

## SHORT REPORT

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

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Recently, observational evidence has been suggested to show a causal association between various “psychosocial” exposures, including psychological stress, and heart disease.<sup>1,2</sup> Much of this evidence derives from studies in which a self reported psychosocial exposure is related to an outcome dependent on the subjective experience of coronary heart disease (CHD) symptoms. Such outcomes may be measured using standard symptom questionnaires (like the Rose angina schedule). Alternatively they may use diagnoses of disease from medical records, which depend on an individual perceiving symptoms and reporting them to a health worker. In these situations, reporting bias may generate spurious exposure-outcome associations.<sup>3</sup> For example if people who perceive and report their life as most stressful also over-report symptoms of cardiovascular disease then an artefactual association between stress and heart disease will result.

### METHODS

We investigated this phenomenon among 5577 middle aged men recruited from 27 Scottish workplaces in 1970–73. Measurements at recruitment included psychological stress (Reeder Stress Inventory (RSI)), questions on the experience of a range of physical symptoms, “Rose” angina and six lead, resting electrocardiograph. There is good evidence of construct and concurrent validity of the RSI and the instrument seems reliable.<sup>4</sup> A total of 2608 men were re-screened five years after recruitment. During 21 years of follow up 1590 men died, 602 from coronary heart disease (ICD9 410–414).

Stress scores were categorised as high (6–8), medium (4–5) and low (1–3). In an attempt to quantify tendency to over-report physical symptoms we took five symptoms (polydipsia, polyuria, pruritus, blurred vision and increased skin infections). These showed no relation to mortality over 21 years suggesting that it was unlikely they were related to serious disease. We constructed a reporting tendency variable

(0–5) based on a count of reports of these. Known diabetics (n=29) were excluded on the assumption that their reporting of these hyperglycaemic symptoms was more likely to reflect their diabetes than any reporting tendency.

We examined the relation between stress exposure, incident angina and incident ECG ischaemia (new angina or ischaemia at second screening) through logistic regression and the relation between perceived stress and mortality through Cox regression. Estimates were adjusted for age, occupational class, coronary risk factors and reporting tendency. Results are for men with complete data on all measures. Morbidity analyses are confined to men screened twice, mortality analyses are for all men. Results of mortality analyses were essentially unchanged with analysis confined to men screened twice (available on request). A full description of subjects and methods is published elsewhere.<sup>4</sup>

### 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.<sup>2</sup> 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

Perceived stress	Mean reporting tendency score	Incident angina* (odds ratio (95% CI))		Incident ischaemia† (odds ratio (95% CI))		All cause mortality (hazard ratio (95% CI))		CHD mortality (hazard ratio (95% CI))	
		Adjustment A	Adjustment B	Adjustment A	Adjustment B	Adjustment A	Adjustment B	Adjustment A	Adjustment B
High (n=739)	0.77	2.63 (1.59 to 4.33)	2.28 (1.37 to 3.80)	0.58 (0.31 to 1.08)	0.62 (0.33 to 1.16)	0.94 (0.80 to 1.10)	0.97 (0.82 to 1.14)	0.96 (0.74 to 1.25)	0.98 (0.75 to 1.27)
Medium (n=3017)	0.52	1.36 (0.90 to 2.05)	1.27 (0.84 to 1.92)	0.88 (0.60 to 1.27)	0.90 (0.62 to 1.31)	0.91 (0.82 to 1.02)	0.92 (0.83 to 1.03)	0.95 (0.80 to 1.14)	0.96 (0.80 to 1.15)
Low (n=1821)	0.41	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
p for trend	<0.001	<0.001	0.003	0.10	0.23	0.42	0.16	0.69	0.76

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/m<sup>2</sup>), lung function (observed FEV<sub>1</sub> 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<sup>5</sup> 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.<sup>5,6</sup>

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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. Funding: a grant from the Phase 2 of the Economic and Social Research Council's Health Variations Programme. Conflicts of interest: none.

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