

# Contribution of job control and other risk factors to social variations in coronary heart disease incidence

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## Summary

**Background** The first Whitehall Study showed an inverse social gradient in mortality from coronary heart disease (CHD) among British civil servants—namely, that there were higher rates in men of lower employment grade. About a quarter of this gradient could be attributed to coronary risk factors. We analysed 5-year CHD incidence rates from the Whitehall II study to assess the contribution to the social gradient of psychosocial work environment, social support, coronary risk factors, and physical height.

**Methods** Data were collected in the first three phases of examination of men and women in the Whitehall II study. 7372 people were contacted on all three occasions. Mean length of follow-up was 5.3 years. Characteristics from the baseline, phase 1, questionnaire, and examination were related to newly reported CHD in people without CHD at baseline. Three self-reported CHD outcomes were examined: angina and chest pain from the Rose questionnaire, and doctor-diagnosed ischaemia. The contribution of different factors to the socioeconomic differences in incident CHD was assessed by adjustment of odds ratios.

**Findings** Compared with men in the highest grade (administrators), men in the lowest grade (clerical and office-support staff) had an age-adjusted odds ratio of developing any new CHD of 1.50. The largest difference was for doctor-diagnosed ischaemia (odds ratio for the lowest compared with the highest grade 2.27). For women, the odds ratio in the lowest grade was 1.47 for any CHD. Of factors examined, the largest contribution to the socioeconomic gradient in CHD frequency was from low control at work. Height and standard coronary risk factors made smaller contributions. Adjustment for all these factors reduced the odds ratios for newly reported CHD in the lowest grade from 1.5 to 0.95 in men, and from 1.47 to 1.07 in women.

**Interpretation** Much of the inverse social gradient in CHD incidence can be attributed to differences in psychosocial work environment. Additional contributions were made by coronary risk factors—mainly smoking—and from factors that act early in life, as represented by physical height.

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## Introduction

Inequalities in health are a matter for concern in the UK, USA, and many European countries. In the UK, such inequalities have been labelled “variations”, and have been subject to government enquiry from an NHS viewpoint.<sup>1</sup> But the causes and remedies of social inequalities in health go beyond differentials in health services,<sup>2–4</sup> and beyond lifestyle. The first Whitehall study of British civil servants clearly showed an inverse social gradient in mortality from coronary heart disease (CHD); the lower the grade of employment, the higher the age-adjusted mortality rate. About a quarter of this gradient could be explained by social differentials in smoking, plasma cholesterol, blood pressure, height, obesity, and physical activity.<sup>5</sup> Better measurement might have explained the gradient further, but much was unexplained by these established risk factors. We proposed that psychosocial factors, particularly related to work, may be important in the generation of the social gradient.<sup>6</sup>

The Whitehall II study of a new cohort of male and female civil servants was devised to test this hypothesis.<sup>7</sup> The job-strain model has been most influential as a method to characterise the psychosocial work environment.<sup>8</sup> The model postulates that a combination of high psychosocial demand and low control is related to cardiovascular risk. However, a 1994 review provided only partial support for this two-factor model;<sup>9</sup> the control dimension is consistently related to cardiovascular risk, but the demand dimension is not. This finding is consistent with analyses of data for occupational mortality in England and Wales from 1970 to 1972. Occupations characterised by low control were associated with increased CHD mortality, but those characterised by high demand were not.<sup>6</sup>

Analyses of longitudinal data from Whitehall II confirm the prediction that low control, but not high demand, at work is associated with increased incidence of CHD, independently of measures of socioeconomic status,<sup>10</sup> and that low control is associated with higher plasma fibrinogen concentrations.<sup>11</sup> Since the importance of low control at work has been established, the purpose of this study was to test the hypothesis that low control makes an important contribution to the generation of the expected social gradient in incident CHD, in addition to the potential contribution made by social supports, height (a possible marker of early life influences), and established risk factors.

## Methods

### Study population

The Whitehall II study is based on a cohort of civil servants examined between 1985 and 1988 (phase 1). All male and female civil servants, aged between 35 and 55 years in 20

	Angina pectoris			Severe chest pain			Doctor-diagnosed ischaemia			Any CHD event		
	Total number	Number of cases	Odds ratio (95% CI)	Total number	Number of cases	Odds ratio (95% CI)	Total number	Number of cases	Odds ratio (95% CI)	Total number	Number of cases	Odds ratio (95% CI)
<b>Men</b>												
High grade	1888	61 (3.2%)	1.00*	1840	92 (5.2%)	1.00	1971	49 (2.5%)	1.00*	1785	139 (7.8%)	1.00*
Intermediate grade	2405	84 (3.5%)	1.28 (0.91-1.81)	2342	131 (5.6%)	1.11 (0.85-1.47)	2500	52 (2.1%)	1.06 (0.71-1.58)	2264	203 (9.0%)	1.25 (1.00-1.57)
Low grade	292	15 (5.1%)	1.74 (0.97-3.11)	277	19 (7.2%)	1.44 (0.87-2.37)	311	15 (5.1%)	2.27 (1.27-4.08)	264	29 (11.0%)	1.50 (0.98-2.29)
Total	4585	160 (3.5%)		4459	245 (5.5%)		4782	116 (2.4%)		4313	371 (8.6%)	
<b>Women</b>												
High grade	268	17 (6.3%)	1.00	257	13 (5.1%)	1.00	275	4 (1.5%)	1.00	252	24 (9.5%)	1.00*
Intermediate grade	853	54 (6.4%)	1.00 (0.57-1.75)	866	45 (5.2%)	1.02 (0.54-1.92)	907	10 (1.1%)	0.67 (0.21-2.17)	822	87 (10.6%)	1.12 (0.69-1.79)
Low grade	850	61 (7.2%)	1.04 (0.59-1.83)	840	69 (8.3%)	1.62 (0.88-3.01)	905	25 (2.8%)	1.30 (0.44-3.84)	810	113 (14.0%)	1.47 (0.92-2.35)
Total	1971	132 (6.7%)		1963	127 (6.5%)		2087	39 (1.9%)		1884	224 (11.9%)	

\*Trend test:  $p < 0.05$ .

Table 1: Age-adjusted odds ratios of new reports of CHD according to employment grade in the Civil Service

London-based Civil Service departments were sent an introductory letter and screening questionnaire, and were offered a screening examination for cardiovascular disease. The overall response rate was 73%, though the true rate is likely to be higher because 4% of those on the employee list had moved before the study began, and were thus not eligible for inclusion. Full details of the examination have been reported elsewhere.<sup>7</sup> A total of 10 308 people were examined, 6895 men and 3413 women. Participants were approached again in 1989/90 (phase 2: postal questionnaire), and in 1991/93 (phase 3: postal questionnaire and screening examination). The participation rate at these two phases was 79% and 83%, respectively; 7372 (72%) participated at all three phases. The length of follow-up was 5.3 years on average (range 3.7-7.6).

#### CHD and risk factors

Three indicators of CHD were analysed: angina pectoris from the Rose questionnaire,<sup>12</sup> severe pain across the chest that lasted half an hour or more, and doctor-diagnosed ischaemia (ie, a participant's report that a doctor had diagnosed or suspected a heart attack or angina pectoris). To be included as a new case, the participant had to have been free of disease at phase 1, but to respond positively to one or more of the categories at phase 2 or 3.

Coronary risk factors were measured in standard ways:<sup>7</sup> smoking (never, former, current), serum cholesterol (mmol/L), body-mass index ( $\text{kg}/\text{m}^2$ ), hypertension (diastolic blood pressure  $\geq 95$  mm Hg, or systolic blood pressure  $\geq 160$  mm Hg, or antihypertensive drugs), and physical activity ( $\geq 1.5$  h vigorous activity per week,  $< 1.5$  h either vigorous or moderate activity per week).

#### Employment grade, psychosocial work characteristics, and social support

Employment grades were grouped into three categories: Unified Grades 1-7 (administrators in Whitehall I), executive officers, and clerical and office support staff. Professional grades were

classified with the equivalent administrative or executive grade. Job control was measured by a self-completed questionnaire at phase 1. Fifteen items deal with decision authority and skill discretion, and these were combined into an index of decision latitude or control.<sup>10</sup>

Social-support measures were derived from the three scales of the close persons questionnaire:<sup>13</sup> confiding/emotional support, practical support, and negative aspects of close relationships from the closest person. The network measures were summarised by a scale that measured beyond the household; this network includes frequency of contact and number of contacts with friends and relatives, and participation in social groups.

At baseline, while screening continued, there were additions to the questionnaire, and 2983 people did not have complete measures of social support. For these participants, the mean score of confiding/emotional support, practical support, and negative aspects of support was substituted for the complete index. When all missing data were excluded, the (relative) contribution of work and support measures to the grade gradient was roughly the same as presented in this paper. Furthermore, use of phase 2 data with fewer missing values did not result in different findings.

#### Statistical analysis

For self-reported job control and social support, we used tertiles; for height, sex-specific quartiles. Logistic regression analyses were used to calculate odds ratios of developing new CHD. Administrators were assigned an odds ratio of developing CHD of 1, and the other grades were compared with them. All analyses excluded baseline CHD cases and controlled for age. The effect of work on the social gradient in CHD incidence was assessed by inclusion of low control in the model. The effect of social supports was similarly assessed without work in the model. The effect of coronary risk factors was assessed without either work or social supports in the model. Similarly, the effect was assessed of controlling for height alone. Finally, the effect

Factor	Men			Women		
	High grade	Intermediate grade	Low grade	High grade	Intermediate grade	Low grade
% of participants reporting low job control	8.7	26.6	77.9	10.1	34.8	75.3
<b>Social support (% of participants)</b>						
Low confiding/emotional	30.2	36.0	38.4	24.6	29.0	32.4
Low practical support	26.3	30.5	38.9	31.6	39.8	35.7
High negative characteristics	28.8	34.9	39.1	32.7	32.9	35.2
Small social network	24.0	28.8	36.4	19.3	31.6	34.7
Mean height (cm)	177.4	176.2	173.0	165.1	163.4	160.8
<b>Coronary risk factors</b>						
Current smokers (%)	9.2	16.0	28.9	13.7	19.9	28.3
Low physical activity (%)	27.6	28.4	47.9	46.1	42.3	52.5
Hypertension (%)	6.9	7.3	13.5	5.9	6.1	9.6
Mean cholesterol (mmol/L)	6.0	5.9	6.0	5.8	5.8	6.0
Mean body-mass index ( $\text{kg}/\text{m}^2$ )	24.5	24.5	25.1	23.6	24.3	25.3

Table 2: Employment grade and work characteristics, social support, physical height, and coronary risk factors at phase 1

	Odds ratio for new CHD (95% CI)	
	Men	Women
<b>Height quartile (unadjusted)*</b>		
1 (short)	1.00	1.00
2	1.02 (0.77–1.36)	0.85 (0.59–1.23)
3	0.88 (0.66–1.18)	0.52 (0.35–0.78)
4 (tall)	0.65 (0.47–0.88)	0.65 (0.45–0.96)
<b>Height quartile (adjusted for employment grade)*</b>		
1 (short)	1.00	1.00
2	1.05 (0.79–1.41)	0.87 (0.60–1.27)
3	0.91 (0.68–1.23)	0.54 (0.36–0.81)
4 (tall)	0.68 (0.49–0.93)	0.70 (0.47–1.04)

\*Quartiles of height (cm): 172.0, 176.2, and 180.9 (men); 158.0, 162.3, and 166.6 (women).

**Table 3: Age-adjusted odds ratios of new reports of CHD at phase 2 or 3 by sex-specific quartiles of height, unadjusted and adjusted for employment grade at phase 1**

on the grade differences in CHD was measured by inclusion of all four groups of variables—work, social supports, coronary risk factors, and height—in the model. Tests for trends were calculated by estimation of the odds ratio for employment grade, modelled as a continuous variable (six categories).

## Results

Table 1 shows the number of men and women by employment grade, the proportion who developed newly reported CHD during the follow-up period, and the odds ratios with high grades assigned a CHD risk of 1. Among men, all three CHD endpoints show an inverse gradient in risk by grade of employment. For the combined endpoint of any CHD event, the odds ratio in the lowest grade compared with the highest was 1.5. Similarly, in women, the odds of developing any CHD in the lowest grade compared with the highest was 1.47. There was no social gradient in new angina for women. The steepest gradient was in doctor-diagnosed ischaemia in men.

Table 2 shows the distribution by grade of potential explanations for the social gradient in CHD frequency.

Low job control was closely linked to position in the employment hierarchy. In addition, various features of social support and social networks show less favourable patterns in lower grades. Among coronary risk factors, there were clear social gradients in smoking, low physical activity, and hypertension; small differences in mean body-mass index; and no grade difference in cholesterol.

Higher-grade men are taller than men in lower grades. Short height predicted CHD incidence even after controlling for grade (table 3).

Among men, adjustment for support made little difference to the grade differences in CHD frequency; and the grade difference in CHD was largely independent of height (table 4). Adjustment for job control reduced the gradient substantially. For example, the odds of development of any CHD in the lowest compared with the highest grade was reduced from 1.50 to 1.18. The risk factors also contributed to the CHD gradient, as shown by the reduction in odds ratio from 1.50 to 1.30 for any CHD in the lowest grade. In the fully adjusted model, the effects of work, social support, height, and risk factors were assessed simultaneously. For any CHD event the odds ratio was reduced from 1.50 to 0.95. For doctor-diagnosed ischaemia, the odds ratio in the lowest grade after full adjustment was 1.4—a substantial reduction from 2.27 in the model with age as the only adjustment.

The results for women were similar to those for men (table 4). Height made some contribution, but social support did not contribute to the explanation of the CHD gradient. For the combined category of any CHD, the odds ratio in the lowest grade was reduced from 1.47 to 1.23, by adjustment only for work in addition to age, and to 1.35 by adjustment for risk factors.

Adjustment for all these factors reduced the odds ratio to 1.07. The results for doctor-diagnosed ischaemia in women were probably caused by the small number of such events in women (n=39).

Employment grade	Odds ratio for new CHD event in men (95% CI)				Odds ratio for new CHD event in women (95% CI)			
	Angina pectoris	Severe chest pain	Diagnosed ischaemia	Any CHD event	Angina pectoris	Severe chest pain	Diagnosed ischaemia	Any CHD event
<b>Age-adjusted</b>								
High	1.00*	1.00	1.00*	1.00*	1.00	1.00	1.00	1.00*
Intermediate	1.28 (0.91–1.81)	1.11 (0.85–1.47)	1.06 (0.71–1.58)	1.25 (1.00–1.57)	1.00 (0.57–1.75)	1.02 (0.54–1.92)	0.67 (0.21–2.17)	1.12 (0.69–1.79)
Low	1.74 (0.97–3.11)	1.44 (0.87–2.37)	2.27 (1.27–4.08)	1.50 (0.98–2.29)	1.04 (0.59–1.83)	1.62 (0.88–3.01)	1.30 (0.44–3.84)	1.47 (0.92–2.35)
<b>Work adjusted†</b>								
High	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate	1.20 (0.84–1.72)	1.08 (0.81–1.44)	0.96 (0.63–1.46)	1.16 (0.92–1.48)	1.04 (0.55–1.98)	0.90 (0.47–1.74)	0.83 (0.24–2.86)	0.98 (0.59–1.60)
Low	1.48 (0.77–2.86)	1.18 (0.68–2.06)	1.88 (0.95–3.73)	1.18 (0.74–1.88)	0.99 (0.55–1.78)	1.42 (0.71–2.83)	1.57 (0.42–5.77)	1.23 (0.72–2.09)
<b>Support adjusted‡</b>								
High	1.00*	1.00	1.00*	1.00*	1.00	1.00*	1.00	1.00*
Intermediate	1.19 (0.84–1.68)	1.12 (0.85–1.47)	1.02 (0.68–1.53)	1.22 (0.97–1.53)	1.02 (0.58–1.81)	1.09 (0.57–2.06)	0.73 (0.22–2.40)	1.16 (0.72–1.88)
Low	1.61 (0.89–2.89)	1.40 (0.84–2.31)	2.17 (1.20–3.91)	1.43 (0.93–2.19)	1.07 (0.61–1.90)	1.74 (0.93–3.26)	1.37 (0.46–2.20)	1.54 (0.96–2.49)
<b>Height adjusted</b>								
High	1.00*	1.00	1.00	1.00*	1.00	1.00	1.00	1.00
Intermediate	1.25 (0.89–1.76)	1.08 (0.82–1.42)	1.02 (0.68–1.52)	1.22 (0.97–1.53)	0.96 (0.55–1.69)	1.00 (0.53–1.90)	0.63 (0.19–2.04)	1.08 (0.67–1.75)
Low	1.63 (0.90–2.95)	1.32 (0.79–2.19)	2.06 (1.13–3.76)	1.40 (0.91–2.15)	0.94 (0.53–1.67)	1.55 (0.82–2.91)	1.12 (0.37–3.37)	1.34 (0.83–2.18)
<b>Risk-factor adjusted§</b>								
High	1.00*	1.00	1.00	1.00*	1.00	1.00	1.00	1.00
Intermediate	1.25 (0.88–1.76)	1.06 (0.81–1.40)	1.01 (0.68–1.52)	1.21 (0.96–1.52)	0.92 (0.52–1.62)	0.99 (0.52–1.87)	0.67 (0.20–2.20)	1.06 (0.66–1.72)
Low	1.45 (0.80–2.64)	1.27 (0.76–2.11)	2.05 (1.11–3.79)	1.30 (0.85–2.01)	0.92 (0.52–1.62)	1.56 (0.84–2.91)	1.20 (0.40–3.62)	1.35 (0.84–2.18)
<b>Fully adjusted¶</b>								
High	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Intermediate	1.07 (0.74–1.54)	1.01 (0.75–1.35)	0.84 (0.55–1.30)	1.07 (0.84–1.37)	0.89 (0.49–1.62)	0.91 (0.47–1.77)	0.71 (0.20–2.55)	0.94 (0.57–1.55)
Low	1.12 (0.56–2.23)	0.97 (0.55–1.72)	1.49 (0.72–3.07)	0.95 (0.59–1.54)	0.85 (0.44–1.66)	1.36 (0.67–2.76)	1.18 (0.30–4.62)	1.07 (0.62–1.86)

\*p (trend test) <0.05. †Job control and effort-reward imbalance. ‡Confiding/emotional support, practical support, negative characteristics, social network. §Smoking, serum cholesterol, body-mass index, hypertension, and physical activity. ¶Early life, work, support, and risk factors.

**Table 4: Age-adjusted odds ratios of new reports of CHD at phase 2 or 3 by employment grade at phase 1, adjusted for work characteristics, social support, height, and coronary risk factors**

## Discussion

As predicted, the specific psychosocial work characteristic of low control made an important contribution to the social gradient in incident CHD in the Whitehall II study. We showed previously that low control in the workplace was related—independently of employment grade—to 5-year CHD incidence.<sup>10</sup> The analyses given here show that low control is related to employment grade, and appears to account for much of the grade difference in CHD frequency in both men and women. Taken together, these results support the hypothesis that low control is involved in the process that links socioeconomic status with CHD.

The first Whitehall study, started in the late 1960s, showed an inverse social gradient in CHD mortality: clerical officers (low grade) had more than twice the mortality rate of administrators (high grade).<sup>5</sup> In Whitehall II, begun 20 years later, the lowest grade had age-adjusted CHD incidence 1.5 times higher than the highest grade. During comparison of the cross-sectional baseline findings from Whitehall I and Whitehall II, we commented that the social inequalities in prevalent CHD had not narrowed in the 20 years that separated the two studies.<sup>7</sup> Does the smaller gradient in Whitehall II compared with the Whitehall study indicate that social differences are narrowing? This seems unlikely, since mortality data from England and Wales<sup>14,15</sup> and from Scotland<sup>16</sup> show a continued widening of the social gap in mortality that was evident between the 1970s and 1980s.<sup>17</sup> The apparently smaller relative difference in Whitehall II may be a result of the difference between incidence and mortality rates. The cohort is being followed up for mortality. Future analyses of Whitehall II data will provide more detailed insight into the grade-related differences in incidence, mortality, and case-fatality.

As in the first Whitehall study, the established coronary risk factors seem to account for less than half the social gradient. We used height as a marker of persisting influences—genetic and environmental—from early life; though, since height is a crude indicator, it may understate the early life influence.<sup>18</sup> The data suggest, as in the first Whitehall study, that height and social position are independently related to CHD risk. Taken all together, work, height, and coronary risk factors appear to provide a fairly complete account of the reasons for the higher rates of CHD in men and women in lower grades.

Our findings should be regarded as preliminary because of the provisional nature of the CHD endpoints, and their basis on self-reports. However, we have preliminary evidence that validates the reported doctor-diagnosed ischaemia. Angina diagnosed by the Rose questionnaire may be a less reliable indication of CHD in women than it is in men,<sup>19,20</sup> which may account for the discrepant findings for angina in women. As expected, there were few cases in women of doctor-diagnosed ischaemia, so the confidence intervals around the odds ratios were wide. The (relative) contribution of work to the grade/gradient for severe chest pain in women is also similar to the findings in men. Since both the exposure variable (low control) and the outcome (CHD frequency) are based on self-reports, reporting bias is possible. This bias is most likely to apply if both measurements are made simultaneously, but exclusions of baseline CHD cases makes it less likely. In addition, we showed that low job control was related to new CHD independently of a measure of negative affectivity.<sup>10</sup> Reporting bias is

therefore unlikely to have overestimated the contribution of work to the social gradient.

In Whitehall II, we addressed two questions related to psychosocial work environment. First, is low control at work related to CHD? Our previously reported analysis suggests that it is.<sup>10</sup> Second, the analysis reported here suggests that low control contributes to the socioeconomic gradient in CHD. Both questions are subject to the same drawback: if low control were nothing other than an alternative measure of socioeconomic position, our two conclusions could be wrong. However, evidence shows that, although they are correlated, low control and low socioeconomic status are separable.

The association between work and CHD does not simply result from confounding by socioeconomic status; a review<sup>9,21–23</sup> of 17 studies found the low control–CHD association in 14. Of the three studies that revealed no association, Reed and colleagues' study<sup>24</sup> was consistent with the confounding hypothesis in that there was no association between socioeconomic status and CHD. However, in the two remaining negative studies (in Denmark<sup>22</sup> and the USA<sup>21</sup>), there was a strong inverse socioeconomic gradient of CHD. Despite this gradient, low control at work was not associated with CHD. Moreover, three studies, in England (Whitehall II),<sup>10</sup> the Czech Republic,<sup>25</sup> and Sweden<sup>26</sup> have explicitly adjusted for socioeconomic status and show an independent control effect on CHD. The control–CHD relation, therefore, is unlikely to be confounded by socioeconomic status.

The fact that low control predicts CHD independently of its relation to socioeconomic status decreases the likelihood that it “explains” the socioeconomic gradient simply because it is an alternative measure of social status. There are two other relevant points. First, a whole set of factors was related to employment grade<sup>1</sup>—low social supports, hostility, sedentary lifestyle, and smoking. The criticism that these factors may be indicators of socioeconomic status may also be made, but none of these measures explains the gradient as well as low control does. Of course, low control might be a better measure of socioeconomic status than the other factors. But, second, if this were true, why should low control account for more of the social gradient in CHD than it does for sickness absence rates<sup>27</sup> or plasma fibrinogen?<sup>11</sup>

Residual confounding is possible—ie, people with low control at work are predisposed in other ways to CHD. People arrive at their job positions after a lifetime in the educational and wider social systems, a process that may be related to development of risk.<sup>28,29</sup> Similarly, people in different employment grades have different social environments. In this analysis, and others from Whitehall II, we have controlled for these factors as much as possible: physical height, education, social supports, housing tenure, and car access were taken into account; we have also shown that work conditions and low control are related to CHD independently of both these and coronary risk factors.<sup>10</sup> Even if there is tentative acceptance that work is the major cause of the social gradient in CHD incidence in this cohort, some might argue that civil servants are atypical—a poor argument given the results of the case-control study from the Czech Republic.<sup>25</sup> This study applied the Whitehall II questions on work to Czech men, and showed that low control was related to acute myocardial infarction in middle-age, and that a combination of low control and coronary risk factors accounted for the higher risk of myocardial

infarction in men with low education. These results confirm the importance of psychosocial work characteristics to the social gradient.

If subsequent incidence data confirm these findings, they raise a problem: how can psychosocial work conditions account for much of the socioeconomic difference in CHD rates, when such differences are also observed in people beyond working age and in housewives classified by their husbands' occupations? The putative pathophysiological mechanisms by which psychosocial factors cause CHD involve activation of the autonomic nervous system and the hypothalamic pituitary adrenal axis, which in turn leads to metabolic changes that increase cardiovascular risk.<sup>30</sup> Work conditions are not the only way to activate these neuroendocrine pathways: low control in other areas of life, a self-image of low efficiency, and hostility may be other social or psychological factors that activate these pathways. This argument is supported by the finding that atherosclerosis in cynomolgus monkeys is accelerated in those of low status,<sup>31</sup> and that baboons of low status have higher basal concentrations of plasma cortisol and lipid disturbances, which suggest cardiovascular risk.<sup>32</sup> Similar metabolic changes have been observed in Whitehall II (unpublished).

Our findings build on evidence that psychosocial factors are important in the aetiology of CHD, since they suggest that such factors have an important role in the generation of social inequalities in CHD. Greater attention to the design of work environments<sup>33</sup> may be one important way to reduce inequalities in health.

#### Contributors

M G Marmot was the principal investigator and author of this paper, responsible for inception and design. H Bosma did all analyses, and helped with writing. H Hemingway was the principal adviser on the cardiovascular endpoints, responded to referees' comments, and helped with revisions. E Brunner was a senior investigator, responsible for biochemical analyses. S Stansfeld, co-principal investigator, gave critical comments on the paper, and was responsible for organisation of data collection.

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