. *et al.* 1982. Occupational exposure and 12 year spirometric changes among Paris area workers. *Br. J. Indust. Med.* 39: 221–232.
- 93. Kryzyzanowski, M., W. Jedrychowski & M. Wysocki. 1986. Factors associated with the change in ventilatory function and the development of chronic obstructive pulmonary disease in a 13-year follow-up of the Cracow study. *Am. Rev. Resp. Dis.* **134:** 1101–1019.
- 94. Xu, X. *et al.* 1992. Exposure-response relationships between occupational exposures and chronic respiratory

illness: a community based study. Am. Rev. Resp. Dis. 146: 413–418.

- American Thoracic Society. 2003. American Thoracic Society statement: occupational contribution to the burden of airway disease. *Am. J. Respir. Crit. Care Med.* 167: 787–797.
- Bianchi, C. & T. Bianchi. 2007. Malignant mesothelioma: global incidence and relationship with asbestos. *Indust. Health* 45: 379–387.
- Barone-Adesi, F., L. Richiardi & F. Merletti. 2005. Population attributable risk for occupational cancer in Italy. *Int. J. Occup. Environ. Health* 11: 23–31.
- Rushton, L., S. Hutchings & T. Brown. 2008. The burden of cancer at work: estimation as the first step to prevention. *Occup. Environ. Med.* 65: 789–800.
- 99. Doll, R. & R. Peto. 1981. *The Causes of Cancer*. Oxford University Press. Oxford.
- 100. Kristal-Boneh, E. *et al.* 1995. Acute and chronic effects of noise exposure on blood pressure and heart rate among industrial employees: the Cordis study. *Arch. Environ. Health* **50**: 298–304.
- 101. Ortiz, G.A. *et al.* 1974. Modification of epinephrine, norepinephrine, blood lipid fractions and the cardiovascular system produced by noise in an industrial medium. *Horm. Res.* 5: 57–64.
- Redmond, C.K. *et al.* 1979. Mortality of steelworkers employed in hot jobs. *J. Environ. Pathol. Toxicol.* 2: 75– 96.
- Wild, P. *et al.* 1995. Mortality from cardiovascular diseases among potash workers exposed to heat. *Epidemiology* 6: 243–247.
- 104. Paffenbarger, R.S.J. *et al.* 1970. Work activity of longshoremen as related to death from coronary heart disease and stroke. *New. Engl. J. Med.* 282: 1109–1114.
- 105. Kales, S.N. *et al.* 2007. Emergency duties and deaths from heart disease among firefighters in the United States. *New. Engl. J. Med.* **356:** 1207–1215.
- 106. Boggild, H. & A. Knutsson. 1999. Shift work, risk factors and cardiovascular disease. *Scan. J. Work Environ. Health* 25: 85–99.
- Knuttson, A. 2003. Health disorders of shift workers. Occup. Med. 53: 103–108.
- 108. Boggild, H., P. Saudicani & H.O. Hein. 1999. Shift work, social class and ischaemic heart disease in middle aged and elderly men; a 22 year follow-up in the Copenhagen male study. Occup. Environ. Med. 56: 45–64.
- 109. Tenkanen, L., T. Sjoblom & R. Kalimo. 1997. Shift work, occupation and coronary heart disease over six years of follow-up in the Helsinki Heart Study. *Scand. Work Environ. Health* 23: 257–265.

- 110. Kawachi, I., G.A. Colditz & M.J. Stampfer. 1995. Prospective study of shift work and risk of coronary heart disease in women. *Circulation* 92: 3178–3182.
- 111. Miller, G. & E. Chen. 2007. Unfavorable socioeconomic conditions in early life presage expression of proinflammatory phenotype in adolescence. *Psychosom. Med.* 69: 402–409.
- 112. Pope, C.A., M. Ezzati & D.W. Dockery. 2009. Fineparticulate air pollution and life expectancy in the United States. *New. Eng. J. Med.* **360**: 376–386.
- 113. Brook, R.D. *et al.* 2004. Air pollution and cardiovascular disease. A statement for healthcare professionals from the expert panel on population and prevention science of the American Heart Association. *Circulation* 109: 2655–2671.
- 114. Guldenmund, F.W. 2000. The nature of safety culture: a review of theory and research. *Safety Sci.* **34**: 215–257.
- Clark, G. 2002. Organizational culture and safety: an interdependent relationship. *Aust. Health Rev.* 25: 181– 189.
- Clarke, S.P., D.M. Sloane & L.H. Aiken. 2002. Needlestick injuries to nurses, in context. *LDI Issue Brief* 8: 1–4.
- 117. Stone, P.W. & R.R. Gershon. 2006. Nurse work environments and occupational safety in intensive care units. *Policy Polit. Nurs. Pract.* 7: 240–247.
- 118. Quinlan, M., C. Mayhew & P. Bohle. 2001. The global expansion of precarious employment, work disorganization, and the consequences for occupational health: a review of the recent literature. *Int. J. Health Serv.* 31: 335–341.
- 119. Eloivainio, M. *et al.* 2004. Job decision latitude, organizational justice and health: multivariate covariance structure analysis. *Soc. Sci. Med.* **58**: 1659–1669.
- Leyland, A.H. & Groenewegen. 2003. Multilevel modeling and public health policy. *Scand. J. Public Health* 31: 267–274.
- 121. Soderfeldt, B. *et al.* 1997. Does organization matter? A multilevel analysis of the demand-control model applied to human services. *Soc. Sci. Med.* 44: 527–534.
- Amick, B. 1998. Relationship of job strain and iso-strain to health status in a cohort of women in the U.S. Scand. *J. Work. Environ Health* 24: 54–61.
- 123. Laine, S. *et al.* 2008. Job strain as a predictor of disability pension: the Finnish Public Sector Study. *J. Epidemiol. Community Health* 63: 24–30.
- Kivimaki, M. *et al.* 2003. Temporary employment and risk of overall and cause-specific mortality. *Am. J. Epidemiol.* 158: 663–668.
- 125. Virtanen, P. et al. 2008. Employment trajectory as

determinant of change in health-related lifestyle: the prospective HeSSup study. *Eur. J. Public Health* **18:** 504–508.

- 126. Kopp, M.S. *et al.* 2007. Chronic stress and social changes: socioeconomic determination of chronic stress. *Ann. N.Y. Acad. Sci.* 1113: 325–338.
- 127. Ruhm, C.J. 2003. Good times make you sick. *J. Health Econ.* **22:** 637–658.
- 128. Granados, J.A.T. 2005. Increasing mortality during the expansions of the US economy 1900–96. *Int. J. Epidemiol.* **34**: 1194–1202.
- 129. Ruhm, C.J. 2005. Healthy living in hard times. *J. Health Econ.* **24**: 341–363.
- Kasl, S.V., S. Gore & S. Cobb. 1975. The experience of losing a job: reported changes in health, symptoms and illness behavior. *Psychosom. Med.* 37: 106–122.
- 131. Keefe, V. *et al.* 2002. Serious health events following involuntary job loss in New Zealand meat processing workers. *Int. J. Epidemiol.* **31**: 1155–1161.
- 132. Hamilton, V.L. *et al.* 1990. Hard times and vulnerable people: initial effects of plant closings on autoworkers' mental health. *J. Health Soc. Behav.* 31: 123–140.
- 133. Jin, R.L., C.P. Shah & T.J. Svoboda. 1995. The impact of unemployment on health: a review of the evidence. *Can. Med. Assoc. J.* 153: 529–540.
- 134. Nylén, L., M. Voss & B. Floderus. 2001. Mortality among women and men relative to unemployment, part time work, overtime work, and extra work: a study based on data from the Swedish twin registry. *Occup. Environ. Med.* 58: 52–57.
- 135. Voss, M. *et al.* 2004. Unemployment and early causespecific mortality: a study based on the Swedish twin registry. *Am. J. Public Health* **94:** 2155–2161.
- Gallo, W.T. *et al.* 2005. Health effects of involuntary job loss among older workers: findings from the health and retirement survey. *J. Geront. Ser. B: Psych. Sci. Soc. Sci.* 55: S131–S140.
- 137. Gallo, W.T. *et al.* 2006. The impact of late career job loss on myocardial infarction and stroke: a 10 year follow up using the health and retirement survey. *Occup. Environ. Med.* **63**: 683–687.
- 138. Lynge, E. 1997. Unemployment and cancer: a literature review. *IARC Sci. Publ.* **138**: 343–351.
- Mathers, C.D. & D.J. Schofield. 1998. The health consequences of unemployment: the evidence. *Med. J. Aust.* 168: 178–182.
- 140. Roberts, H. *et al.* 1997. Unemployment and health: the quality of social support among residents in the Trent region of England. *J. Epidemiol. Community Health* 51: 41–45.

- 141. Kessler, R.C., J.B. Turner & J.S. House. 1988. Effects of unemployment on health in a community survey: main, modifying, and mediating effects. *J. Social Issues* 44: 69–85.
- 142. Feather, N.T. & G.E. O'Brien. 1986. A longitudinal study of the effects of employment and unemployment on school-leavers. *J. Occup. Psych.* **59**: 121–144.
- 143. Janicki-Deverts, D. *et al.* 2008. History of unemployment predicts future elevations in C-reactive protein among male participants in CARDIA. *Ann. Behav. Med.* DOI:10.1007/S12160-008-9056-5.
- Aronsson, G. 1999. Contingent workers and health and safety. Work Employ. Soc. 13: 439–460.
- 145. Heaney, C.A., B.A. Israel & J.S. House. 1994. Chronic job insecurity among automobile workers: effects on job satisfaction and health. *Soc. Sci. Med.* 38: 1431– 1437.
- 146. De Witte, H. 1999. Job insecurity and psychological well being: review of the literature and exploration of some unresolved issues. *Eur. J. Work. Org. Psych.* 8: 155–177.
- 147. Ferrie, J. *et al.* 1998. Health effects of anticipation of job change and non-employment: longitudinal data from the Whitehall II study. *BMJ* **311**: 1264–1269.
- 148. Mattiasson, I. *et al.* 1990. Threat of unemployment and cardiovascular risk factors: longitudinal study of quality of sleep and serum cholesterol in men threatened with redundancy. *BMJ* **301:** 461–466.
- Arnetz, B. 1991. Neuroendocrine and immunologic effects of unemployment and job insecurity. *Psychother. Psychosom.* 55: 76–80.
- 150. Price, R.H. & S.A. Burgard. 2008. The new employment contract and worker health in the Unites States. In *Making Americans Healthier. Social and Economic Policy as Health Policy*, Vol. 201–207. Schoeni, R.F. *et al.* Eds. Russle Sage Press. New York.
- Hipple, S. 2001. Contingent work in the late 1990's. Monthly Labor. Rev. 124: 3–27.
- 152. Benach, J. *et al.* 2002. The consequences of flexible work for health. *J. Epidemiol. Community Health* **56**: 405–406.
- 153. Benach, J. *et al.* 2000. The health damaging potential of new types of flexible employment: a challenge to public health researchers. *Am. J. Public Health* **90:** 1316–1317.
- 154. Blank, V. 1995. Hidden accident rates and patterns in the Swedish mining industry due to the involvement of contract workers. *Safety Sci.* **21**: 23–35.
- 155. Mayhew, C. & M. Quinlan. 1999. The effects of outsourcing on occupational health and safety: a comparative study of factory-based and outworkers in the Australian clothing industry. *Int. J. Health Serv.* 29: 83– 107.

- Meyer, J. & C. Muntaner. 1999. Injuries in home health care workers: an analysis of occupational morbidity from a state compensation database. *Am. J. Indust. Med.* 35: 295–301.
- Morris, J. 1999. Injury experience of temporary workers in a manufacturing setting: factors that increase vulnerability. *Am. Assoc. Occup. Health Nurses J.* 47: 470–478.
- Silverstein, B. 1998. Claims incidence of work-related disorders of the upper extremities: Washington state 1987 through 1995. *Am. J. Public Health* 88: 1827– 1833.
- Aronsson, G. & S. Goransson. 1999. Permanent employees but not in a preferred occupation: psychological and medical aspects. *J. Occup. Health Psychol.* 4: 152–163.
- Johansson, J. 1995. Psychosocial work factors, physical work load and associated musculoskeletal symptoms among home care workers. *Scand. J. Psychol.* 36: 113– 129.
- 161. Benach, J. *et al.* 2004. Types of emplyment and health in the European Union: changes from 1995–2000. *Eur. J. Public Health* 14: 314–321.
- Dooley, D., R. Catalano & G. Wilson. 1994. Depression and unemployment: panel findings from the epidemiologic catchment area study. *Am. J. Community Psychol.* 22: 745–765.
- 163. Friedland, D.S. & R.H. Price. 2003. Underemployment: consequences for the health and well-being of workers. *Am. J. Community Psych.* **32:** 33–45.
- 164. Kalleberg, A.L., B.F. Reskin & K. Hudson. 2000. Bad jobs in America: standard and nonstandard employment relations and job quality in the United States. *Am. Sociol. Rev.* 65: 256–278.
- 165. Alexanderson, K. & P. Ostlin. 2001. Work and ill-health among men and women in Sweden. In *Worklife and Health in Sweden*. S., Marklund, Ed. National Institute for Working Life. Stockholm.
- 166. United States Department of Labor. 2002. http://www.dol.gov.
- London, L. *et al.* 2002. Pesticide usage and health consequences for women in developing countries: out of sight, out of mind? *Int. J. Occup. Environ. Health* 8: 46–59.
- Silverstein, B.A., L.J. Fine & T.J. Armstrong. 1986. Hand wrist cumulative trauma disorders in industry. *Br. J. Ind. Med.* 43: 779–784.
- Salminen, S. *et al.* 1992. Risk factors for women in serious occupational accidents. *Occup. Health Safety* (*Australia, New Zealand*). 8: 341–347.

- Hall, E.M. 1989. Gender, work control, and stress: a theoretical discussion and an empirical test. *Int. J. Health Serv.* 19: 725–745.
- LaCroix, A. 1994. Psychosocial factors and risk of coronary heart disease in women: an epidemiologic perspective. *Fertil. Steril.* 62: 1335–1395.
- Hall, E., J. Johnson & T. Tsou. 1993. Women, occupation, and risk of cardiovascular morbidity and mortality. *Occup. Med.* 8: 709–719.
- 173. Baigi, A. *et al.* 2002. Cardiovascular mortality focusing on socio-economic influence: the low-risk population of Halland compared to the population of Sweden as a whole. *Public Health* 116: 285–288.
- 174. Pollack, K.S. *et al.* 2007. The association of body mass index and acute traumatic workplace injury in hourly manufacturing workers. *AJE* **166**: 204–211.
- 175. Hill, J.J. et al. Unpublished analysis.
- 176. Hill, J.J. *et al.* 2008. The relationship between lost work time and duration of absence spells. Proposal for a payroll driven measure of absenteeism. *J. Occup. Environ. Med.* 50: 840–851.
- 177. Krieger, N. 2003. Genders, sexes, and health: what are the differences, and why does it matter? *Int. J. Epidemiol.* 32: 652–657.
- 178. Messing, K. & J.M. Stellman. 2006. Sex, gender, and women's occupational health: the importance of considering mechanism. *Environ. Res.* **101**: 146–162.
- 179. Wizemann, T.M. & M.L. Pardue. 2001. *Exploring the Biological Contributions to Human Health: Does Sex Matter*? National Academy Press. Washington, DC.
- Courville, J., N. Vezina & K. Messing. 1991. Comparison of the work activity of two mechanics: a woman and a man. *Int. J. Ind. Ergon.* 7: 163–174.
- Punnett, L. & U. Bergqvist. 1999. Musculoskeletal disorder in visual display unit work: gender and work demands. Occup. Med. State. Art. Rev. 14: 113–124.
- 182. Stevenson, J.M. *et al.* 1996. Selection test fairness and the incremental lifting machine. *Appl. Ergon.* **27:** 45–52.

- 183. Gutek, B.A. 2001. Women and paid work. *Psychol. Women Q.* 25: 379–393.
- 184. Arcand, R. *et al.* 2000. Work environment and health (environnement de travail et santé). In *Enquete sociale et de sante 1998 (Social and Health Survey 1998)*: 525–570. Institute de la statistique du Quebec. Quebec.
- 185. Bourbonnais, R. *et al.* 2000. Environnement psychosocial du travail (The psychosocial environment of work). In *Enquete sociale et de sante*. Daveluy, C., R. Audet & F. Lapointe, Eds.: 571–583. Institut de la statistique. Quebec.
- 186. Blau, F., M. Ferber & A. Winkler. 2002. *The Economics of Women, Men, and Work*. Prentice Hall. Upper Saddle River, NY.
- Bergqvist, U. *et al.* 1995. Musculoskeletal disorders among visual display terminal workers: individual, ergonomic and work organizational factors. *Ergonomics*. 38: 763–776.
- Brisson, C. *et al.* 1999. Effect of family responsibilities and job strain on ambulatory blood pressure among white-collar women. *Psychosom. Med.* 61: 205– 213.
- Feeney, A. *et al.* 1998. Socioeconomic and sex differentials in reason for sickness absence from the Whitehall II study. *Occup. Environ. Med.* 55: 91–98.
- Katz, J.N. *et al.* 1998. Prevalence and predictors of longterm work disability due to carpal tunnel syndrome. *Am. J. Ind. Med.* 33: 543–550.
- 191. Islam, S.S. *et al.* 2001. Gender differences in workrelated injury/illness: analysis of workers' compensation claims. *Am. J. Ind. Med.* **39:** 84–91.
- 192. Statistics Canada. 2001. Labour force 15 years and over by detailed occupation.
- 193. Houtman, I. *et al.* 1994. Psychosocial stressors at work and musculoskeletal problems. *Scand. J. Work Environ. Health* 20: 139–145.
- 194. Wilkins, K. & S. Mackenzie. 2007. Work injuries. *Health Report* **18**: 25–42.