Job Strain, Work Place Social Support, and Cardiovascular Disease: A Cross-Sectional Study of a Random Sample of the Swedish Working Population

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Abstract: This cross-sectional study investigates the relationship between the psychosocial work environment and cardiovascular disease (CVD) prevalence in a randomly selected, representative sample of 13,779 Swedish male and female workers. It was found that self-reported psychological job demands, work control, and co-worker social support combined greater then multiplicatively in relation to CVD prevalence. An age-adjusted prevalence ratio (PR) of 2.17 (95% CI-1.32, 3.56) was observed among workers with high demands, low control, and low social support compared to a low demand, high control, and high social support reference group. PRs of approximately 2.00 were observed in this group after consecutively controlling for the effects of age together with 11 other potential confounding factors. The magnitude of the age-adjusted PRs was greatest for blue collar males. Due to the cross-sectional nature of the study design, causal inferences cannot be made. The limitations of design and measurement are discussed in the context of the methodological weaknesses of the work stress field. (*Am J Public Health* 1988; 78:1336–1342.)

Introduction

The role of a stressful work environment in the development of cardiovascular disease (CVD) is a matter of current interest.[1–6] A model of job stress—the demand control model—has been proposed by Karasek.[7] The model predicts that biologically aversive strain will occur when the psychological demands of the job exceed the resources for control over task content. The research of Karasek, Theorell and colleagues suggests that it is this combination of high demands and low control that produces job strain.[7–12] Workers in high strain jobs have been shown to have greater risk of developing CVD.[7–9,11]

However, important methodological challenges to etiological inference remain in the occupational stress field[13,14] despite 20 years of research. These include: an over-reliance on cross-sectional as opposed to prospective designs; lack of generalizability due to the frequent restriction of samples to healthy, employed males; lack of valid and reliable measures of chronic disease outcomes; lack of exposure data with stress being evaluated on the basis of only a single measure in time; and incomplete models of the stress process. Investigations using the demand-control model have addressed a number of these problems.[15] However, the model itself has been criticized for not including other, equally important, psychosocial work characteristics.[9]

Although the cross-sectional design of the present study does not permit causal inferences, it does address two of the difficulties mentioned above: restricted samples amd incomplete modeling. Our study group consists of a random sample of both men and women which is representative of the entire working population of Sweden. Also, the demand-control model has been redefined by the addition of work related social support. Previous research has suggested that social support may serve to modify the impact of psychological demands both on and off the job.[2,6] A number of prospective studies have found an association between general social network

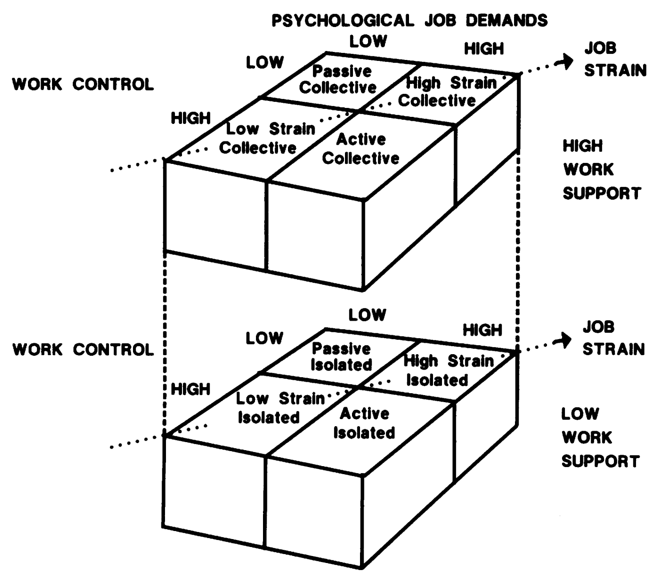

FIGURE 1—Demand-Control-Support Model

interaction and total mortality incidence.[16-21] Inadequate work place social support and social isolation has been shown to be associated with a higher incidence of angina pectoris among male workers in Israel;[22] a greater incidence of coronary heart disease among female clerks;[23] psychological problems among air traffic controllers;[24] higher cholesterol values among those whose work mates were constantly changing;[25] higher levels of illness among the unemployed;[26,27] a greater physical health impact from perceived stress among male petrochemical workers[28,29] and increased job stress and psychological strain among men in 23 occupations.[30] Studies which have looked at the moderating or so-called "buffering" effect of social support have found that it ameliorates the impact of perceived stress and job strain on physical and mental health.[30-32]

None of the research on social support and CVD to date has been linked with the demand-control formulation. This was an impetus to the present study and to the development of the model shown in Figure 1.

In the Demand-Control-Support model work-related social support has been dichotomized into isolated or collective conditions, thereby redefining the process of job strain (indicated by the diagonal arrows in Figure 1). This model

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permits us to examine whether the lack of social support combines with job strain to further increase the likelihood of CVD prevalence.

Methods

Study Sample

The data used were the Swedish Central Bureau of Statistics (SCB) Survey of Living Conditions ("ULF" in Swedish). This ongoing survey was mandated in the early 1970s by the Swedish Parliament as the major social accounting system to investigate the distribution of health status, income, education, and various aspects of the social and work environment. Over the past 14 years, considerable effort has been devoted to constructing reliable and valid indicators which, according to the survey's director, "are not colored by the respondent's ambitions, and reference frames. Among other matters this means that wishes, demands, opinions, etc. are not in principle surveyed."*

The Living Conditions Survey is conducted annually. A systematic random sample of individuals born on the 15th of each month was obtained from the National Registry of Births. Spouses were also interviewed. The present study combined two annual samples. In 1976, the sample consisted of 14,000 subjects and in 1977 of 14,500. Response rates were 79 per cent in 1976 and 81 per cent in 1977. Later studies on the effects of non-response on variables concerning illness were found to be minimal.[33,34]

The survey data were collected in a one-hour personal or telephone interview performed by a trained SCB interviewer. The present investigation used a sub-sample of 13,779 subjects consisting of employed persons from the ages of 16 to 65 (mean age 39); 52 per cent were males, 48 per cent females.

Measurement of Job Characteristics

The psychological job demands indicator was constructed from two items:

- Is your job hectic?
- Is your job psychologically demanding?

A Guttman scale was constructed using the combined responses to these questions. The proportions reporting low, medium, and high demands were: 34.7, 36.7 and 28.6 per cent, respectively. The Guttman coefficient of reproducibility for the scale was .92 and the coefficient of scalability was .79.

This measure can be criticized as providing an overly simplistic assessment of actual levels and types of demands, and for measuring subjective appraisals as opposed to objective psychosocial conditions. However, it correlates highly with other known occupational stressors such as lack of rest breaks, anticipation of job loss, and piece rate work,[10] and, it has been used in earlier prospective studies of the relation between job strain and coronary heart disease.[7]

The work control scale is a linear composite which measures decision-making authority, task variety, and personal freedom on the job. The following items were scored according to whether the subject had never (0), sometimes (1), or often (2) any of the following:

- Influence over the planning of work
- Influence over the setting of the work pace
- Influence over how time is used in work
- The planning of work breaks
- The planning of vacations
- Flexible working hours
- Freedom to receive a phone call during working hours
- Freedom to receive a private visitor at work
- Varied task content
- Varied work procedures
- Possibilities for on-going education as part of the job

Scores range from 0 to 22 with a mean of 12. The standard deviation was 4.4; scores were normally distributed. The Cronbach's alpha for the scale was .70; the average item to total scale correlation was .52. This measure is similar to indicators used by Swedish and American investigators to measure comparable aspects of job content.[7,10,12]

Work-related social support was measured using a scale consisting of five dichotomous items. Respondents were asked whether they:

- Could talk to co-workers during breaks
- Could leave their job to talk with co-workers
- Could interact with co-workers as part of their work
- Met with co-workers, outside of the work place
- Had met with a co-worker during the last six months

The total scale was constructed by adding the responses, across items. The scores ranged from 0–5, with a mean of 3, and a standard deviation of 1.5. The Cronbach's alpha was .75; the average item-to-total correlation being .70. This scale measures two aspects of support: opportunity to interact at work and if co-worker interaction is carried over into non-work life. It measures the basic prerequisites for social support but does not evaluate whether social contact at work is positive or negative and therefore may tend to overestimate the degree of social support found in Swedish work places. However, in a separate analysis we found that it correlated highly with more sensitive measures of social support available in the 1976 ULF, which included instrumental aid from co-workers, free time activities performed with co-workers, and supportive discussions between co-workers.[35]

The three psychological work environmental scales are weakly associated with each other. The Pearson correlation coefficients between the measures are: demands and control = .01, demands and support = .09, control and support = .12; indicating that these are relatively independent characteristics.

Cardiovascular Disease Indicator

The measure of CVD prevalence was based on the SCB's health classification system. As part of the larger SCB effort information on all types of long-term illness and disability was obtained in a personal interview. The respondent was asked a general question concerning health status: "Do you suffer any longstanding illness, effects of an injury, any disability or weakness?" Given an affirmative response, the interviewer probes: "Could you explain that a little more?", "What did the doctor say it was?", "What part of the body or organ system is affected?" The respondents were also questioned concerning use of regular medication, in order to include symptom free conditions, not covered by the previous questions. The description of any illness was coded by a central unit of the SCB, using a coding system developed and tested by two Swedish physicians, who worked as consultants to the SCB. They matched symptoms with the International Classification of Disease, 8th revision. Based on the total set of responses, SCB coders classified 804 subjects with ICD codes 3900–4589 as having CVD. The prevalence rate for the entire study group was 5.83 per cent,

*Vogel J: The Swedish annual Level of Living Survey: Social indicators and social reporting as an official statistics program. Paper presented at the Tenth World Congress of Sociology Mexico City, August 16, 1982, p. 30.

TABLE 1—Prevalence Rates for Cardiovascular Disease in the Demand-Control-Support Model

	Psychological Job Demands		
	Low Demand	Medium Demand	High Demand
High Work Support			
% Low Work Control (n)	[1] 5.48 (675)	[2] 7.26 (822)	[3] 6.36 (566)
% Medium Work Control (n)	[4] 4.95 (808)	[5] 4.33 (876)	[6] 5.66 (672)
% High Work Control (n)	[7] 2.88 (729)	[8] 3.25 (862)	[9] 5.11 (880)
Medium Work Support			
% Low Work Control (n)	[10] 4.46 (493)	[11] 7.39 (555)	[12] 7.44 (363)
% Medium Work Control (n)	[13] 7.51 (506)	[14] 6.50 (523)	[15] 7.25 (331)
% High Work Control (n)	[16] 3.99 (376)	[17] 5.84 (411)	[18] 5.34 (393)
Low Work Support			
% Low Work Control (n)	[19] 8.22 (426)	[20] 8.33 (492)	[21] 9.25 (400)
% Medium Work Control (n)	[22] 5.59 (420)	[23] 8.53 (305)	[24] 6.81 (191)
% High Work Control (n)	[25] 6.25 (352)	[26] 6.94 (216)	[27] 11.03 (136)

NOTE: Numbers 1 through 27 in brackets correspond to "cells" referred to in text.

395 females with CVD with a prevalence rate of 6.03 per cent and 409 males with a prevalence rate of 5.66 per cent. These differences in sex-specific rates are similar to those reported in other studies of cardiovascular prevalence in Sweden and Britain.** Karasek found that the CVD prevalence rate in Swedish employed males was 5.9 per cent, as measured by having two or more self-reported symptoms on the Karasek-Theorell CVD indicator.[7] The difference in prevalence estimations between the SCB and the Karasek-Theorell indicators may be due to the much larger sample size of the present study, and/or to the divergence in diagnostic methods.

The predictive validity of the CVD indicator was examined by observing whether or not individuals who report symptoms were later at risk for CVD mortality. The survey group was linked to the National Mortality Registry using the individual person number assigned to all Swedish residents. Incidence of cardiovascular related mortality was identified by combining all deaths for arteriosclerotic heart disease, cerebrovascular and peripheral vascular disease (ICD codes, 8th Revision, 400–404, 410–414, 427, 430–436, 440–445). During the six-year follow-up period, individuals classified as having CVD had an age- and sex-adjusted relative risk ratio of 3.32 (95% CI-3:00, 3.64) for cardiovascular-related mortality, as compared to persons who were classified as not having CVD. In order to test the discriminant validity of the CVD prevalence measure, an indicator of all noncardiovascular mortality was constructed. CVD prevalence cases had a much lower, and age- and sex-adjusted relative risk ratio of 1.15 (95% CI-.71, 1.59) for noncardiovascular-related mortality.

Potential Confounding Factors and Effect Modifiers

Twelve variables were examined to see if they confounded or otherwise modified the relationship between work environment characteristics and CVD. These factors are displayed in Appendix I and are described in more detail elsewhere.[21,35]

Statistical Analysis

The data were analyzed using a series of epidemiologic programs developed by Rothman and Boice.[36] The most frequent measure of association was the relative prevalence ratio. The Mantel-Haenszel Chi was used for hypothesis testing[37] and confidence limits were constructed from the point estimates of the PR and the Mantel-Haenszel test statistic, using test-based interval estimation.[38–40]

When controlling for confounding factors the data were first stratified into categories of the confounding factor, and a weighted average of the stratum specific rate ratios was obtained using Mantel-Haenszel procedures.[36,37] Hypothesis testing and confidence interval estimation in the stratified analysis were performed using Mantel-Haenszel[36] and Miettinen's techniques.[40]

To analyze for interactions, an index of synergy—the Rothman Interaction Ratio—was used to evaluate whether two or more factors combine greater than additively in relation to risk.[41,42] This expression is the ratio of the observed effect of combined exposure to two or more risk factors, divided by that effect which would be expected if the factors were acting independently. If the interaction ratio exceeds 1, it is an index of synergism and if the value of the ratio is less than 1, it is an index of antagonism. A slight modification of Rothman's approach has been used in this study in that the independent contribution of each risk factor is arrived at by simultaneously controlling for the other factors following the modeling procedure proposed by Breslow and Day.[43]

Results

The combined effects of psychological job demands, work control, and work-related social support on CVD are shown for prevalence rates in Table 1 and for age-adjusted PRs in Table 2. The demand-control model is reproduced in each of these tables for high, medium, and low levels of social support. The reference category used for the age-adjusted PR calculations is the low demand, high control, and high support group. These workers were identified as the low exposure group based on the theoretical criteria of the model which distinguishes this as having the fewest demands combined with the maximum psychosocial resources.

We can first examine the impact of the introduction of social support on the demand-control model by comparing the overall magnitude of the prevalence rates and ratios at each level of social support. With few exceptions for each demand-control combination prevalence rates and ratios increase with decreasing levels of social support. Within the high social support category, PRs relative to the reference category are highest in high strain combinations where the level of job demands exceed that of work control (i.e., cells

**Lundberg O: Social class and inequalities in morbidity—Some recent Swedish findings. Paper presented at the Fourth Nordic Social Policy Research Seminar, Hasselby Slott, Sweden, October 1984.

TABLE 2—Age-Adjusted Prevalence Ratios for Cardiovascular Disease in the Demand-Control-Support Model

	Psychological Job Demands					
	Low Demand		Medium Demand		High Demand	
High Work Support						
% Low Work Control	[1]	1.44	[2]	1.69	[3]	1.82
95% CI		(.87, 2.39)		(1.05, 2.73)		(1.10, 3.01)
% Medium Work Control	[4]	1.59	[5]	1.34	[6]	1.74
95% CI		(.96, 2.62)		(.80, 2.25)		(1.06, 2.87)
% High Work Control	[7]	1.00	[8]	1.11	[9]	1.58
95% CI		(Reference Category)		(.64, 1.92)		(.97, 2.58)
Medium Work Support						
% Low Work Control	[10]	1.69	[11]	1.88	[12]	1.94
95% CI		(1.00, 2.85)		(1.14, 3.09)		(1.14, 3.31)
% Medium Work Control	[13]	1.85	[14]	1.70	[15]	1.86
95% CI		(1.12, 3.06)		(1.02, 2.84)		(1.07, 3.22)
% High Work Control	[16]	1.14	[17]	1.80	[18]	1.30
95% CI		(.60, 2.17)		(1.04, 3.12)		(.72, 2.29)
Low Work Support						
% Low Work Control	[19]	1.95	[20]	2.10	[21]	2.17
95% CI		(1.18, 3.23)		(1.30, 3.38)		(1.32, 3.56)
% Medium Work Control	[22]	1.49	[23]	2.08	[24]	1.57
95% CI		(.85, 2.60)		(1.22, 3.56)		(.81, 3.06)
% High Work Control	[25]	1.43	[26]	1.77	[27]	2.55
95% CI		(.81, 2.53)		(.94, 3.30)		(1.38, 4.71)

NOTE: Numbers 1 through 27 in brackets correspond to "cells" referred to in text.

2, 3, and 6 in Table 2). Moreover, the job combinations predicted by the model to be the highest strain groups (high demands and low control) demonstrate an increase in the magnitude of the PRs with decreasing levels of social support.

The demand-control model hypothesizes that a pattern of increasing strain will occur along a strain diagonal, shown for high and low levels of social support in Figure 1. The results for CVD prevalence along this diagonal are shown in Table 1 and 2. As the job combinations change from low demand and high control (cells 7, 16, 25) to medium demands and medium control (cells 5, 14, 23) to high demands and low control (cells 3, 12, 21) the prevalence rates and ratios increase. However, as the level of social support decreases, CVD prevalence rates and PRs increase at each point along the strain diagonal.

The potential modifying effect of work control on the relation between job demands and cardiovascular prevalence is also evident in the low social support condition of the high demand-high control group (cell 27). The PR for this group is the highest observed. It was not predicted by the model. These results suggest that the modifying effect of work control on the job demand and CVD relationship is evident only under a particular contingency: when social support from co-workers is present. If workers have few social interaction opportunities, there is an elevation in cardiovascular prevalence in the high demand-high control combination.

Other job combinations exhibit elevated prevalence rates and ratios relative to the reference category, not predicted by the basic demand-control formulation. The low demand-low control cells characterized in Figure 1 as passive work situations (cells 1, 10, 19) show substantially elevated PRs when social support is medium (PR = 1.69) or low (PR = 1.95).

TABLE 3—Rothman's Interaction Analysis for Cardiovascular Disease

Excess Prevalence Risk Analysis:

Job Variable or Combination	Controlling For:	Prevalence Ratio (95% CI)	% Excess Prevalence Risk
Job Demands	Age Control Support	1.24 (1.06, 1.47)	24
Work Control	Age Demands Support	1.25 (1.06, 1.47)	25
Work Support	Age Demands Control	1.29 (1.10, 1.52)	29
High Demand, Low Control Combination	Age Support	1.69 (1.25,2.28)	19
High Demand, Low Support Combination	Age Control	1.56 (1.15, 2.10)	3
Low Control, Low Support Combination	Age Demands	1.71 (1.33, 2.25)	17

Interaction Analysis:
Additive Expected Excess Prevalence Risk: 78%
Observed Combined Excess Prevalence Risk: 117% (From Table 4, cell 21)
Rothman's Interaction Ratio (observed/expected): 1.50
Multiplicative Expected Prevalence Ratio: 2.00
Multiplicative Interaction Ratio: 1.09

TABLE 4—Controlling for Potential Confounding Factors and Effect Modifiers in the Demand-Control-Support and Cardiovascular Disease (CVD) Relationship[a]

Confounding Factor or Effect Modifier	Prevalence Ratio for CVD	95% CI
Crude	3.12	1.96–5.26
Age	2.17	1.32–3.56
(All subsequent analyses are age-adjusted)		
Sex	1.87	1.13–3.11
Physical Exercise[b]	1.96	1.04–3.70
Intergenerational Class Mobility	1.97	1.12–3.46
Marital Status	2.00	1.21–3.29
Non-work Social Support	2.16	1.32–3.53
Immigrant Status	2.16	1.30–3.56
Occupational Class Level	2.17	1.12–4.19
Rural vs Urban	2.19	1.33–3.56
Smoking[b]	2.31	1.20–4.48
Household Disposable Income	2.35	1.40–3.95
Physical Job Demands	3.41	1.59–7.36

[a]High demand, low control, low support workers are compared with low demand, high control, and high support workers.
[b]Information on smoking and exercise available only in 1977 survey group.

The statistical interactions of the three job characteristics were examined using Rothman's technique.[42,43]

Each factor, taken individually, contributed about the same amount of excess prevalence for CVD with PRs ranging from 1.24 to 1.29 (Table 3). The additive expected excess prevalence risk was 78 per cent and the observed combined excess prevalence risk was 117 per cent. By dividing the observed excess prevalence risk by the expected prevalence risk the Rothman's interaction ratio of 1.50 was obtained. This finding indicates that demands, control, and support combine 50 per cent more than additively. Indeed, as the multiplicative interaction ratio of 1.09 indicates, the three-factor interaction is 9 per cent more than multiplicative.

The influence of potential confounding factors such as health behaviors, other types of job demands, and sociodemographic factors was considered. In this series of analyses, the theoretically lowest exposure group (low demands-high control-high social support) was compared to the theoretically highest exposure category (high demands, low control, low social support) within each level or strata of the potential confounding factor. After testing for age and finding that this decreased the crude relative risk from 3.21 to 2.17, age was included in all subsequent analysis.

In Table 4, it can be seen that after controlling for the various potential confounding factors the PR remains elevated above 1.00; the potential confounding factors have been ordered along a continuum according to whether they decrease, have no effect on, or increase the PR. Controlling for sex and physical exercise reduces the PR to some degree. Adjusting for occupational mobility, marital status, social class level, etc. did not alter the PR. When controlling for the smoking, income, and physical job demand variables the association between the adverse work combination and CVD increased. This was particularly pronounced when controlling for physical job demands.

The individual and combined effects of demands, control and support were examined within four sex-class groups: blue collar males (n = 4,242), blue collar females (n = 3,661), white collar males (n = 2,987), and white collar females (n = 2,889). These results are shown in Table 5.

In all four sex-class groups the three work factors taken individually were only slightly associated with cardiovascular prevalence, while the combinations of work characteristics show the highest PRs.

There is a differential pattern of association among the various sex-class groups. The blue collar male group is most affected by the combination of the three job characteristics, particularly in the high demand-low control combination, with a PR 3.55 (95% CI-1.64, 7.69); and in the high demand-low control-low support combination with a PR 7.72 (95% CI-1.60, 37.39).

TABLE 5—Individual and Combined Cardiovascular Disease (CVD) Prevalence Ratios of Job Demands, Work Control, and Work Social Support for Sex and Class Groups

Job Variable or Combination	Blue Collar Males	White Collar Males	Blue Collar Females	White Collar Females
High Job Demands[a]	1.36	1.32	1.21	1.14
95% CI	(.99, 1.86)	(.93, 1.86)	(.88, 1.66)	(.76, 1.70)
Low Work Control[b]	1.42	1.03	1.12	1.07
95% CI	(.96, 2.09)	(.60, 1.75)	(.77, 1.62)	(.70, 1.66)
Low Social Support[c]	1.13	1.16	1.27	1.33
95% CI	(.82, 1.57)	(.77, 1.74)	(.97, 1.67)	(.89, 2.01)
High Job Demands Low Work Control[d]	3.55	1.03	1.43	1.13
95% CI	(1.64, 7.69)	(.36, 2.91)	(.88, 2.30)	(.36, 2.91)
High Job Demands, Low Social Support[e]	1.82	1.81	1.68	2.06
95% CI	(1.06, 3.15)	(1.02, 3.22)	(1.07, 2.63)	(1.05, 4.01)
Low Work Control, Low Social Support[f]	1.97	1.86	1.86	1.44
95% CI	(1.04, 3.73)	(.98, 3.51)	(.93, 3.71)	(.75, 2.77)
High Job Demands, Low Work Control, Low Social Support[g]	7.22	2.44	2.19	1.95
95% CI	(1.60, 37.39)	(.95, 6.28)	(.77, 6.23)	(.74, 5.12)

[a]Compared to low demand group, controlling for age, control, support.
[b]Compared to high control group, controlling for age, demand, support.
[c]Compared to high support group, controlling for age, demand, control.
[d]Compared to low demand, high control group, controlling for age, support.
[e]Compared to low demand, high support group, controlling for age, control.
[f]Compared to high control, high support group, controlling for age, demand.
[g]Compared to low demand, high control, high support group, controlling for age.

The combined effects are less evident in white collar males, although the basic hypothesized relations are still discernible.

For females the combined effects can also be seen but are less pronounced than among blue collar males and there is no obvious difference between blue and white collar workers.

Discussion

We are able to corroborate some of the basic predictions of the demand-control formulation in this study. As Karasek and others have previously reported, increasing levels of job strain are associated with increasing rates of cardiovascular prevalence. Work-related social support appears to accentuate the impact of job strain, in that workers with the lowest levels of social support had higher prevalence rates and ratios at each level of job strain. What was not predicted by the earlier demand-control model, however, was the elevated PRs found among active-isolated workers. Although we can only speculate as to the explanation for this finding, it is possible that in some working situations, high levels of control may accentuate rather than reduce the impact of demands. Our indicator of control may actually be measuring responsibility pressure, which in some occupations might constitute another component of job demands. This suggests the need to further refine our measurement of work control in order to at least distinguish control as a resource from responsibility as a demand.

Low social support in combination with low control was also associated with increased PRs, even in the absence of psychological job demands. This suggests that earlier insights concerning the negative impact of understimulation and qualitative underload on the cardiovascular system may still be relevant.[44]

The findings for the various sex and class groups clearly demonstrate that blue collar male workers have the strongest associations between adverse work combinations and CVD. However, it should be noted that one effect of reproducing the analysis performed in the total population for subgroups is to restrict the overall variance of work characteristics. This probably had the greatest effect on restricting the subgroup variation of work control, most strongly associated with sex and class, thereby diminishing its association with CVD.

Many of the work stress studies to date have been restricted to males.[45] It is often assumed that job characteristics that are important predictors for males are important for females as well. The findings reported here indicate that there may be sex as well as class differences. The sex-class analysis suggests that among women social support may be a more important predictor for CVD prevalence than work control. However, these findings can only be interpreted as suggestive and as an indication of the need to focus more attention on the specific differences betwen the various sex and class groups in the working population.

While this investigation supports the other research linking psychosocial work organization and CVD, it shares certain methodological weaknesses common to the field as a whole. As noted, because of the cross-sectional design, it is not possible to discern causal relationships. Since we cannot temporally separate work exposure from the manifestation of the disease, it is impossible to rule out alternative explanations to the observed associations. Although we have adjusted for a variety of individual characteristics, including class mobility, differential selection factors could provide alternative explanations to the observed findings. Although, as Theorell and his colleagues point out,[15] it is unlikely that individuals with CVD would select themselves into high strain occupations, it is plausible that individuals previously employed in high strain jobs who have experienced cardiovascular symptoms could move into passive isolated jobs.

Due to lack of information in the ULF data, it was not possible to examine the potential confounding effects of diet. It is unlikely, however, that diet would explain the associations observed in this study for dietary practices observed in Sweden are markedly homogenous. Although men in lower level occupations may tend to have a higher relative weight than those in upper level occupations,[15] since we have controlled for occupational class level in our analysis, the effects of class differences in diet and weight would at least be partly accounted for.

Another threat to causal interpretation arises from the fact that the presence of cardiovascular illness might affect the way in which individuals perceive their working situation, leading them to report it as being more psychologically demanding.

Although there is evidence of the predictive and discriminant validity of the CVD prevalence indicators, a more objective measure would be preferable. The measures of demand, control, and support used in this study also could be further refined. In the present study the evaluation of psychological job demands, in particular, is determined by the subjective perception of employees. Also, it is not clear that the social support measure reflects more than the frequency and opportunity for interaction. In general, research into the effects of the psychosocial environment would be strengthened if there were more objective exposure data.

In a recent review discussing many of the problems in research methodology which retard the development of the occupational stress field, Kasl notes a dilemma faced by many researchers: "better research designs are more a function of the resources available to the investigator and less a reflection of his/her level of methodological sophistication."[14] Large data sets, such as the one used in this study, are resources, but within these extensive samples, one must make use of the information as it exists. Certain strengths, i.e., a large, representative sample, a good response rate, and the ability to identify and control for a variety of confounding factors are important advantages; relying on survey questions which were not specifically formulated to meet the requirements of stress research has distinct disadvantages.

In conclusion, the addition of social support expands the demand-control formulation from an emphasis on the individual connection between a person and their job into the domain of collective relationships between people. Although recent reviews have pointed out that the findings concerning work place social support have been weak and confusing,[46] our results can only suggest that social support may have to be linked to influence processes such as work control to have any substantial effect on cardiovascular health.

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REFERENCES

1. Levi L, Frankenhaeuser M, Gardell B: Work stress related to social structures and processes: *In:* Elliott G, Eisdorfer C (eds): Stress and Human Health. New York: Springer, 1982; 119–146.
2. House J, Cottington E: Health and the workplace. *In:* Aiken L, Mechanic D (eds): Applications of Social Science to Clinical Medicine and Health Policy. New Brunswick: Rutgers University Press, 1986; 392–416.
3. Kasl S: The challenge of studying the disease effects of stressful work conditions. Am J Public Health 1981; 71:628–630.
4. Kasl S: Epidemiological contributions to the study of work stress. *In:* Cooper C, Payne R (eds): Stress at Work. New York: Wiley, 1977; 3–48.
5. Frankenhaeuser M: Psychoneuroendocrine approaches to the study of stressful person-environment transactions. *In:* Selye H (ed): Selye's Guide to Stress Research. New York: Van Nostrand Reinhold, 1980; 46–70.
6. House J, Jackson M: Occupational stress and health. *In:* Ahmed P, Coelho C (eds): Towards a New Definition of Health. New York: Plenum, 1979.
7. Karasek R, 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.
8. Alfredsson L, Karasek R, Theorell T: Myocardial infarction risk and psychosocial work environment characteristics: An analysis of the male Swedish work force. Soc Sci Med 1982; 16:463–467.
9. Baker D: The study of stress at work. Annu Rev Public Health 1985; 6:367–381.
10. Karasek R: Job demands, job decision latitude, and mental strain: Implications for job redesign. Admin Sci Q 1979; 24:285–308.
11. Karasek R, Theorell T, Schwartz J, Pieper C, Alfredsson A: Job psychological factors and coronary heart disease. Adv Cardiol 1982; 29:62–76.
12. Karasek R: Job socialization and job strain: Implications of two related psychosocial mechanisms for job design. *In:* Gardell B, Johansson G (eds): Working Life. Chichester, England: Johns Wiley and Sons, 1981.
13. House J, Strecher V, Metzner H, *et al:* Occupational stress and health among men and women in the Tecumseh Community Health Study. J Health Soc Behav 1986; 27:62–77.
14. Kasl SV: Methodologies in stress and health: Past difficulties, present dilemmas, future directions. *In:* Kasl S, Cooper C (eds): Stress and Health Issues in Research Methodology. Chichester, England: John Wiley and Sons, 1987.
15. Theorell T, Alfredsson L, Knox S, Perski A, Svensson J, Waller D: On the interplay between socioeconomic factors, personality and work environment in the pathogenesis of cardiovascular disease. Scan J Work Environ Health 1984; 10:373–380.
16. Berkman L: The relationshp of social networks and social support to morbidity and mortality. *In:* Cohen S, Syme SL (eds): Social Support and Health. New York: Academic Press, 1985; 240–259.
17. Berkman L, Syme SL: Social networks, host resistance and mortality: A nine year study of Alameda county residents. Am J Epidemiol 1979; 109: 186–204.
18. House J, Robbins C, Metzner H: The association of social relationships and activities with mortality. Prospective evidence from Tecumseh community health study. Am J Epidemiol 1982; 116:123–140.
19. Blazer D: Social support and mortality in an elderly community population. Am J Epidemiol 1982; 115:684–694.
20. Schoenbach V, Kaplan BH, Fredman L, Kleinbaum D: Social ties and mortality in Evans County, Georgia. Am J Epidemiol 1986; 123:577–591.
21. Orth-Gomer K, Johnson JV: Social network interaction and mortality: A six year follow-up of a random sample of the Swedish Population. J Chronic Dis 1987; 40:949–957.
22. Medalie J, Kahn HA, Neufeld H, Rise E, Goldbourt U: Five year myocardial infarction incidence: 2. Association of single variables to age and birthplace. J Chronic Dis 1973; 26:329–349.
23. Haynes S, Feinleib M: Women, work and coronary heart disease: Prospective findings from the Framingham Heart Study. Am J Public Health 1980; 70:133–141.
24. Rose R, Hurst M, Herd A: Cardiovascular and endocrine responses to work and the risk of psychiatric symptoms among air traffic controllers. *In:* Barrett J (ed): Stress and Mental Disorder. New York: Raven Press, 1979.
25. Cassel J: The use of medical records: Opportunities for epidemiological studies. JOM 1963; 5:185–190.
26. Cobb S, Kasl S: Termination: The consequences of job loss. NIOSH Pub. No. 77-224. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1977.
27. Jahoda M: Employment and Unemployment: A Socio-Psychological Analysis. Cambridge: Cambridge University Press, 1982.
28. House J: Work Stress and Social Support. Reading, Mass: Addison, Wesley, 1981.
29. House J, McMichael A, Wells J, Kaplan B, Landerman L: Occupational stress and health among factory workers. J Health Soc Behav 1979; 20: 139–160.
30. Pinneau S: Effect of social support on psychological and physiological strain. Doctoral dissertation, University of Michigan, 1975.
31. LaRocco J, House J, French J: Social support, occuptional stress and health. J Health Soc Behav 1980; 21:202–218.
32. Karasek R, Triantis K, Chaudhry S: Co-worker and supervisor support as moderators of associations between task characteristics and mental strain. J Occ Behav 1982; 3:147–160.
33. Bergman L, Hanve R, Rappe R: Why do some people refuse to particpate in interview surveys? Stat Rev 1978; 5:286–301.
34. Lindstrom H: Non-response estimates due to illness: S.C.B. Report No. 24. Stockholm: Swedish Statistical Bureau, 1981.
35. Johnson J: The impact of workplace social support, job demands and work control. Environmental and Organizational Psychology Research Reports, 1, Stockholm: University of Stockholm, 1986.
36. Rothman K, Boice J: Epidemiologic Analysis with a Programmable Calculator. Boston: Epidemiology Resources, 1982.
37. Mantel N, Haenszel W: Statistical aspects of the analysis of data from retrospective studies of disease. JNCI 1959; 22:719–748.
38. Miettinen O: Confounding and effect modification. Am J Epidemiol 1974; 100:350–535.
39. Miettinen O: Simple interval estimation of risk ratio. Am J Epdiemiol 1974; 100:515–516.
40. Miettinen O: Estimability and estimation in case-referent studies. Am J Epidemiol 1976; 103:226–235.
41. Rothman K: Synergy and antagonism in cause-effect relationships. Am J Epidemiol 1974; 99:385–388.
42. Rothman K: The estimation of synergy or antagonism. Am J Epidemiol 1976; 103:506–511.
43. Breslow N, Day N: Statistical methods in cancer research. Lyon: International Agency for Research on Cancer, 1980.
44. Frankenhaeuser M, Gardell B: Overload and underload in working life: Outline of a multidisciplinary approach. J Human Stress 1976; 2:35–46.
45. Haw M: Women, work and stress: A review and agenda for the future. J Hlth Soc Behav 1982; 123:132–144.
46. Kasl S, Wells J: Social support and health in the middle years. *In:* Cohen S, Syme SL (eds): Social Support and Health. New York: Academic Press, 1985; 143–165.

APPENDIX

Potential Confounding Factors and Effect Modifiers

Factor	Categories	Percentage
1. Age	16–44	66
	45–65	34
2. Sex	Female	48
	Male	52
3. Marital Status	Married	64
	Single	36
4. Income	Low	33
	Medium	34
	High	32
5. Intergenerational Class Mobility	Upwardly mobile	35
	Downwardly mobile	35
	No change in class	30
6. Occupational Class Level	Lower level manual	39
	Upper level manual	18
	Lower level office	18
	Upper level office	25
7. Immigrant Status	Native Swedish	87
	Immigrant	13
8. Region	Urban	65
	Rural	35
9. Smoking	Nonsmoker	61
	Smoker	39
10. Physical Exercise	Sedentary	60
	Active	40
11. Physical Job Demands	Low	29
	Medium	32
	High	39
12. Non-work Related Social Support	Low	33
	Medium	32
	High	34