

A prospective study of job strain and coronary heart disease in US women

Sunmin Lee,^{a,c} Graham Colditz,^{b,c,d} Lisa Berkman^{a,e} and Ichiro Kawachi^{a,c,e}

Background Previous studies of job strain and coronary heart disease (CHD) in men have established job strain as a predictor of CHD risk. Despite the wealth of convincing evidence in men for an association between job strain and CHD, data in women remain sparse.

Methods We prospectively evaluated the relation between job strain and CHD risk in the Nurses' Health Study. In this analysis, we followed a sample of 35 038 US female nurses aged 46–71 years, who completed questions about job strain in 1992 and who were free of diagnosed CHD, stroke, and cancer at baseline. The main outcome measure was the incidence of CHD occurring between baseline (1 June 1992) and 31 May 1996.

Results During 4 years of follow-up, we documented 146 incident cases of CHD (108 non-fatal cases of myocardial infarction and 38 CHD deaths). No evidence was found for a relationship between job strain and risk of CHD. In multivariate analyses controlling for age, smoking, alcohol intake, body mass index, history of hypertension, diabetes mellitus, and other covariates, women in high strain jobs did not have an increased risk of CHD (relative risk [RR] = 0.71, 95% CI: 0.42–1.19) compared with women in low strain jobs. Neither women in passive jobs (RR = 1.08, 95% CI: 0.69–1.68) nor those in active jobs (RR = 0.91, 95% CI: 0.54–1.53) had an increased risk of CHD.

Conclusions Job strain was not related to an increase in the incidence of CHD in the present cohort of nurses.

Keywords Job strain, coronary heart disease, prospective study, work organization, women

Accepted 17 January 2002

More US women than ever before are in paid employment. According to data from the US Bureau of Labor Statistics, 61% of women over age 20 are in paid employment.¹ Exposure to adverse job conditions and other occupational factors are therefore increasingly important considerations in the aetiology of coronary heart disease (CHD) in women. Among psychosocial risk factors for CHD, studies in men have established that a particular form of job stress, job strain, is predictive of CHD risk. The job strain model, developed by Karasek, posits that a

combination of high psychological demands and low control increases the risk of CHD.²

With few exceptions,^{3–7} the majority of more than a dozen case-control and cohort studies in men have established an association between job strain and cardiovascular disease, across a variety of occupations and settings.^{8–23} This association seems to be mediated in part through a more adverse profile of cardiovascular risk factors, such as smoking,²⁴ higher body mass index (BMI),²⁴ risk of hypertension,²⁵ and negative emotions such as hostility and depression.²⁶ However, the association between job strain and cardiovascular disease in men has also been found to be independent of these risk factors.

Despite the wealth of evidence in men for an association between job strain and CHD, data in women are sparse and not totally consistent. To date, five cohort studies have examined the association in women. Of these, one study found no significant association,²⁷ one found an association only with job control,¹⁷ and the remaining three reported a statistically significant association between job strain and CHD.^{14–16} The purpose of our study was therefore to examine prospectively the association between job strain and incidence of CHD in a cohort

^a Departments of Health and Social Behavior, Harvard School of Public Health, Boston, MA, USA.

^b Department of Epidemiology, Harvard School of Public Health, Boston, MA, USA.

^c Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA.

^d Harvard Center for Cancer Prevention, Boston, MA, USA.

^e Harvard Center for Society and Health, Boston, MA, USA.

Correspondence: Ichiro Kawachi, Department of Health and Social Behavior, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115, USA. E-mail: ichiro.kawachi@channing.harvard.edu

of middle-aged and older female registered nurses. We hypothesized women in high strain jobs or passive jobs would be at increased risk for CHD compared to women in low strain jobs or active jobs.

Methods

Study population

The study took place within the Nurses' Health Study, which is an ongoing cohort of US female registered nurses. The Nurses' Health Study was established in 1976, when 121 701 female registered nurses aged 30–55 years completed a mailed questionnaire providing information about risk factors for cardiovascular disease, cancer, and other major health conditions. Since then, follow-up questionnaires have been mailed to the cohort every 2 years to update information on exposures and the occurrence of major illnesses.

Assessment of job strain

Psychological demands, job control, and work-related social support were assessed in 1992 by the Karasek Job Content Questionnaire. The Karasek demands/control model of job strain categorizes workers into four distinct kinds of psychosocial work experience, based on the interaction between the level of demands and the level of control in the job. The four categories are referred to as: passive jobs (low job demand and low job control), low strain jobs (low job demand and high job control), high strain jobs (high job demand and low job control), and active jobs (high job demand and high job control). The job demands scale is based on the sum of five items that measure the level of psychological demands at work (excessive work, conflicting demands, insufficient time to work, fast work pace, and working hard). The job control scale is based on the sum of two sub-scales: *skill discretion* as measured by six items (continual learning of new things on the job, ability to develop new skills, job requiring skill, task variety, work not repetitious, and job requiring creativity) and *decision authority* as measured by three items (freedom to make decisions, choice about how to perform work, and having a lot of say on the job). We also administered the work related social support scale, made up of two sub-scales: support from co-workers (they take a personal interest in me, are friendly, helpful in getting the job done, and are competent in doing work) and support from supervisors (concerned about the welfare of employees, attentive, helpful in getting the job done, successful in getting people to work together). For each item, the respondents could choose from one of four responses; strongly disagree, disagree, agree, and strongly agree. A 'not applicable' response was added to the supervisor support questions.

We dichotomized the demand and control scales (high versus low), based on the median score in the cohort. Women were assigned to high strain jobs if they reported high demand and low control, active jobs if they reported high demand and high control, passive jobs if they reported low demand and low control, and low strain jobs if they reported low demand and high control.

Ascertainment of CHD

The end point for this study comprised incident cases of non-fatal myocardial infarction (MI) and fatal CHD that occurred

after the return of the 1992 questionnaire but before 1 June 1996. Each woman who reported a new case of non-fatal MI was asked for permission to review her medical records. Cases were confirmed if they met the diagnostic criteria of the World Health Organization (i.e. symptoms plus either cardiac enzyme level elevations or diagnostic ECG changes).²⁸ Medical records were reviewed by a physician who was blinded to exposure status. An MI was defined as probable if medical records were not available but hospitalization was required and confirmatory information was obtained by interview or letter. The present analyses included both definite and probable cases. Ninety-two per cent of the CHD cases included in our analyses were 'definite' by these criteria.

Most deaths were reported by next of kin or postal authorities. The ascertainment of death also included systematic searches of the National Death Index to identify deaths among participants who did not respond during each questionnaire cycle. More than 98% of deaths in the cohort are estimated to have been identified by this method.²⁹ If death appeared to be from vascular causes, written permission was requested from the next of kin (subject to the regulations of vital records offices) to review medical records. Fatal CHD was defined as fatal MI confirmed by hospital records or at autopsy or as CHD recorded on the death certificate, if this was the underlying and most probable cause given and there was previous evidence of CHD. In no instance were the causes on the death certificate accepted without corroboration. Total CHD was defined as non-fatal MI plus fatal CHD.

Of the 121 701 women in the original cohort in 1976, we excluded participants who had died ($n = 2888$) or who had a diagnosis of CHD ($n = 7728$), stroke ($n = 1087$), or cancer ($n = 13\ 068$) (except non-melanoma skin cancer) prior to the beginning of follow-up (1 June 1992). The study population was therefore free of diagnosed CHD, stroke, and cancer (except non-melanoma skin cancer) at the beginning of follow-up. Women with these major illnesses were excluded in order to minimize the possibility of self-selection into different job conditions prior to the assessment of job strain. An additional 11 322 women were excluded due to loss to follow-up between 1976 and 1992. The cumulative loss to follow-up during the 16-year period leading up to the start of this analysis (1976–1992) was therefore 9.3%. Questions related to job strain were asked on the last page (p.6) of the 1992 questionnaire, which was mailed just once to the entire cohort. The response rate to this single mailing was 78%, i.e. 10 466 women did not provide information on job strain. Although we mail up to five times to non-respondents in any given questionnaire cycle, the job strain items were dropped from repeat mailings in order to minimize respondent burden. Finally, we excluded 38 515 women who reported that they were no longer in paid work in 1992. In all 1589 women were excluded in the second cycle only due to loss to follow-up or non-response in 1994. The study sample therefore consisted of 35 038 working women who provided information on job conditions. Women who were excluded either because of loss to follow-up or missing information on job strain were generally similar to the women included in the present study with the following exceptions: those excluded had a slightly higher smoking rate (16.6% versus 14.6%). They were also less likely to be current users of postmenopausal hormones (32.3% versus 36.0%), and somewhat less

likely to be in the highest quintile of vitamin E intake (15.4% versus 18.4%) as well as regular aspirin use (12.2% versus 14.4%). They were also less likely to have attained a college degree or higher educational qualifications (32.0% versus 35.5%).

Data analysis

The major focus of the data analyses was the relationship between job strain and incidence of CHD, controlling other known risk factors for CHD. Job strain was assessed just once, at baseline in 1992. Questions on all other major coronary risk factors, such as personal history of hypertension, hypercholesterolaemia, diabetes, physical activity, smoking, cholesterol intake, and post-menopausal hormone use were asked in 1992 and updated in 1994 according to the information provided by the participants on the biennial questionnaire.

In all models, the job strain measure was treated as a categorical variable with four job strain categories. The relative risk (RR) was defined as the CHD incidence rate among women in one of three categories of work (passive, high strain, and active jobs) divided by the corresponding rate among women who had low job strain (the reference category). Relative risks were adjusted for age, categorized in 5-year groups, and 95% CI were calculated. We used pooled logistic regression to approximate a Cox proportional hazards model. Pooled logistic regression has been shown to approximate Cox models if the intervals between follow-up questionnaires are short and the probability of an event within an interval is small.³⁰ The following coronary risk factors were adjusted in our multivariable analyses: smoking (never, past, current 1–14 cig/day, current 14–25 cig/day, current 25+ cig/day); alcohol intake (0, 0.1–4.9 g/day [equivalent to one-third to one-half drink per day]: one drink was considered a can of beer [12.8 g] or a glass of wine [11.0 g], or a standard drink of spirits [14.0 g]; 5.0–14.9 g/day [about one drink]; 15.0+ g/day [more than one drink per day]); BMI (kg/m², in quintiles); history of hypertension, diabetes mellitus, or hypercholesterolaemia; menopausal status; current use of post-menopausal hormones; average aspirin use (<1, 1–6, and ≥7 tablets/wk); past use of oral contraceptives; quintiles of saturated fat intake; quintiles of vitamin E intake; quintiles of physical activity. Recreational physical activity was assessed from the responses to questions about the frequency of engagement in eight common activities (walking, jogging, running, lap swimming, bicycling, rowing, aerobics, and racket sports). The score is measured in metabolic equivalent hours (MET-h) per week. One MET-h is equivalent to the energy expenditure during one hour of rest. For example, walking at an average pace for one hour is estimated to consume about 3.0 MET units, while jogging or bicycling is estimated to consume about 7.0 MET units. In this analysis, physical activity was divided into quintiles based on MET units. We also adjusted for parental history of MI before age of 60 years; education (registered nurse, bachelor's degree, graduate degree); marital status (currently married, divorced, widowed); and husband's education (high school graduate, bachelor's degree, graduate degree, missing information). Husband's education was used as an additional measure of socioeconomic position, given the restricted range in other measures of socioeconomic status (i.e. all women were registered nurses with a similar degree of educational attainment).

We also examined the separate associations between job demand, job control, and workplace social support and CHD. We divided job demand and job control into tertiles (tertile cut-points for demands: <28.0, 28.1–33, >33.0; for control: <61.0, 61.1–69, >69.0). The workplace social support scale was dichotomized due to the narrower range of scores (<24.0 versus ≥24.0).

All analyses were conducted with the Statistical Analysis System (SAS) program.

Results

Table 1 shows the age-standardized distribution of risk factors for CHD and other characteristics according to job strain categories. In our study, 20.6% of women were in the low strain, 32.1% in passive, 21.5% in active, and 25.8% in high strain category. Women in high strain jobs were the youngest; women in passive jobs were the oldest. Women in high strain jobs were more likely to report a personal history of hypertension and hypercholesterolaemia than were those in other categories. They were more likely to smoke, tended to exercise less than women in low strain jobs, and consumed more saturated fat. However, they also reported slightly higher use of post-menopausal hormones. Women in active jobs were more likely to smoke but were also more likely to engage in physical activity. Women in passive jobs were more likely to be in part-time work than were women in other job categories. Women in low strain jobs and active jobs had higher levels of education than other groups. Women in the active job category included a high proportion of nurses in administrative jobs, while women in high strain jobs included the highest percentage of nurses in operating room (OR) or inpatient care. These differences were controlled for in examining the association of job strain and risk of CHD.

In all 146 incident cases of CHD (including 108 non-fatal MI and 38 CHD deaths) occurred during 4 years of follow-up.

Compared to women in low strain jobs, the age-adjusted RR of total incident CHD was 0.80 (95% CI: 0.48–1.34) for women in high strain jobs, 1.16 (95% CI: 0.75–1.81) for women in passive jobs, and 0.98 (95% CI: 0.58–1.65) for women in active jobs (Table 2). Adjusting for other risk factors for CHD did not alter these estimates appreciably. Iso-strain jobs (high demand—low control—low workplace social support) were similarly not associated with increased risk of CHD.

When we examined the various dimensions of work environment separately, we found no significant associations between job demands, job control, or social support and CHD (Table 3). About one-fifth of women in the cohort reported working in occupations other than nursing in 1992. We therefore examined the association between job conditions and CHD just within nurses (84.3% of the whole sample). Compared with nurses in low strain jobs, nurses in high strain jobs had a multivariate relative risk of CHD of 0.66 (95% CI: 0.38–1.14). We also examined the association between job strain and CHD within full time workers (67.6% of the whole sample). Full time workers in high strain jobs had a multivariate relative risk of CHD of 0.86 (95% CI: 0.45–1.64) compared with full time workers in low strain jobs. As a marker of stress within the home environment, we also stratified the analyses according to caregiving duties outside of work (i.e. caring for children and

Table 1 Mean ages and age-standardized distribution of cardiovascular risk factors and other characteristics according to job strain

Characteristics	Low strain	Passive	Active	High strain
N	7224	11 260	7519	9035
(%)	(20.6)	(32.1)	(21.5)	(25.8)
Mean age (years)	55.2	56.3	54.4	53.8
Hypertension (%)	26.2	26.4	25.5	27.5
Diabetes mellitus (%)	3.5	3.9	3.6	3.6
Hypercholesterolaemia (%)	39.1	39.3	38.4	40.3
Smoking (%)				
Never	44.7	45.0	43.3	44.1
Past	41.8	41.4	40.3	40.5
Current	13.5	13.7	16.4	15.3
Current use of postmenopausal hormones (%)	35.9	34.5	36.6	37.5
Past use of oral contraceptives (%)	62.3	59.6	62.6	60.2
Parental history of MI^a before age 60 years (%)	13.4	13.8	14.2	13.9
Alcohol intake (g/day)	5.3	4.9	5.2	4.6
Highest quintile of exercise >40 MET^b h (%)	20.0	18.1	20.9	17.7
Highest quintile of vitamin E intake (%)	18.7	18.5	17.2	18.8
Highest quintile of saturated fat intake (%)	17.3	18.8	18.5	19.6
Mean body mass index (kg/m²)	26.3	26.1	26.0	26.0
Aspirin use, ≥7+ tablets /wk (%)	14.1	15.2	14.1	14.2
Marital status (%)				
Currently married	81.3	81.8	81.3	81.0
Divorced or separated	11.4	9.5	12.5	12.5
Widowed	7.3	8.7	6.5	6.5
Education (%)				
RN	53.2	72.5	53.4	72.9
Bachelor's degree	26.0	20.0	25.6	20.0
Graduate degree	20.8	7.4	21.1	7.0
Husband education (%)				
High school graduate	33.6	39.2	33.6	38.2
Bachelor's degree	25.5	24.2	24.2	25.2
Graduate degree	23.3	19.6	22.5	17.8
Missing	17.6	17.6	19.8	18.9
Employment status (%)				
Full-time nurse	54.3	39.3	69.14	55.4
Part-time nurse	21.7	39.2	15.5	35.0
Full-time other	13.3	7.5	12.6	5.2
Part-time other	10.7	14.0	2.8	4.5
Nursing type				
Administrative	13.3	5.4	21.2	6.4
OR ^c or inpatient	7.9	12.6	16.6	30.8
Outpatient	13.0	11.5	9.7	10.0
Others	44.4	50.9	38.8	44.7
Former nurses not currently active	21.4	19.6	13.7	8.1

^a Myocardial infarction.

^b Metabolic equivalents.

^c Operating room.

sick relatives). In neither stratum was job strain associated with risk of CHD. Among women free of caregiving burdens outside of work, those with high strain jobs had a relative risk of 0.75 (95% CI: 0.32–1.76) compared with women in low strain jobs. Among women providing care outside of work, the RR was 0.69 (95% CI: 0.35–1.33).

Discussion

In this 4-year prospective study we found no association between job strain (measured by the Karasek Job Content Questionnaire) and incidence of CHD among women. The results of the present study are not consistent with the majority

Table 2 Relative risk (RR) of coronary heart disease (CHD)^a by job strain

	Low strain	Passive	Active	High strain
Total CHD				
Cases	30	61	27	28
Age-adjusted RR	1.00	1.16 (0.75–1.81)	0.98 (0.58–1.65)	0.80 (0.48–1.34)
Multivariate RR ^b	1.00	1.08 (0.69–1.68)	0.91 (0.54–1.53)	0.71 (0.42–1.19)
Non-fatal myocardial infarction				
Cases	23	49	17	19
Age-adjusted RR	1.00	1.21 (0.74–1.99)	0.81 (0.43–1.52)	0.71 (0.39–1.31)
Multivariate RR ^b	1.00	1.12 (0.67–1.84)	0.75 (0.40–1.42)	0.63 (0.34–1.17)
Fatal CHD				
Cases	7	12	10	9
Age-adjusted RR	1.00	1.01 (0.40–2.57)	1.51 (0.57–3.98)	1.09 (0.40–2.92)

^a Coronary heart disease.

^b Adjusted for age in 5-year intervals; follow-up period (1992–1994, 1994–1996); smoking (never, past, current 1–14, current 14–25, current 25+ cig/day); alcohol intake (0, 0.1–4.9 g/day, 5.0–14.9 g/day, 15.0+ g/day; body mass index (in quintiles); history of hypertension, diabetes mellitus, and hypercholesterolaemia; menopausal status; current use of postmenopausal hormones; average aspirin use (<1, 1–6, and ≥7 tablets/wk); past use of oral contraceptives; quintiles of saturated fat intake; quintiles of vitamin E intake; quintiles of physical activity; parental history of myocardial infarction before age of 60 years; education (registered nurse, bachelor's degree, graduate degree); marital status (currently married, divorced, widowed); husband's education (high school graduate, bachelor's degree, graduate degree, missing information). There were too few cases of fatal CHD to carry out multivariate analyses.

95% CI are shown in parentheses.

Table 3 Relative risk (RR) of coronary heart disease by demand, control, and work related social support

	Cases	Age-adjusted RR	Multivariate RR ^a
Demand	Low	51	1.00
	Intermediate	59	1.35 (0.93–1.97)
	High	36	0.85 (0.55–1.32)
Control	High	49	1.00
	Intermediate	46	0.85 (0.57–1.28)
	Low	51	1.06 (0.72–1.58)
Social support	High	54	1.00
	Low	73	1.28 (0.90–1.83)

^a See footnote to Table 2.

95% CI are shown in parentheses.

of previous reports among men. Among five cohort studies that included women, the results have been inconsistent. In the Whitehall II Study, which included 3413 women aged 35–55 years, Bosma *et al.*¹³ reported an odds ratio of 1.73 (95% CI: 1.15–2.64) for women with low job control compared to women with high job control. In that study, neither job demands nor social support at work were related to the risk of CHD. The finding for low job control was also based on the occurrence of any coronary event (any of the following: angina, severe pain across the chest, and diagnosed ischaemic heart disease). When these outcomes were examined separately, neither angina nor diagnosed ischaemic heart disease was significantly associated with job control. In other words, the positive association with low control was mainly driven by self-reported chest pain.¹⁷

Alfredsson and colleagues¹⁴ followed 4191 Swedish working women aged 20–64 for 4 years, and found a relative hospitalization ratio for MI of 1.6 (95% CI: 1.1–2.3) for those who reported a combination of hectic and monotonous work. In that study, job strain was assessed by the job title method, and among risk factors for MI, only smoking was controlled.¹⁶ The job title method, also referred to as job exposure matrices (JEM), is based on national surveys of the working population

that are then used to impute job stress exposures for individual occupations by means of three-digit occupational codes. This method 'is not the best choice if it is possible to make more direct assessments' of job conditions, such as via self-administered questionnaires.³¹

Another study of 5921 Swedish women aged 45 and 74 years, by Hall and colleagues¹² did not find an association between job strain and cardiovascular disease. That study also used the job title method to assign workers to job strain categories based on the past five occupations. The outcome measure in that study was a combination of the prevalence of cardiovascular disease based on symptoms and descriptions of illness, and the cardiovascular mortality during the 11-year follow-up period.²⁷

The only previous US cohort study of job strain and CHD to include women, by LaCroix and colleagues,¹⁶ found a significant RR of 2.9 for developing CHD in women with high strain jobs compared to women in low strain jobs during 10 years of follow-up of 389 women in the Framingham Heart Study. The authors adjusted for age, smoking, blood pressure, and cholesterol in their analyses.

There may be several reasons for our null findings. Our study, based on a single occupation (registered nurses), may have had

insufficient variability across categories of job conditions. In this regard, our study resembles another US study based on a single occupation (bus drivers), which found no association between job strain and hypertension.³² As suggested by Kristensen,²¹ the different positions in the demands/control matrix should be ideally represented by a variety of occupations to provide optimal statistical power to detect the effects of job strain in health outcomes.³³ On the other hand, others have argued that job strain is inextricably confounded by socioeconomic position.³⁴ To the extent that our cohort consisted predominantly of a single occupation (nurses), we were able to control for such confounding. Moreover, despite the presumed lack of variability in job conditions, we have nevertheless previously found a prospective association between job strain and decline in health functioning in the same cohort.²⁵

Our assessment of job strain was based on self-report, a method in which the assessment of job conditions may themselves be contaminated by worker characteristics, such as personality, negative affectivity, and worker attitudes towards their jobs. However, our inability to control for these factors would have likely led to a bias away from the null, since variables such as negative emotions are themselves risk factors for CHD.³⁵ We had limited data on stress in the home environment. In considering the effects of job stress on women's health, it is important to consider the interaction between work stress and home stress. This is because women are much more likely than men to be exposed to the adverse effects of working the 'second shift'.³⁶ As more women have entered the paid workforce, their responsibilities in the home environment (caregiving, household chores, child-rearing) have not diminished. Neglecting the effects of concurrent stresses in the home environment may therefore result in considerable misclassification of the overall stress burden for working women. Limited data from previous studies suggest that the combination of job stress and home environment stress may increase the risk of CHD in women.³⁷ Findings from the Framingham Heart Study showed that women who are working and have three or more children have a 1.7-fold increased risk of CHD compared with women who are working and have no children, and a 2.5-fold increased risk of CHD compared with women who are not working and have three or more children. In the present study, we had some limited assessment of stress in the home environment (number of hours of caregiving outside of work for children and sick relatives). However, we found no interaction between job stress and caregiving burden. Nonetheless, it is possible that a more complete assessment of stress in the home environment would have yielded an increased risk of CHD in certain sub-groups of women.

Our job strain assessment was restricted to a one-time self-report at the beginning of the follow-up, and thus the effect of duration of exposure could not be assessed. Current exposure to job conditions, combined with the relatively short period

of follow-up (4 years), may not reflect accurately the effects of cumulative exposure over an entire career. In the Cornell Worksite Blood Pressure Study (WSBPS), 22% of participants changed their dichotomous job strain status in 3 years, resulting in a more than 50% turnover in the initial high-strain group.³⁸ We have repeated the job strain assessment in 1996, although we could not use these data in the present analyses due to the incomplete assessment of incident CHD cases beyond 1996 at the time of writing. According to the job strain assessment in 1996, 49% of women who reported as being in high strain jobs in 1992 changed their job strain status in 1996 (high strain job to active job, 18%; high strain job to passive job, 22%; high strain job to low strain job, 9%). Restricting the measurement of job strain to baseline assessment in our study could have missed the cumulative effect of adverse psychosocial work environment on CHD risk.

A 'healthy worker effect' may have also diluted the findings in our study. In other words, healthier employees tend to remain in the work force longer while those with frail health may self-select into low strain jobs or quit work altogether. Over time, it is possible that high strain jobs become over-represented by women with more robust health. To the extent this occurs, it may lead to an underestimation of the effects of job strain on CHD.

Finally, our findings do not preclude the adverse effects of other psychosocial work conditions. An alternative model of job stress, developed by Siegrist, posits that an imbalance between work effort and three types of reward (financial, self-esteem, and social) results in increased risk of CHD.³⁹ Although the effort/reward imbalance model was not tested in our study, other prospective studies have found that the model predicts risk of CHD.^{18,40} In addition, nurses may experience stress from hierarchical structures or gender discrimination in their work setting. Nurses are less likely to have control in decision making in their work, usually taking orders from doctors, who are higher in the hospital hierarchy and mostly male.

Although we could not find an association between job strain and CHD in this cohort, the association between job strain and diminished health functioning we reported previously in this cohort⁴¹ means that work stress remains an important determinant of working women's quality of life.

Acknowledgements

This research was funded by a grant from the National Institute of Aging Research (R01-AG-12806) and by the Dana Foundation. The Nurses' Health Study was funded by a grant from the National Institute of Health (NIH) (R01-CA-87969). Dr Kawachi was supported by a NIH Career Development Award (HL-03453). The authors wish to thank the members of the Nurses' Health Study for their continuing participation and support.

KEY MESSAGES

- In previous cohort studies, job strain (high demands, low control) at work has been linked to increased risk of coronary heart disease (CHD). Few studies have included women, however.
- In the present cohort of female nurses, neither high psychological demands, nor low job control was prospectively associated with increased CHD incidence. Nor was high strain work associated with increased CHD risk.

References

- ¹ Bureau of Labor Statistics. *Employment Status of Civilian Population by Sex and Age*. Washington DC: United States Department of Labor, 2001.
- ² Karasek R, Theorell T. *Healthy Work—Stress, Productivity, and the Reconstruction of Working Life*. New York: Basic Books, 1990.
- ³ Alterman T, Shekelle R, Vernon S, Burau K. Decision latitude, psychologic demand, job strain, and coronary heart disease in the Western Electric Study. *Am J Epidemiol* 1994;**139**:620–27.
- ⁴ Reed D, LaCroix A, Karasek R, Miller D, MacLean C. Occupational strain and the incidence of coronary heart disease. *Am J Epidemiol* 1989;**129**:495–502.
- ⁵ Hlatky M, Lam L, Lee K *et al*. Coronary heart disease/myocardial infarction/systemic vascular responses: job strain and the prevalence and outcome of coronary artery disease. *Circulation* 1995;**92**:327–33.
- ⁶ Netterstrom B, Suadicani P. Self-assessed job satisfaction and ischaemic heart disease mortality: a 10-year follow-up of urban bus drivers. *Int J Epidemiol* 1993;**22**:51–56.
- ⁷ Suadicani P, Hein H, Gynnetelberg F. Are social inequalities associated with the risk ischaemic heart disease a result of psychosocial working conditions? *Atherosclerosis* 1993;**101**:165–75.
- ⁸ Alfredsson L, Karasek R, Theorell T. Myocardial infarction risk and psychosocial work environment: An analysis of the male Swedish working force. *Soc Sci Med* 1982;**16**:463–67.
- ⁹ Alfredsson L, Theorell T. Job characteristics of occupations and myocardial infarction risk: Effect of possible confounding factors. *Soc Sci Med* 1983;**17**:1497–503.
- ¹⁰ Billing E, Hjemdahl P, Rehnqvist N. Psychosocial variables in female versus male patients with stable angina pectoris and matched healthy controls. *Eur Heart J* 1997;**18**:911–18.
- ¹¹ Bobak M, Hertzman C, Skodova Z, Marmot M. Association between psychosocial factors at work and non-fatal MI in a population based case-control study in Czech men. *Epidemiology* 1998;**9**:43–47.
- ¹² Hallqvist J, Diderichsen E, Theorell T, Reuterwall C, Ahlbom A. The SHEEP Study Group: Is the effect of job strain on myocardial infarction due to interaction between high psychological demands and low decision latitude? Results from Stockholm Heart Epidemiology Program (SHEEP). *Soc Sci Med* 1998;**46**:1405–15.
- ¹³ Hammar M, Alfredsson L, Theorell T. Job characteristics and the incidence of myocardial infarction: A study of men and women in Sweden, with particular reference to job strain. *Int J Epidemiol* 1994;**23**:277–84.
- ¹⁴ Sihm I, Delholm G, Hansen E, Gerdes L, Faergeman O. The psychosocial work environment of younger men surviving acute myocardial infarction. *Eur Heart J* 1991;**12**:203–09.
- ¹⁵ Theorell T, Hamsten A, de Faire U. Psychosocial work conditions before myocardial infarction in young men. *Int J Cardiol* 1987;**15**:33–46.
- ¹⁶ Alfredsson L, Spetz C, Theorell T. Type of occupation and near-future hospitalization for myocardial infarction and some other diagnoses. *Int J Epidemiol* 1985;**14**:378–88.
- ¹⁷ Bosma H, Marmot M, Hemingway H, Nicholson A, Brunner E, Stansfeld S. Low job control and risk of coronary heart disease in Whitehall II (prospective cohort) study. *BMJ* 1997;**314**:558–65.
- ¹⁸ Bosma H, Peter R, Siegrist J, Marmot M. Two alternative job stress models and the risk of coronary heart disease. *Am J Public Health* 1998;**88**:68–74.
- ¹⁹ Johnson J, Hall E, Theorell T. Combined effects of job strain and social isolation on cardiovascular disease morbidity and mortality in a random sample of the Swedish male working population. *Scand J Work Environ Health* 1989;**15**:271–79.
- ²⁰ Johnson J, Stewart W, Hall E, Fredlund P, Theorell T. Long term psychosocial work environment and cardiovascular mortality among Swedish men. *Am J Public Health* 1996;**86**:342–31.
- ²¹ Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T. Job decision latitude, job demands, and cardiovascular disease: A prospective study of Swedish men. *Am J Public Health* 1981;**71**:694–705.
- ²² Theorell T, Perski A, Orth-Gomer K, Hamsten A, de Faire U. The effects of the strain of returning to work on the risk of cardiac death after an MI before age 45. *Int J Cardiol* 1991;**30**:61–67.
- ²³ Steenland K, Johnson J, Nowlin S. A follow-up study of job strain and heart disease among males in the NHANES1 population. *Am J Ind Med* 1997;**31**:256–59.
- ²⁴ Hellerstedt W, Jeffery R. The association of job strain and health behaviors in men and women. *Int J Epidemiol* 1997;**26**:575–83.
- ²⁵ Schnall P, Schwartz J, Landbergis P, Warren K, Pickering T. Relation between job strain, alcohol, and ambulatory blood pressure. *Hypertension* 1992;**19**:488–94.
- ²⁶ Williams R, Barefoot J, Blumenthal J *et al*. Psychosocial correlates of job strain in a sample of working women. *Arch Gen Psychiatry* 1997;**54**:543–48.
- ²⁷ Hall E, Johnson J, Tsou T. Women, occupation, and risk of cardiovascular morbidity and mortality. *Occup Med* 1993;**8**:709–19.
- ²⁸ Rose G, Blackburn H. *Cardiovascular Survey Methods, 2nd Edn*. Geneva, Switzerland: World Health Organization, 1982.
- ²⁹ Stampfer M, Willett W, Speizer F, Dysert D, Lipnick R, Rosner B. Test of the National Death Index. *Am J Epidemiol* 1984;**119**:837–39.
- ³⁰ D'agostino R, Belanger A. Relation of pooled logistic regression to time dependent Cox regression analysis: the Framingham Heart Study. *Stat Med* 1990;**9**:1501–15.
- ³¹ Theorell T. Working conditions and health. In: Berkman L, Kawachi I (eds). *Social Epidemiology*. New York: Oxford University Press, 2000, pp.95–117.
- ³² Albright C, Winkleby M, Ragland D, Fisher J, Syme S. Job strain and prevalence of hypertension in a biracial population of urban bus drivers. *Am J Public Health* 1992;**82**:984–89.
- ³³ Kristensen T. The demand-control-support model: methodological challenges for future research. *Stress Med* 1995;**11**:17–26.
- ³⁴ Smith G, Harding S. Is control at work the key to socioeconomic gradients in mortality? (letter). *Lancet* 1997;**350**:1369–70.
- ³⁵ Kubzansky L, Kawachi I. Affective state and health. In: Berkman L, Kawachi I (eds). *Social Epidemiology*. New York: Oxford University Press, 2000, pp.213–41.
- ³⁶ Hochschild A. *The Second Shift*. New York: Avon Books, Inc., 1989.
- ³⁷ Haynes S, Manning F. Women, work and coronary heart disease: prospective findings from the Framingham Heart Study. *Am J Public Health* 1980;**70**:133–41.
- ³⁸ Landbergis P, Theorell T. Measurement of psychosocial workplace exposure variables. *Occup Med* 2000;**15**:163–88.
- ³⁹ Siegrist J. Adverse effects of high effort/low reward conditions. *J Occup Health Psychol* 1996;**1**:27–41.
- ⁴⁰ Siegrist J, Peter R, Junge A, Cremer A, Siedel D. Low status control, high effort at work and ischemic heart disease: prospective evidence from blue-collar men. *Soc Sci Med* 1990;**31**:1127–34.
- ⁴¹ Cheng Y, Kawachi I, Coakley E, Schwartz J, Colditz G. Association between psychosocial work characteristics and health functioning in American women: prospective study. *BMJ* 2000;**320**:1432–36.

Commentary: Work stress and coronary heart disease—a gender (role) specific association?

Johannes Siegrist

In his *Logic of Scientific Discovery* Karl R Popper argued that a theory is based on risky assumptions that deviate from what is already known.¹ Therefore, a (preliminary) confirmation of a theory has the potential of creating new knowledge while its refutation by empirical data produces some pressure towards reconsidering the theoretical formulation. It is one of the advantages of the job strain model proposed by Karasek² that risky assumptions can be derived from it. Up to now, there is impressive empirical support in favour of this model, especially so with respect to cardiovascular risk and disease.³ It is true that the majority of investigations were concerned with men. Therefore, a test of the model in a large prospective study of women, as the one reported in this issue of the *International Journal of Epidemiology* by Lee *et al.*,⁴ is particularly instructive. Its negative results give rise to several considerations.

First, one may ask whether the study design, the measures used or the sample were adequate to test the model. Even when taking the authors' own arguments about limitations of their study into account it is difficult to question the strengths of this report. The Nurses' Health Study covers a large sample of a homogeneous professional group. Despite considerable sample loss its size provides adequate statistical power to test the hypothesis and to adjust for multiple confounders. Moreover, confounding of findings by socioeconomic status is excluded given the rather homogeneous professional background. Restriction of health outcomes to medically diagnosed incident cases of coronary heart disease must be considered an additional strength of the study.

A second consideration concerns the specific work stress profile of nursing. One may argue that for many nurses a low level of task control in combination with high job demands and low workplace social support is perhaps less stressful than coping daily with patients' needs and expectations or exposure to their suffering and ill health.⁵ Moreover, lack of esteem by physicians, inadequate salaries and restricted promotion prospects may contribute to work stress among nurses.⁶ These aspects have not been measured in the present study. Similarly, as briefly discussed by the authors, the work-non work interface, and particularly stressful conditions at home, have not been adequately assessed in the current investigation, thus giving rise to potential underestimation of the contribution of psychosocial stressors to cardiovascular risk and disease in nurses (and women in general).

However, in keeping with Popper's argument the negative findings of this study might challenge the job strain model in a productive way as they urge us to consider gender (or gender role) differences more carefully. It may well be that a substantial part of stressful experience at work is contingent on the perceived threats associated with one's occupational position, and that men, as a result of socialized gender roles, are generally

more vulnerable to these threats than women. For instance, in terms of the social cognitive theory of gender differentiation,⁷ women may be better suited to combine different roles or to change roles with more flexibility and thus to profit from multiple sources of self-efficacy and self-esteem. Men, on the contrary, often stick more exclusively to their occupational role as it provides a major source of their self-reliance.

Broadly speaking, sociocultural factors influence the appraisal of demands and threats in salient social roles in adult life, and these influences are embedded in gender-based practices of coping. Models of work stress might be designed in a way that takes these considerations into account. Interestingly, two recent studies of occupational stress and cardiovascular risk comparing men and women in Sweden, found some evidence along these lines.^{8,9} In men, the threats to occupational status in terms of low control and low reward were more strongly associated with cardiovascular risk than in women. Conversely in women, inadequate or excessive ways of personal coping with the demands at work predicted disease risk more strongly than in men.

It is certainly premature to evaluate the relevance of gender or gender roles in explaining links between psychosocial stress at work and health. Yet, the negative findings reported by Lee *et al.*,⁴ rather than being discouraging, stimulate new thoughts in an area of research that continues to be seminal and important.

References

- 1 Popper KR. *The Logic of Scientific Discovery*. London: Routledge, 1959.
- 2 Karasek R, Theorell T. *Healthy Work. Stress, Productivity, and the Reconstruction of Working Life*. New York: Basic Books, 1990.
- 3 Schnall PL, Belkic K, Landsbergis P, Baker D (eds). The workplace and cardiovascular disease. *Occupational Medicine: State of the Art Reviews* 2000;**15**:1–334.
- 4 Lee S, Colditz G, Berkman L, Kawachi I. A prospective study of job strain and coronary heart disease in US women. *Int J Epidemiol* 2002;**31**:1147–53.
- 5 Dewe PJ. Identifying the causes of nurses' stress: a survey of New Zealand nurses. *Work & Stress* 1987;**1**:15–24.
- 6 Bakker AB, Killmer CH, Siegrist J, Schaufeli WB. Effort-reward imbalance and burnout among nurses. *J Adv Nurs* 2000;**31**:884–91.
- 7 Bussey K, Bandura A. Social cognitive theory of gender development and differentiation. *Psychol Rev* 1999;**106**:676–713.
- 8 Peter R, Alfredson L, Hammer N, Siegrist J, Theorell T, Westerholm P. High effort, low reward, and cardiovascular risk factors in employed Swedish men and women: Baseline results from the WOLF study. *J Epidemiol Community Health* 1998;**52**:540–47.
- 9 Peter R, Siegrist J, Hallqvist J, Reuterwall C, Theorell T & the SHEEP study group. Psychosocial work environment and myocardial infarction: improving risk estimation by combining two alternative job stress models in the SHEEP study. *J Epidemiol Community Health* 2002;**56**:294–300.