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Stress at work—an independent risk factor for coronary heart disease?

John Yarnell*

Queen’s University Belfast, Institute of Clinical Science, Department of Epidemiology and Public Health, Mulhouse Building, Grosvenor Road, Belfast BT12 6BJ, UK

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This editorial refers to ‘Work stress and coronary heart disease: what are the mechanisms?’ by T. Chandola et al.,† on page 640

In their recent study, Chandola et al. add to the body of evidence linking work stress with the development of coronary heart disease (CHD) in British civil servants, and examine potential mechanisms.† Similar findings have been reported in cohort studies across Europe; for example, in the Job Stress, Absenteeism and Coronary Heart Disease in Europe study (JACE), a multicentre study in four European countries, in working populations from a broad range of occupational groups and also from the general population.† In 2004, a review showed that fewer than half of the 17 longitudinal studies and eight cross-sectional studies included showed a significant association between perceived work stress and subsequent CHD.† In 2006, a meta-analysis of 14 prospective cohort studies examined the contributions of three models of work stress—job strain, effort–reward imbalance, and organizational injustice—to subsequent risk of CHD. Overall the studies suggested an excess risk of 50% for subsequent CHD following exposure to chronic work stress, but only the organizational injustice model remained statistically significant following adjustment for classical risk factors and potential mediators.†

Most of us would experience stress at work at some point in time, but we would recognize its subjective nature and variability. Devising an instrument to capture the experience, particularly of chronic work stress, is a challenge for psychologists and sociologists, and studies use a variety of instruments. Differences between the instruments used and periods of exposure may account for some of the variations in study findings thus far. The development of a standardized instrument that could be used across Europe and elsewhere, particularly in newly emerging economies where work stress is likely to be more prevalent, and working hours may be the longest, would represent a step forward in the epidemiological investigation of work stress.

One well-recognized coping behaviour for work stress is sickness absence, also analysed in the Whitehall II study of British civil servants.† The authors noted the moderating effect of social support and chronic domestic stressors (family, financial, housing, etc.), and other major studies† have raised the possibility that domestic stressors contribute to the pathogenesis of acute coronary events. In the INTERHEART study, the authors noted that stress at work and at home were highly correlated. The authors devised four psychosocial variables—general stress (home and work), financial stress, locus of control (related to life events), and depression, which were used in 52 countries. The authors noted large variations in the reporting of adverse levels of these variables between world regions, but consistency in their differences between myocardial infarction cases (first event) and controls. The evidence suggests that there is likely to be an interaction between the stressors in the domestic and working environments, and studies should consider the complete psychosocial environment for an individual. Composite indicators of psychosocial disadvantage, closely linked with exposure to chronic stressors, have been shown to have a graded relationship with subsequent risk of CHD in a long-term follow-up study.

Certain personality types may be particularly prone to the effects of work stress. Studies in cardiac patients have implicated type D (‘distressed’) personality, which tends to experience negative emotions such as depressed mood, anxiety, anger, and hostile feelings, as an important prognostic factor for long-term mortality. Links have also been shown between personality type (including type D) and physiological responses such as cortisol secretion in experimental studies of laboratory stressors.† Epidemiological studies which incorporate personality traits may be helpful in defining a group at particularly high risk from work stress.

Evidence from observational epidemiological studies may be of limited value to the clinician unless followed up by intervention studies showing evidence of benefit. Evidence from psychosocial interventions in the workplace is limited to small-scale studies with intermediary outcome variables,† and evidence from a large
multicentre trial in cardiac patients is equivocal. In this study, psychological therapy, counselling, relaxation training, and stress management training were provided to unselected myocardial infarction cases and their spouses over a 7-week period; follow-up events were limited to 12 months. However, a recent randomized controlled trial of exercise and stress management in 134 patients with stable ischaemic heart disease reported significant changes in both psychological and physiological measures—including baroreflex sensitivity and heart rate variability (HRV)—in response to the stress management.

Physiological variables may provide the key to understanding the relationship between chronic stressors and subsequent cardiovascular disease or other health outcomes. In the report by Chandola et al., exposure to chronic work stress at both recruitment and the first follow-up examination is associated with low HRV in a graded relationship at 13 years of follow-up. This provides support for the possibility that chronic work stress may increase the level of dominance of the sympathetic nervous system associated with low HRV.

Adaptive physiological responses to stress have been reviewed in detail elsewhere, but three such responses will be briefly discussed. Hypertension has been linked with work stress in a large cohort of Canadian civil servants, but a recent report from Whitehall II indicates that newly diagnosed hypertension was not directly linked with the development of CHD following chronic exposure to work stress. Nonetheless, in the Atherosclerosis Risk in Communities Study (ARIC), HRV was measured at baseline examination, and low HRV was associated with new-onset hypertension, incident CHD, and all-cause mortality. Clearly further prospective studies with serial measurements of both HRV and blood pressure will be required to examine the pathogenesis and relevance of each risk factor for subsequent cardiovascular disease.

Finally, in their report Chandola et al. reported a cross-sectional association between work stress and morning cortisol levels, in agreement with findings from a large number of small experimental studies. Few prospective studies have examined the contribution of morning cortisol levels to subsequent development of CHD, and further studies would be useful.

In conclusion, there is increasing evidence of the potential role of work stress in the pathogenesis of premature coronary disease, but improved instruments and more controlled trials using intermediary markers may be helpful. Physiological markers such as HRV may prove to be useful predictors of subsequent coronary events in the general population, but more evidence is clearly needed.

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References


