In a recent article, Eaker et al. (1) presented convincing evidence that the increased psychosocial “stress” presumed to be a consequence of a low level of job control is not associated with increased risk of heart disease in either men or women in the Framingham Offspring Study. In fact, they found a paradoxical association between a higher level of job control and greater risk of heart disease in women (1). Some may see these results as essentially negative; however, the authors advance a causal explanation for their findings in psychosocial terms: Perhaps women with higher job control during the historical period in which the study was conducted experienced particular resentment (and thus higher psychosocial stress) in a society resistant to the progress exemplified by better working conditions for women. This explanatory paradigm seems at odds with past claims that low job control is the key etiologic factor in social gradients in heart disease (2, 3).

Perhaps there is a more parsimonious explanation for these findings. Job control, in itself, may have no causal relation to risk of heart disease. Associations between job control (and other socially patterned “psychosocial exposures”) in men and women may arise through noncausal mechanisms reflecting the limitations of observational epidemiology and the changing apparent social distribution of heart disease (4, 5). In the Framingham offspring cohort, total mortality was highest among low-status women; however, rates of heart disease were highest among women in higher-status employment (1). Women in higher-status employment would be expected to have higher levels of job control. The association between job control and heart disease reported could simply reflect the social patterning of both factors in this cohort. The fact that the association changed little after adjustment for current income, education, and occupational class may largely reflect the limitations of statistical adjustment in cases where the adjustment factors are poor indices of the etiologically relevant confounders (6).

When higher perceived stress is associated with social advantage, it appears to protect against smoking-related cancers, despite its association with heavier smoking, even in analyses with adjustment for several indices of social position (7). In this situation, it is clearly implausible that higher stress causes reduced risk of cancer. It seems similarly implausible to us that the improved working conditions women have struggled so long and hard for are really damaging to their health.

Eaker et al. (1) also make the point that associations between psychosocial factors and self-reported heart disease should be interpreted differently from associations between such factors and objectively diagnosed heart disease. We agree that the importance of this distinction cannot be overemphasized. It relates to the issue of reporting bias, which we have discussed and illustrated with empirical data elsewhere (8). Indeed, we were surprised that Eaker et al. appeared unaware of the particular relevance of this point to evidence of associations between job control and heart disease. This is illustrated in table 1, which contrasts associations between job control and subjective heart disease with associations between job control and objectively diagnosed coronary heart disease (electrocardiographic ischemia according to the Minnesota system) in the Whitehall II Study.

<table>
<thead>
<tr>
<th>Level of job control</th>
<th>Subjective CHD† outcome‡</th>
<th>Objective CHD outcome§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>2.02</td>
<td>1.17</td>
</tr>
<tr>
<td>Medium</td>
<td>1.44</td>
<td>1.16</td>
</tr>
<tr>
<td>High</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

* Baseline examinations for the Whitehall II Study were carried out in 1985–1988. The mean duration of follow-up was 5.3 years.
† CHD, coronary heart disease; RR, relative risk; CI, confidence interval.
‡ Data were obtained from Bosma et al. (3).
§ Data were obtained from Stansfeld et al. (9).

REFERENCES

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