CLINICAL EVIDENCE

Psychosocial factors in the etiology and prognosis of coronary heart disease: systematic review of prospective cohort studies

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Do psychosocial factors cause coronary heart disease or affect survival among patients with coronary heart disease? Here we use an explicit methodological quality filter to review systematically prospective cohort studies that test specific psychosocial hypotheses. This review of the epidemiological literature identifies the psychosocial factors that have been most rigorously tested. Only four psychosocial factors met the quality filter: type A/hostility, depression and anxiety, work characteristics, and social supports. The importance of other study designs (for example, ecological[1] or nested case-control[2-4] studies) is acknowledged. The review should be seen as complementary to existing reviews[5-8] on single psychosocial factors and as a challenge to investigators in the field to ensure that the systematic review is made unbiased, kept up-to-date, and used to guide future hypothesis testing.

WHAT IS A PSYCHOSOCIAL FACTOR?

A psychosocial factor may be defined as a measurement that potentially relates psychological phenomena to the social environment and to pathophysiologic changes. The validity and reliability (precision) of the questionnaire-based instruments used to measure psychosocial factors have been improved through the use of psychometric techniques. By avoiding the unhelpful general term of "stress," recent work has developed theoretical models (for example, the job control-demands-support model of psychosocial work characteristics) which generate specific hypotheses that can be tested.

Summary points

- In healthy populations, prospective cohort studies show a possible etiological role for type A/hostility (6/14 studies), depression and anxiety (11/11 studies), psychosocial work characteristics (6/10 studies), and social support (5/8 studies)
- In populations of patients with coronary heart disease, prospective studies show a prognostic role for depression and anxiety (6/6 studies), psychosocial work characteristics (1/2 studies), and social support (9/10 studies); 0 of 5 studies showed a prognostic role for type A/hostility
- Although this review cannot discount the possibility of publication bias, prospective cohort studies provide strong evidence that psychosocial factors, particularly depression and social support, are independent etiological and prognostic factors for coronary heart disease

HOW MIGHT PSYCHOSOCIAL FACTORS BE LINKED TO CORONARY HEART DISEASE?

Evidence of mechanisms linking psychosocial factors with coronary heart disease (reviewed elsewhere[9,10]) is important in making causal inferences and, therefore, in designing preventive interventions. Psychosocial factors may act alone or combine in clusters[11] and may exert effects at different stages of life.[12] Broadly, three interrelated pathways may be considered. First, psychosocial factors may affect health-related behaviors such as smoking, diet, alcohol consumption, or physical activity that in turn may influence the risk of coronary heart disease.[13] If such behaviors lie on the causal pathway between psychosocial factors and coronary heart disease, then treating them as confounding variables, as some studies do, must be questioned. Second, psychosocial factors may cause direct acute or chronic pathophysiologic changes. Third, access to and the content of medical care may plausibly be influenced by, for example, social supports (but there is little direct evidence for this). Although it is beyond the scope of this review to consider the determinants of adverse psychosocial factors, socioeconomic status is inversely associated with coronary heart disease[14] and also with certain psychosocial factors, and it has been proposed that psychosocial pathways may play a mediating role.[15,16]

METHOD OF SYSTEMATIC REVIEW

A methodologic quality filter was used to select studies for inclusion in the systematic review so that the strength of evidence could be compared across psychosocial factors. Prospective cohort studies are the best observational design for questions of etiology and prognosis. The studies included had a prospective cohort design; a population-based sample (etiological studies in healthy populations); at least 500 participants (etiological studies) or 100 participants (prognostic studies in populations of patients with coronary heart disease); measurements of a psychosocial factor used in at least 2 different study populations; and outcomes of fatal coronary heart disease or nonfatal myocardial infarction or (prognostic studies only) all-cause mortality.

Articles were identified by MEDLINE search (1966-1997), manually searching the bibliographies of retrieved articles and previous review articles, writing to researchers in the field, and checking an in-house bibliographic database. No register of published and unpublished studies

with psychosocial exposures exists, and hand searching of journals was not performed, so there is a serious potential for publication bias. For this reason, as well as the lack of standardized methods for measuring psychosocial factors, we carried out a narrative, rather than a quantitative, systematic review. Given that randomized, controlled trials, at least for primary prevention, are rarely feasible, observational studies are likely to remain the main type of evidence on which to base preventive action.

EVIDENCE FOR SPECIFIC PSYCHOSOCIAL FACTORS

Largely on the basis of studies of middle-aged men (Table 1), four groups of psychosocial factors were identified by using the predefined quality filter: psychological traits (type A behavior, hostility), psychological states (depression, anxiety), psychological interaction with the organization of work (job control-demands-support), and social networks and social support. In simple terms, this corresponds to a spectrum, with mainly psychological components at one end and a stronger social component at the other.

Hostility and type A behavior

Type A behavior pattern, the only personality trait that met the criteria of our review, is characterized by hard-driving and competitive behavior, a potential for hostility, pronounced impatience, and a vigorous speech style. The instruments for measuring type A behavior and hostility (the Jenkins Activity Scale, the structured interview, the Minnesota Multiphasic Personality Inventory, and the Bortner Hostility Scale) have been subjected to psychometric testing and incorporated into many cardiovascular cohort studies, including some that have not reported results. Unlike other psychosocial factors, the type A behavior pattern is distinguished by being the subject of numerous intervention trials.[17] On the basis of early positive findings in the Framingham study[18] and the Western Collaborative Group's 8-year follow-up,[19] among other evidence, the National Institutes of Health declared type A an independent risk factor for coronary heart disease. However, with the publication of negative findings,[20-22] it was proposed that a more specific component of type A, namely hostility, might be etiological, although there are conflicting studies. None of the five studies that examined type A or hostility in relation to prognosis among patients with coronary heart disease showed an increased risk; indeed, one suggested a protective effect.

Depression and anxiety

The relation between depression and anxiety and coronary heart disease differs from that of other psychosocial factors for several reasons. First, unlike other psychosocial factors, depression and anxiety represent well-defined psychiatric disorders, with standardized instruments of measurement. Second, depression and anxiety are commonly the consequence of coronary heart disease, and determining the extent to which they are also the cause poses important methodological issues. Third, the ability to diagnose and treat such disorders makes them attractive points for intervention. Finally, depression and coronary heart disease could share common antecedents—for example, environmental stressors and social supports.

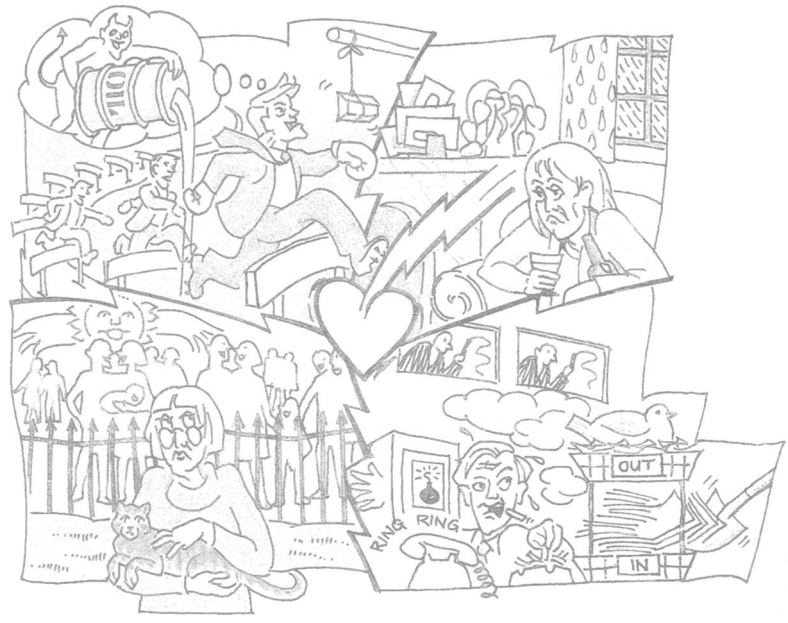

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Table 2 shows the results from 11 prospective studies that investigated depression or anxiety in the etiology of coronary heart disease, all of which were positive. All three of the prospective studies examining the effect of anxiety in the etiology of coronary heart disease had positive results. Intriguingly, there is some evidence that this effect is strongest specifically for phobic anxiety and sudden cardiac death. Wassertheil-Smoller et al[23] reported the effect of depression in relation to cardiovascular events among 4367 healthy older people. An increase in depression symptoms (but not the baseline scores) predicted events, even when multiple covariates were controlled for. Such findings are compatible with the hypothesis that premonitory signs of coronary heart disease such as angina or breathlessness may have led to the increase in depression. Studies with longer periods of follow-up are less likely to be confounded by the possibility of early disease causing depression but raise further questions about the time course of exposure. For example, it is possible that there is a common trigger (such as viral illness) that precipitates both symptoms of depression and atherothrombotic processes. By examination of subclinical manifestations of coronary heart disease (using noninvasive measures of ar-

Table 1 *Studies of type A behavior, hostility, and coronary heart disease*

Author, year, country	Total sample (% women)	Age at entry, y	Exposure	Follow-up, y	Number of events	Type of events	Adjustments	Relative risk	Summary†
Prospective etiological studies									
Jenkins, 1974[1] USA	2750 (0)	39–59	Type A	4	120	Nonfatal MI and angina	Age	1.8*	+
Rosenman, 1976,[2] USA	3154 (0)	39–59	Type A	8.5	257	Fatal CHD and nonfatal MI	Age, smoking, cholesterol, family history, corneal arcus, schooling, β:α-lipoprotein ratio	2.16*	++
Haynes, 1980[3] USA	1674 (57)	45–77	Type A (Framingham)	8	170	Fatal CHD and nonfatal MI and coronary insufficiency and angina	Age, smoking, blood pressure, cholesterol, glucose intolerance, and other psychosocial factors	1.8*; among men, the effect was confined to white-collar workers	
Shekelle, 1983,[4] USA	1877 (0)	40–55	Hostility (MMPI)	10	139	Fatal CHD and nonfatal MI	Age, smoking, blood pressure, cholesterol, alcohol	1.47*, but effect not linear	+
Cohen, 1985,[3] USA	2187 (0)	57.8 (mean)	Type A (JAS)	8	190	Fatal CHD and nonfatal MI and angina	Smoking, blood pressure, cholesterol, body mass index, alcohol, and other biological factors	1.43; type A associated with prevalence, not incidence or postmortem findings	0
Shekelle, 1985,[6] USA	3110 (0)	46 (mean)	Type A (JAS)	7.1	554	Fatal CHD and nonfatal MI	Age, smoking, blood pressure, cholesterol, alcohol, education	0.87	0
Johnston, 1987,[7] UK	5936 (0)	40–59	Type A (Bortner)	6.2	255	Fatal CHD and nonfatal MI	Age, social class	0.89	0
Ragland, 1988,[8] USA	3154 (0)	39–59	Type A (SI)	22	214	Fatal CHD	Age, smoking, blood pressure, cholesterol	0.98	0
Hearn, 1989,[9] USA	1399 (0)	19	Hostility (MMPI)	33	54	Fatal CHD and nonfatal MI and angina and coronary surgery	Smoking, hypertension, family history	1.1; no association in crude or risk factor adjusted analyses	0
Barefoot, 1995,[10] USA	730 (44)	50	Hostility (Cook-Medley)	27	122	Nonfatal MI	Age, sex, smoking, blood pressure, triglycerides, exercise	1.26 (men) 2.95* (women)	0 (men) ++ (women)
Bosma, 1995,[11] Lithuania and Netherlands	5817 (0)	45–60	Type A (JAS)	9.5	394	Fatal CHD and nonfatal MI	Age	No association	0
Kawachi, 1996,[12] USA	1305 (0)	40–90	MMPI-2 (anger content scale)	7	110	Fatal CHD and nonfatal MI and angina	Age, smoking, blood pressure, cholesterol, body mass index, family history, alcohol	2.66*	++
Everson, 1997,[13] Finland	1599 (0)	42–60	Cynical hostility (Cook-Medley)	6	60	First MI	Age, and biological, socioeconomic, behavioral, and social support factors, prevalent diseases	1.43 (2.18* when adjusted for age only)	0
Tunstall-Pedoe, 1997,[14] Scotland	11,659 (50)	40–59	Type A (Bortner)	7.6	581	Fatal CHD and nonfatal MI and coronary surgery	Age	0.82* in women, ie, type A protective	0
Prognostic studies									
Case, 1985,[15] USA	516 (18) patients <14 days post-MI	<70	Type A (JAS)	2	53	Fatal CHD and all-cause mortality	Age, sex, education, rales, ejection fraction, NYHA functional class, ventricular premature beats	0.8	0
Shekelle, 1985,[16] USA	2314 (11) patients post MI	30–69	Type A (JAS)	3	294	Nonfatal MI and fatal CHD	Smoking, previous MI, angina, fasting glucose level	No association	0
Ragland, 1988,[17] USA	257 (0) with MI or angina	39–70	Type A (SI)	11.5	91	Fatal CHD	Age at initial event, follow-up time, type of initial coronary event, smoking, blood pressure, cholesterol	0.58*; type A protective	0
Barefoot, 1989,[18] USA	1467 (18) patients with angiographic disease	mean 52 (SD 9)	Type A (SI)	5	315	Fatal CVD and non fatal MI	Stratified on clinical prognostic factors	No association with nonfatal MI	0
Jenkinson, 1993,[19] UK	1376 (22) 7 days post-MI	25–84	Type A	3	247	All-cause mortality	Age, previous MI, hospital complications, diabetes, hypertension, car ownership, sex	No association	0

References in this table are given on the BMJ website.
CHD = coronary heart disease; MI = myocardial infarction; JAS = Jenkins Activity Survey; MMPI = Minnesota Multiphasic Personality Inventory; NYHA = New York Heart Association; SI = structured interview.
*$P < .05$.
†0 = no association (relative risk not significantly different from unity); + = moderate association (relative risk, 1.0≤2.0); ++ = strong association (relative risk >2.0).

Table 2 *Studies of depression and anxiety and coronary heart disease*

Author, year, country	Total sample (% women)	Age at entry, y	Exposure	Follow-up, y	Number of events	Type of events	Adjustments	Relative risk	Summary†
Prospective etiological studies									
Hallstrom, 1986,[20] Sweden	795 (100)	38–54	Depression (Hamilton and psychiatric interview)	12	75	Nonfatal MI, angina, and ischemic changes on ECG	Age, social class, marital status, conventional risk factors	5.4*; severity of depression predicted angina but not other outcomes	++
Hagman, 1987,[21] Sweden	5735 (0)	55 (mean)	Anxiety ("stress")	2–7	162	Angina with or without MI	Age, smoking, blood pressure, cholesterol, relative weight	Strong predictor for angina alone	+
Haines, 1987,[22] UK	1457 (0)	40–64	Phobic anxiety (Crown-Crisp)	10	113	Fatal CHD and nonfatal MI	Fibrinogen, cholesterol, factor VII, systolic blood pressure	3.77* for fatal CHD	++
Appels, 1990,[23] Netherlands	3877 (0)	39–65	Depression	4.2	59	Nonfatal MI, unstable angina, and angina	Age, smoking, blood pressure, cholesterol	1.86* for unstable angina for combination of low mood, low energy, hopelessness, poor sleep ("vital exhaustion")	+
Anda, 1993,[24] USA	2832 (52)	45–77	Depression (general well-being)	12	394	Fatal CHD and nonfatal CHD hospitalizations	Age, sex, race, education, marital status, smoking, blood pressure, cholesterol, body mass index, alcohol, exercise	1.6*	+
Aromaa, 1994,[25] Finland	5355 (55)	40–64	Depression (GHQ and PSE)	6.6	91	Fatal CHD	Age, preexisting cardiovascular disease	3.36* (5.52 in those with preexisting cardiovascular disease)	++
Kawachi, 1994,[26] USA	33,999 (0)	42–77	Phobic anxiety (Crown Crisp)	2	168	Fatal CHD and nonfatal MI	Age, smoking, blood pressure, cholesterol, body mass index, diabetes, parental history of MI, alcohol, exercise	3.01* (6.08 when sudden cardiac death examined)	++
Everson, 1996,[27] Finland	2428 (0)	42–60	Hopelessness	6	95	Nonfatal MI	Age, smoking, blood pressure, cholesterol education, income, exercise, alcohol, lipids, social supports, depression	2.05*	++
Wassertheil-Smoller, 1996,[28] USA	4367 (53)	72 (mean)	Depression (CES-D)	4.5	321	Nonfatal MI and nonfatal strokes	Age, smoking, baseline depression, sex, race, randomization group, education, history of stroke, MI, diabetes, and baseline ADL	1.18* per 5-unit increase in depression score (baseline scores) alone did not predict events)	+
Barefoot, 1996,[29] Denmark	730 (44)	50 or 60	Depression (MMPI, obvious depression scale)	27	122	Nonfatal MI	Age, conventional CHD risk factors, baseline CHD	1.7* for 2 SD difference in depression score	+
Kubzansky, 1997,[30] USA	1759 (0)	21–80	Social conditions worry scale	20	323	Fatal CHD, nonfatal MI, and angina	Age, smoking, blood pressure, cholesterol, body mass index, family history, alcohol	1.23* per 1-point increase in social conditions worry scale	+
Prognostic studies									
Ahern, 1990,[31] USA	353		Depression (Beck), anxiety (Spielberger)	12		Fatal CHD	Age, left ventricular dysfunction, previous MI	1.3* for depression	+
Kop, 1994,[32] Netherlands	127 (17) patients 2 weeks after coronary angioplasty	56 (SD 9)	Maastricht questionnaire for vital exhaustion	1.5	29	Fatal CHD, nonfatal MI, further revascularization, increase in coronary atherosclerosis, and new angina	Age, sex, smoking, blood pressure, cholesterol, severity of coronary artery disease, clinical presentation	2.34 ($P = .06$)	+
Ladwig, 1994,[33] Germany	377 (0) 17–21 days after acute MI	29–65	Depression (interview)	0.5		Angina, not returning to work, continuing to smoke	Age, social class, recurrent infarction, rehabilitation, cardiac events, helplessness	2.31* for the effect on angina; depression predicted all outcomes	++
Frasure-Smith, 1995,[34] USA	222 (21) patients 5–15 days after acute MI	24–88	Depression (diagnostic interview schedule)	1.5	21	All-cause mortality and fatal CHD	Age, Killip class, premature ventricular contractions, previous MI	6.64* effect of depression higher in those with (10 premature contractions per hour	++
Barefoot, 1996,[35] USA	1250 (18) patients with angiographic disease	52 (mean)	Depression (Zung)	19.4	604	All-cause mortality and fatal CHD	Disease severity, initial treatment	1.66*, 1.84*, and 1.72* in 3 follow-up periods (year 1, 5–10, and >10, respectively)	+
Denollet, 1996,[36] Belgium	303 (12) patients with angiographic disease	31–79	Type D personality (suppression of emotional distress), depression, social alienation	7.9	38	All-cause mortality and fatal CHD	Left ventricular function, number of diseased vessels, low exercise tolerance, lack of thrombolytic treatment	4.1* for type D and 2.7* for depression	++

References in this table are given on the BMJ website.
ADL = activities of daily living; CES-D = Center for Epidemiological Studies-Depression Scale; CHD = coronary heart disease; ECG = electrocardiogram; GHQ = General Health Questionnaire; MI = myocardial infarction; MMPI = Minnesota Multiphasic Personality Inventory; PSE = Present State Examination.
*$P < .05$.
†0 = no association (relative risk not significantly different from unity); + = moderate association (relative risk, 1.0≤2.0); and ++ = strong association (relative risk >2.0).

terial structure and function, for example) before the onset of symptoms, the temporal sequence of the relation might be better understood.

Depression in patients after myocardial infarction seems to be of prognostic importance beyond the severity of coronary artery disease. Although discrete major depressive episodes are not uncommon after a myocardial infarction, depressive symptoms are more prevalent. Given the graded relation between depression scores and risk, the long-lasting nature of the effect, and the stability of the depression measured across time, it has been proposed that depression is a continuously distributed chronic psychological characteristic.

Psychosocial work characteristics

The long-standing observation that rates of coronary heart disease vary markedly among occupations (more than can be accounted for by conventional risk factors for coronary heart disease) has generated a quest for specific components of work that might be of etiological importance. The dominant "job strain" model of psychosocial work characteristics, as proposed by Karasek and Theorell, grew out of secondary analyses of existing survey data on the labor force. This model proposes that jobs characterized by low control over work and high conflicting demands might be high strain. A subsequent addition to the model was the idea that social support might buffer this effect. The advantage of the model is that it generates specific hypotheses for testing.

Table 3 shows prospective cohort studies that have examined the relation between job strain and coronary heart disease. Both self-reports and ecological measurements (assigning a score on the basis of job title) of job strain have been made. Self-reports may be biased by early manifestations of disease, and ecological measurements may lack precision. The finding that these methods tend to give reasonably consistent results suggests that they are complementary. Six of the 10 studies had positive results. There is growing emphasis on the importance of low job control rather than on conflicting demands,[24] and it seems likely that these empirical results will lead to a reformulation of the model. Alternative models of psychosocial work characteristics involve an imbalance between the effort at work and rewards received.[25,26]

Social network structure and quality of social support

Social supports and networks relate to both the number of a person's social contacts and their quality (including emotional support and confiding support). Marital status, information that is routinely sought in clinical practice, is a simple measure of social support, and the ability of low social support to predict all-cause mortality has long been recognized. It has been proposed that social supports may act to buffer the effect of various environmental stressors and, hence, increase susceptibility to disease,[27] but most of the evidence supports a direct role.

Five of the 8 prospective cohort studies that investigated aspects of social support in relation to the incidence of coronary heart disease were positive (Table 4). Nine of the 10 prognostic studies were positive, and the relative risks for 3 of these studies exceeded 3.0. Despite the strength and consistency of these findings, the relative effect of structural and functional aspects of social supports has yet to be delineated.

MODIFICATION OF PSYCHOSOCIAL FACTORS

The main implications of these findings for clinical practice are summarized in the box. A recent meta-analysis found that psychosocial interventions are associated with improved survival after myocardial infarction.[28] However, two large, randomized, controlled trials of psychological rehabilitation after myocardial infarction found no difference in anxiety and depression, and this may in part explain the lack of effect on mortality.[29,30] Randomized, controlled trials of modification of social supports after myocardial infarction show a decrease in cardiac death or reinfarction rates.[31] A patient's social circumstances should be elicited as part of the history, and the physician may have a role in mobilizing social support. A multicenter trial of 3000 patients after myocardial infarction (Enhancing Recovery in Coronary Heart Disease) is currently under way in the United States. It will target patients at high psychosocial risk (those who are depressed or socially isolated) and enroll large numbers of women and people from ethnic minority groups.

The potential for primary prevention in relation to psychosocial factors lies largely outside the purview of clinicians. Psychosocial factors themselves are determined largely by social, political, and economic factors, and it is, therefore, policymakers who influence the structure and function of communities--in the public and private domains--who may have scope for primary prevention.

CONCLUSION

Of the large number of psychosocial factors that have been studied, only four met the quality filter criteria: type

Psychosocial components of secondary prevention

Clinicians should consider

Detecting and treating depression

Mobilizing social support

Using socioeconomic status and psychosocial factors to stratify patients by risk

Table 3 *Studies of psychosocial work characteristics and coronary heart disease*

Author, year, country	Total sample (% women)	Age at entry, y	Exposure	Follow-up, y	Number of events	Type of events	Adjustments	Relative risk	Summary†
Prospective etiological studies									
LaCroix, 1984,[37] USA	876 (37)	45–64	Job control or demands (individual and ecological)	10	Not stated	Fatal CHD, nonfatal MI, coronary insufficiency and angina	Age, smoking, blood pressure, cholesterol	2.9* all women (clerical women, RR = 5.2), no association in men; ecological exposure was associated with risk in men and women	+
Alfredsson, 1985,[38] Sweden	958,096 (51)	20-64	Hectic work and few possibilities for learning (ecological)	1	1201	Nonfatal MI (hospitalization)	Age, 10 sociodemographic factors, smoking, heavy lifting	1.5*	+
Haan, 1988,[39] Finland	902 (33) factory workers	20–62	Job control, physical strain, variety (individual)	10	60	Fatal and nonfatal CHD	Age, smoking, blood pressure, cholesterol, alcohol, relative weight	4.95* for low control, low variety, high physical strain	++
Reed, 1989,[40] Hawaii (Japanese ancestry)	4737 (0)	45–65	Job control, demands, and their interaction (ecological)	18	359	Fatal CHD and nonfatal MI	Age	No effect of control, demands or their interaction (nonsignificant trend for lower strain men to have higher CHD)	o
Netterstrom, 1993,[41] Denmark	2045 (0) bus drivers	21–64	Job variety, satisfaction	10	59	Fatal CHD	Age	2.1*; high job variety and satisfaction associated with CHD risk	o
Suadicani, 1993,[42] Denmark	1752 (0)	59 (mean)	Job influence, monotony, pace, satisfaction, ability to relax	3	46	Fatal CHD and nonfatal MI	None	Only inability to relax after work associated with CHD	o
Alterman, 1994,[43] USA	1683 (0)	38–56	Job control, demands, and their interaction (ecological)	25	283	Fatal CHD	Age	1.4 for job strain	o
Bosma, 1997,[44] UK	10,308 (33) civil servants	35–55	Job control, demands (individual, assessed twice 3 years apart, and ecological)	5	654	Angina and physician-diagnosed ischemia	Age, smoking, blood pressure, cholesterol, body mass index, employment grade	1.93*; self-reported or externally assessed low job control predicted CHD	+
Lynch, 1997,[45] Finland	1727 (0)	42–60	Job demands, resources, and income	8.1	89	Fatal CHD and nonfatal MI	Age, behavioral, biological, and psychosocial covariates	1.57* for the effect of high demands, low resources, and low income; 2.59 when adjusted for age only	+
Steenland, 1997,[46] USA	3575 (0)	25–74	Job control and demands (ecological)	14	519	Fatal CHD and nonfatal MI	Age, smoking, blood pressure, cholesterol, education, body mass index, self-reported diabetes	1.41* for low control	+
Prognostic studies									
Hlatky 1995,[47] USA	1489 (24) employed patients undergoing coronary angiography	41–59	Job control, demands (individual)	5	112	Fatal CHD + non-fatal MI prevalence of coronary artery disease	Ejection fraction, extent of coronary atherosclerosis, myocardial ischemia	0.96 for effect of job strain on events. Job strain was associated with normal coronary arteries	–
Hoffman 1995,[48] Switzerland	222 (0) after first MI	30–60	Job work load, locus of control, social supports	1	19	All cause mortality, reinfarction, severe symptoms or poor exercise capacity	Age, severity of MI, exercise	High workload and low external locus of control associated with outcome	+

References in this table are available on the BMJ's website.
CHD = coronary heart disease; MI = myocardial infarction.
*$P < .05$.
†o = no association (relative risk not significantly different from unity); + = moderate association (relative risk, 1.0≤2.0); ++ = strong association (relative risk >2.0).

A/hostility, depression and anxiety, work characteristics, and social supports. Although this review cannot discount the possibility of publication bias, the prospective observational studies show etiological roles for social supports, depression and anxiety, and work characteristics and prognostic roles for social supports and depression. Further evidence of a causal role is provided by human and other primate evidence of biological and behavioral pathways mediating these effects. However, conflicting data exist on whether psychosocial interventions reduce mortality after myocardial infarction. This systematic review should be updated and expanded to include other observational study designs and other end points (for example, all-cause mortality) to focus future research and, ultimately, policy.

Table 4 *Studies of social networks and social supports and coronary heart disease*

Author, year, country	Total sample (% women)	Age at entry, y	Exposure	Follow-up, y	Number of events	Type of events	Adjustments	Relative risk	Summary†
Prospective etiological studies									
Medalie, 1976,[49] Israel	10,000 (0)	>40	Perceived love and support from spouse	5	300	Angina	Age, blood pressure, cholesterol, diabetes, ECG abnormalities	1.8*	+
House, 1982,[50] USA	2754 (52)	35–69	Social relationships and activities	11	114	Fatal CHD	Age, baseline CHD, smoking, forced expiratory volume at 1 second	Not stated	+
Berkman, 1983,[51] USA	4725 (53)	30–69	Social network index	9	120	Fatal CHD	Age	2.13*	++
Reed, 1983,[52] USA	4653 (0)	52–71	Social network index	6	218	Fatal CHD and nonfatal	Age, blood pressure, cholesterol, glucose, uric acid, forced vital capacity, body mass index, exercise, alcohol, complex carbohydrate	Social network associated with CHD prevalence, but not incidence	0
Kaplan, 1988,[53] Finland	13,301	39–59	Social network index	5	223	Fatal CHD	Age, smoking, blood pressure, cholesterol, prevalent illness, urban or rural residence	1.34 for men but not women	0
Vogt, 1992,[54] USA	2603 (54)	18–75+	Network scope, network frequency, and network size	15	Not stated	Fatal CHD and nonfatal CHD	Age, sex, socioeconomic status, smoking, and subjective health status at baseline	1.5* for effect of network scope on CHD incidence; all 3 measures predicted survival in those with CHD	+
Orth-Gomer, 1993,[55] Sweden	736 (0)	50	Emotional support from close people and support from extended network (social integration)	6	25	Fatal and nonfatal CHD	Age, cholesterol treatment of hypertension, diabetes, body mass index, smoking physicial activity	3.8* for social integration 3.1 for emotional support	++
Kawachi, 1996,[56] USA	36,624 (0)	42–77	Social network index	4	403	Fatal CHD and nonfatal MI	Age, time period, smoking, blood pressure, cholesterol, diabetes, angina, body mass index, family history, alcohol, exercise	1.14; some evidence for association with fatal CHD (particularly nonsudden cardiac death) rather than nonfatal MI	0
Prognostic studies									
Chandra, 1983,[57] USA	1401	Not stated	Marital status	10	Not stated	All-cause mortality	Age, race, smoking, severity of MI, medical care factors	Married men and women had better in-hospital and 10-year survival	+
Ruberman, 1984,[58] USA	2320 (0) patients with MI	30–69	Social support, life stress	3	128	All-cause mortality, sudden cardiac death	Age, myocardial function, ventricular arrhythmia, smoking	4.5* for the effect of social isolation and high life stress on all-cause mortality; 5.62 for sudden cardiac death	++
Wiklund, 1988,[59] Sweden,	201 (0) patients with first MI	32–60	Social support, depression, and other psychosocial factors	8.3	85	All-cause mortality and recurrent nonfatal MI	Hypertension, smoking, angina	Being single increased risk of death	+
Case, 1992,[60] USA	1234 (38) participants in diltiazem post-MI trial	25–75	Living alone, disrupted marriage	2	226	Fatal CHD and recurrent nonfatal MI	NYHA functional class, ejection fraction, education, no β-blockers, ventricular premature complexes, prior infarction	1.54* for effect of living alone; no effect of marital disruption	+
Hedblad, 1992,[61] Sweden	98 (0) men with ischemic 24-hour ECG	68	Social support and social network	5	17	Fatal CHD and nonfatal MI	Age, smoking, blood pressure, cholesterol, alcohol, exercise, body mass index, triglycerides	5.6* and 4.1* for low informational support and low emotional support, respectively	++
Williams, 1992,[62] USA	1368 (18) patients with angiographic disease	52 (median)	Structural social support (marital status) and functional social support	9	249	All-cause mortality	Age, ejection fraction, noninvasive myocardial damage index, conduction disturbance, pain or ischemic index, mitral regurgitation, number of diseased vessels, % stenosis of left main stem and left anterior descending artery	3.34* for effect of unmarried patients without confidant	++
Berkman, 1992,[63] USA	194 (48) patients with acute MI	65–85+	Emotional support	0.5	76	All-cause mortality	Age, sex, Killip class, ejection fraction, reinfarction, comorbidity, functional disability, previous MI, ventricular tachycardia	2.9* for lack of emotional support	+

Table 4 *Continued*

Author, year, country	Total sample (% women)	Age at entry, y	Exposure	Follow-up, y	Number of events	Type of events	Adjustments	Relative risk	Summary†
Gorkin, 1993,[64] USA	1322 (17) patients with previous MI plus ventricular premature complexes	60.8 (SD 9.9)	Social support	0.8	Not stated	All-cause mortality	Ejection fraction, arrhythmia rates, CHD risk factors	1.46* for 1-point decrease in social support	+
Jenkinson, 1993,[19] UK	1376 (22) 7 days after MI	25–84	Social isolation, life stress, depression, type A personality	3	247	All-cause mortality	Age, previous MI, hospital complications, diabetes, hypertension, car ownership, sex	1.33 for social support; no effect of type A or depression	0
Friedman, 1995,[65] USA	369 (15) patients after acute MI with ventricular arrhythmias in the CAST	63 (SD 9)	Social support, life events, depression, anxiety, type A, anger	1	20	All-cause mortality	Physiological severity, demographic, and other psychosocial factors	Not stated	+

References in this table are given on the BMJ website.
CAST = Cardiac Arrhythmia Suppression Trial; CHD = coronary heart disease; ECG = electrocardiogram; MI = myocardial infarction; NYHA = New York Heart Association.
*$P < .05$.
†0 = no association (relative risk not significantly different from unity); + = moderate association (relative risk, 1.0≤2.0); ++ = strong association (relative risk >2.0).

In this expanding area, future primary research might investigate:

- Interrelationships between different psychosocial factors,
- Effect of change in and cumulative exposure to psychosocial factors,
- Short- and long-term effects throughout life,
- Differences by sex, ethnic group, and country,
- Behavioral and biological mechanisms involved,
- Effect of psychosocial factors on different clinical and subclinical outcomes, and
- Appropriate primary and secondary preventive measures.

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The Last Simple Yo-Yo

Yo-yos are back. Of course, they're high tech now, with shiny plastic, lights, bells, and whistles. Trying to teach my niece the basics of yo-yoing brought back memories. It was 1950—the "happy days." A few of us had televisions, but for most, the entertainment was simple and inexpensive: choose-up touch football, baseball, and basketball games on the school gravel playground and, in the spring, the local yo-yo contests.

For 10 cents, you could purchase a simple Duncan wooden yo-yo at the corner drugstore. When the strings wore out, you could get three new ones for a nickel. We would practice for hours on the playground or at home. Mom always wanted to know where those new, parallel colored marks on the floor, wall, and ceiling came from. The marks on the floor came from practicing "walk the dog," those on the wall from "rock the cradle," and the particularly annoying marks on the ceiling from "around the world."

Remember the first time you tried making the yo-yo "sleep"? No matter what you did, it came right back up to your hand without spinning at the end of the string. It took perfecting a smooth overhand wrist snap to make the yo-yo come to a rest just above the ground. The trick, of course, was to then get it back to your hand. You would try that gentle, reverse wrist motion, but the yo-yo would stop spinning and just lie on the ground. An even harder task was to rewind the yo-yo—not so easy with the "sleeper" string that surrounded the short wooden axle but did not attach (thus allowing the yo-yo to sleep). You soon learned to wrap the string around the axle a few times before attempting the rewind. These days, with the axle made of smooth plastic material, anybody can make the yo-yo sleep.

Having conquered the sleep maneuver, you could try all sorts of other stuff with the yo-yo. To walk the dog, you made the yo-yo sleep and then just touch the floor, where it "walked" forward. A gentle upward wrist movement, and back it came.

Next came around the world. This was a real test of yo-yopersonship. You threw the yo-yo straight out in front of you, and while it was at the end of the string, you swirled it around in a giant circle with a radius equal to the string length. At the end of the circle, you pulled your hand back, and the yo-yo returned home.

The highest level of achievement was rocking the cradle. You made the yo-yo sleep with your right hand, grabbed the string first with the left hand and then the right hand at the one-third and two-thirds marks, respectively, and formed a triangular cradle with the yo-yo hanging down and still sleeping. If you were really good, you released the cradle and allowed the yo-yo to snap back to your hand.

Thus prepared, you entered the school Duncan Yo-Yo Contest. There were nine of us at the start. Four couldn't get their yo-yos to sleep, and they were out. When it came time to walk the dog, two contestants couldn't get the dog back. Two of us remained. The dreaded rock the cradle would determine it all. I snapped the yo-yo into the sleep position and carefully formed the cradle. The yo-yo slept like never before. With a quick snap of my wrist, it came back to my hand—almost perfect. The last yo-yoer made his moves. Everything went perfect. The yo-yo came back up to his hand . . . and he dropped it.

I had won. The Duncan man gave me an emblem, three regular, one small gold, and a magnificent, gleaming black-painted wooden yo-yo with three "diamond cut" glass studs in a row on each side. I was the school champion, the neighborhood hero, the joy of my parents—all for 10 cents and a lot of hard work and practice.

You can buy that expensive high-tech plastic stuff, but I still prefer my inexpensive low-tech wooden one from the mid-20th century! All that fun on a few pennies per day. Somehow, life—and the practice of medicine—was simpler and better.

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