

# Two Alternative Job Stress Models and the Risk of Coronary Heart Disease

## ABSTRACT

**Objectives.** This study examined the association between two alternative job stress models—the effort-reward imbalance model and the job strain model—and the risk of coronary heart disease among male and female British civil servants.

**Methods.** The logistic regression analyses were based on a prospective cohort study (Whitehall II study) comprising 6895 men and 3413 women aged 35 to 55 years. Baseline measures of both job stress models were related to new reports of coronary heart disease over a mean 5.3 years of follow-up.

**Results.** The imbalance between personal efforts (competitiveness, work-related overcommitment, and hostility) and rewards (poor promotion prospects and a blocked career) was associated with a 2.15-fold higher risk of new coronary heart disease. Job strain and high job demands were not related to coronary heart disease; however, low job control was strongly associated with new disease. The odds ratios for low job control were 2.38 and 1.56 for self-reported and externally assessed job control, respectively. Work characteristics were simultaneously adjusted and controlled for employment grade level, negative affectivity, and coronary risk factors.

**Conclusions.** This is apparently the first report showing independent effects of components of two alternative job stress models—the effort-reward imbalance model and the job strain model (job control only)—on coronary heart disease. (*Am J Public Health.* 1998;88:68–74)

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## Introduction

There is a cumulating body of evidence on the association between psychosocial hazards at work and the physical health of workers. The job stress model most frequently used is that of job strain.<sup>1-3</sup> This model postulates that job strain results from the joint effects of high job demands and low job control. Recent publications increasingly underscore the special importance of low job control for a range of outcomes, including cardiovascular disease and sickness absence.<sup>4-7</sup> Another recently developed job stress model is that of effort-reward imbalance.<sup>8</sup> In this model, there is an explicit emphasis on individual attributes—that is, coping characteristics of high intrinsic effort, as defined by the concept of “need for control.”<sup>9</sup> Extrinsic efforts, as defined by a high workload, are also specified. On the other side, this model also takes three different sources of rewards into account: money, esteem, and occupational status control (promotion prospects, job security). The focus is on a negative trade-off between experienced “costs” and “gains” at work rather than on specific job task characteristics, as in the job strain model.

In a prospective study among German blue-collar men, poor promotion prospects and job insecurity (low rewards) in men having a high workload and a high need for control (high efforts) predicted new cardiovascular events.<sup>10</sup> Thus, worksite-specific stressful experience has been linked with stressful experience related to broader socioeconomic influences of the labor market and income distribution. While the two job stress models clearly differ, there is also some overlap with respect to the dimensions of extrinsic effort (job demands) and esteem reward (social support at work).<sup>11</sup>

Given both the promising results and the different analytical perspectives of the

two job stress models, more information is greatly needed on how they compare in their association with coronary heart disease, especially if the potentially overlapping dimensions (i.e., extrinsic effort and esteem reward) are excluded from the comparative analysis. The longitudinal phase of the Whitehall II study of British civil servants<sup>12</sup> allowed us to examine whether crucial components of effort-reward imbalance and job strain are independently associated with new reports of coronary heart disease.

## Subjects and Methods

### Study Population

The Whitehall II study was set up to investigate the degree and causes of the social gradient in morbidity; to study work characteristics, social support, and additional factors related to the gradient in mortality; and, importantly, to include women. In the study, a new cohort of civil servants was established between 1985 and 1988 (phase 1). All male and female civil servants between 35 and 55 years of age in 20 London-based civil service departments were sent an introductory letter and screening questionnaire and were offered a screening examination for cardiovascular

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diseases. The response rate was 73% and probably would have been higher, had about 4% of the civil servants on the lists provided by the civil service not moved before the study and thus become ineligible for inclusion. In total, 10 308 civil servants were examined: 6895 men (67%) and 3413 women (33%). The participants were approached again in 1989/1990 (phase 2: postal questionnaire) and in 1991/1993 (phase 3: postal questionnaire and screening examination). The participation rates at these two phases were 79% and 83%, respectively; 7372 subjects (72%) participated in all three phases and 9302 subjects (90%) participated in either phase 2 or phase 3. The length of follow-up was 5.3 years on average, with a range of 3.7 to 7.6 years. Full details of the screening examinations are reported elsewhere.<sup>12,13</sup>

### Coronary Heart Disease

Three indicators of coronary heart disease were analyzed: angina pectoris, doctor-diagnosed ischemia, or either of these outcomes. Angina pectoris was measured by the Rose questionnaire and defined as pain located over the sternum or in both the left chest and left arm that is precipitated by exertion, causes the person to stop, and goes away in 10 minutes or less.<sup>14</sup> Doctor-diagnosed ischemia depended on whether the subject reported that a general practitioner or hospital doctor ever suspected or confirmed a heart attack or angina pectoris.

The outcomes were assessed at all three phases. Excluding 230 men and 144 women with any coronary heart disease at phase 1, there were 239 men and 174 women reporting any new coronary heart disease outcome at phases 2 and 3.

### Effort-Reward Imbalance

As there was no original measurement of effort-reward imbalance at phase 1, proxy measures (available from the authors) had to be constructed for the crucial components of the model. In the original measurement, adverse personal characteristics of high need for control (high intrinsic effort) were assessed using a well-tested scale that contains 29 items.<sup>9</sup> These items measure characteristics such as competitiveness, latent hostility, a high need for approval, and an excessive work commitment. On the basis of the highest face validity compared with the original "need for control" items, 10 items were selected from the Framingham type A questionnaire<sup>15</sup> and the Cook-Medley hostility questionnaire<sup>16</sup>

for assessment at phase 1. The dimensionality of these 10 items was confirmed with factor analysis. The three identified scales—competitiveness (3 items), work-related overcommitment (4 items), and hostility (3 items)—were considered valid proxy measures of the original "need for control" scale.

On the reward side, information was available on core aspects of occupational status control, especially on perceived poor promotion prospects (1 item) and on objectively determined restricted occupational mobility ("poor promotion prospects" and "blocked career"). A blocked career was determined by a lower than expected current employment grade level. The expectation was based on the mean current grade level for the different grade levels in which subjects started their careers. Job insecurity, a further important aspect of occupational status control, was not assessed at phase 1 because most civil servants were not (yet) threatened by unemployment (see "Discussion"). Information on financial rewards was not available. Thus, the effort-reward imbalance in our study reflected a mismatch between personal characteristics (high efforts) and occupational career characteristics (low rewards).

Each effort scale was constructed by summing the scores on the individual items and dichotomizing the resulting score (0 = bottom two tertiles and 1 = highest tertile). The highest tertile indicated high efforts. Poor promotion prospects were also dichotomized using the most adverse tertile to indicate low rewards. Blocked career was already a dichotomous variable (low rewards). The indicators were used to create the effort-reward imbalance indicator, which had three categories: 1 = neither high efforts nor low rewards; 2 = either high efforts or low rewards; and 3 = both high efforts and low rewards. This indicator reflects the theoretically postulated cumulative effect of high efforts (adverse personal characteristics) and low rewards (adverse occupational career).

Because the hostility questionnaire was included in the questionnaire at a later stage of the first phase, 3790 subjects (37%) had no hostility score. However, these subjects did not differ from the other group in their risk of newly reported coronary heart disease. Furthermore, using an imputation method did not result in substantially different odds ratios (ORs) for the effort-reward imbalance indicator.

### Job Strain

At all three phases, the subjects were given a questionnaire asking them about the

central components of the job strain model: high job demands, low job control, and low work support. The items, which were based on the job strain questionnaire,<sup>1,2,17,18</sup> can be found elsewhere.<sup>5</sup> Furthermore, jobs were externally assessed at phase 1: in 18 of 20 departments, 140 well-informed personnel managers provided information on work pace ("How often does the job involve working very fast?") and on the level of control ("How often does the job permit complete discretion and independence in determining how, and when, the work is to be done?"). Detailed information was obtained on individual jobs because 5766 different jobs were rated and 8838 subjects occupied them. Both self-reported and externally assessed work characteristics were grouped into tertiles.

Job strain was modeled by assigning subjects who simultaneously scored above the median of job demands and below the median of job control to the group with job strain. All others were assigned to the "no job strain" category. In an alternative strategy, interaction terms of job demands and job control were introduced into a logistic regression model with the main components. This was done separately for the categorical and continuous work scales.

### Statistical Analysis

Logistic regression analyses were used to determine whether effort-reward imbalance and job strain at phase 1 were related to new reports of coronary heart disease during follow-up (phases 2 and 3). In all analyses, coronary heart disease cases at phase 1 were excluded and age and length of follow-up were controlled for. In a subsequent analysis, effort-reward imbalance and job strain were controlled for each other. This model was successively adjusted for employment grade level, negative affectivity, and classical coronary risk factors, including smoking (never, stopped, or smoked 1 to 10, 11 to 20, or 21 or more cigarettes per day), cholesterol (mmol/L), hypertension according to diastolic ( $\geq 95$  mm Hg) or systolic ( $\geq 160$  mm Hg) blood pressure or drug treatment for hypertension, and body mass index ( $\text{kg}/\text{m}^2$ ), which were all assessed at phase 1. Negative affectivity, which is the disposition to respond negatively to questionnaires and which may inflate correlations between self-reported work characteristics and self-reported disease,<sup>19-22</sup> was measured with the negative affect subscale of the affect balance scale.<sup>23,24</sup> Ordinal variables, such as employment grade level, were represented by dummy indicators in the analyses.

**TABLE 1—Number and Percentage of Men and Women Reporting Effort-Reward Imbalance and Job Strain at Phase I, by Employment Grade Level<sup>a</sup>**

	Number	Effort-Reward Imbalance (%)	Job Strain: Self-Report (%)	Job Strain: External Assessment (%)
<b>Men</b>	6895	41.2	14.7	11.9
Grade level				
High	2647	33.3	10.5	7.5
Intermediate	3607	44.4	17.6	13.4
Low	641	55.6	15.8	20.3
<b>Women</b>	3413	48.1	17.2	18.8
Grade level				
High	381	36.9	12.4	9.2
Intermediate	1336	37.5	20.4	15.7
Low	1696	60.8	15.7	23.4

<sup>a</sup>Employment grades were grouped into three levels: unified grades 1–7 (administrators in Whitehall I), executive officers, and clerical and office support grades. Professional grades were classified with the equivalent administrative or executive grade.

## Results

Table 1 shows that women reported high effort and low reward conditions more often than men (48% and 41%, respectively). Moreover, men and women in lower employment grades reported effort-reward imbalance more often than subjects in higher grades. This was mainly owing to a higher prevalence of hostility, a blocked career, and poor promotion prospects in the lower grades; work-related overcommitment and competitiveness were more prevalent in the higher grades. High job strain was also somewhat more prevalent among women than among men (self-reported job strain: 17% and 15%, respectively).

The lowest prevalence of job strain was found in the highest employment grades.

This was primarily owing to the strong association between high job control and high employment grade level. In the highest grade level, 60% reported high job control compared with 5% in the lowest grade. Job demands were also highest in the higher grades, reported by 45% compared with 10% in the lowest grade level. Similar results were found with the external assessments.

Table 2 shows that effort-reward imbalance is associated with elevated risks of subsequent coronary heart disease. The risk of coronary heart disease for men and women who have both high efforts and low rewards is about three times as high as that for subjects with low efforts and high rewards (ORs varied from 2.59 to 3.63). Job strain was not consistently related to new coronary

heart disease reports; only the association between self-reported job strain and any coronary heart disease outcome in men was statistically significant (OR = 1.45). The interaction terms of job demands and job control were not statistically significant and not consistently in the expected direction; this is primarily because (strong) adverse effects of high job demands were absent. Nor did low work support affect the coronary heart disease outcomes. These negative findings did not change when additional information from phase 2 was used. Low job control was, however, consistently related to new coronary heart disease reports. Because low job control, as measured on two occasions (phases 1 and 2), was earlier found to have cumulative effects on new coronary heart disease at phase 3,<sup>5</sup> subsequent analyses will use the mean of phases 1 and 2 job control to predict new coronary heart disease outcomes at phase 3. (Coronary heart disease cases at phases 1 and 2 were excluded in these analyses.)

The interaction terms between sex and effort-reward imbalance or job control were not statistically significant, so all further analyses were based on the total sample and sex was controlled for in each logistic regression model. Table 3 shows that both effort-reward imbalance and low job control have strong and significant associations with the coronary heart disease outcomes in the total sample. Odds ratios of any coronary heart disease outcome were 3.14, 2.04, and 1.57 for effort-reward imbalance (high efforts and low rewards), self-reported low job control, and externally assessed low job control, respectively. The association between job control and doctor-diagnosed ischemia was somewhat smaller.

**TABLE 2—Odds Ratios (ORs)<sup>a</sup> and 95% Confidence Intervals (CIs) of New Coronary Heart Disease Reports, by Effort-Reward Imbalance and Job Strain at Phase I**

	Men			Women		
	Angina Pectoris OR (95% CI)	Diagnosed Ischemia OR (95% CI)	Any Coronary Heart Disease Outcome OR (95% CI)	Angina Pectoris OR (95% CI)	Diagnosed Ischemia OR (95% CI)	Any Coronary Heart Disease Outcome OR (95% CI)
<b>Effort-reward imbalance</b>						
Low efforts and high rewards	1.00	1.00	1.00	1.00	1.00	1.00
High efforts or low rewards	2.13 (0.97, 4.70)	2.13 (0.75, 6.03)	2.12 (1.05, 4.27)	2.08 (0.63, 6.84)	1.45 (0.18, 11.6)	2.41 (0.74, 7.91)
High efforts and low rewards	2.59 (1.17, 5.73)	3.63 (1.30, 10.2)	2.98 (1.48, 5.99)	3.14 (0.96, 10.3)	3.10 (0.40, 23.8)	3.59 (1.10, 11.7)
Number (events)	3751 (129)	3910 (97)	3724 (178)	1589 (110)	1687 (30)	1588 (125)
<b>Job strain (self-reported)</b>						
Number (events)	1.40 (0.93, 2.10) 4817 (168)	1.16 (0.70, 1.94) 5027 (118)	1.45 (1.03, 2.06) 4784 (227)	1.01 (0.65, 1.58) 2116 (141)	1.89 (0.90, 3.99) 2247 (35)	1.14 (0.76, 1.72) 2116 (160)
<b>Job strain (external assessment)</b>						
Number (events)	0.91 (0.53, 1.57) 4169 (149)	1.18 (0.65, 2.14) 4351 (106)	1.03 (0.66, 1.61) 4143 (204)	1.27 (0.81, 1.98) 1839 (122)	0.97 (0.40, 2.39) 1958 (32)	1.22 (0.80, 1.86) 1841 (140)

<sup>a</sup>Adjusted for age and length of period between phases 1 and 3; coronary heart disease cases at phase 1 were excluded; new coronary heart disease reports at phases 2 or 3 were the outcome.

**TABLE 3—Odds Ratios (ORs)<sup>a</sup> and 95% Confidence Intervals (CIs) of New Coronary Heart Disease Reports by Effort-Reward Imbalance at Phase 1, Self-Reported Job Control (Mean Phases 1 and 2) and Externally Assessed Job Control at Phase 1 in the Total Sample**

	Angina Pectoris OR (95% CI)	Diagnosed Ischemia OR (95% CI)	Any Coronary Heart Disease Outcome OR (95% CI)
<b>Effort-reward imbalance<sup>b</sup></b>			
Low efforts and high rewards	1.00	1.00	1.00
High efforts or low rewards	2.06 (1.07, 3.98)	2.00 (0.79, 5.06)	2.17 (1.19, 3.95)
High efforts and low rewards	2.78 (1.44, 5.37)	3.55 (1.42, 8.90)	3.14 (1.72, 5.71)
Number (events)	5340 (239)	5597 (127)	5312 (303)
<b>Self-reported job control<sup>c</sup></b>			
High job control	1.00	1.00	1.00
Intermediate job control	1.36 (0.83, 2.23)	1.39 (0.79, 2.45)	1.61 (1.04, 2.48)
Low job control	2.09 (1.29, 3.37)	1.49 (0.81, 2.74)	2.04 (1.32, 3.16)
Number (events)	6565 (132)	6982 (73)	6489 (163)
<b>Externally assessed job control<sup>d</sup></b>			
High job control	1.00	1.00	1.00
Intermediate job control	1.28 (0.91, 1.81)	1.08 (0.68, 1.71)	1.26 (0.92, 1.71)
Low job control	1.47 (1.77, 2.02)	1.38 (0.74, 2.09)	1.57 (1.17, 2.08)
Number (events)	6003 (271)	6303 (137)	5979 (343)

<sup>a</sup>Adjusted for age, sex, and length of period between phases 1 and 3.

<sup>b</sup>Coronary heart disease cases at phase 1 were excluded; new coronary heart disease reports at phase 2 or phase 3 were the outcome.

<sup>c</sup>Mean phases 1 and 2 job control; coronary heart disease cases at phases 1 and 2 were excluded; new coronary heart disease reports at phase 3 were the outcome.

Subjects with low job control reported effort-reward imbalance conditions more often than subjects with high job control (51% and 36%, respectively). Despite this association, both characteristics were partially independently associated with new reports of any coronary heart disease outcome (Table 4). The odds ratios only marginally decreased when job control and effort-reward imbalance were simultaneously adjusted for (model 2). Additional adjustments for employment grade level, negative affectivity, and coronary risk factors only marginally affected the odds ratios for the work characteristics. When grade was controlled for, the odds ratios for self-reported low job control increased strikingly (from 2.04 to 2.44). This was caused not by any unexpected direction of the underlying associations but by the combination of a relatively small number of events (115) with strong associations between low job control, low grade, and effort-reward imbalance. In the fully adjusted model, subjects with high effort-low reward conditions had more than twice the risk of any new coronary heart disease outcome compared with their counterparts without such conditions (OR = 2.15). Low job control had independent effects on new coronary heart disease reports because the odds ratios in the fully adjusted model were 2.38 and 1.56 for self-reported low job con-

trol and externally assessed low job control, respectively.

## Discussion

The findings in the Whitehall II study further support the predictive validity of components of two alternative job stress models—the effort-reward imbalance model and the job strain model—for coronary heart disease morbidity. In the latter model, only low job control was related to new reports of coronary heart disease. Hence, subjects experiencing high effort and low reward conditions and subjects with low job control had higher risks of new coronary heart disease than their counterparts in less adverse psychosocial work environments.

### Effort-Reward Imbalance

Subjects experiencing a mismatch between their personal characteristics and characteristics of their occupational career had strongly elevated risks of subsequent coronary heart disease. More specifically, competitive, hostile, and overcommitted subjects experiencing poor promotion prospects and blocked careers had the highest risks. The association between this effort-reward imbalance indicator and the

coronary heart disease outcomes was present after adjustment for employment grade level, negative affectivity, and coronary risk factors and was not significantly different in men and women. These findings corroborate the results found in male German blue-collar and middle-management populations.<sup>8</sup> In a previous paper based on the Whitehall II study, Ferrie and colleagues found adverse health effects from anticipation of job loss or job change.<sup>25</sup> Their results may be interpreted as providing further evidence for the importance of “status control” (job insecurity, poor promotion prospects) in the effort-reward imbalance model.

### Low Job Control

Low job control also increased the risks of coronary heart disease. However, neither high job demands nor low social support nor the interactions between work characteristics (job strain) were related to the coronary heart disease outcomes. It is possible that specific characteristics of our sample of white-collar workers contributed to the negative findings for high job demands and high job strain. High job demands were more common in the higher employment grade levels and were positively associated with high job control, resulting in a relatively small number of high strain jobs. Our finding corresponds to that in the review by Schnall and colleagues, in which they concluded that 17 of 25 studies that examined main effects found significant associations between job control and cardiovascular outcome, whereas only 8 of 23 studies found significant associations with job demands.<sup>3</sup>

The importance of (job) control is further elaborated upon by several investigators.<sup>26–28</sup> More details on the association between self-reported and externally assessed low job control and coronary heart disease can be found in another Whitehall II paper.<sup>5</sup> Other reports based on the Whitehall II study have shown the wider predictive validity of low job control as it relates to sickness absence,<sup>7</sup> fibrinogen,<sup>29</sup> and psychiatric disorder.<sup>24</sup> These results underscore the view expressed by Johnson and colleagues that in the job strain model, it is control over the work process rather than high job demands or job strain that increasingly emerges as the main critical component of a healthy work environment.<sup>6</sup>

### Alternative Job Stress Models

To our knowledge, this is the first report that compares components of the effort-reward imbalance model and the job

**TABLE 4—Odds Ratios (ORs) and 95% Confidence Intervals (CIs) of Any New Coronary Heart Disease Outcome by Effort-Reward Imbalance at Phase 1, Self-Reported Job Control (Mean Phases 1 and 2), and Externally Assessed Job Control at Phase 1 in the Total Sample, Adjusted for Age, Sex, and Length of Period between Phases 1 and 3 (Model 1), Additionally Adjusted for Other Work Characteristic (Model 2), and Model 2 Adjusted for Employment Grade Level, Negative Affectivity, and Coronary Risk Factors.**

	Model 1 <sup>a</sup> OR (95% CI)	Model 2 <sup>b</sup> OR (95% CI)	Model 2 Separately Adjusted for			
			Employment Grade Level	Negative Affectivity	Coronary Risk Factors	Fully Adjusted
<b>Effort-reward imbalance<sup>c</sup></b>						
Low efforts and high rewards	1.00	1.00	1.00	1.00	1.00	1.00
High efforts or low rewards	1.93 (1.05, 3.55)	1.88 (1.02, 3.45)	1.87 (1.01, 3.44)	1.70 (0.92, 3.13)	1.93 (1.05, 3.57)	1.77 (0.95, 3.28)
High efforts and low rewards	2.68 (1.46, 4.91)	2.54 (1.38, 4.67)	2.52 (1.36, 4.65)	2.12 (1.15, 3.91)	2.56 (1.39, 4.72)	2.15 (1.15, 4.01)
N = 4393 (251 events)						
<b>Self-reported job control<sup>d</sup></b>						
High job control	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate job control	2.05 (1.22, 3.44)	2.02 (1.20, 3.39)	2.08 (1.22, 3.53)	1.96 (1.17, 3.37)	2.05 (1.22, 3.44)	2.08 (1.22, 3.55)
Low job control	2.15 (1.26, 3.67)	2.04 (1.19, 3.49)	2.44 (1.37, 4.38)	1.94 (1.13, 3.34)	1.97 (1.15, 3.40)	2.38 (1.32, 4.29)
N = 4702 (115 events)						
<b>Externally assessed job control<sup>e</sup></b>						
High job control	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate job control	1.53 (1.07, 2.14)	1.52 (1.06, 2.17)	1.52 (1.06, 2.19)	1.51 (1.06, 2.17)	1.52 (1.06, 2.18)	1.53 (1.06, 2.20)
Low job control	1.72 (1.23, 2.02)	1.63 (1.16, 2.28)	1.61 (1.11, 2.33)	1.62 (1.16, 2.27)	1.53 (1.09, 2.15)	1.56 (1.08, 2.27)
N = 4393 (251 events)						

Note. Subjects with missing values on any of the variables were excluded from the separate analyses.

<sup>a</sup>Adjusted for age, sex, and length of period between phases 1 and 3.

<sup>b</sup>Model 1 additionally adjusted for other work characteristics. Effort-reward imbalance was adjusted for externally assessed job control (findings were similar when mean self-reported job control was adjusted for); mean self-reported job control was adjusted for effort-reward imbalance; externally assessed job control was adjusted for effort-reward imbalance.

<sup>c</sup>Coronary heart disease cases at phase 1 were excluded; new coronary heart disease reports at phase 2 or phase 3 were the outcome.

<sup>d</sup>Mean phases 1 and 2 job control; coronary heart disease cases at phases 1 and 2 were excluded; new coronary heart disease reports at phase 3 were the outcome.

strain model. Effort-reward imbalance and low job control were independently related to the coronary heart disease outcomes. When these were controlled for one another and for other potential confounders, the odds ratio for effort-reward imbalance was 2.15 whereas those for low job control were 2.38 and 1.56 for self-reported and externally assessed job control, respectively. This suggests that the further refinement of job stress theories may benefit from integrating theories on control-related personal attributes and theories on actual control over environmental factors, such as daily tasks (job control) and occupational career (status control). The cumulative adverse health impact of low job control and effort-reward imbalance indicates that both job stress factors provide supplementary information on relevant stressors in the psychosocial work environment.

#### Conceptual Overlap between Job Stress Models

Possible conceptual overlap between the effort-reward imbalance model and other models needs to be explored in further studies. First, job demands and work support from the job strain model closely

resemble extrinsic efforts and esteem rewards from the effort-reward imbalance model. However, the effort-reward imbalance model attaches much importance to the perception and appraisal of adverse work conditions, whereas the job strain model focuses attention primarily on the "objective" psychosocial work environment (we used both self-reported and objective measures of job control).<sup>2</sup>

Second, there might be overlap with hostility, which on its own has been shown to be strongly related to future coronary heart disease.<sup>30</sup> Although the odds ratios of our imbalance indicator did not change substantially when the total 38-item hostility scale was controlled for, the theoretical and empirical contribution of hostility to effort-reward imbalance is worth some further elaboration. Third, it has also been suggested that the adverse personal characteristics (e.g., competitiveness or hostility) in the effort-reward imbalance model could be the result of low job control and high job demands. If this were the case, controlling effort-reward imbalance for job control should have resulted in decreased odds ratios of the former. However, the odds ratio for high efforts and low rewards hardly decreased (from 2.68 to 2.54). More-

over, in a 6.5-year prospective study on blue-collar workers, an impressive degree of stability over time was reported for the "need for control" measures.<sup>9,10</sup> Fourth, proponents of the job strain model have often referred to the need to expand the job strain model by including information on job insecurity (macro-level decision latitude).<sup>2</sup> In further studies, the links between low job control (job strain model) and low occupational status control (effort-reward imbalance model) should be elaborated upon and tested more vigorously.

#### Further Considerations

Despite our promising findings, some further considerations need to be taken into account. First, given the known variability in angina reporting,<sup>31</sup> which has been replicated in our data set, using new reports of angina pectoris as an indicator of incident coronary pathology may be problematic. One might speculate that the underlying condition has not altered but that the tendency to report it has changed. A new report of a doctor diagnosis is likely to be a better indicator of new disease, although other factors may also influence both recall of diagnosis or access to medical care. Preliminary

results show that 87% of subjects reporting a myocardial infarction at phase 3 had documented coronary heart disease. Furthermore, the classic coronary risk factors were related to both doctor-diagnosed ischemia and angina pectoris, suggesting that the endpoints do reflect coronary heart disease, not just reporting bias. Despite the likely different levels of sensitivity and specificity of both endpoints, effort-reward imbalance and low job control show consistent associations with both angina and doctor-diagnosed ischemia. This supports an etiological hypothesis. Future analyses will examine the effects of effort-reward imbalance and low job control on fatal and non-fatal myocardial infarction and biological mechanisms.

Second, small numbers did not permit extensive analyses of the associations between employment grade level, job control, effort-reward imbalance, and coronary heart disease. Because the interaction between grade and any of the job stress factors was never statistically significant, we would not expect different effects of work characteristics among grades. The absence of "confounding by" or "interaction with" employment grade level suggests that the association between work and coronary heart disease does not depend on employment grade level. A related issue is the extent to which work contributes to the inverse association between employment grade level and coronary heart disease in Whitehall II. This is further investigated in another paper, which concludes that much of the inverse social gradient in coronary heart disease incidence in Whitehall II can be attributed to differences in the psychosocial work environment.<sup>32</sup>

Third, some further methodological issues need to be addressed. Although the three-category effort-reward imbalance indicator reflects a mismatch between efforts and rewards, there was no significant (multiplicative) interaction term between our proxy measures of efforts and rewards in the logistic regression analysis. This should be further elaborated upon using original effort and reward measures. The high number of subjects whose effort-reward imbalance scores were not available, mainly owing to a delayed inclusion of the hostility scale in the phase 1 questionnaire, probably did not bias the results because these subjects did not differ from the other group in their risk of newly reported coronary heart disease. Moreover, using an imputation method did not result in substantially different odds ratios for the effort-reward imbalance indicator. Given that individuals with effort-reward imbalance, low job control, angina pectoris, and

doctor-diagnosed ischemia at phase 1 had somewhat lower participation rates at phases 2 and 3, it is likely that the impact of effort-reward imbalance and low job control on newly reported coronary heart disease is somewhat underestimated in these analyses. Controlling for whether individuals had left the civil service did not affect the results. Theoretically, information bias may have caused overestimated odds ratios in our analyses because a complaining attitude regarding work and health (negative affectivity) may have resulted in negative reports about job control, effort-reward imbalance, and coronary heart disease.<sup>33-36</sup> However, because baseline cases were excluded in a longitudinal setting and negative affect balance was controlled for, it is unlikely that negative affectivity biased the results.<sup>19-22</sup>

## Conclusion

Low job control and high cost/low gain conditions influence the development of heart disease among men and women working in British government offices. The finding that competitive, overcommitted, and hostile subjects with a less successful occupational career and low job control have higher risks of coronary heart disease underscores the advantage of a job stress model combining personal and environmental factors. To our knowledge, this is the first report showing independent effects on coronary heart disease of components of two alternative job stress models: the effort-reward imbalance model and the job strain model (job control). □

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## References

1. Karasek RA. Job demands, job decision latitude, and mental strain: implications for job redesign. *Admin Sci Q.* 1979;24:285-308.
2. Karasek RA, Theorell T. *Healthy Work: Stress, Productivity, and the Reconstruction of Working Life.* New York, NY: Basic Books; 1990.
3. Schnall PL, Landsbergis PA, Baker D. Job strain and cardiovascular disease. *Ann Rev Public Health.* 1994;15:381-411.
4. Alterman T, Shekelle RB, Vernon SW, Burau KD. Decision latitude, psychologic demand, job strain, and coronary heart disease in the Western Electric Study. *Am J Epidemiol.* 1994;139:620-627.
5. Bosma H, Marmot MG, Hemingway H, Nicholson AC, Brunner E, Stansfeld SA. Low job control and the risk of coronary heart disease in the Whitehall II (prospective cohort) study. *BMJ.* 1997;314:558-564.
6. Johnson JV, Stewart W, Hall EM, Fredlund P, Theorell T. Long-term psychosocial work environment and cardiovascular mortality among Swedish men. *Am J Public Health.* 1996;86:324-331.
7. North FM, Syme SL, Feeney A, Shipley M, Marmot M. Psychosocial work environment and sickness absence among British civil servants: the Whitehall II study. *Am J Public Health.* 1996;86:332-340.
8. Siegrist J. Adverse health effects of high effort/low reward conditions. *J Occup Health Psychol.* 1996;1:27-41.
9. Matschinger H, Siegrist J, Siegrist K, Dittmann K. Type A as a coping career: towards a conceptual and methodological redefinition. In: Schmidt H, Dembroski TM, Blümchen G, eds. *Biological and Psychological Factors in Cardiovascular Disease.* Heidelberg, Germany: Springer; 1986:104-126.
10. Siegrist J, Peter R, Junge A, Cremer P, Seidel D. Low status control, high effort at work and ischemic heart disease: prospective evidence from blue-collar men. *Soc Sci Med.* 1990;31:1127-1134.
11. Kasl SV. The influence of the work environment on cardiovascular health: a historical, conceptual, and methodological perspective. *J Occup Health Psychol.* 1996;1:42-56.
12. Marmot MG, Davey Smith G, Stansfeld S, et al. Health inequalities among British civil servants: the Whitehall II study. *Lancet.* 1991;337:1387-1393.
13. Beksinska M, Yea L, Brunner E. *Whitehall II Study. Operations Manual for Screening Examination 1991-93 (Phase s3).* London, England: University College London, Department of Epidemiology and Public Health; 1995.
14. Rose G, Reid DD, Hamilton PS, McCartney P, Jarrett RJ. Myocardial ischaemia risk factors and death from coronary heart disease. *Lancet.* 1977;i:105-109.
15. Haynes SG, Levine S, Scotch N, Feinleib M, Kannel WB. The relationship of psychosocial factors to coronary heart disease in the Framingham study, I: methods and risk factors. *Am J Epidemiol.* 1978;107:362-383.
16. Cook WW, Medley DM. Proposed hostility and pharisaic-virtue scales for the MMPI. *J Appl Psychol.* 1954;38:414-418.

17. Karasek RA, Baker D, Marxer F, Ahlbom A, Theorell T. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *Am J Public Health*. 1981;71:694-705.
18. Quinn RP, Staines GL. *The 1977 Quality of Employment Survey*. Ann Arbor, Mich: Survey Research Center, Institute for Social Research, University of Michigan; 1979.
19. Brief AP, Burke MJ, George JM, Robinson BS, Webster J. Should negative affectivity remain an unmeasured variable in the study of job stress? *J Appl Psychol*. 1988;73:193-198.
20. Chen PY, Spector PK. Negative affectivity as the underlying cause of correlations between stressors and strains. *J Appl Psychol*. 1991;76:398-407.
21. McCrea G. Controlling neuroticism in the measurement of stress. *Stress Med*. 1990;6:237-241.
22. Spector PE. A consideration of the validity and meaning of self-report measures of job conditions. *Int Rev Ind Org Psychol*. 1992;123-151.
23. Bradburn NM. *The Structure of Psychological Wellbeing*. Chicago, Ill: Aldine; 1969.
24. Stansfeld S, North FM, White I, Marmot MG. Work characteristics and psychiatric disorder in civil servants in London. *J Epidemiol Community Health*. 1995;49:48-53.
25. Ferrie JE, Shipley MJ, Marmot MG, Stansfeld S, Davey Smith G. Health effects of anticipation of job change and non-employment: longitudinal data from the Whitehall II study. *BMJ*. 1995;311:1264-1269.
26. deJonge J. *Job Autonomy, Well-Being, and Health*. Maastricht, The Netherlands: Datawyse b.v. Universitaire Pers Maastricht; 1996. Master's thesis.
27. Sauter SL, Hurrell JJ, Cooper CL, eds. *Job Control and Worker Health*. New York, NY: John Wiley & Sons; 1989.
28. Steptoe A, Appels A, eds. *Stress, Personal Control and Health*. New York, NY: John Wiley & Sons, 1989.
29. Brunner E, Davey Smith G, Marmot M, Canner R, Beksinska M, O'Brien J. Childhood social circumstances and psychosocial and behavioural factors as determinants of plasma fibrinogen. *Lancet*. 1996;347:1008-1013.
30. Miller TQ, Smith TW, Turner CW, Guizarro ML, Hallet AJ. A meta-analytic review of research on hostility and physical health. *Psychol Bull*. 1996;119:322-348.
31. Rose G. Variability of angina. Some implications for epidemiology. *Br J Prev Soc Med*. 1968;22:12-15.
32. Marmot M, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet*. 1997;350:235-239.
33. Frese M. Stress at work and psychosomatic complaints: a causal interpretation. *J Appl Psychol*. 1985;70:314-328.
34. Frese M, Zapf D. Methodological issues in the study of work stress: objective vs subjective measurement of work stress and the question of longitudinal studies. In: Cooper CL, Payne R, eds. *Causes, Coping and Consequences of Stress at Work*. New York, NY: John Wiley & Sons, 1988:375-411.
35. Kasl SV. The challenge of studying the disease effects of stressful work conditions. *Am J Public Health*. 1981;71:682-684.
36. Kasl SV. Methodologies in stress and health: past difficulties, present dilemmas, future directions. In: Kasl SV, Cooper CL, eds. *Stress and Health: Issues in Research Methodology*. New York, NY: John Wiley & Sons, 1987:307-318.