A cocktail fallacy in research with composite variables: a commentary on job strain and effort–reward imbalance (ERI) theories with updated meta-analyses on health outcomes

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Abstract

Job strain and effort–reward imbalance are two widely used theories in occupational stress research. In this paper we show that a large body of research based on these theories needs to be reevaluated. We take a non-technical approach to show that the use of *single parameter composite variable models* to test the job strain and effort–reward imbalance theories is insufficient. Further, we describe how to use standard two or three parameter models to investigate associations proposed by these theories. We present a method for recalculating estimates from a particular composite variable model so they represent proper estimates from a three parameter interaction model, and apply that method on estimates from five large studies on health outcomes. We also present an updated meta-analysis on job strain and coronary heart disease (CHD) with over 400k subjects and >6.8k CHD cases using such recalculated estimates. The results show no support for the job strain theory, indicating instead that low job control alone is associated with an increased risk of CHD. We conclude that further research on the job strain and effort-reward imbalance theories using single parameter composite variable models is untenable and that a constructive avenue for occupational stress research is to better understand the association between low job control and adverse health outcomes.

Introduction

There is a need to reevaluate a large body of occupational stress research concerning two widely used theories in the field: the job strain¹ and effort–reward imbalance² theories. In research on these theories a standard practice has emerged to estimate complex effects, which depend on two or more factors, from a *single parameter composite variable model*, instead of using standard two or three parameter models. The use of such *composite variable models* has obscured conclusions about what reported associations mean; despite more than 40 years of research on job strain, with thousands of published papers, a debate continues about whether job strain is an interaction between work demand and work control, or merely an additive effect of both demand and control^{3–5}.

While occupational stress researchers debate whether job strain is an interaction or an additive effect of the two constituent factors, there is also the possibility that it is neither⁶. Composite variable models are susceptible to spurious associations, and may show an association if only one of the constituent variables is associated with the outcome. Simple univariate associations can therefore erroneously be offered as evidence for a more complex theory, such as the job strain and effort–reward imbalance theories. This problem has been discussed before^{6–13}, but the practice of using single parameter composite variable models continues unabated, as indicated by our current review presented below, where we found that none of 20 eligible studies published on job strain and coronary heart disease had used proper interaction models.

The primary aim of this paper was to demonstrate that continued use of single parameter composite variable models to test theories such as the job strain and effort–reward imbalance theories is untenable. We take a non-technical approach, using illustrative examples and figures as well as providing fully annotated R code, to demonstrate a common (cocktail) fallacy in job strain and effort–reward imbalance research using composite variable models. We also present proper two and three parameter models that should be used to provide evidence for (or against) the job strain and effort–reward imbalance theories.

Not all composite variable models are the same, and this is particularly true for job strain research where a common approach has been to split data into four quadrants, and fit a model that is *equivalent to* (but not the same as) the fully specified three parameter interaction model. This means that some published estimates can be re-calculated to represent (unbiased) estimates from the proper interaction model. A secondary aim of this study was to present the tools for recalculating such estimates. No meta-analysis to date has recognized the need to correct this bias in published estimates. Here, we provide the first publication of such estimates of previously published large studies on several different health outcomes, as well as an updated meta-analysis on job strain and coronary heart disease.

The job strain and effort–reward imbalance theories

The job strain and effort–reward imbalance theories are similar in many ways. Both are occupational stress theories used to predict health outcomes and both are based on two independent factors that *interact* to produce a negative effect of exposure. The job strain theory was originally formulated in 1979¹ by Robert Karasek, who proposed (p 285):

This model predicts that mental strain results from the interaction of job demands and job decision latitude.

We will refer to *decision latitude* as *job control* throughout this text. Job strain theory predicts that high job demands produce more adverse health effects when job control is low than when it is high. Thus, job control is proposed to *modify* the association between job demand and health outcomes. This is why Karasek modeled the effect using the standard three parameter interaction model in his original paper. However, the interaction was found to *not* be statistically significant. Karasek then created two custom “interaction” terms, using a more complex mathematical function, with which he could demonstrate a significant effect, but these formulations of the job strain theory are never seen in contemporary research.

The effort–reward imbalance theory (ERI)² is methodologically similar to job strain theory but the functional form of the proposed interaction is a ratio of effort/reward¹⁴. Here, *effort* represents the workload leading to adverse health outcomes, modified by *reward*. The suggested mechanism is more elaborate, with roots in social psychology and the theory of reciprocity (p 1484)¹⁹:

This model assumes that effort at work is spent as part of a contract based on the norm of social reciprocity where rewards are provided in terms of money, esteem, and career opportunities including job security.

To illustrate the methodological similarities between ERI and the job strain model, we illustrate both in figure 1 (top and middle panel) assuming two-level/binary data. We will later discuss models with continuous data, which is a more common approach in ERI research.

A cocktail fallacy in job strain and ERI research

A main challenge in interpreting results from composite variable models is that an observed association may depend on a single univariate association with just one of the constituent variables. Here we will use a non-technical illustration of the problem, and consider the popular cocktail *Gin & Tonic*. The cocktail has two main constituent ingredients, gin and tonic, and is a composite, the cocktail *Gin & Tonic*. If someone drinks *Gin & Tonic* and then feels a little intoxicated, what is the cause? Is there an interaction between *Gin & Tonic* that makes them intoxicated? Is it the combined (additive) effect of both gin and tonic? Or is it just one of the constituent ingredients on its own, e.g. gin, that is the active ingredient causing intoxication?

The problem is similar in job strain and ERI research. Studies on coronary heart disease (CHD) indicate that *low job control* but not high job demand^{6,12,15,16}, and *low reward* but not high effort^{9,17}, are associated with CHD. Thus, the job strain and ERI theories can also be hypothesized to have one “active ingredient”, *control* and *reward*, similar to the *gin* in the *Gin & Tonic* model discussed above.

To see why this is a problem let's first consider the *binary job strain* composite variable model, as it was defined by Kivimäki et al (p 1492)¹⁵:

We defined exposure as job strain (high demands and low control) versus no strain (all other combinations) according to the job-strain model.

As we can see in figure 1, this model is equivalent to comparing a group drinking Gin & Tonic to a mixed group drinking only gin, only tonic, or nothing at all. It would not be surprising to find the Gin & Tonic group more intoxicated on average, because all of them consumed gin while most in the reference group did not. There is no need for an *interaction* between gin and tonic to be present, and no need for both gin and tonic to *additively* contribute to intoxication, in order to observe such an outcome.

As illustrated in figure 1, the above discussion also applies to a binary ERI composite variable (p 621)¹⁷:

We calculated the effort–reward imbalance score as the ratio of the harmonized effort scale divided by the harmonized reward scale. Values >1 indicate that effort exceeds reward (an effort–reward imbalance). The score was then dichotomized (effort–reward imbalance versus no effort–reward imbalance).

Another popular model in job strain research is the *quadrant* model (p 149)¹⁸:

Four quadrants of job strain (high demand and high decision latitude; high demand and low decision latitude; low demand and high decision latitude; low demand and low decision latitude) were constructed, by cross tabulating job demands and decision latitude, both divided in two groups at the median.

This is the model that the IPD-Work Consortium proposed when defending against criticism¹⁹:

We report separate risk estimates for each combination of occupational demands and control, taking the combination of “neither high demands nor low control” as the reference.

The “low demand and high control” group (i.e. the *relaxed* quadrant) was used as the reference when estimating the association with “job strain”. In figure 1, we can see that this is equivalent to comparing a group drinking Gin & Tonic with a group that had nothing to drink at all. Again, one would not be surprised to find the Gin & Tonic group more intoxicated, but it would be fallacious to attribute intoxication to both gin and tonic, or to an interaction between gin and tonic.

Continuous models

The principles discussed above also apply to continuous data. Figure 2 illustrates various composites based on subtraction, addition, multiplication and division, where one of the constituent variables is correlated with the outcome (A) and one is not (B). In all cases the correlation of A transfers to the composite variable. This demonstrates that composite variables are susceptible to bias, and that observed associations can be fully explained by a single univariate association with one of the constituent variables.

The ratio variable used in effort–reward imbalance (ERI) research¹⁴ is no exception (figure 2, bottom panels). If we consider the Gin & Tonic model discussed above, the “active ingredient” is represented by variable A, and the bottom right panel would represent ERI when the association is fully explained by a univariate association with *reward*, as has been recently shown in a large meta-study⁹. Dichotomising this ratio variable at $ERI > 1$ ¹⁷ in order to produce a binary exposure that is popular in the literature (similar to figure 1) does not remove this bias.

How to avoid the cocktail fallacy when estimating combined effects of two exposures

In order to estimate interactions or combined effects of two exposures without introducing bias, we need to include the constituent main effects in the model^{20,21}. Associations of main effects and interactions are then estimated simultaneously in the same model, making it possible to determine if any, or both, of the main effects are associated with the outcome, and whether an interaction is present.

The job strain model is straightforward and described in equation 1 below, where $f(y)$ is a generic link function that should be adapted to the data at hand (e.g. a logit link for logistic regression or an identity link for linear regression), and β are regression parameters. The ERI model may at first glance appear different because it is a ratio, but a ratio is a product where the denominator has been inverted; so, inverting *reward* produces a standard product interaction term (equation 2) that we can use to specify the standard three parameter interaction model (equation 3).

$$f(y) = \beta_0 + \beta_1 demand + \beta_2 control + \beta_3 (demand \times control) \quad (\text{equation 1})$$

$$ERI = effort / reward = effort \times reward^{-1} \quad (\text{equation 2})$$

$$f(y) = \beta_0 + \beta_1 effort + \beta_2 reward^{-1} + \beta_3 (effort \times reward^{-1}) \quad (\text{equation 3})$$

If no interaction is present, we can remove the interaction term in order to estimate associations of the two main effects only. This model benefits from fewer parameters which means less variance in estimates and better statistical power, and can be used to test the alternative hypothesis in job strain (and ERI) research, of two independent *additive* effects of job demand and job control:

$$f(y) = \beta_0 + \beta_1 demand + \beta_2 control \quad (\text{equation 4})$$

Recalculating estimates from the quadrant model to represent the standard three parameter interaction model

A popular model in job strain research is the *quadrant* model discussed above. Some researchers argue that this model is the solution to the bias problem presented by the *binary* job strain model discussed earlier¹⁹, but this model actually produces a larger bias than the binary job strain model¹².

In a response to a discussion in *Epidemiology*¹⁹, the IPD-Work Consortium published estimates from the quadrant model to defend their use of the binary job strain model, and argue that their estimates of risk were (p e28):

..highest in individuals with job strain, whereas any effects of high occupational demands in the absence of low control, and of low job control in the absence of high demands, were weaker.

However, simply comparing the magnitude of point estimates is not sufficient for inference; we also need to know if observed group differences are likely to be explained by chance. In this particular case all confidence intervals overlapped on all reported outcomes (table 1); thus, the estimates presented by the IPD-Work Consortium cannot be used to support the claim that the groups had different risks. In addition, the way the *quadrant* model is defined, confidence intervals compare the *job strain*, *active* and *passive* quadrants with the *relaxed* quadrant only. Looking at figure 1, we can see that they do provide valid estimates of the two main effects, on the *active* and *passive* quadrants respectively, where individuals were exposed only to either high job *demands* or low job *control*; but that estimates on the *job strain* quadrant are composite estimates, equivalent to comparing a group drinking Gin & Tonic with a group that had nothing to drink at all, i.e. an observed association on the job strain quadrant can be explained by any of the following: an interaction between demand and control, a combined additive effect of both, or a single univariate association with just one of the constituent variables: demand or control.

The quadrant model does have one big advantage; it is equivalent to (but not the same as) the standard interaction model (equation 1). We can exploit this equivalence and calculate *unbiased* estimates of the standard three parameter model with two main effects and an interaction term^{6,12}. To do this we subtract the two main effects from the job strain quadrant estimate; the remainder then describes any effect in data that is specific to being exposed to both high job demand and low job control (i.e. an interaction). On median split data, the variance of the interaction term is then calculated by adding the variances of the two main effects¹². Equations 7–11 below show how estimates are transformed from the quadrant model (equation 5) to the standard interaction model (equation 6)¹²:

$$f(y) = \beta_{10} + \beta_{11} \text{active} + \beta_{12} \text{passive} + \beta_{13} \text{job strain} \quad (\text{equation 5})$$

$$f(y) = \beta_{20} + \beta_{21} \text{demand} + \beta_{22} \text{control} + \beta_{23} (\text{demand} \times \text{control}) \quad (\text{equation 6})$$

Where:

$$\beta_{20} = \beta_{10} \quad (\text{equation 7})$$

$$\beta_{21} = \beta_{11} \quad (\text{equation 8})$$

$$\beta_{22} = \beta_{12} \quad (\text{equation 9})$$

$$\beta_{23} = \beta_{13} - \beta_{12} - \beta_{11} \quad (\text{equation 10})$$

$$Var(\beta_{23}) = Var(\beta_{21}) + Var(\beta_{22}) \quad (\text{equation 11})$$

Table 1 summarizes the original estimates published by the IPD-Work Consortium¹⁹ together with recalculated estimates (equation 5–11) that represent the standard interaction model (equation 1). It shows that all confidence intervals on the interaction term included unity. Thus, contrary to the authors claim that was based on the observed association on the composite, there was no evidence for an interaction, nor any evidence for additive effects of both job demand and job control on any of the presented outcomes. Thus, contrary to the authors' claim¹⁹, their data do not support the job strain theory.

Updated meta-analysis on job strain and coronary heart disease

We examined published meta-analyses on job strain and coronary heart disease to see if any of them had recognized the problem with composite variables. We found nine published meta-analyses^{15,22–29}, and one unpublished meta-analysis in a PhD thesis by the first author of this paper¹². None of the published meta-analyses recognised any problems with using composite variable models in the original research and they all concluded that the job strain model was supported by data. The unpublished meta-analysis did recognise the problem, presented estimates corrected for bias, and found no support in data for the job strain theory.

To obtain an indication of the combined published support for the job strain theory we took a closer look at all prospective cohort studies on non-clinical groups included in previously published meta-analyses on job strain and coronary heart disease^{12,15,22–29}. A total of 61 studies were screened (see supplemental material) of which 27 were prospective cohort studies on non clinical groups, of those 7 studies were excluded because they were also part of a larger study by the IPD-work consortium that was included instead, leaving us with 20 independent studies. None of the 20 studies had fitted the proper three parameter interaction model (equation 1) and only two studies had fitted a model to evaluate additive effects of both demand and control (equation 4). The remainder reported some version of a composite variable model. For a summary of the review see table S2 in the supplemental material.

Twelve studies reported one of the proper models (equation 1 or 4) or the quadrant model (equation 5). We picked the most developed or fully adjusted model published by the authors and recalculated estimates when needed to represent the proper estimates using equations 5–11. When studies reported separate estimates for more than one cohort (e.g. men and women), a meta analysis was performed on these cohorts to obtain a single summary estimate for each study. For a summary of these studies see table S3 in the supplemental material.

The results of a meta-analysis on twelve studies representing 462,220 persons and 6,836 coronary heart disease (CHD) events, making it the largest meta-analysis on job strain and CHD to date, are summarized in figure 3. The results show that there was no evidence of a job strain interaction (RR=1.00; 95% CI: 0.89–1.11) nor any evidence that job demand contributed to the association (RR=1.03; 95% CI: 0.97–1.11). The meta-analysis could only confirm an increased risk of CHD for low job control (RR=1.12; 95% CI: 1.03–1.21). Thus, it appears that previously reported job strain interactions in these meta-analyses were

explained by a spurious association, caused by a single univariate association with job control, and that the job strain theory is not supported by data on CHD outcomes.

Discussion

It is well known that use of composite variables as *dependent* variables can cause confusion about exactly what the effect of an intervention is³⁰. As we have shown here, using composite variables as *independent* variables similarly creates confusion about what is causing an observed effect. In job strain and effort–reward imbalance research, researchers have primarily chosen to interpret observed effects as an interaction between the two constituent factors, which, as shown here, is a (cocktail) fallacy and similar to concluding that an interaction between gin and tonic is responsible for people becoming intoxicated after consuming a few cocktails.

The short methodological review and recalculated estimates from several meta-analyses presented here raise serious doubts concerning the validity of published evidence in support of the job strain and effort reward imbalance (ERI) theories. They suggest that the apparent support for these theories may be fully explained by inappropriate use of single parameter composite variable methods.

Looking closer at published evidence on coronary heart disease (CHD), which is one of the most studied outcomes and perhaps *the* outcome that has lent most credibility to both theories with highly cited publications in *The Lancet*¹⁵ and *Epidemiology*¹⁷, we find that *low job control* but not high job demand^{6,12,15,16}, and *low reward* but not high effort^{9,17}, are associated with CHD. This was also confirmed above in the largest meta-analysis on this association performed to date, with >400k subjects and >6.8k CHD events. The assumed job strain interaction was estimated at perfect unity to two decimal points, indicating no evidence for the interaction that the job strain theory predicts. Together, these observations suggest that the simplest explanation is likely to be the best explanation: associations observed on the job strain and ERI composites are explained by a simple univariate association with one of the constituent factors (job control and reward, respectively).

Here we have demonstrated that the evidence used to argue support for the job strain and effort-reward imbalance theories is flawed, but that the flaw is easily remedied. We have also presented new evidence indicating that many previously reported associations that are claimed to offer support for the job strain theory are spurious, and in fact do not support the theory. We suggest the following alternative hypotheses to explain findings published in these fields, and hope to see the research community focusing on providing evidence that can properly test, falsify and differentiate between them (equations 1, 3 & 4):

- There are no interaction effects in data supporting job strain and ERI theories
- There are no additive effects of both constituent variables in either theory
- Associations observed on widely used job strain and ERI composites are explained by univariate associations with one of the constituent variables

Our findings call into question whether reported associations between job strain and other adverse health outcomes are similarly due to univariate associations to either low job control or high job demands. A reevaluation of a large body of research is called for. The association

between low job control and CHD reported here requires further research to rule out residual confounding by factors such as socioeconomic status³¹ and better understand any causal interrelationships.

Tables

Table 1: Recent estimates from the "job strain" quadrant composite variable model reported by the IPD-Work Consortium, recalculated to represent the main effects and interaction of the standard interaction model (equation 1)

Outcome	Composite estimate (job strain quadrant)			Job demand (High) (active quadrant)			Job control (low) (passive quadrant)			Job strain interaction (demand × control)		
	HR	95% CI		HR	95% CI		HR	95% CI		HR	95% CI	
CHD	1.21	1.05	1.39	1.09	0.97	1.23	1.07	0.90	1.27	1.04	0.84	1.28
Depression	1.29	1.06	1.56	1.04	0.86	1.25	1.18	0.99	1.41	1.05	0.81	1.36
Diabetes	1.13	1.02	1.25	0.98	0.90	1.08	0.97	0.81	1.15	1.19	0.98	1.45
Mortality	1.69	1.19	2.42	0.98	0.72	1.34	1.20	0.88	1.64	1.44	0.93	2.23
Stroke	1.16	0.94	1.42	1.03	0.86	1.24	1.09	0.89	1.33	1.03	0.79	1.36

Note: The table shows estimates from the *quadrant* model reported by Kivimäki et al (2019). The leftmost column shows the original *composite* "job strain" quadrant estimate, followed by the *Active* and *Passive* quadrants, corresponding to the main effects of the *unbiased* standard interaction model (equation 1 and 6), i.e. high job demand and low job control (equation 8-9). The rightmost column shows the recalculated *unbiased* interaction term of high job demands × low job control (equations 10-11). HR=Hazard Ratio. 95% CI=95% confidence intervals.

Figures

Figure 1. The top panel shows an adaptation of figure 1 from the original publication on the job strain model¹ assuming binary (e.g. median split) exposure data with labels assigned to each quadrant; the middle panel is an adaptation of effort–reward imbalance (ERI) on a similar diagram assuming binary data. The bottom panel shows a similar model using the example of Gin & Tonic and intoxication. Note: It has been demonstrated in large studies that *low job control*^{6,15,16}, but not high demand; and *low reward*^{9,17} but not high effort, is associated with coronary heart disease. Thus, *low job control* and *low reward* are similar to *gin*, which is the “active ingredient” in the Gin & Tonic model, and they are all represented on the y-axis. The “passive ingredient” of each model is represented on the x-axis. The focus of analysis is on the top right quadrant, i.e. job strain, ERI and Gin & Tonic. Comparing this quadrant with all the other (i.e. the *binary* job strain and ERI models) or with only the bottom left quadrant (i.e. the *quadrant* model uses the *relaxed* job strain quadrant as reference) produces an expected association in all models due to the “active ingredient” alone – which may be readily apparent in the Gin & Tonic model but is true also for job strain and ERI – without the need for an interaction or a combined effect that depends on both exposures to be present; it is a logical fallacy to use such observations in support for the job strain and ERI theories.

Figure 2. Top panels show simulated data of two normally distributed independent variables (A & B) where A is correlated with a hypothetical outcome ($r=0.70$) and B is completely uncorrelated ($r=0.00$). A and B are also uncorrelated ($r=0.00$). The panels below show associations after the two variables have been combined into various composites. The second row shows subtracting B from A and A from B, followed by adding and multiplying the two (row 3) and taking a ratio (row 4). In all cases a substantial correlation is present on the composite variable, which is completely determined by only one of the constituent variables (A). Thus, these models are all susceptible to bias and cannot be used as evidence for an interaction, or a combined effect that depends on both constituent variables.

Figure 3. Forest plots summarizing meta-analyses on job strain and coronary heart disease, based on 12 studies, 462,220 subjects and 6,836 CHD events. Top panel: job strain interaction (demand×control). Middle panel: high job demand; lower panel: low job control.

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