Overtime is bad for the heart

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This editorial refers to ‘Overtime work and incident coronary heart disease: the Whitehall II prospective cohort study’, by M. Virtanen et al., on page 1737

‘work, work, work till you die’

C. S. Lewis, Surprised by Joy, 1955

The English author, whose most widely read works include the children’s series ‘The Chronicles of Narnia’, seems to have recognized the danger of work more than half a century ago. Supporting evidence appears in the study of Virtanen et al. This is a study from the Whitehall II project examining the association between overtime work and incident coronary heart disease as assessed by the incidence of fatal coronary heart disease, clinically verified myocardial infarction, or definite angina in a large prospectively followed-up cohort of civil servants studied for an average of 11 years. After adjustment for different recognized cardiovascular risk factors, the results indicate a significant influence on fatal and non-fatal coronary heart disease in individuals working 3–4 h overtime per working day. Results were consistent for fatal coronary heart disease, non-fatal myocardial infarction, definite angina pectoris, and all-cause mortality, although the influence was not always statistically significant. Furthermore, for each of these variables, there was a trend for risk to be increased as the number of hours per day of overtime work increased, at least if examined categorically (1 h, 2 h, and 3–4 h). These findings may have implications for cardiovascular risk assessment in the western population and beyond.

A small proportion of the association appears to be explained by type A behavioural pattern which is recognized as a risk factor for coronary heart disease. Employees who undertake overtime may also be more likely to work while ill, a behaviour previously associated with increased risk of myocardial infarction in the Whitehall II cohort.

Overtime work is probably best considered as a form of work stress. Chronic work-related stress is associated with a 2- to 4-fold increase in cardiovascular events, particularly when there is little control over the work environment. Although this study did not suggest a significant relationship, the excess risk of coronary heart disease in employees with high decision latitude was smaller than in those with lesser decision latitude.

The mechanism by which work stress increases the risk of coronary heart disease remains to be determined, but is likely to be complex. However, most of the burden of coronary heart disease can be explained by conventional risk factors such as smoking, diabetes mellitus, hypercholesterolaemia, and hypertension. Cardiovascular disease is unusual in the absence of one or more of these risk predictors. Cardiovascular events due to work stress have been correlated with an increase in total cholesterol, but the most frequently reported association is with high blood pressure. Suboptimal blood pressure is the single most important correctable risk factor for the development of cardiovascular disease, being responsible for about half of all strokes, coronary heart disease, and heart failure. High blood pressure appears to be a cardiovascular risk factor more powerful than suggested previously.

Previous work has pointed to a relationship between extended working hours and high blood pressure. In the population reported by Virtanen et al., baseline readings did not support elevated blood pressure as a mechanism. However, blood pressure was assessed only at a single point, with no readings during follow-up. It seems possible that blood pressure levels increased more depending on the hours of overtime worked.

Earlier studies have also indicated an association between average working hours and increased risk of coronary heart disease. A major concern of such epidemiological observations is reverse causality, i.e. factors that predispose to coronary heart disease, such as early life experience, may influence an individual’s work behaviour. At baseline, the characteristics of employees who did or did not work overtime were little different, but the possibility of residual confounding by other unmeasured or imprecisely measured predictors of coronary heart disease cannot be ruled out.

The authors acknowledge these and other limitations of their study. Modelling of potential confounders as time-independent variables does not allow for the possible impact of changes in these factors and risk of coronary heart disease events. As a predictor of cardiovascular outcomes, baseline blood pressure is far
weaker than achieved blood pressure, and recent evidence suggests that visit to visit variability in blood pressure may be crucial in determining cardiovascular risk. Working hours appeared stable during follow-up, but no information is provided on the work pattern before the start of follow-up. Since this was likely to be similar to that during the observation period, the overtime group at baseline may have reflected ‘survivors’ without coronary heart disease. This, together with a lack of blue collar workers in the study population, limits its generalizability.

Finally, employees with highest risk of coronary heart disease claimed to work 11–12 h per day, a most unusual work pattern certainly in the European context. Also, no information is provided on cardioprotective medication intake by participants. This might well have confounded interpretation of the data generated.

Despite the above reservations, these data from a large occupational cohort reinforce the notion that work stress attributable to overtime is associated, apparently independently, with an increased risk of coronary heart disease. A trend for risk to be related to hours of overtime worked supports this conclusion. If the effect is truly causal, the importance is much greater than commonly recognized. Overtime-induced work stress might contribute to a substantial proportion of cardiovascular disease. However, a crucial question remains. If more comprehensive risk factor assessment were available, would the excess risk associated with overtime be explained or does overtime truly exert an additional independent effect? These are important considerations for health policy and for potential modification through clinical intervention.

A growing body of evidence suggests that working overtime may be associated with adverse health outcomes including hypertension and, now, coronary heart disease. There are strong consistent associations between work stress, cardiovascular risk factors, and outcomes. Therefore, physicians should be aware of the risks of overtime and take seriously symptoms such as chest pain, monitor and treat recognized cardiovascular risk factors, particularly blood pressure, and advise an appropriate lifestyle modification. There is little evidence that strategies to modify stress reduce cardiovascular risk, but further research should examine whether interventions designed to reduce overtime work could alter the risk of cardiovascular disease.

In the long term, the solution may lie in genetics. The identification of genes that affect both the neurological processing of stress and peripheral responses to stress illustrates an individualized stress response and suggests molecular pathways that could provide important therapeutic targets. This raises the attractive prospect of evidence-based interventions. In the meantime, we must rely on government interventions, such as the European Working Time Directive, to reduce hours of work.

Understanding of the relationship between the work environment and health is evolving rapidly, and recent findings suggest an important influence. Those involved in the Whitehall studies have made critical contributions, the latest being the relationship between overtime work and coronary heart disease. A suitable summation is suggested by the words of the great English philosopher, Bertrand Russell: ‘If I were a medical man, I should prescribe a holiday to any patient who considers work important’.

Conflict of interest: none declared.

References


