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(Oct 27, p 1491)[1] has several strengths, among which are: analyses of population-based studies, inclusion of ongoing unpublished studies, a large sample, subgroup analyses, and efforts to reduce possible bias owing to movement to less stressful work resulting from subclinical disease.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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1 Kivimäki M, Nyberg ST, Batty GD, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. *Lancet* 2012; **380:** 1491–97.
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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.

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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