Coronary heart disease: a review of the role of psychosocial stress and social support


Abstract

Background The role of psychosocial factors in the aetiology of coronary heart disease continues to be debated. Despite public perception of a major role for their effect, scientific opinion on their relevance remains divided. This paper reviews the literature on the influence of social support and life stress on coronary heart disease incidence and mortality.

Methods Observational studies published in English, based on over 100 human subjects from the general population, investigating life stress or social support were considered. Fourteen studies derived from MEDLINE searches on MeSH headings: coronary disease; stress, psychological; social support; social isolation; life change events. An equivalent search of BIDS and studies referenced by papers identified using these sources was carried out.

Results and conclusions The review concludes that both life stress and social support were found to have an influence on coronary heart disease, social support more so than stress. Both have a stronger influence on coronary heart disease mortality than on initial incidence of clinical disease. Measures of the quality of support, in particular emotional support, show the largest effects. The review highlights problems in drawing conclusions from the available literature; in particular, the inconsistency in measures used to define the psychosocial factors. Further studies are needed to investigate interrelationships between stress and social support, and a recommendation is made to adopt pragmatic measures in future studies, which if proven to have an effect, may be open to modification.

Keywords: psychosocial stress, social support, coronary heart disease

Introduction

The role of psychosocial risk factors in coronary heart disease aetiology has often been investigated. Ever since Type A behaviour pattern was described by Friedman and Rosenman in 1959, there has been considerable interest in the role that psychosocial variables play in the development of coronary heart disease. Within this broad heading, research has included the influence of factors such as personality type, life stress, social support, education and socioeconomic status. Even with our modern understanding of the natural history of coronary heart disease, accurate prediction of disease incidence, and of subsequent prognosis, is not yet possible. It has been suggested that research into psychosocial factors could improve our ability to predict disease so as to intervene and potentially prevent its occurrence. This paper reviews the evidence concerning the magnitude of the effects of these factors.

Social support is the name given to the supportive interaction between humans, and may refer to structural aspects of a social network (e.g. size of network) or the different functions of support: the areas of life in which support is needed (e.g. emotional support, practical support). Life stress may be described as upset caused by external influences such as the work environment or home situation.

The role of psychosocial factors in the natural history of coronary heart disease may be investigated at different stages in the process of the disease. It is commonly related to measurable events such as the onset of clinical symptoms or mortality. It is necessary to review the evidence linking stress, social support and outcome separately for both clinical disease and death because the role that psychosocial factors have may be different at each stage in the natural history of the disease. Most studies focus on only one issue, but by recognizing each as separate some of the apparent variation in results from studies may be explained. This review considers incidence of clinical disease and mortality separately, examining the role of stress and...
social support in each, including methodological points where relevant. It is important to examine the literature because from the point of view of primary prevention it is useful for health professionals to recognize those at risk, and the nature of the support that may be required to reduce that risk. Given that the majority of patients suffering myocardial infarction would name stress as a major cause there is clearly a need to assess the role of stress and social support on coronary disease.

**Method of identification of included papers**

Potential papers for review were identified from four sources: (1) a MEDLINE search covering papers published since 1966 on MeSH headings: coronary disease; stress, psychological; social support; social isolation; life change events; (2) a Bath Information and Data Services (BIDS) search covering papers published since 1981 on equivalent words in the title, abstract or keywords; (3) the group’s knowledge in the field; (4) an extensive search of studies referenced in papers identified from the previous three sources. Observational studies on over 100 human subjects from the general population (either men or women), published in English, investigating life stress or social support were considered. Other studies not meeting these criteria are not the subject of this review.

**Measures of stress and social support**

Measuring stress and social support is not a straightforward task. There are many different dimensions that could be measured and as no ‘gold standard’ measure exists for either stress or social support, many different instruments are used. This complicates any comparison between papers using different measures, so the type of measure used in each paper is highlighted in this review.

For life stress, broadly two approaches exist to measurement: the objective measures (e.g. life events scales) and the subjective measures (e.g. ones involving the individual’s emotive response to stress). The objective measures benefit from their apparent objectivity but fail to measure any effect on the individual and are therefore crude measures. Instruments that measure the individual’s response are inherently subjective and also subject to more potential bias in recall and other external influences, and cannot be independently validated. Both approaches can be abused if an inappropriate time point is chosen at which to measure stress or social support. Methods of assessing social support in population surveys have been reviewed by Orth-Gomér and Undén.

**The buffer hypothesis**

Emerging from the research into stress and social support, a plausible framework for how social support might influence health has been suggested. The concept, known as the ‘buffer hypothesis’, is that social support interacts with stress causing people who have high levels of social support to be protected from the adverse effects of stress. Efforts to explain this hypothesis through biological mechanisms have concentrated on demonstrating the effects of psychologic stimuli on the autonomic nervous system.

**Evidence from studies**

**Studies investigating incidence of clinical disease**

Associations between social support, life stress and the development of coronary heart disease are most appropriately investigated through prospective studies. Furthermore, studies assessing disease incidence rather than prevalence are also preferable. The prospective or cohort study design offers the advantage of ensuring that risk factor exposure precedes disease occurrence. The inclusion of only incident, as opposed to prevalent cases further allows accurate assessment of the temporal relationships of exposure–disease relationships. The prospective design is also less open to recall bias, as is potentially likely to occur in retrospective or case–control studies.

**Life stress and incidence of clinical disease**

The first prospective study of life stress and coronary heart disease incidence was based on a sample of 1822 subjects from the Framingham Heart Study, and did not find any association between a four-item perceived daily stress score and coronary heart disease incidence (coronary insufficiency, angina pectoris, myocardial infarction) in multivariate analyses. More recently, however, Rosengren et al. have demonstrated prospectively that middle-aged men with high levels of self-perceived psychological stress have up to a 50 per cent higher incidence rate of coronary artery disease adjusting for all established risk factors, though the outcome included coronary artery disease death so it is not possible to separate morbidity from mortality. Incidence of angina pectoris has also been demonstrated to be associated with perceived stress in a cohort of 5735 Swedish men, and with life events. Hollis et al. found that the relative risk (adjusted for several risk factors) of angina pectoris associated with each additional life event was 1.08 [95 per cent confidence interval (CI) 1.03–1.13], but they did not demonstrate a statistically significant association between life events.
and fatal or non-fatal myocardial infarction. Studies investigating the association between life stress and clinical disease incidence are shown in Table 1. Elliott has reviewed the evidence connecting women’s psychosocial stress with coronary disease in women, though the definition of psychosocial stress was broad and the potentially important buffer of social support was not considered, nor were the effects in men. The review concluded that evidence was mixed and no clear patterns have emerged.

Social support and incidence of clinical disease
Several prospective cohort studies assessing the influence of social support on coronary heart disease have been reported. Medalie et al. found that family problems (which could be interpreted as both stressful and damaging to perceived familial support) were found to be associated with five-year incidence of angina pectoris among 10 000 men in Israel. In particular, men perceiving themselves to have a loving and supportive wife were at a lower risk of angina pectoris than the other men. When this association was further investigated, men with high levels of anxiety benefited most from love and support from a spouse, being associated with a halving in the risk of angina pectoris compared with those categorized as having a low degree of perceived support. Medalie et al., however, did not adjust for any confounders in their analysis of support and so a causal interpretation cannot be made.

Investigating a different aspect of social support, a small study of 159 patients found social resources to be associated with severity of coronary heart disease, suggesting that the supportive aspects of social networks are more protective than their structure and size. Furthermore, Vogt et al. used three scales to investigate the influence of different aspects of social support on first occurrence of a coronary heart disease episode. Their study concluded that, once confounders were allowed for, network scope (based on the range of functions the support performs, e.g. providing emotional or practical support) had greater influence than network size.

Most recently, Orth-Gomer et al. have demonstrated that low emotional support and poor social integration predicted incidence of major coronary events (nonfatal myocardial infarction or CHD death) independently of other risk factors. By pooling nonfatal myocardial infarction and CHD death, however, no comparison can be made between the two.

The above results must be taken together with the inconclusive results from the Honolulu Heart Project, which found prevalence rates for fatal myocardial infarction, non-fatal myocardial infarction, angina pectoris and total coronary heart disease in 4653 men of Japanese ancestry in Hawaii were all inversely associated with the two social network scales used. More importantly, they were also not associated with respective incidence rates. Only non-fatal myocardial infarction and total coronary heart disease were associated with social network allowing only for age, and no associations were found when adjusting for other risk factors in multivariate analyses. This study also raises the question of the relevancy of results derived from populations with different cultures.

The main characteristics of the studies investigating the association between social support and incidence of clinical disease are shown in Table 2.

Studies investigating onset of myocardial infarction
Many studies have investigated psychosocial influences on the precipitation of myocardial infarction in sufferers of coronary heart disease. The evidence for this has been reviewed by Jenkins, and before that by Mai. As Jenkins described, there have been several retrospective studies which have claimed to show that the incidence of stressful life events increases in the months immediately preceding infarct. These studies suggest that suddenly increased levels of stress precipitate a significant infarct followed by sudden death, though the deciding factor may be an individual’s rating of how stressful life events are to them. Other retrospective studies have since been carried out and also claim to show that people suffer an increased measure of stress in the period preceding myocardial infarct.

The only large prospective study, however, has found no association at all between stressful life events and onset of myocardial infarction in the subsequent 12–15 months.

The paucity of prospective studies confirming and quantifying the importance of stress in the precipitation of myocardial infarction leads to the conclusion that the role of stress remains in question. In particular, the time interval between experiencing life stress and onset of myocardial infarction requires further attention.

Studies investigating coronary heart disease mortality
The influences of life stress and social support on coronary heart disease mortality may be different from their effects on incidence of clinical disease, as they may act at different stages and in different ways in the progression of coronary heart disease. The influence of these factors on CHD mortality is now considered.
<table>
<thead>
<tr>
<th>Study</th>
<th>Size and design</th>
<th>Study population</th>
<th>Aspect of life stress measured</th>
<th>Main results</th>
<th>Results adjusted for</th>
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</thead>
<tbody>
<tr>
<td>Haynes et al.</td>
<td>1822 prospective</td>
<td>Men and women aged 45–77, members of Framingham Heart Study; followed for 8 years</td>
<td>Perceived daily stress</td>
<td>CHD prevalence associated with stress only in men aged 45–64; no association between stress and CHD incidence (angina, coronary insufficiency, MI) in any age group</td>
<td>Age, systolic blood pressure, cholesterol, smoking, Type A behaviour, ageing worries</td>
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<tr>
<td>Hagman et al.</td>
<td>5735 cross-sectional and prospective</td>
<td>Swedish men in a prevention trial, followed for 4 years</td>
<td>Perceived stress</td>
<td>Perceived stress associated with prevalence of uncomplicated angina $p &lt; 0.001$, and with incidence of uncomplicated angina, OR = 1.3, $p &lt; 0.001$, but not complicated angina pectoris, following MI</td>
<td>Systolic blood pressure, diastolic blood pressure, cholesterol, diabetes, dyspnea, relative body weight, smoking, physical activity – work, physical activity – leisure</td>
</tr>
<tr>
<td>Hollis et al.</td>
<td>12866 prospective</td>
<td>High CHD risk men aged 35–97 in an intervention trial, followed for 6 years</td>
<td>Life events</td>
<td>Incidence of angina pectoris associated with life events: HR = 1.08 (95% CI 1.03–1.13) $p &lt; 0.002$ for each additional life event, but not for fatal or non-fatal MI</td>
<td>Age, diastolic blood pressure, cholesterol, smoking, Type A behaviour, intervention group</td>
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<tr>
<td>Rosengren et al.</td>
<td>6935 prospective</td>
<td>Swedish men aged 47–55 in the intervention arm of trial, mean follow-up of 11.8 years</td>
<td>Perceived stress</td>
<td>Incidence of coronary artery disease (nonfatal MI or CHD death; not angina) OR = 1.5 (95% CI 1.2–1.9) for low perceived stress vs high</td>
<td>Age, systolic blood pressure, cholesterol, body mass index, diabetes, family history of MI, smoking, physical activity – leisure, occupational class, marital status, alcohol abuse</td>
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CHD, coronary heart disease; MI, myocardial infarction; OR, odds ratio; HR, hazard ratio; CI, confidence interval.
<table>
<thead>
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<tbody>
<tr>
<td>Medalie et al. 1973,</td>
<td>10000 prospective</td>
<td>Men in Israel aged over 40 followed for 5 years</td>
<td>Perceived love/support from spouse</td>
<td>Angina incidence associated with perceived support ($\chi^2$ test for trend $p = 0.01$; low vs high support crude RR = 1.5); with high anxiety levels, crude RR = 1.8; with low anxiety score, no association</td>
<td>Unadjusted in analysis of love/support from spouse</td>
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<td>1976$^{20,21}$</td>
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<td>Reed et al. 1983$^{25}$</td>
<td>4653 cross-sectional and prospective</td>
<td>Japanese men in Hawaii followed for 6 years</td>
<td>(1) 9-item network score (2) Factor-derived network score</td>
<td>Social network associated with CHD prevalence (fatal or nonfatal MI, angina), but not incidence once adjusted for confounders</td>
<td>Age, systolic blood pressure, cholesterol, serum glucose, serum uric acid, forced vital capacity, complex carbohydrate, body mass index, smoking, physical activity, alcohol intake, socioeconomic status</td>
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<td>Vogt et al. 1992$^{23}$</td>
<td>2603 prospective</td>
<td>Members of a health maintenance organization followed for 15 years</td>
<td>(1) Network scope (2) Network frequency (3) Network size</td>
<td>For first occurrence of CHD episode: low vs high tertiles (1) HR = 1.5 (95% CI 1.0–2.3) $p = 0.03$ (2) HR = 1.1 (95% CI 0.8–1.5) $p = 0.54$ (3) HR = 1.2 (95% CI 0.9–1.6) $p = 0.26$</td>
<td>Age, sex, smoking socioeconomic status, baseline health</td>
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<tr>
<td>Orth-Gomér et al. 1993$^{24}$</td>
<td>736 prospective</td>
<td>Middle-aged Swedish men followed for 6 years</td>
<td>(1) Emotional support (2) Social integration</td>
<td>For nonfatal MI or CHD death: (1) OR = 3.1 (95% CI 1.3–7.6) lower vs upper quartile (2) OR = 3.8 (95% CI 1.1–13.9) lower vs upper 3 quartiles</td>
<td>Age, cholesterol, treatment for hypertension, diabetes, body mass index, smoking, physical activity</td>
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RR, relative risk.
Life stress and CHD mortality

Rosengren et al. found that, in addition to being associated with an increased risk of developing coronary heart disease, high self-perceived stress in their middle-aged men led to a 70 per cent higher death rate from cardiovascular disease even after allowing for the effects of established prognostic indicators. Rosengren et al. also found, in the same prospective study that Orth-Gomér et al. reported on, that men experiencing over three life events in a year were at a significantly higher risk of all-cause death (odds ratio =3·6; 95 per cent CI 1·5–8·5), again after adjusting for several prognostic indicators. A similar (but not statistically significant) trend was found for death from coronary heart disease (odds ratio =2·0; 95 per cent CI 0·5–8·5). Socioeconomic status was allowed for, but direct measures of income or alcohol abuse, which may both influence mortality, were not. An interesting additional finding was that men with adequate emotional support seem to be protected from the effects of stressful life events, which may be taken as evidence supporting the ‘buffer hypothesis’.

The only published study demonstrating non-significant results for the association between life stress and coronary heart disease mortality was by Hollis et al. Though they found angina pectoris associated with stressful life events, they did not make the same finding for coronary heart disease mortality. As CHD is a major public health problem and interest in stress is high it is unlikely that there will be very large negative publication bias in this field.

The main characteristics of the studies investigating the association between life stress and coronary heart disease mortality are listed in Table 3.

Social support and CHD mortality

Large prospective cohort studies in Alameda County, California, Tecumseh in Michigan, and a study in Eastern Finland have all found that poor social support is associated with all-cause mortality and in particular mortality from heart disease, with the last two studies adjusting for confounding factors.

Smaller studies, including that by Blumenthal et al. on 113 patients undergoing diagnostic coronary angiography, found that a perceived social support scale was inversely associated with degree of coronary artery disease severity in Type A personalities, but not Type B. Orth-Gomér et al., in another small study, also found relative social isolation was a predictor of all-cause and ischaemic heart disease mortality, and subgroup analysis suggested social support may have a greater impact on the survival of Type A men than Type B men. Though statistical tests of the interactions declared a priori would be preferred, these analyses provide interesting hypotheses for further investigation, and suggest that there may exist a psychosocially high-risk subgroup of heart disease patients. Both their personality type and lack of social support mean they are vulnerable and more susceptible to external stressors.

Pooling the results from seven relevant studies indicates that the proportion of deaths that would not occur if everybody had adequately developed social networks is estimated to be in the range of 20–40 per cent, by assuming that cardiovascular mortality responds to social support in a similar way to all-cause mortality.

The characteristics of the studies investigating the association between social support and coronary heart disease mortality are given in Table 4.

One study has specifically examined the influence of social resources on both incidence of clinical disease and ischaemic heart disease mortality in 2603 members of a health maintenance organization over 15 years, as well as cause-specific and all-cause mortality among the incident disease cases. By examining disease incidence, and cause-specific morality among the incident cases, the authors claim that social networks may be more effective in supporting recovery after illness has occurred than in preventing its occurrence.

Discussion

Evidence from large, well-designed prospective studies that supports a relationship between acute stressful life events and subsequent precipitation of acute myocardial infarction is lacking. The role of psychosocial factors in these events in particular is also unclear.

Longer-term psychosocial stresses and lack of support may be influential in the development of coronary heart disease in the first place, but the magnitudes of these effects also remain unreliably defined. In the studies that have been done, participants suggested to have inadequate support had up to 380 per cent higher risk of coronary heart disease (Table 2), with measures of the quality, rather than quantity, of support tending to demonstrate a larger effect. Subjects with high life stress levels were at up to 50 per cent higher risk (Table 1). The impact of stress may be less than that of social support. The studies and their results are inconsistent and it is not possible to sensibly estimate the influence of life stress and social support by pooling results.

The influence of stress and psychosocial factors on mortality is clearer. Higher all-cause and cause-specific mortality in samples from the general population has
<table>
<thead>
<tr>
<th>Study</th>
<th>Size and design</th>
<th>Study population</th>
<th>Aspect of life stress measured</th>
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<tr>
<td>Hollis et al. 1990(^{18})</td>
<td>12866 prospective</td>
<td>High CHD risk men aged 35–57 in intervention trial, followed for 6 years</td>
<td>Life events</td>
<td>Life events not significantly related to CHD death, or fatal or nonfatal MI</td>
<td>Age, cholesterol, diastolic blood pressure, smoking, Type A behaviour, intervention group</td>
</tr>
</tbody>
</table>
| Rosengren et al. 1991\(^{16}\) | 6935 prospective | Swedish men aged 47–55 in the intervention arm of trial; mean follow-up 11.8 years | Perceived stress               | Cardiovascular death is related to self-perceived stress (low stress vs high): OR = 1.7  
  (95% CI 1.2–2.4)                                                                 | Age, systolic blood pressure, cholesterol, body mass index, diabetes, family history of MI, occupational class, marital status, physical activity, alcohol abuse |
| Rosengren et al. 1993\(^{42}\) | 752 prospective  | Random population sample of Swedish men aged 50; 7 year follow-up                 | Life event                     | 0 events vs 3 or more: OR = 2.0  
  (95% CI 0.5–8.5)                                                                                     | Smoking self-perceived health, emotional support                              |
<table>
<thead>
<tr>
<th>Study</th>
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<th>Aspect of social support measured</th>
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<tbody>
<tr>
<td>Berkman and Syme, 1979</td>
<td>4725 prospective</td>
<td>Men and women aged 30–69 from Alameda county, followed for 9 years</td>
<td>Marital status and formal social contacts</td>
<td>IHD mortality similar to all-cause For men age-adjusted RR = 2.3, age-adjusted RR = 2.8 for women. Fewest contact vs most</td>
<td>Age</td>
</tr>
<tr>
<td>House et al., 1982, 1988</td>
<td>2754 prospective</td>
<td>Men and women in Tecumseh, MI, aged 35–69, followed for 10–12 years</td>
<td>Marital status, social contacts and activities</td>
<td>Social contacts and activities generally related to all-cause and IHD mortality, but RR estimates for IHD mortality not presented</td>
<td>Age, FEV₁; males: smoker, retired/disabled, farmer/labourer; females: bronchitis, hypertension</td>
</tr>
<tr>
<td>Orth-Gomér et al., 1987</td>
<td>17433 prospective</td>
<td>Swedish men and women aged 29–74, followed for 6 years</td>
<td>Available social contact</td>
<td>For cardiovascular mortality, upper two vs lower tertile: age-adjusted RR 1.38 (95% CI 1.12–1.72). Adjusted for all confounders: RR = 1.37 (95% CI 0.97–1.96)</td>
<td>Age, cardiovascular disease, smoking, exercise</td>
</tr>
<tr>
<td>Orth-Gomér et al., 1988</td>
<td>150 prospective</td>
<td>150 middle-aged Swedish men: 50 with IHD, 50 at high IHD risk, 50 healthy; followed for 10 years</td>
<td>Social activities</td>
<td>Social isolation is predictive of all-cause and IHD mortality</td>
<td>Age, smoking, marital status, social class, alcohol</td>
</tr>
<tr>
<td>Kaplan et al., 1988</td>
<td>13301 prospective</td>
<td>Men and women in Finland aged 39–59, followed for 5 years</td>
<td>Social contacts</td>
<td>For the men: OR (cardiovascular death) = 1.54 (95% CI 1.11–2.13); OR (IHD death) = 1.34 (95% CI 0.94–1.90)</td>
<td>Age province, urban/rural, mean weighted blood pressure, cholesterol, body mass index, family history, prevalent MI, angina, diabetes, cardiac insufficiency, emphysema, cardiovascular medication, smoking, education, cohort</td>
</tr>
<tr>
<td>Vogt et al., 1992</td>
<td>2603 prospective</td>
<td>Members of a health maintenance organization over 18 followed for 15 years</td>
<td>(1) Network scope (2) Network frequency (3) Network size</td>
<td>Among incident IHD cases, for 1 unit decrease in score: (1) HR = 1.47 (95% CI 1.18–1.85) (2) HR = 1.43 (95% CI 1.14–1.82) (3) HR = 1.54 (95% CI 1.22–1.92)</td>
<td>Age, sex, smoking, socioeconomic status, baseline health status</td>
</tr>
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</table>
been demonstrated to be associated with poor social support. Lack of social support is a very strong factor in mortality from coronary heart disease, increasing mortality by up to four times compared with that of normal people (see Table 4). The emotional aspects of support appear more important than the size of the support network as a protective element. Life stress appears a much weaker factor than social support, but nevertheless the balance of evidence shown in Table 3 suggests it does have a small effect, and that social support and life stress are independent risk factors.

An important rider to the above conclusions is indicated by the fact that any review of previously published work is open to the potential bias induced by the possibility that small studies with non-significant findings are potentially less likely to be published. The magnitude of this possible 'publication bias' is unknown.

Strong evidence supporting a statistical interaction between stress and social support in coronary heart disease incidence is lacking but remains central to further study to explain the effects observed. The suggested 'buffer hypothesis', i.e. that the social support acts as a buffer to the effect of stressors, remains an attractive proposal for their likely interaction but as yet it remains unproven. The data of Rosengren et al. that emotional support did protect from stressful life events provide some evidence, but further data are required. A very large prospective study would be required to show a statistically significant interaction between stress and social support given the expected magnitude of the effects. Further major studies are therefore required if the above or other possible biological mechanisms are to be confirmed. If the mode of action was understood more fully then future interventions could be proposed more effectively.

The public health importance of life stress and social support in the natural history of coronary heart disease is unclear, because the magnitude of any effects cannot be accurately estimated using the available evidence. The relative risks from the different studies cannot be compared because the different prevalence rates of high life stress and social support defined by the different scales used in each study mean that a different high-risk population is identified in each study. In this situation, the concept of Population Proportional Attributable Risk (PPAR) provides a more appropriate approach to compare studies by, and thereby to assess the implications for the general population.

Comparing the PPAR for life stress and social support with the PPAR for other risk factors would assist in the decision on whether to proceed to psychosocial interventions. As yet, very little has been done by way of estimating PPAR for social support and life stress, after appropriate adjustment for other risk factors. If the PPAR for a psychosocial factor was as large as for established risk factors this would theoretically justify intervention strategies. However, it may not be possible to find practical interventions to take full advantage of any potential reduction in coronary heart disease or mortality.

We conclude that further work is required to adequately quantify the magnitude and usefulness of stress and psychosocial risk factors in the development of heart disease. Such studies should be large and prospective in design and utilize questions relating to pragmatically defined factors which, if confirmed, are open to modification. Such a measure would have validity, but it would also be useful from the point of view that one would be able to intervene on it in the future. Only such pragmatic factors will be useful in designing interventions to reduce life stress levels and increase social support and thereby reduce the considerable burden of ill health and premature death caused by coronary heart disease in future populations.

Acknowledgements

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