SHORT REPORT

Is the effect of work stress on cardiovascular mortality confounded by socioeconomic factors in the Valmet study?

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J Epidemiol Community Health 2004;**58**:1019–1020. doi: 10.1136/jech.2003.016881

The link between work stress and cardiovascular disease (CVD) is robust, but derived exclusively from observational studies.¹ The work stress effect may thus not be causal, but attributable to confounding with low socioeconomic position.² This report examines the confounding explanation for the effects of work stress on CVD mortality in the Valmet study,³ using measures of socioeconomic circumstances across the life course.

PARTICIPANTS, METHODS, AND RESULTS

Participants were staff at a metal working company in Finland. The baseline survey (1973) identified those who were free from CVD at baseline (812 employees: 545 men, 267 women) and determined stressful characteristics of work and socioeconomic factors. The questionnaire based evaluation of work stress has been described in detail.³ Principal component analysis was used to derive internally consistent scales from existing questionnaire items on work demands, job control, effort at work, and material and psychosocial rewards. Content validity was high compared with the current instruments. Missing values for father's occupation ($n = 14$) and participant's salary ($n = 40$) were imputed (SAS PROC MI and PROC MIANALYZE) in relevant analyses using five imputations based on maximum likelihood estimates. The

outcome was cardiovascular mortality 1973–2000 from the national mortality register.

One hundred and eighty deaths, of which 73 were from CVD (60 among men), occurred over 27 years of follow up (mean 25.6 years). A third ($n = 24$) of CVD deaths were among non-manual employees. Socioeconomic circumstances were associated with both measures of work stress (table 1). Job strain and effort-reward imbalance at baseline were each associated with a doubling of CVD mortality, after adjusting for age and sex (table 2). After excluding non-manual workers, age and sex adjusted hazard ratios for high job strain and high effort-reward imbalance were 1.9 (95% confidence intervals: 0.7 to 5.1) and 2.9 (0.7 to 12.3), respectively.

Of the childhood factors, controlling for education reduced work stress effects slightly (25%–30%), but controlling for father's occupation and height did not. After simultaneous adjustment for these three childhood factors, the hazard ratio for high job strain was 1.93 (1.0 to 3.8) and for high effort-reward imbalance 2.02 (1.0 to 4.1). Of the adult factors, controlling for occupational group reduced the work stress effects slightly, while adjustment for salary did not. After simultaneous adjustment for occupation and income the hazard ratio for high job strain was 1.95 (0.9 to 4.0) and for high effort-reward imbalance 2.50 (1.1 to 5.8). After

Table 1 Hazard ratios (95% CI) for cardiovascular mortality and mean scores of work characteristics by measures of socioeconomic position at baseline in the Valmet study (adjusted for age and sex)

Confounder	Number of participants (number of deaths)†	CVD mortality Adjusted HR (95% CI)	Work stress indicators	
			Job strain score‡	Effort-reward imbalance score‡
Father's occupational group				
White collar	131 (12)	1.00	1.8*	1.6*
Blue collar	423 (37)	1.04 (0.5 to 2.0)	2.1	2.0
Farmer	244 (21)	1.17 (0.6 to 2.4)	2.1	2.3
Height (cm, men/women)				
>176/>163	275 (16)	1.00	2.0 NS	2.0 NS
171–176/158–163	267 (21)	1.08 (0.6 to 2.1)	2.1	2.1
<171/<158	270 (36)	1.49 (0.8 to 2.7)	2.1	2.1
Education				
Secondary or more	77 (2)	1.00	1.7*	1.2*
Intermediate level	409 (28)	3.05 (0.7 to 12.9)	1.9	1.8
Primary or less	326 (43)	4.04 (1.0 to 16.8)	2.3	2.4
Occupational group				
Managers	94 (5)	1.00	1.6*	1.2*
Other office staff	269 (19)	1.29 (0.5 to 3.5)	1.8	1.5
Skilled workers	165 (24)	2.86 (1.1 to 7.5)	2.2	2.4
Semiskilled workers	284 (25)	1.63 (0.6 to 4.3)	2.4	2.6
Salary				
Top quartile	161 (14)	1.00	1.7*	1.3*
3rd/2nd	407 (34)	1.44 (0.8 to 2.7)	2.1	2.1
Bottom quartile	204 (21)	1.53 (0.8 to 3.0)	2.3	2.4

*Test of heterogeneity $p < 0.05$ (analysis of variance). †Numbers before imputation. ‡1 = low, 2 = intermediate, 3 = high.

Table 2 Hazard ratios (95% CI) for cardiovascular mortality by levels of work characteristics in the Valmet study (adjusted for age and sex, and separately for measures of socioeconomic position at study baseline)

	Number of participants (number of deaths)	Adjusted for age and sex	Covariate in addition to age and sex				
			Father's occupational group	Height	Education	Occupational group	Income (salary)
Job strain*							
Low	215 (16)	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate	389 (32)	1.53 (0.8 to 2.8)	1.54 (0.8 to 2.9)	1.49 (0.8 to 2.7)	1.39 (0.8 to 2.6)	1.36 (0.7 to 2.6)	1.51 (0.8 to 2.9)
High	201 (25)	2.20 (1.2 to 4.2)	2.22 (1.2 to 4.3)	2.20 (1.2 to 4.2)	1.85 (1.0 to 3.6)	1.89 (0.9 to 3.8)	2.15 (1.1 to 4.4)
Effort-reward imbalance†							
Low	269 (15)	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate	272 (29)	2.21 (1.2 to 4.2)	2.28 (1.2 to 4.4)	2.09 (1.1 to 4.0)	1.94 (1.0 to 3.7)	2.16 (1.0 to 4.5)	2.35 (1.2 to 4.7)
High	271 (29)	2.36 (1.3 to 4.4)	2.44 (1.3 to 4.7)	2.29 (1.2 to 4.3)	2.01 (1.0 to 4.0)	2.40 (1.1 to 5.5)	2.56 (1.2 to 5.3)

*Low job strain (low demands combined with high or intermediate control), high job strain (high or intermediate demands combined with low control), and intermediate job strain (all other combinations of demands and control). †Tertiles.

controlling for height and all four of the socioeconomic measures, the hazard ratio for high job strain was 2.04 (1.0 to 4.3) and for effort-reward imbalance 2.54 (1.1 to 5.9). The corresponding estimates for all cause mortality were 0.95 (0.6 to 1.5) and 1.08 (0.7 to 1.8) respectively. The association between effort-reward imbalance and non-CVD mortality was non-significant, but, unexpectedly, high job strain was related to lowered non-CVD mortality (hazard ratio 0.51 (0.3 to 1.0)).

COMMENT

This study shows that work stress was a robust predictor of cardiovascular mortality after controlling for socioeconomic circumstances in childhood and adulthood, adding evidence that stress may be causally related to risk of CVD.¹ Reporting bias is most unlikely to account for our findings. The outcome was CVD mortality obtained from the reliable Finnish national register. There was a comparatively long follow up period of 27 years and so sub-clinical disease is unlikely to lie behind reports of high work stress. Indeed, exposure was measured once and therefore the effects observed may be underestimated. Work circumstances seem quite stable in this cohort. Of the 674 participants in the five year follow up, 87% remained in the same occupational group as at baseline. At 10 years the most common reason for change in employment status was retirement. At the end of follow up (2000) 53% reported Valmet as their current or last employer.

Most but not all of previously published prospective studies of work stress and CVD show a similar relation to that published here.¹ However, several of these were not able to take adequate account of socioeconomic circumstances, as we have done. The effect of job strain on CVD risk may be limited to those on low incomes in some populations,⁴ but in the Whitehall II cohort, for example, the effect tended if anything to be greater in higher civil service grades.⁵ In our study, salary was the purest available measure of current material circumstances and controlling for it did not weaken the effects of workplace stress.

Educational attainment is a predictor of future income and psychosocial resources, and was linked both to work stress exposures and to CVD risk in this study. In models that included both education and work stress, work stress was the stronger predictor of mortality, and may thus lie on the pathway between education and CVD.

In conclusion, the Valmet study provides evidence for a link between work stress and mortality with cardiovascular causes. This effect does not seem to be explained entirely by confounding by socioeconomic circumstances over the life course. Further studies are needed to assess the generalisability and specificity of our findings and to link the

epidemiological evidence with the physiological and behavioural mechanisms that may underlie the effects of work stress on cardiovascular disease.⁶

ACKNOWLEDGEMENTS

We gratefully acknowledge the role of Professors Jeddi Hasan and Risto Telama in founding the Valmet cohort, and the financial support of the Academy of Finland (projects 70139, 77560, and 105195) during successive phases of the study programme.

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Conflicts of interest: none declared.

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Accepted for publication 22 March 2004

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