



Job strain and clinical depression

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The IPD-Work consortium recently published a study in Psychological Medicine entitled ‘Job strain as a risk factor for clinical depression: systematic review and meta-analysis with additional individual participant data’ (Madsen *et al.* 2017). The authors concluded that ‘Job strain may precipitate clinical depression among employees’. We question this conclusion and the job strain definition used by the IPD-Work consortium.

Does job strain precipitate clinical depression among employees?

The meta-analysis in the IPD-Work study included six prospective studies. Odds ratios (OR) of incident clinical depressions ranged from 1.04 to 2.52 with a pooled OR estimate of 1.77 [95% confidence interval (CI) 1.47–2.13]. None of the studies adjusted for symptoms of depression at baseline or previous clinical depression, most likely causing inflated risk estimates. Four of the studies adjusted for no more than age, gender, and marital status, and only two of the studies adjusted for socioeconomic status (SES). Furthermore, they used four different job strain definitions. In our view, a meta-analysis of these six heterogeneous studies is not very meaningful, even if there was no statistically significant heterogeneity in the pooled estimate. Furthermore, this estimate was most likely inflated.

In the analyses of unpublished IPD-Work data job strain was associated with hospitalization for depression with hazard ratios (HR) ranging from 0.42 to 2.14 and a pooled HR estimate of 1.27 (95% CI 1.04–1.55), adjusted for age, sex, cohabitation, and SES. When further adjusted for depressive symptoms at baseline, this association between job strain and clinical depression disappeared (HR=1.03, 95% CI 0.81–1.32). Owing to a bi-directional relation between job strain and depressive symptoms the authors argue that ‘the observed association might overestimate the causal effect of job strain on depression, although the association is unlikely to be fully attributable to confounding’. However, considering the small extra risk associated with job strain and that the

lower 95% confidence limit was close to unity, even a small overestimation of the observed association would leave little if any support for a ‘causal effect of job strain on depression’.

The quadrant strain model

The hallmark of Karasek’s job strain theory is a *joint* effect of demands and control, originally described as an interaction between the two variables (Karasek, 1979; Karasek *et al.* 1988), later modified to be satisfied with only additive effects (Karasek, 1989). Thus, according to the job strain theory work-related stress does not result from a single factor, e.g. demands or control separately, but from the joint effects of both factors in conjunction (Karasek *et al.* 1988).

The IPD-Work protocols rely on the so-called quadrant definition of job strain. The demands and the control scales are dichotomized at their medians to form four combinations, of which the group with high demands and low control is defined as high strain and the rest as low strain.

The quadrant approach does not examine if there is an interaction between demands and control and only formally examines if there is a joint effect of demands and control. It accepts that an effect of job strain may be due to an effect of only demands or of only control. In our view it is meaningless to talk about a joint effect of demands and control, if the effect is only due to one of the two factors.

Thus, the quadrant approach is not consistent with neither the original interaction model of job strain nor with a reduced model claiming only an additive effect of demands and control.

The IPD-Work consortium made an alternative analysis that assessed the independent effects of demands and control and their interaction. This analysis showed no interaction effect between demands and control, demands had no significant effect, but control was associated with an increased risk of clinical depression. Thus, there was no joint effect of demands and control, and consequently no effect of job strain according to the job strain theory. The result of this analysis was not mentioned in the discussion.

The operationalization of job strain is not only a matter of academic interest but may have important practical and economic implications in terms of prevention. Thus, according to the IPD-Work conclusion, preventive measures should be implemented and directed toward high demands as well as low control. We question this conclusion as the analysis of separate

independent effects showed that it may be more efficient to direct preventive measures only toward low control. However, since the latter analyses were not adjusted for depressive symptoms at baseline the justification for any intervention is uncertain.

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Declaration of Interest

None declared.

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