We were surprised to read that longstanding debates on the association between job strain (high-demand and low-control work) and coronary heart disease “were resolved” as the result of 1 meta-analysis of 13 cohort studies using “an individual-participant data meta-analysis approach” (1, p. 1). In fact, the conservative estimate of association provided was likely the consequence of biases towards the null in the 13 studies that were unacknowledged in the meta-analysis (2). A previous review (3) documented that 15 of 17 cohort studies of job strain and cardiovascular disease were dominated by biases towards the null.

First, restriction of the range of exposure created a bias towards the null. Only 3 of the 13 cohort studies in the meta-analysis (2) were randomly selected from general working populations in which the participation rates were greater than 50%; most of the others were recruited from white-collar organizations (4). The prevalence of job strain is generally lower in white-collar occupations than in blue-collar occupations (5). Workers in the most stressful jobs (industrial, sweatshop, and temporary workers and undocumented immigrants) were much less likely to be included in the 13 studies. Workers with job strain are also less likely to participate in research studies (6). Although previous studies suggested that there was a stronger association between job strain and coronary heart disease in lower (vs. higher) socioeconomic groups (7, 8), the meta-analysis by Kivimäki et al. (2) suggested that there was a stronger association (although no significant effect modification) in higher socioeconomic groups, raising questions about the potential impact of restricted occupational variance in the 13 cohorts. Moreover, the meta-analysis included studies only from Northern Europe, thus severely limiting its external validity. The focus on Scandinavian cohorts (51% of person-years in the meta-analysis) is problematic, given the weaker associations observed between work stressors and depression symptoms in Scandinavian countries compared with other European countries (9). Therefore, the summary effect estimate of 1.23 (95% confidence interval: 1.10, 1.37) and population attributable risk of 3.4% are likely to be underestimates of the true effect not only in Northern European countries but also in other European countries and even more so in industrializing countries such as China, which are likely to have a much higher prevalence and severity of job strain.

Second, all 13 cohort studies suffered from 2 forms of exposure misclassification: the use of median cut points (which are arbitrary) for job demands and job control to define job strain and the measurement of job strain, an exposure that can change over time, only at baseline and not at follow-up. Additionally, in 9 of 11 studies in the meta-analysis in which such data were available, a proportion of the sample became 65 years of age or older during follow-up. Because job strain is associated with earlier retirement (10, 11), this creates an additional bias toward the null. Although the authors (1) cited the Whitehall II study (1 of the 13 cohort studies) as an example of weaker associations between job strain and coronary heart disease associations in older (vs. younger) age groups (12), in fact, Whitehall II is an example of risk attenuation due to retirement. There is a much weaker association in the group aged approximately 62–72 years at the end of follow-up than in the group aged 49–61 years at the end of follow-up, a limitation acknowledged by the authors of that Whitehall II paper (12).

In summary, despite the promise of individual-participant data meta-analyses, such analyses are limited by the limitations of the included studies and may simply not be affordable or feasible in many areas of the world. Therefore, the longstanding debates in the job strain literature remain unresolved, awaiting a more comprehensive meta-analysis of the many existing cohort studies of this association that were not included in the current meta-analysis (3, 13).

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References


2. Kivimäki M, Nyberg ST, Batty GD, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis

THE AUTHORS REPLY

We are delighted to respond to the points raised by Choi et al. (1) and Landsbergis et al. (2) in their letters regarding our article (3). The commentators suggested that a meta-analysis including previously published studies, in addition to data from the Individual-Participant-Data Meta-analysis of Working Populations (IPD-Work) Consortium (n = 197,473) (4), would be essential to evaluate the association between job strain and coronary heart disease. This meta-analysis has already been published (5). The summary hazard ratio for developing coronary heart disease for job strain versus no job strain based on 26 independent prospective cohort studies from Europe, the United States, and Asia was 1.3 (95% confidence interval: 1.2, 1.5), that is, consistent with that from the IPD-Work Consortium alone (1.2; 95% confidence interval: 1.1, 1.4) (4).

Our commentators noted that the use of the median split to define job strain in the IPD-Work Study was arbitrary. Although this is certainly true, it is also the case that this measure is the most widely used approach to operationalize job strain in the literature. Landsbergis et al. have previously introduced multiple alternative (though equally arbitrary) measures of job strain: the quotient, the quadrant term, the quadrant term using national means, and linear term formulations (6). However, they do not indicate which measure, in their opinion, should be used.

The lack of standard measures, even after 3 decades of research on the job-strain theory, is indeed a major problem potentially encouraging post hoc decisions, such as selective reporting of findings based on the operationalization that provides the strongest associations. This would lead to an evidence base over-represented by false positive findings. To minimize this bias in the IPD-Work Consortium, we published our harmonized operationalization of job strain before obtaining data on coronary heart disease from the participating studies (7).

The lack of standard definitions for job strain also complicates the interpretation of null findings. In any new publication, a null finding is not necessarily viewed as adding to scientific knowledge, as it may be interpreted as a “false negative” because of the use of nonoptimal measures. It seems that our commentators followed this reasoning, implying that a stronger association with coronary heart disease would have been found if some alternative measures of job strain were used. Our reviewers for the IPD-Work paper (4) requested subsidiary analyses with the quadrant method and separately for job demand and job control components. As reported in the appendix of that article, the associations with coronary heart disease were not stronger for these alternative measures (4).

Our commentators’ argument that the use of white-collar cohorts in addition to those from the general working population caused underestimation of the job strain association with coronary heart disease is not supported by the meta-analysis; the hazard ratios for job strain were similar for those in high versus low socioeconomic positions (4). One way of illustrating the relatively modest role of job strain in coronary heart disease etiology is to compare the associations with other risk factors within the same cohort. In the IPD-Work cohort, the population attributable risk for an unhealthy lifestyle (i.e., 2 or more of the following: smoking, heavy alcohol consumption, obesity, and physical inactivity) was 7 times greater (26.4%) than the population attributable risk for job strain (3.8%) (8). That IPD-Work includes some white-collar cohorts cannot explain the relative difference in population attributable risks between job strain and lifestyle factors because the better health of...