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Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees

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Abstract

Objective To examine the association between work stress, according to the job strain model and the effort-reward imbalance model, and the risk of death from cardiovascular disease.

Design Prospective cohort study. Baseline examination in 1973 determined cases of cardiovascular disease, behavioural and biological risks, and stressful characteristics of work. Biological risks were measured at 5 year and 10 year follow up.

Setting Staff of a company in the metal industry in Finland.

Participants 812 employees (545 men, 267 women) who were free from cardiovascular diseases at baseline.

Main outcome measure Cardiovascular mortality 1973-2001 from the national mortality register.

Results Mean length of follow up was 25.6 years. After adjustment for age and sex, employees with high job strain, a combination of high demands at work and low job control, had a 2.2-fold (95% confidence interval 1.2 to 4.2) cardiovascular mortality risk compared with their colleagues with low job strain. The corresponding risk ratio for employees with effort-reward imbalance (low salary, lack of social approval, and few career opportunities relative to efforts required at work) was 2.4 (1.3 to 4.4). These ratios remained significant after additional adjustment for occupational group and biological and behavioural risks at baseline. High job strain was associated with increased serum total cholesterol at the 5 year follow up. Effort-reward imbalance predicted increased body mass index at the 10 year follow up.

Conclusions High job strain and effort-reward imbalance seem to increase the risk of cardiovascular mortality. The evidence from industrial employees suggests that attention should be paid to the prevention of work stress.

Introduction

Concern is increasing about the adverse effects that work stress may have on health, particularly the risk of cardiovascular disease. Two models identifying stressful components of the psychosocial work environment have received particular attention: the job strain model[1 2] and, more recently, the effort-reward imbalance model.[3] In spite of the large body of research on these models,[3–7] no previous study has tested them simultaneously in relation to cardiovascular mortality.

The job strain model posits that a combination of high work demands and low job control at work, called job strain, is a health risk for employees.[2] The few studies on cardiovascular mortality partly support the model. Alterman et al showed a moderate prospective association between job strain and fatal cardiovascular disease.[8] Other investigations have linked cardiovascular mortality to a combination of high demands, low resources, and low income,[9] to job control only,[10] and to neither job control, work demands, nor their interaction.[11]

The effort-reward imbalance model considers the impact of labour market conditions on health in addition to the more proximal job conditions.[3] Health risk derives from the mismatch between high efforts at work and low reward received in turn. Rewards concern money, social approval, job security, and career opportunities. Direct evidence of cardiovascular mortality has been lacking. Results from the Whitehall II study showed an association between effort-reward imbalance and incidence of coronary heart disease, as indicated by self reports.[12] Cross sectional findings have revealed associations of effort-reward imbalance with precursors of cardiovascular disease, such as hypertension, high concentrations of low density lipoprotein cholesterol, lowered vagal tone, and impaired fibrinolytic capacity.[13–15]

Cardiovascular disease is the leading cause of death in modern civilisations. Work stress models focusing on aspects of the workplace, work organisation, and labour market conditions may offer promising opportunities for theory based intervention. We aimed to test the extent to which the work stress models can explain deaths from cardiovascular disease.

Methods

Study population

The study sample was drawn from the employees (n=4570 in 1973) of the Valmet factories in Jyväskylä, central Finland, which manufacture paper machines, tractors, firearms, gauges, and so on. The work tasks varied from foundry work and heavy engineering to precision engineering and clerical and administrative work. The study population comprised people who had been employed by Valmet for at least 15 months in

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January 1973 (n=2653). We selected a systematic sample of 902 participants in strata by sex, age (born in 1925 or earlier, born in 1926-45, born in 1946 or later), and occupational group (managers, other office staff, skilled workers, semiskilled workers).[16] People who refused to participate were replaced by new participants from a reserve list. We gathered data by questionnaire, interviews, and clinical examinations. After exclusion of people with cardiovascular disease at baseline, the study cohort comprised 812 employees. Follow up examinations included measurement of blood pressure, cholesterol concentration, and body mass index in 1978 (n=674) and in 1983 (n=594). We obtained ethical approval for the study from the Hospital district of Helsinki and Uusimaa ethics committee for research in occupational health and safety.

Work stress questionnaire

We used self assessment scales used to measure the components of the job strain model and the effort-reward imbalance model.[16] The four questions on work demands deal with the degree of responsibility at work, task difficulty, and mental load (Cronbach's α reliability=0.67), and the 12 questions on job control concern decision authority and skill discretion (α=0.78). (Sample questions: "How mentally straining do you consider your work?" "Do you learn new things in your work?") The nine questions on effort at work indicate pace of work and physical and mental load (α=0.72), and the 16 questions on rewards measure satisfaction with income, fairness of supervision, job security, and promotion prospects (α=0.80). (Sample questions: "How great is the strain due to haste in your work?" "If changes or reorganisation take place at your workplace, how great is your risk of getting laid off?") All the questions required responses on Likert-type response formats (for example, 1="no strain" to 5="very great strain"). Each scale was constructed by summing the response scores on the individual questions. We divided the resulting scores into thirds to indicate low, intermediate, and high levels on each scale.

We used the work demand and job control scales to create the job strain indicator. This indicator had three categories: high job strain (high or intermediate demands combined with low control), low job strain (low demands combined with high or intermediate control), and intermediate job strain (all other combinations of demands and control).

We created the indicator of effort-reward imbalance by calculating the ratio between the sum response score in the effort scale and the sum response score in the reward scale.[13–15] We divided the resulting quotient into thirds to indicate low, intermediate, and high effort-reward imbalance.

We observed an association between job strain and effort-reward imbalance. Employees with high job strain reported high effort-reward imbalance more often than employees with intermediate and low job strain (60% *v* 28% *v* 13%; weighted κ=0.32, $P<0.001$).

Cardiovascular mortality

We collected mortality data from the Statistics Finland national mortality register, using the participants' personal identification codes. We obtained the date and cause of death for all participants who died between the date of their clinical examination (which took place between 5 February and 30 June 1973) and 1 November 2000. The causes of death were coded according to the ICD-8 (international classification of diseases, eighth revision) in 1973-86, the ICD-9 in 1987-95, and the ICD-10 in 1996-2000. Statistics Finland provided a classification that converted the different codes (up to 1997; subsequent deaths were classified on the basis of the death certificates) to the following categories: ischaemic heart diseases (I20-I25 in ICD-10), other heart diseases (I30-I52), cerebrovascular diseases (I60-I69), and other diseases of the cardiovascular system (I00-I19, I26-I29, I70-I99). We pooled these categories to indicate death due to cardiovascular diseases. We used information on the basic cause of death.

Demographic, behavioural, and biological factors

We measured baseline covariates in standard ways: sex, age, occupational group (managers, other office staff, skilled workers, semiskilled workers),[16] smoking status (current smoker, non-smoker), physical activity, systolic blood pressure (mm Hg), serum total cholesterol concentration (mmol/l), and body mass index (kg/m^2). We divided the distributions of the last four measures into thirds. We based the assessment of physical activity and diabetes on a detailed interview described elsewhere.[17]

At the five year follow up we measured systolic blood pressure and serum total cholesterol concentration. We measured body mass at the 10 year follow up.

Statistical analysis

We used Cox proportional hazard models to assess associations between baseline characteristics and cardiovascular mortality. The first step studied associations of age (18-27 years, 28-47 years, 48 years or over), sex, occupational group, smoking status, and other behavioural and biological factors (physical activity, systolic blood pressure, serum total cholesterol, and body mass index; all indicators divided into thirds) with cardiovascular mortality.

In the second step, we explored the associations of each work stress variable (job strain, effort-reward imbalance, and their components) with cardiovascular mortality. As the interactions between sex and work stress variables were not significant ($P>0.10$), we based all analyses on a combined sample of male and female employees. We used the employees in the most favourable third for each work stress variable as the reference group. We adjusted hazard ratios and 95% confidence intervals for age, sex, occupational group, and significant behavioural and biological predictors of cardiovascular mortality. We repeated analyses with two subsamples: employees free from diabetes at baseline and employees whose occupational group remained unchanged at least five years after the assessment of work stress.

Finally, in order to identify potential biological linking mechanisms, we assessed associations of work stress variables with blood pressure, cholesterol concentration, and body mass index at follow up by using analysis of variance with adjustment for baseline values of these biological factors, age, and sex. We treated biological factors as continuous variables here. We used the SAS 8.12 statistical program package to perform all analyses.

Results

During the mean follow up of 25.6 (range 0.9-27.8) years, 73 deaths from cardiovascular causes occurred among the participants who had been free from overt cardiovascular diseases at baseline. Table 1 shows the associations of demographic, behavioural, and biological factors with cardiovascular mortality. As expected, higher age, male sex, low worker status, smoking, sedentary lifestyle, high blood pressure, high cholesterol concentration, and high body mass index increased the risk of death.

Table 2 presents the relative hazards for cardiovascular mortality by levels of the components of the work stress models. After adjustment for age and sex, we found excess risks for high job strain, low job control, high effort-reward imbalance, and low reward, but not for high demands or high efforts.

After additional adjustment for occupational group, job control no longer predicted mortality, as shown in table 3. The hazard ratio for the reward component also became non-significant after adjustment for behavioural risk factors. Adjustment for baseline covariates had little effect on the associations of job strain and effort-reward imbalance with cardiovascular mortality. The fit was the same for both these models (−2 log likelihood statistics were 786.4 for job strain and 787.8 for effort-reward imbalance). Employees scoring high on job strain and effort-reward imbalance had a twofold risk of death compared with their colleagues with low strain and low effort-reward imbalance (table 3). Exclusion from the analyses of the four participants who had diabetes at baseline did not change these results.

A stratified analysis of the 584 employees (55 deaths) whose occupational group remained unchanged five years after the assessment of work stress revealed a strengthened association between job strain and cardiovascular mortality—the hazard ratio for high job strain was 2.90 (95% confidence interval 1.25 to 6.71). The association between reward-effort imbalance and mortality remained unaltered—hazard ratio for high imbalance of 2.59 (1.18 to 5.68). The individual components of the work stress models were not significantly associated with death from cardiovascular disease.

Table 4 shows increased concentrations of total cholesterol at follow up for employees with high job strain and low job control, and increased body mass index for employees with low job control and high effort-reward imbalance. Work stress was not associated with blood pressure at follow up.

Table 1 Hazard ratios for cardiovascular mortality with demographic, behavioural, and biological factors

Characteristic	No of participants (No of deaths)	Hazard ratio (95% CI)
Age at baseline (years):		
⩽27	251 (3)	1.00
28-47	330 (13)	3.37 (0.96 to 11.82)
>47	231 (57)	26.48 (8.29 to 84.61)
Sex:		
Women	267 (13)	1.00
Men	545 (60)	2.41 (1.32 to 4.39)
Occupational group:		
Managers	94 (5)	1.00
Other office staff	269 (19)	1.29 (0.48 to 3.46)
Skilled workers	165 (24)	2.86 (1.09 to 7.49)
Semiskilled workers	284 (25)	1.63 (0.62 to 4.26)
Smoking status:		
Non-smoker	560 (39)	1.00
Current smoker	251 (34)	2.17 (1.37 to 3.44)
Physical activity level:		
Low	364 (44)	1.00
Intermediate	171 (15)	0.71 (0.39 to 1.27)
High	277 (14)	0.39 (0.21 to 0.71)
Systolic blood pressure (mm Hg):		
<127	255 (9)	1.00
127-143	301 (14)	1.31 (0.57 to 3.03)
>143	256 (50)	6.21 (3.05 to 12.63)
Cholesterol (mmol/l):		
<5.6	286 (8)	1.00
5.6-6.8	268 (26)	3.68 (1.66 to 8.12)
>6.8	258 (39)	5.94 (2.77 to 12.70)
Body mass index (kg/m^2):		
<23	267 (13)	1.00
23-25.5	268 (24)	1.92 (0.98 to 3.77)
>25.5	277 (36)	2.88 (1.53 to 5.42)

Table 2 Hazard ratios for cardiovascular mortality by levels of work characteristics. Adjusted for age and sex

Characteristic	No of participants (No of deaths)	Hazard ratio (95% CI)
Job strain:		
Low	215 (16)	1.00
Intermediate	389 (32)	1.53 (0.83 to 2.82)
High	201 (25)	2.20 (1.16 to 4.17)
Demands (a component of job strain):		
Low	298 (23)	1.00
Intermediate	232 (22)	1.58 (0.88 to 2.84)
High	282 (28)	1.35 (0.77 to 2.36)
Job control (a component of job strain):		
High	264 (20)	1.00
Intermediate	259 (21)	1.17 (0.63 to 2.16)
Low	282 (32)	1.90 (1.08 to 3.37)
Effort-reward imbalance:		
Low	269 (15)	1.00
Intermediate	272 (29)	2.21 (1.17 to 4.15)
High	271 (29)	2.36 (1.26 to 4.42)
Effort (a component of effort-reward imbalance):		
Low	263 (17)	1.00
Intermediate	277 (24)	1.54 (0.82 to 2.90)
High	272 (32)	1.63 (0.90 to 2.96)
Reward (a component of effort-reward imbalance):		
High	280 (32)	1.00
Intermediate	252 (14)	0.79 (0.41 to 1.51)
Low	280 (27)	2.04 (1.21 to 3.43)

Discussion

To our knowledge, this is the first study of cardiovascular mortality that tests the validity of the two main work stress models in a single working population. We found that employees reporting high job strain and high effort-reward imbalance had a twofold higher risk of death from cardiovascular disease than their colleagues scoring low in these dimensions. The specific strengths of our investigation were a long follow up period, comprehensive questionnaires to indicate stressful work characteristics, control for a large set of potential confounding factors, and the use of reliable mortality registers.

In this study of initially healthy employees adjustment for baseline variation in smoking, seden-

Table 3 Adjusted hazard ratios (95% confidence intervals) for cardiovascular mortality by levels of work characteristics

Characteristic	Covariates in addition to age and sex			
	Occupational group	Behavioural risk factors*	Biological risk factors†	All aforementioned
Job strain:				
Low	1.00	1.00	1.00	1.00
Intermediate	1.36 (0.72 to 2.57)	1.71 (0.92 to 3.17)	1.58 (0.84 to 2.95)	1.64 (0.85 to 3.19)
High	1.89 (0.93 to 3.81)	2.20 (1.12 to 4.32)	2.35 (1.22 to 4.52)	2.22 (1.04 to 4.73)
Job control (a component of job strain):				
High	1.00	1.00	1.00	1.00
Intermediate	0.94 (0.48 to 1.82)	1.06 (0.57 to 1.98)	1.14 (0.62 to 2.11)	0.74 (0.39 to 1.50)
Low	1.55 (0.80 to 3.01)	1.79 (0.98 to 3.27)	1.89 (1.06 to 3.38)	1.42 (0.72 to 2.82)
Effort-reward imbalance:				
Low	1.00	1.00	1.00	1.00
Intermediate	2.16 (1.04 to 4.49)	2.00 (1.06 to 3.78)	2.07 (1.09 to 3.91)	1.91 (0.90 to 4.05)
High	2.36 (1.06 to 5.46)	2.18 (1.15 to 4.13)	2.29 (1.21 to 4.35)	2.42 (1.02 to 5.73)

*Smoking and physical activity.
†Systolic blood pressure, cholesterol concentration, and body mass index.

tary lifestyle, blood pressure, serum total cholesterol, body mass index, and history of diabetes resulted in no or only a minor attenuation of hazard ratios. However, high job strain and high effort-reward imbalance were associated with increased cholesterol concentration and body mass index at follow up. These associations may reflect pathophysiological changes related to the development of cardiovascular disease.

The work stress models are aimed at identifying characteristics of work life that are likely to cause frequent and longlasting stress and therefore be predictive of disease endpoints.[18] Stable levels of work stress are more likely among employees who do not change their job or workplace. In line with this, we found the adverse effects of high job strain to be greatest for employees who remained with the same employer and in the same occupation during the five years after assessment of their work stress. The results for effort-reward imbalance remained unaltered in this stratified analysis.

Many recent studies on the job strain model emphasise the importance of low job control rather than its interaction with high demands.[19] We found low job control to be associated with cardiovascular mortality before, but not after, adjustment for occupational group. The 40% attenuation in the hazard ratio after the adjustment suggests that job control varies according to occupational group or that part of the effect of job control may be attributable to its relation to low socioeconomic status, a major determinant of public health.[20 21] Our results are in line with those of Lynch et al, who reported that the effect of low resources (a construct close to job control) on mortality and acute myocardial infarction was evident only for employees with low socioeconomic status.[9]

High work demands and high mobilisation of efforts at work may not always indicate harmful stress. In this study, the levels of demands and effort did not independently predict cardiovascular mortality, and corresponding results have been reported previously.[8 10–12 22] However, people with a very high workload, as indicated by working continuously over 11 hours a day, may be at increased risk of cardiovascular disease.[23]

Assessment of work stress with self reports is apparently not a source of major bias in our study. Previous studies using subjective and objective methods have tended to give reasonably consistent results,[19] and the correlations between subjective assessments and expert ratings of job conditions are high.[5]

Table 4 Serum total cholesterol concentration and body mass index at follow up* by levels of work characteristics. Means are adjusted for baseline value, sex, and age

Characteristic	Serum total cholesterol (mmol/l×100)		Body mass index (kg/m²)	
	Mean (SE)	P value	Mean (SE)	P value
Job strain:		0.050		0.151
Low	634.6 (6.28)		24.8 (0.14)	
Intermediate	649.0 (4.34)		25.1 (0.10)	
High	655.8 (6.07)		25.2 (0.15)	
Job control (a component of job strain):		0.033		0.008
High	635.2 (5.54)		24.7 (0.13)	
Intermediate	653.8 (5.35)		25.1 (0.13)	
Low	651.7 (5.16)		25.3 (0.12)	
Effort-reward imbalance:		0.168		0.002
Low	639.0 (5.48)		24.8 (0.12)	
Intermediate	652.0 (5.25)		25.1 (0.12)	
High	651.1 (5.15)		25.4 (0.13)	

*Five year follow up of cholesterol and 10 year follow up of body mass.

Policy implications

In the promotion of cardiovascular health, the traditional advice for people to stop smoking, cut down drinking, eat less fat, and engage in physical activity has been shifted towards a more holistic view. Structural and psychosocial factors, in addition to behavioural ones, are increasingly seen as important determinants of public health. Our findings on work stress are consistent with this interpretation.

However, excess health risk in employees with high stress might not exclusively reflect a causal relation. For example, a selection into a stressful work environment may partly reflect early risk factors and adverse environments during childhood and adolescence.[24] Research on organisational interventions is needed to evaluate the additional gains achievable from efforts to change work life.

Conclusions

This prospective cohort study on industrial employees sheds more light on the potential causative role of work stress in cardiovascular disease. The finding of a twofold higher cardiovascular mortality risk among people with high job strain and effort-reward imbalance supports the theoretical models of Karasek, Theorell,[1 2] and Siegrist.[3]

What is already known on this topic

Job strain (high demands and low job control) and effort-reward imbalance (high demands, low security, few career opportunities) elicit stress at work

Their status as risk factors for cardiovascular mortality has, however, remained uncertain

What this study adds

Job strain and effort-reward imbalance were each associated with a doubling of the risk of cardiovascular death among employees who were free from overt cardiovascular diseases at baseline

Job strain and effort-reward imbalance also predicted adverse changes in biological factors such as cholesterol concentration and body mass index

MK is also a senior researcher at the Finnish Institute of Occupational Health, Helsinki. We thank Hans Helenius for his help with statistical analysis.

Contributors: MK formulated the hypotheses, designed the study, carried out the data analyses, and was the principal author of the paper. PL-A is the coordinator of the project and the second principal investigator; she helped in developing the hypotheses, designing the study, data analysis, interpreting the results, and writing the paper. MK and PL-A will act as guarantors for the paper. RL, HR, JV, and JK took part in the study design, contributed to interpretation of the data, and edited the manuscript.

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What is already known on this topic

The most recent systematic review evidence suggests that self help interventions designed specifically for pregnant smokers can be effective in increasing cessation rates

These reviews, however, are based mainly on efficacy trials involving staff who are specifically employed to provide the intervention

In other attempts to assess the effectiveness of such an approach within routine antenatal care, it has been difficult to implement scientifically rigorous evaluations

What this study adds

A low cost, self help intervention was ineffective when implemented during routine antenatal care, even though it was acceptable to midwives and pregnant women

Validated smoking cessation rates among pregnant women are substantially lower than the self reported rates on which current smoking policy is based

associations between social inequality and continued smoking by pregnant women show that more complex interventions that take full account of the social and cultural circumstances of this target group are required.[13]

Implications for policy

Midwives will always have an important role in encouraging pregnant women to stop smoking, but if the government's target of a reduction from 23% to 15% in the percentage of women who smoke during pregnancy is to be met by the year 2010, more intensive interventions or interventions provided by dedicated staff will be required.[14] The discrepancy between biochemically validated and self reported quit rates highlights the importance of biochemical validation. This calls into question the adequacy of monitoring of the government's target for smoking in pregnancy, which currently relies on retrospective self reported smoking behaviour.[15]

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Corrections and clarifications

Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees
A lapse in concentration at proof stage of this paper by Mika Kivimäki and colleagues (19 October, pp 857-60) led us to assign the wrong address to some authors. The correct affiliation for Päivi Leino-Arjas, Ritva Luukkonen, and Hilkka Riihimäki is the Department of Epidemiology and Biostatistics, Finnish Institute of Occupational Health, Helsinki, Finland, and for Jussi Vahtera is the Turku Regional Institute of Occupational Health, Finland. Our apologies for getting these wrong.

Career focus
Two editorial errors crept into the article "Induction courses for international doctors" by Martha Swierczynski (16 November, p s159). In trying to clarify the meaning of the phrase "international doctors," we added (in the opening paragraph) "doctors who have trained in the United Kingdom." This is clearly wrong; what we had intended to add was "doctors who have trained outside the United Kingdom." Also, in the last paragraph of the section "Eligibility for induction courses" the penultimate sentence should read "Trusts [not deaneries] are advised to make the courses as accessible as possible."

Nurse led follow up and conventional medical follow up in management of patients with lung cancer: randomised trial
In this paper by Sally Moore and colleagues (16 November, pp 1145-7), the affiliation for Mary Wells was out of date. She has informed us that for the past three years she has been a clinical research fellow in cancer nursing at the School of Nursing and Midwifery, University of Dundee.