Update on Work-Related Psychosocial Factors and the Development of Ischemic Heart Disease

A Systematic Review

Jan Hyld Pejtersen, MSc, PhD, Hermann Burr, MA, PhD,† Harald Hannerz, MSc, PhD,‡ Alba Fishta, MD, MPH,† and Nanna Hurwitz Eller, MD, DMSc§*

Abstract: The present review deals with the relationship between occupational psychosocial factors and the incidence of ischemic heart disease (IHD) with special regard to the statistical power of the findings. This review with 4 inclusion criteria is an update of a 2009 review of which the first 3 criteria were included in the original review: (1) Study: a prospective or case–control study if exposure was not self-reported (prognostic studies excluded); (2) Outcome: definite IHD determined externally; (3) Exposure: psychosocial factors at work (excluding shift work, trauma, violence or accidents, and social capital); and (4) Statistical power: acceptable to detect a 20% increased risk in IHD. Eleven new papers met the inclusion criteria 1–3; a total of 44 papers were evaluated regarding inclusion criteria 4. Of 169 statistical analyses, only 10 analyses in 2 papers had acceptable statistical power. The results of the 2 papers pointed in the same direction, namely that only the control dimension of job strain explained the excess risk for myocardial infarction for job strain. The large number of underpowered studies and the focus on psychosocial models, such as the job strain models, make it difficult to determine to what extent psychosocial factors at work are risk factors of IHD. There is a need for considering statistical power when planning studies.

Key Words: ischemic heart disease, job strain, skill discretion, decision authority, social support, psychosocial factors, work stress, work, epidemiology, statistical power

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The present review deals with occupational psychosocial risk factors for ischemic heart disease (IHD). It aims to discuss critically the psychosocial risk factors for IHD based on the statistical power of the reviewed papers. As a critical discussion of psychosocial factors has been done by Eller et al[1] in 2009, the present review is an update of that review, with the additional evaluation of statistical power.

Eller et al[1] concluded in 2009 that for males, there was moderate evidence for high psychological demands, low social support, and iso-strain being risk factors for IHD. As a number of studies in the review by Eller et al[1] did not find influence at work to be a risk factor for IHD, the review suggested that the association of job strain with IHD could be explained by the demands dimension of job strain. Additionally, the review concluded that there was insufficient evidence that effort reward imbalance, job insecurity, and long working hours were risk factors for IHD, due to a limited number of studies.[1] Among women, the review was inconclusive regarding psychosocial risk factors as too few studies were carried out.

It has been suggested that many studies of IHD and psychosocial factors might be too small to detect a possible association,[2] as the random error can be as relevant for examining scientific evidence as the systematic error. The standard error (Stderr) is an approximate measure of the expected distance between the obtained estimate and the true value. If the Stderrs are large, then we can expect our study results to be far from the truth. The larger the Stderr, the lower the statistical power (the probability that the test will yield a statistically significant result if the hypothesis is true.). The lower the statistical power, the higher the probability that the finding will remain unpublished due to being inconclusive. Hence, if the Stderrs are large and the statistical power is low, and the results are published, then, due to the combined effect of the random and the systematic error (publication bias), we can expect the study results to be far from the truth.[3] Thus, statistical power is important to take into account when we evaluate published research results—even if they are statistically significant. In the review by Eller et al,[1] the systematic errors were the main focus. Therefore, in the present review we will also consider the robustness of the findings.

MATERIALS AND METHODS

Inclusion Criteria

We considered papers with the first 3 inclusion criteria, as in Eller et al,[1] and a fourth criteria set up for the present review. The inclusion criteria were (1) the study should be a prospective or case–control study if exposure assessment was not self-reported (prognostic studies excluded); (2) the outcome should be definite IHD determined externally; (3) exposure should be an occupational psychosocial factor (excluding the factors of shift work, night work, unemployment, trauma, violence, accidents at work, or social capital); and (4) the analyses in the papers should have acceptable statistical power (80%) to detect an increased risk of IHD of 20% due to the work environment.

First, we carried out a literature search for papers meeting the first 3 criteria above to update the pool of the papers from the original review.[1] Second, we determined all analyses in the new papers the same way as in the original review, including giving each paper a point score. Third, we calculated the statistical power of the analyses of all papers in the updated pool to determine which of the papers should be included in the review according to inclusion criteria 4.

Literature Search

In the Thomson Reuters Web of Knowledge, we searched all original papers in English since January 1, 2007 (search date April 28, 2013). We did 7 searches. First, we made 5 searches of papers which cited reviews on the topic since 2004[1,4–6] or 1 meta-analysis.[7]

From the *The Danish National Centre for Social Research, Copenhagen, Denmark; †Federal Institute for Occupational Safety and Health (BAuA), Berlin, Germany; ‡National Research Centre for the Working Environment, Copenhagen, Denmark; and §Department of Occupational and Environmental Medicine, Bispebjerg University Hospital, Copenhagen, Denmark.

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Correspondence: Jan Hyld Pejtersen, MSc, PhD, The Danish National Centre for Social Research, Herluf Trolles Gade 11, DK-1052 Copenhagen K, Denmark. E-mail: jhp@sfi.dk.

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Second, we made 2 additional searches: all papers mentioned in the latest review[6] and in the most recent meta-analysis.[8] We included only papers that met inclusion criteria 1–3 regarding study type, outcome, and exposure. In all, we found 526 papers of which 124 were duplicates, resulting in 402 unique papers. The abstracts of these papers were scrutinized and 369 papers were excluded as they did not meet the inclusion criteria. We obtained the full text of 33 papers; of these, 20 did not meet the inclusion criteria. Of the remaining 13 papers, 2 were already included in the original review by Eller et al,[1] thus 11 papers were added to review. In the review by Eller et al,[1] 33 papers were considered, therefore a total of 44 papers were included in the updated review.

Of the 44 included papers, 26 (6 new) originated in the Nordic countries, 8 (1 new) were from the United States, and 5 (3 new) were from England. Germany, Belgium, and Japan were represented by 1 paper each (no new), and 2 papers (1 new) were based on populations from several countries, the first used data from Belgium, France, Spain, and Sweden, the second—the new paper—used data from Finland, the Netherlands, England, Sweden, Denmark, and Belgium.

Evaluation of Papers

Each new paper was evaluated by means of a scale going up to 11 for prospective papers and up to 10 for case–control papers as in the original review.[1] The following criteria were evaluated: validity of exposure assessment, validity of end point assessment, treatment of prevalent cases, heterogeneity of the patient population, age composition of the population, length of follow-up, gender separation, and richness of adjustment for confounding variables.

Power Calculations

We considered a 20% increase in risk of IHD due to work environmental exposures to be an important effect, and we wanted to know the power to detect such an effect in the 44 individual papers found. In our opinion, a 95% power is desirable and an 80% power is acceptable. For each paper, we calculated the power to detect a rate ratio of 1.2 through the following procedure.

When available, the confidence interval of a published rate ratio was used to estimate the Stderr of its logarithm by means of the equation

$$\text{Stderr} = \frac{\log(\text{UpperCL}) - \log(\text{LowerCL})}{2\Phi^{-1}(1-\alpha)} \quad (1)$$

where UpperCL is the upper confidence limit and LowerCL is the lower confidence limit of a $100(1 - 2\alpha)\%$ confidence interval. The power to detect a rate ratio of 1.2 as a function of Stderr and α was thereafter approximated by the equation

$$\text{Power} = \Phi\left(\frac{\log(1.2)}{\text{Stderr}} - \Phi^{-1}(1-\alpha)\right) \quad (2)$$

where Φ is the standard normal distribution function.

For papers, which gave the expected number of cases but no confidence interval, we replaced Stderr in Equation 2 with

$$E[\text{Stderr}] = \sqrt{\frac{1}{e_1} + \frac{1}{e_2}} \quad (3)$$

where e_1 is the expected number of cases among the exposed and e_2 is the expected number of cases in the comparison group. Three of the papers contained neither of the above,[9–11] but it was obvious from the extraordinary low number of cases that the power would be <0.1.

In some cases the expected direction of the relationship between compared groups was reversed: for example, the rate ratio between workers with low vs high control was expected to be >1, but the rate ratio of high vs low control was expected to be <1. In such cases, a rate ratio was deemed clinically important if it was ≤1/1.2. Because the standard normal probability density function is symmetric around zero, the power to detect a rate ratio of 1/1.2 would be equal to the power to detect a rate ratio of 1.2. The equations are based on the central limit theorem and Gauss' propagation of error formulas. The derivation of the power formula is given by Bickel and Doksum.[12]

RESULTS

We were able to calculate the power for 169 significance tests out of 170 tests in 44 papers (an overview of the power analyses can be obtained from the authors). It was not possible to calculate the power in 1 test due to insufficient information.[13] Thirty-six tests were found in 9 prospective studies with aggregated data,[13–21] 111 were found in 28 prospective studies with self-reported data,[8–11,22–45] and 22 were found in 7 case-control studies.[46–52] Sixty-six of the tests in 31 papers[8,14,15,17,19–23,25,27,29–32,34–36,38–46,48,50–52] were statistically significant and 63 of these pointed in the hypothesized direction. Only 2 tests in 1 paper pointed in the opposite direction of the hypothesized.[19]

The statistical power to detect a rate ratio of 1.2 ranged from 0.04 to 0.99. The median power was 0.11. The power was acceptable (≥80%) in 10 of the tests in 2 papers[8,50] and exceeded 95% in 7 of them. Four of the acceptably powered tests were performed in a multicenter study by Kivimäki et al,[8] whereas the remaining 6 tests were performed in a case-control study by Hammar et al[50] using a job exposure matrix.

In the papers meeting the first 3 inclusion criteria regarding study type, outcome and exposure, many analyses dealt with job strain, some with effort reward imbalance and working hours, a few studies were looking at job insecurity, leadership quality, and predictability. However, in the 2 papers also meeting the inclusion criteria regarding statistical power, only job strain, demands, job control, influence at work, and social support were investigated (Table 1).

In the acceptably powered analyses, Kivimäki et al[8] found that self-reported job strain was a risk factor for incident myocardial infarction (MI) when controlled for gender and age in a pooled cohort from of 197.000 employees from Finland, the Netherlands, England, Sweden, Denmark, and Belgium (Table 1). The association remained significant when further controlled for socioeconomic status. Analyses controlling also for health behaviors and the Framingham score did not cover all the cohorts involved, and were thus underpowered. The paper also looked into the separate dimensions constituting job strain, namely demands, and control. The paper found that demands at work were not associated with IHD, but that low control was negatively associated with IHD. The paper also looked at job strain in analyses stratified by gender and publication status of the studies involved. Both these analyses were underpowered.

In the other acceptably powered paper of 10,008 cases and 28,466 controls, Hammar et al[50] found that high demands was not associated with IHD, but that low decision latitude (part of the dimension control) was (Table 1). These analyses of risk factors on the occupational level were only able to control for country of residence and age; the analyses were stratified for gender. Additionally, it was found that low social support among males was associated with incidence of MI.

DISCUSSION

The present review has 2 main findings. First, the overwhelming number of statistical analyses of psychosocial risk factors for MI

TABLE 1. Power Calculations to Detect a 20% Increased Risk of IHD for Analyses Published in the Papers Meeting Inclusion Criteria Regarding (1) Study Type, (2) Outcome, (3) Exposure, and (4) Power

Study	Quality Score	Gender	Risk Factor	Rate Ratio	95% Confidence Interval	Statistical Power
Kivimäki et al 2012[8]	9	M/F	**Job strain**	**1.23**	**1.10–1.37**	**0.90**
		M*	Job strain	1.29	1.13–1.48	0.75
		F*	Job strain	1.46	1.07–1.99	0.21
		M/F*	Only employees aged <50 years	1.29	1.08–1.54	0.52
		M/F*	**Job strain, control for SES**	**1.17**	**1.05–1.31**	**0.90**
		M/F*	Job strain, control for health behaviors	1.21	1.03–1.44	0.57
		M/F*	Job strain, control for Framingham score	1.42	1.16–1.74	0.42
		M/F	*2SD increase in job demands, low*	*1.04*	*0.92–1.17*	*0.86*
		M/F	**2SD increase in job control, high**	**0.86**	**0.79–0.96**	**0.96**
		M/F	High strain vs low strain	1.28	1.11–1.48	0.70
Hammar et al 1998[50]	6	M	**Low decision latitude**	**1.37**	**1.25–1.50**	**0.98**
		M	*High demands*	*0.93*	*0.89–1.02*	*1.00*
		M	**Low social support**	**1.28**	**1.17–1.41**	**0.97**
		M	ISO-strain	1.35	1.16–1.58	0.64
		F	**Low decision latitude**	**1.12**	**1.05–1.19**	**1.00**
		F	*High demands*	*0.95*	*0.89–1.01*	*1.00*
		F	*Low social support*	*1.10*	*0.99–1.17*	*0.99*
		F	ISO-strain	1.31	0.99–1.73	0.25

Risk factors in **bold:** Sufficiently powered significant association. Risk factors in *italic*: Sufficiently powered insignificant association.
SD indicates standard deviation; SES, socio economic status.
*Subsample.

in the 44 studies identified failed to have sufficient power for finding an excess risk of 20%, and only 2 contributions contained acceptable power (>0.8). The results confirm that in occupational psychosocial IHD epidemiology, the populations studied have been small.[2] Second, the results of 2 papers with sufficient power pointed in the same direction, namely that the control dimension of job strain seems to explain excess risk for MI for job strain. In the published literature, the robustness of findings had not been tested.[1] Little is known about the relation between other aspects of psychosocial working conditions and IHD.

In the present study, we defined a critical value of 1.2 for a rate ratio to be clinically significant and we determined that an 80% power to detect such a rate ratio would be acceptable. The 80% power is a convention, but the critical value for clinical significance was an arbitrary decision. To allow for possible effects of selection bias, misclassifications and uncontrolled confounding in observational cohort studies, Monson recommends epidemiologists to interpret rate ratios in the open interval 0.9–1.2 as "no association."[53] In keeping with this recommendation, we did not want our critical value to be <1.2. We recognize that death or hospital treatment due to coronary heart disease is a serious endpoint and that effects may be of clinical importance even if they are smaller than our critical value. For example, in a meta-analysis, Ha et al[54] estimated rate ratios of IHD for occupational factors which they deemed to be important. The rate ratios were thereafter used to estimate the attributable fraction of work environmental exposures upon the incidence of death due to IHD in Korea. For the occupational factors considered among male workers, they estimated the following rate ratios: noise 1.06, environmental tobacco smoke 1.19, shift work 1.12, and low job control 1.15. All of these individual rate ratios were lower than 1.20, yet the combination of the concerned exposures were estimated to be the cause of nearly 10% of all deaths due to IHD among men aged 15–69 years in Korea. If we would have required that, the studies should have had a power to detect 10%, only 2 analyses—for demands and influence at work among women[50]—would have had sufficient power to detect such a low risk. It might also be that the choice of excess risk for the power calculation might be too restrictive if risks for some psychosocial factors should be much higher. However, if we would have required an excess risk for our power calculations of 40%, only 6 further analyses from 2 additional papers[14,38] and 4 analyses in subpopulations of the already included papers[8,50] would have had acceptable power. Alfredsson et al[14] found that hectic and monotonous work was associated with IHD, and Väänänen et al[38] found that low influence at work and low predictability was associated with IHD. The first of these findings is in line with both Hammar et al[50] and Kivimäki et al,[8] whereas the second finding is new. Kivimäki et al[8] found that job strain was a risk factor among males, younger workers (<50 years) and among all workers controlled for the Framingham score. Hammar et al[50] also found isostrain (a combination of high demands, low control, and low support) to be associated with IHD.

We used the same power criteria for the studies with aggregated data as we did for the ones with person-based self-reported data. However, it should be noted that the studies with aggregated data estimated rate ratios between groups with a "high" vs a "low" proportion of exposed workers, whereas the ones with person-based data estimated rate ratios between exposed and nonexposed workers.

If RR_p is the person-based rate ratio, then the rate ratio between groups with p_1 and p_2 percent exposed workers, respectively, is given by the equation

$$RR_G = \frac{p_1 RR_P + (100 - p_1)}{p_2 RR_P + (100 - p_2)}$$

If, for example, the person-based rate ratio equals 1.2 and 60% of the workers in the high group are exposed while 10% in the low group are exposed, then the rate ratio of the aggregated data would equal 1.1. One may therefore argue that we should have used a different cut-point for clinical significance in aggregated analyses than in studies of person-based exposure data. We chose, however, not to put the critical value of any test lower than 1.2, in accordance with the recommendations given by Monson.[53]

We did not find an association between the power of the studies and the scoring of the study quality constructed by Eller et al,[1]

possibly due to 2 issues. The main issue is that the scoring did not prioritize the simplicity needed when power is an issue; first, it favored gender stratified analyses, second, it required elaborated control for a number of risk factors. So the review by Eller et al[1] focused on systematic error, not on random error.

A null finding in an insufficiently powered study does not indicate much. A null finding in a well-powered study, on the other hand, indicates the absence of a clinically important effect. A well-powered study is therefore more likely to be published regardless of its outcome, whereas a low-powered study may be difficult to publish unless it is statistically significant. "Publication bias arises whenever the probability that a study is published depends on the statistical significance of its results."[55] To circumvent the problem of publication bias in small sample studies, we chose to only include well-powered studies in our final analysis.

It is too early to conclude to what extent psychosocial factors at work are associated with IHD. Since our literature search in April 2013, it has been found in a well-powered study that perceived job insecurity was associated with IHD.[56] The focus on psychosocial aspects should be widened to include more than job strain and effort reward imbalance. Also, there is a need for testing the assumptions of the job strain model, namely that there is an interaction between demands and control. The 2 sufficiently powered papers[8,50] in the updated review failed to find that high demands was a risk factor. It might have to do with the complexity of demands as defined by Karasek,[57] where work pace, quantitative demands, role clarity, conflicting demands, and physical demands constitute the demands scale. For example, different types of demands do not occur in the same occupations and do not have the same effects on health.[58]

Three approaches seem to be appropriate for overcoming the power issue. The first is to use large studies, that is, multicenter studies.[2] To make such studies possible, dimensions of the psychosocial working environment should be measured in the same way across studies. This issue should be taken into account when developing questionnaires. The second way is to carry out job exposure matrix-based studies. The problem is that some psychosocial conditions, such as management and social support, are less associated with occupation.[59–62] The third way could be to look for early objective markers for cardiovascular disease, which could increase power of individual studies. This requires that early markers, such as prescribed medications, are validated as indicators of IHD. However, social bias in access to the health system might make use of such indicators less promising. Validations of such markers for IHD are therefore needed.

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