



Psycho-social stress, lifestyle and socio-economic inequalities in morbidity and mortality

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The determinants of health inequalities are only partially understood. It has been suggested that psychosocial factors, such as perceived stress, are important in this regard. This suggestion currently enjoys widespread support, however, the evidence, which comes mainly from observational studies, should be interpreted with caution.

Many studies are cross-sectional, making the direction of causality between stress and disease impossible to determine. Most measurements of stress exposures and many health outcomes include a significant subjective component. Both may be influenced by a common reporting tendency generating apparent, though spurious, associations between stress and health. Similarly, an association between stress and social disadvantage will automatically lead to an apparent association between stress and health due to confounding by other correlates of deprivation.

We examined the relationships between perceived stress, social position, lifestyle, physiological risk factors and both morbidity and mortality within a workplace derived cohort of around 6,000 men and 1,2000 women recruited in Scotland in the early 1970s and followed up for over 20 years. Half this cohort was re-examined 5 years after initial recruitment. Angina and ischaemia were among the health outcomes we examined.

Higher stress was associated with social advantage in men, but not women. Cross-sectionally, greater stress was associated with unhealthy behaviour (greater smoking, greater alcohol consumption and less exercise) but with a mixed profile of physiological risk factors. The pattern was broadly similar for both men and women, although high stress was associated with lower body mass index in men and with higher body mass index in women. In men, stress showed a strong cross-sectional association with self-reported angina but not with ischaemia on ECG. In men, higher stress was associated with a greater than doubling of the risk of incident angina. However, higher stress was also associated with an apparent reduction in the risk of incident ECG ischaemia. Amongst men, higher stress was associated with an apparently reduced risk of both all cause mortality and mortality from coronary heart disease. A similar relationship with mortality was seen for cumulative stress at first and second screening and for stress that increased between first and second screening. After adjustment for social position, all of these relationships were attenuated. Amongst women, there was little difference in the risk of all cause mortality according to perceived stress. Only alcohol related mortality and mortality from respiratory diseases in men showed the association with stress predicted by the relationship between stress and behaviour. However, the suggestion of increase in both these indices with higher stress was weak and of small magnitude. This suggested a primacy of material circumstances in determining health.

The number of male hospital admissions for cardiovascular disease, alcohol-related illness and psychiatric illness increased with increasing stress. There was no convincing evidence of an effect of stress on female hospitalisation, although a similar trend to that of men was found.

Our findings illustrate some of the pitfalls around the interpretation of observational evidence in this area. Amongst men, we found an apparently increased risk of angina and admission to hospital with higher stress. This contrasted with an apparently decreased risk of ischaemic change on ECG and of mortality. We suggest this anomaly arose due to the influence of bias in relation to the former outcomes and confounding in relation to the latter. In the absence of evidence from controlled trials, this suggests that interventions targeting psychosocial factors are not likely to be an effective strategy to reduce health inequalities. A focus on the reduction of material inequalities alongside interventions aimed at established risk factors seems more appropriate.

Background

The policy aim of reducing social inequalities in health is complicated by an incomplete understanding of the processes that generate these inequalities. Psychosocial factors, such as perceived stress, are plausible causes related to health through direct neuro-endocrine pathways or through their promotion of unhealthy behaviour. Since these factors are often related to social position it has been suggested that they may be important determinants of social inequalities in health, particularly inequalities in coronary heart disease. It has been further suggested that interventions targeting these factors may be an effective strategy to reduce health inequalities.

The importance of psychosocial factors, particularly stress, to health enjoys widespread popular credence. However, scientific evidence supporting a causal role for these exposures as important determinants of morbidity and mortality derives almost exclusively from observational studies. Several considerations suggest that such evidence should be interpreted with caution.

First, many studies are cross-sectional, making the direction of causality between exposure and outcome impossible to determine. Causation may run mainly from health to stress, rather than vice versa. For example, it is likely that the experience of ill health will increase perception - and hence reporting - of stress. Second, where the measurement of both exposure and outcome depend substantially on individual subjective perception (both of "stress" and of illness), both may be inflated by a common reporting tendency, producing spurious associations. Third, rather than "explaining" social inequalities in health, the fact that health and psychosocial factors like stress are both related to social position could generate an apparent, but non-causal, association between the two. Confounding of this nature may lead to associations that remain even after adjustment for social position is made in the statistical model. Apparently robust associations would remain if the psycho-social factor captures a dimension of social position which is not adequately measured by the indicator of social position being used in the analysis. Finally, effects of stress on health may be best understood through consideration of

cumulative or changing stress over time and over extended periods of follow-up.

Data and methods

The West of Scotland Collaborative Study involves around 6,000 men and 1,200 women recruited from 27 workplaces in 1971-73. Half this cohort was re-screened in 1977. Screening included measurement of perceived stress (via the Reeder Stress Inventory), physiological risk factors, behavioural risk factors, indicators of social position and indices of coronary morbidity (Rose angina questionnaire and ECG coded according to the Minnesota system). Cause specific mortality data (ICD-9) are available for 21 years of follow-up. Morbidity data from the Scottish Cancer Registry and the Scottish Morbidity Register (hospital admissions) were included from the same period.

Stress at first screening was divided into three categories (high, medium, low). For participants screened twice, stress was divided into the same categories of cumulative stress score and into increasing, stable and decreasing perceived stress. A score variable was derived based on a count of complaints of somatic symptoms showing no relationship to mortality over 21 years of follow-up. Reporting of these symptoms was assumed to relate primarily to reporting tendency rather than underlying somatic pathology.

Cross-sectional relationships between stress, social position, lifestyle, risk factors, reporting tendency and morbidity were examined. Incidence of new coronary heart disease (angina or ischaemia) at second screening by stress category at first screening was calculated in logistic regression models. All cause and cause specific mortality by stress category at first screening, cumulative stress at first and second screening and change in stress over the screening interval was calculated in proportional hazards models.

Findings

Perceived stress showed a graded association with occupational class in men, from highest mean stress in Social Class I to lowest in Social Class V. No association between stress and occupational class was apparent amongst women. Before adjustment for social position, higher stress was associated with an adverse profile of behavioural risk (more smoking and greater alcohol consumption in men and women, less exercise in men only) but a mixed profile of physiological risk (lower blood pressure and higher cholesterol in men and women, lower BMI in men, higher BMI in women). Adjustment for occupational class strengthened the association with unhealthy behaviour. Apart from BMI, none of the physiological risk factors were associated with stress following this adjustment.

Reported stress showed a strong, direct association with reporting tendency in men. Prevalent angina at first screening significantly increased with both increasing perceived stress and increasing reporting tendency score. Table 1 shows the odds for incident angina and incident ischaemia, comparing those with medium and high stress to those with low stress. The odds for incident angina were more than doubled amongst high, compared to low stress men. By contrast, the odds for incident ischaemia were almost halved in high, compared to low stress men (Table 1). Incident angina was also significantly increased amongst those participants with high reporting tendency scores. Adding reporting tendency score to the model attenuated the above effect estimates but in the case of angina they remained very strong. .

Table 2 shows the relative risk of mortality, both for all causes of mortality and for specific causes of death, again comparing men with high and medium stress to those with low stress. It shows that the relative hazard of all cause mortality was reduced in medium and high, compared to low stress participants. These reductions were small and were attenuated following adjustment for current occupational class. Most classes of cause specific mortality followed this pattern. Cumulative stress scores showed a similar pattern with mortality as did change in stress score (i.e. reduced hazard of mortality amongst participants with increasing or stable stress scores compared to decreasing stress scores). Amongst women, there was little difference in the relative hazard of all-cause mortality or cause-specific mortality according to perceived stress, though this analysis was limited due to smaller sample size. Following adjustment for age and socioeconomic status, the number of hospital admissions for cardiovascular

disease, alcohol-related illness and psychiatric illness in men increased with increasing stress. Deaths per admission showed a trend in the opposite direction in the case of cardiovascular disease. No convincing evidence of an effect of stress on hospitalisation was found for women.

Future research

These findings illustrate the pitfalls of observational research on psychosocial exposures and in particular, the influence of bias and confounding. Future research must address these issues. The use of objective exposure and outcome measures reduces the potential for bias. Studying psychosocial factors in populations where they are not proxies for social disadvantage is a partial solution to the problem of confounding. Experimental studies are needed to address this issue fully, and to determine whether psychosocial interventions are a promising strategy to improve population health.

Policy implications

It has been suggested that psychosocial factors may hold the key to reductions in health inequalities; these data cast doubt on this suggestion. Previous studies that have shown an association between stress and objectively poorer health have been undertaken in populations where stress was also related to social disadvantage. It is likely that apparent relationships with health were the product of confounding. In the present study such confounding produced an association between stress and better health because stress was related to social advantage, particularly in men. This was despite an association between stress and unhealthy behaviour.

These findings have important implications for policy. First, though individual behaviours undoubtedly contribute to health, the contribution of material circumstances is more important. Strategies to reduce health inequalities should reflect this. Second, interventions to reduce psychological stress and improve the psychosocial environment are humanitarian imperatives, arguably in need of no further epidemiological justification. However, unless improvements in the psychosocial environment, are accompanied by improvements in the material environment they are unlikely to lead to better health.

Table 1. Stress and incident angina and ischaemia (men only)

		Adjusted for age only	Adjusted for age, social class and risk-factors *	Adjusted for age, social class, risk-factors and reporting tendency
Odds ratio for	High stress	2.33	2.63	2.28
incident angina (95% CI) n=2472 [†]		(1.43-3.80)	(1.59-4.33)	(1.37-3.80)
	Medium stress	1.23	1.36	1.27
		(0.83-1.83)	(0.90-2.05)	(0.84-1.92)
	Low stress	1.00	1.00	1.00
		**p=0.002	p<0.001	p=0.003
Odds ratio for incident	High stress	0.63	0.58	0.62
		(0.34-1.15)	(0.31-1.08)	(0.33-1.16)
ischaemia	Medium stress	1.00	0.88	0.90
(95% CI) n=2487 ^{††}	Low stress	1.00	1.00	1.00
		**p=0.24	p=0.10	p=0.16

Notes:

† n = 2472, excluding participants who had angina at first screening and those not screened twice.

- th n = 2487, excluding participants who had ischaemia at first screening and those not screened twice
- Risk-factors were smoking (cigarettes per day, ex, current, never smokers), alcohol consumption (0, 0-15, 15 or above units weekly), weekly hours of exercise, cholesterol concentration (mmol/l), diastolic blood pressure (mmHg), body mass index (kg/m2), FEV1% participants with missing values excluded.

** p for trend.

Relative risk of mortality (95% CI) associated with perceived stress at first screening (men only)

	Perceived stress	Adjusted for age	Adjusted for age and occupational class	Adjusted for age and all markers of social position	Adjusted, for age social position and risk factors**	Adjusted, for age social position, risk factors and somatisation
All cause (1528 deaths)	High (n=711) Medium (n=2912) Low (n=1764)	0.88 (0.75-1.03) 0.85 (0.76-0.94) 1.00	0.95 (0.81-1.12) 0.93 (0.83-1.04) 1.00	0.95 (0.81-1.12) 0.94 (0.84-1.05) 1.00	0.94 (0.80-1.11) 0.93 (0.83-1.05) 1.00	0.97 (0.83-1.15) 0.95 (0.84-1.06) 1.00
p for trend		0.020	0.34	0.38	0.33	0.55
All cardiovascular (785 deaths)	High Medium Low	0.84 (0.67-1.05) 0.83 (0.71-0.97) 1.00	0.90 (0.72-1.13) 0.91 (0.78-1.06) 1.00	0.90 (0.72-1.13) 0.92 (0.79-1.07) 1.00	0.92 (0.73-1.16) 0.92 (0.79-1.08) 1.00	0.94 (0.74-1.18) 0.93 (0.79-1.09) 1.00
p for trend		0.037	0.26	0.28	0.36	0.44
Coronary heart disease (590 deaths) p for trend	High Medium Low	0.86 (0.67-1.12) 0.86 (0.72-1.03) 1.00 0.14	0.93 (0.71-1.21) 0.94 (0.79-1.13) 1.00 0.50	0.93 (0.71-1.21) 0.95 (0.79-1.14) 1.00 0.52	0.96 (0.74-1.25) 0.97 (0.81-1.16) 1.00 0.72	0.97 (0.74-1.27) 0.97 (0.81-1.17) 1.00 0.78
Smoking-related cancers (269 deaths)	High Medium Low	0.64 (0.42-0.96) 0.79 (0.61-1.02) 1.00	0.71 (0.47-1.08) 0.91 (0.70-1.18) 1.00	0.71 (0.47-1.08) 0.91 (0.70-1.19) 1.00	0.66 (0.44-1.01) 0.89 (0.69-1.16) 1.00	0.69 (0.45-1.06) 0.90 (0.69-1.18) 1.00
p for trend		0.016	0.13	0.13	0.06	0.10
Other Cancers (221 deaths)	High Medium Low	0.91 (0.59-1.40) 0.93 (0.70-1.25) 1.00	0.91 (0.59-1.40) 0.93 (0.69-1.25) 1.00	0.93 (0.60-1.44) 0.94 (0.70-1.27) 1.00	0.94 (0.61-1.46) 0.94 (0.70-1.27) 1.00	1.00 (0.64-1.56) 0.96 (0.71-1.30) 1.00
p for trend		0.61	0.60	0.70	0.73	0.93
Stroke (122 deaths)	High Medium Low	0.85 (0.49-1.48) 0.75 (0.51-1.10) 1.00	0.90 (0.52-1.58) 0.80 (0.54-1.19) 1.00	0.92 (0.52-1.60) 0.81 (0.55-1.20) 1.00	0.94 (0.54-1.65) 0.81 (0.54-1.21) 1.00	0.98 (0.55-1.72) 0.82 (0.55-1.23) 1.00
p for trend		0.32	0.51	0.54	0.59	0.68
Alcohol related (105 deaths)	High Medium Low	1.13 (0.63-2.01) 0.86 (0.56-1.32) 1.00	1.29 (0.72-2.31) 1.01 (0.65-1.56) 1.00	1.26 (0.71-2.26) 1.01 (0.65-1.55) 1.00	1.22 (0.68-2.20) 0.97 (0.63-1.51) 1.00	1.30 (0.72-2.35) 1.00 (0.64-1.54) 1.00
p for trend		0.91	0.51	0.54	0.59	0.68
Respiratory (100 deaths)	High Medium Low	1.23 (0.69-2.22) 0.94 (0.61-1.46) 1.00	1.39 (0.77-2.50) 1.09 (0.69-1.71) 1.00	1.36 (0.75-2.45) 1.11 (0.71-1.74) 1.00	1.22 (0.67-2.22) 0.96 (0.61-1.52) 1.00	1.38 (0.75-2.53) 0.99 (0.63-1.56) 1.00
p for trend		0.62	0.31	0.33	0.61	0.40

** Risk factors as in table 1

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Selected publications:

Heslop, P., Davey Smith, G., Carroll, D., Macleod, J. and Hart, C. (2001) 'Perceived stress and coronary heart disease risk-factors: the contribution of socio-economic' *British Journal of Health Psychology* 6: 167-178.

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Heslop, P., Davey Smith, G., Macleod, J. and Hart, C. (in press 2001) 'The socioeconomic position of employed women, risk factors and mortality' *Social Science and Medicine*.

Heslop, P., Davey Smith, G., Macleod, J., Metcalfe, C. and Hart, C. (in press 2001) 'Job satisfaction, self-reported stress, cardiovascular risk factors and mortality' *Social Science and Medicine*.

Heslop, P., Davey Smith, G., Macleod, J., Metcalfe, C., Carroll, D. and Hart, C. (in press 2001) 'Are the effects of psychosocial exposures attributable to confounding? Evidence from a prospective observational study on psychological stress and mortality' *Journal of Epidemiology and Community Health.*

Information about Programme

The Health Variations Programme was established by the Economic and Social Research Council in 1996 to focus on the causes of health inequalities in Britain. Over the last two decades, Britain has got healthier and richer, but inequalities in health and income have increased. Death rates have fallen but mortality differences between social classes I and V have widened; real incomes have risen but so has the proportion of the population living in poverty. The Programme aims to:

- advance understanding of the social processes which underlie and mediate socioeconomic inequalities in health;
- advance the methodology of health inequalities research;
- contribute to the development of policy and practice to reduce the health gap between socioeconomic groups.

There are 26 projects in the Programme, based in university departments and research units across the UK. The projects have been established in two phases: in 1996/7 and in 1998/9. They address questions at the cutting-edge of health inequalities research, including the influence of material and psycho-social factors across the lifecourse, the influence of gender and ethnicity and whether and how areas have an effect on the socioeconomic gradient over and above the influence of individual socioeconomic status. The potential contribution of policy, at national and local level, is also addressed.



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