Job strain and coronary heart disease

The meta-analysis of 13 European cohort studies of job strain and coronary heart disease by Mika Kivimäki and colleagues (Oct 27, p 1491)[1] has several strengths, among which are: analyses of population-based studies, inclusion of ongoing unpublished studies, a large sample, subgroup analyses, and efforts to reduce possible bias owing to movement to less stressful work resulting from subclinical disease.

Nonetheless, the 13 studies have unacknowledged biases towards the null, a problem recognised in a previous review[2] in which 15 of 17 cohort studies had such biases. One form of bias results from the fact that at least two studies included in the meta-analysis (of government employees) did not include industrial workers, who have a higher prevalence of job strain, thus leading to restriction of range of exposure. All of these studies also suffer from two forms of exposure misclassification: the use of median cut points (which are arbitrary) for job demands and job decision latitude to define job strain; and the fact that job strain, an exposure which can change over time, is measured only at baseline and not during follow-up. Additionally, in nine of 11 studies in this meta-analysis, where such data are available, a proportion of the sample became 65 years or older during follow-up. Since job strain is associated with earlier retirement,[3,4] this creates a bias toward the null.

Therefore, the summary effect estimate of 1·23 and a population-attributable risk of 3·4% are likely to be underestimates of the true effect in Europe, and more so compared with industrialising countries such as China.

We declare that we have no conflicts of interest.

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The paper by Mika Kivimäki and colleagues[1] includes two important errors.

First, the population attributable risk of job strain is underestimated because the prevalence of job strain itself (15%) was underestimated. Kivimäki and colleagues report the prevalence of job strain in the WOLF-N/WOLF-F cohorts as 13–16%. However, in the original WOLF study,[2] in which job strain was measured with the standard 11 items, it was 22–28%, similar to the average prevalence of 25% in 31 European countries.[3] This discrepancy might have occurred because Kivimäki and colleagues harmonised job strain measures with fewer items across the 13 cohorts.[4] Also, only three of the cohorts (COPSOQ-I, POLS, and HeSSup) were randomly selected from general working populations with participation rates of more than 50%; most of the others were recruited from white-collar organisations. The prevalence of job strain is generally lower in white-collar than in blue-collar occupations.[5]

Second, Kivimäki and colleagues do not make it clear that they examined only one, albeit important, work stressor (job strain) in relation to coronary heart disease (CHD). Job strain cannot be equated with "workplace stress". Several other important work stressors (long work hours, poor social support, and job insecurity) are reported to be associated with CHD, independent of job strain. Therefore Kivimäki and colleagues' statement that "our findings suggest that prevention of workplace stress might decrease incidence; however, this strategy would have a much smaller effect than would tackling of standard risk factors" is misleading because the issue of the overall effect of work stress on CHD is not appropriately addressed in their paper.

We declare that we have no conflicts of interest.

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Authors' reply

Paul Landsbergis and Peter Schnall suggest that exposure misclassification, owing to the use of a single measure of job strain, could have underestimated associations. We agree; repeat measurements are best to characterise exposures. By the same token, confounding factors are also best assessed with repeat measurements, as we have shown in a different context.[1] Given that our study of job strain and coronary heart disease (CHD) assessed both the exposure and confounders at baseline, it is unlikely to have led to

underestimation. Landsbergis and Schnall also argue that extending follow-up for CHD events beyond the age of 65 years creates a bias towards the null because people experiencing job strain are more likely to take earlier retirement. Such a bias is likely only for studies with extended follow-up; our mean follow-up was 7·5 years. Censoring at retirement might be even more likely to lead to a bias towards the null, given that the greater early retirement in employees with job strain means their follow-up for CHD would end, on average, at a younger age. We agree with Landsbergis and Schnall that use of the median score as a threshold for job demands or job control is arbitrary even though it is widely used.

BongKyoo Choi and colleagues suggest that we underestimated job strain compared with the WOLF studies. In Alfredsson and colleagues' analyses[2] of the WOLF-N and WOLF-S cohort studies, the prevalence of job strain was 24% because both high demands and low control were coded such that the median value was included in these categories. By contrast, our definition did not include the median value (high demand above median; low job control below median), so producing a lower prevalence of job strain (13–16%). Use of Alfredsson and colleagues' definition[2] of job strain in reanalysis of our WOLF-N and WOLF-S data yielded a slightly lower excess risk (hazard ratio 1·16) than our own definition (hazard ratio 1·3), but the population attributable risk (PAR) based on these two definitions (3·7% *vs* 3·9%) was almost identical.

Standard physiological risk factors, such as high blood pressure and dyslipidaemia, have been shown to have a causal effect on CHD in randomised controlled trials.[3] There is also trial evidence that behavioural change, such as smoking cessation, produces a reduction in CHD risk.[3] By contrast, trials of work stress reduction and CHD have not been done, and the evidence of potential mechanisms underlying the association between job strain and CHD remains inconsistent.

Choi and colleagues assert that job strain, although the most commonly used measure of psychosocial stress in published studies, "cannot be equated with 'workplace stress'." They advocate other indicators, including job insecurity. However, in the largest studies to date,[4,5] no robust association was apparent between job insecurity and CHD.

In conclusion, given the relatively weak association between job strain and CHD in our meta-analysis, the smaller PAR associated with job strain than with classic risk factors, and the uncertainty about causality, we stand by our statement that "prevention of workplace stress might decrease incidence; however, this strategy would have a much smaller effect than would tackling of standard risk factors".

We declare that we have no conflicts of interest.

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Transforming public health specialists into public health leaders


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Matthew Day and colleagues (Oct 6, p 1205)[1] suggest recommendations for developing public health leaders. We agree that leadership is a crucial component in public health. However, the proposed focus on super leaders seems outdated in its assumptions.

The King's Fund report "No more heroes"[2] takes a different stance, as does the US National Public Health Leadership Institute.[3] Strengthening leadership capacity within public health could affect the health of the public substantially through ensuring the maximisation of outcomes of public health programmes. Every public health organisation should therefore be engaged in developing more leaders at every level and creating collaborative organisational cultures. Public health education and training needs to be transformative and interdependent.[4] The teaching of leadership is still not common in public health training programmes around the world and seems particularly rare in countries experiencing intensive public health reforms. There is a pressing need for substantial investment in leadership training for public health professionals.

The Association of Schools of Public Health in the European Region, together with the University of Maastricht, Netherlands; the Lithuanian University of Health Sciences; Sheffield Hallam University, UK; the Medical University of Graz, Austria; and Griffith University, Australia, have launched the Leaders for European Public Health (LEPHIE) project. LEPHIE bridges the gap between current academic programmes and the need for effective public health leaders through development of an EU-centric competencies framework, applying blended and problem-based learning pedagogies and creating cross-cultural educational experiences. The programme aims

For the **LEPHIE project** see http://www.lephie.eu/about-lephie.html