Limitations of adjustment for reporting tendency in observational studies of stress and self reported coronary heart disease

J Macleod, G Davey Smith, P Heslop, C Metcalfe, D Carroll, C Hart

RESULTS AND DISCUSSION

Table 1 shows strong trends of increasing reporting tendency and increasing odds of incident angina with increasing perceived stress. High stress (compared with low stress) is strongly associated with an approximate doubling of the risk of angina. This association is attenuated on adjustment for reporting tendency though remains substantial. Men whose reporting tendency scores were in the top tertile at first screening had similarly increased odds of incident angina (adjusted OR top versus bottom tertile 2.54 (95% CI 1.54 to 4.21) p for trend <0.001). No corresponding relation is seen between stress and any objective disease outcomes. Confining mortality analyses to the first five years of follow up (not shown) did not change this pattern.

Perceived stress has been superseded by other self reported psychosocial exposures in more recent studies. It is probable, however, that measurement of these will be equally prone to the problem of bias. Adjustment for reporting tendency has

Table 1: Reporting tendency, angina, ischaemia and mortality according to level of perceived psychological stress at baseline

<table>
<thead>
<tr>
<th>Perceived stress</th>
<th>Mean reporting tendency score</th>
<th>Incident angina* (odds ratio (95% CI))</th>
<th>Incident ischaemia† (odds ratio (95% CI))</th>
<th>All cause mortality (hazard ratio (95% CI))</th>
<th>CHD mortality (hazard ratio (95% CI))</th>
</tr>
</thead>
<tbody>
<tr>
<td>High (n=739)</td>
<td></td>
<td>0.77</td>
<td>2.63 (1.59 to 4.33)</td>
<td>0.58 (0.31 to 1.08)</td>
<td>0.94 (0.80 to 1.10)</td>
</tr>
<tr>
<td>Medium (n=3017)</td>
<td></td>
<td>0.52</td>
<td>1.36 (0.90 to 2.03)</td>
<td>0.62 (0.33 to 1.16)</td>
<td>0.97 (0.82 to 1.14)</td>
</tr>
<tr>
<td>Low (n=1821)</td>
<td></td>
<td>0.41</td>
<td>1.00 (0.60 to 1.62)</td>
<td>0.90 (0.62 to 1.51)</td>
<td>0.91 (0.83 to 1.10)</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Adjustment A – age, occupational class, smoking (cigarettes smoked daily, ex, current, never smokers), alcohol consumption (0, >0–15, >15 units weekly), weekly hours of exercise, diastolic blood pressure (mm Hg), cholesterol (mmol/l), body mass index (kg/m²), lung function (observed FEV1 as % of expected). Adjustment B – all above and reporting tendency score. *Definite angina taken as pain or discomfort over the sternum or left chest and arm while hurrying or walking uphill causing the participant to slow down or stop, the pain subsiding in 10 minutes or less. Excluding participants with angina at recruitment and those not screened twice. High stress – n=310; Medium stress – n=1328; Low stress – n=834. †Definite ischaemia encompassed by Minnesota codes 1.1–1.3, 4.1–4.4, 5.1–5.3 and 7.1. Excluding participants with ischaemia at recruitment and those not screened twice. High stress – n=335; Medium stress – n=1340; Low stress – n=812.
occasionally been attempted but as the construct is impossible to measure precisely such adjustment is likely to be unsuccessful as we demonstrate.

Ischaemia and angina were often present together (odds ratio for presence of ischaemia given presence of angina 4.09 (95% CI 3.01 to 5.56) and both were predictive of CHD mortality (hazard ratios 2.93 (95% CI 2.41 to 3.56) and 2.15 (95% CI 1.74 to 2.65) respectively). While stress was associated with angina, it was neither associated with ischaemia, nor with subsequent CHD mortality. Some men with CHD would be expected to have a normal resting ECG. However it seems unlikely that among middle aged men followed up for over 20 years, CHD symptoms resulting from cardiovascular disease would not be associated with increased mortality risk. It seems most likely that the isolated association between stress and angina was an artefact. Subjects with a greater tendency to report their lives as stressful were also more inclined to report physical symptoms, including CHD symptoms. We do not suggest that the symptoms associated with stress were less “real” to the men reporting them, however it seems that their basis was often something other than myocardial ischaemia. This has important implications for prevention and treatment. These findings also cast doubt on the conclusions of studies presenting an association between self reported psychosocial exposure measures and substantially subjective outcomes as evidence for a causal relation between psychosocial factors and heart disease.\(^5\)

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CONTRIBUTORS

JM conducted the data analysis and assisted with its interpretation. GDS formulated the hypothesis and assisted with the interpretation of the analysis. PH, CM, DC and CH assisted with the interpretation of the analysis. All authors contributed to writing the paper.

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References


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