

A prospective study of social networks in relation to total mortality and cardiovascular disease in men in the USA

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Abstract

Study objective - Previous studies have established a relationship between low levels of social networks and total mortality, but few have examined cause specific mortality or disease incidence. This study aimed to examine prospectively the relationships between social networks and total and cause specific mortality, as well as cardiovascular disease incidence.

Design - This was a four year follow up study in an ongoing cohort of men, for whom information on social networks was collected at baseline. The main outcome measures were total mortality, further categorised into deaths from cardiovascular disease (stroke and coronary heart disease), total cancer, accidents/suicides, and all other causes; as well as stroke and coronary heart disease incidence.

Participants - Altogether 32 624 US male health professionals aged 42 to 77 years in 1988, who were free of coronary heart disease, stroke, and cancer at baseline.

Results - A total of 511 deaths occurred during 122 911 person years of follow up. Compared with men with the highest level of social networks, socially isolated men (not married, fewer than six friends or relatives, no membership in church or community groups) were at increased risk for cardiovascular disease mortality (age adjusted relative risk, 1.90; 95% CI 1.07, 3.37) and deaths from accidents and suicides (age adjusted relative risk 2.22; 95% CI 0.76, 6.47). No excess risks were found for other causes of death. Socially isolated men were also at increased risk of stroke incidence (relative risk, 2.21; 95% CI, 1.12, 4.35), but not incidence of non-fatal myocardial infarction.

Conclusions - Social networks were associated with lower total mortality by reducing deaths from cardiovascular disease and accidents/suicides. Strong social networks were associated with reduced incidence of stroke, though not of coronary heart disease. However, social networks may assist in prolonging the survival of men with established coronary heart disease.

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The association between social relationships and health was originally described by Durk-

heim,¹ who reported an increased risk of suicide among socially isolated individuals. Subsequent epidemiological research has established that social networks predict not only the risk of suicide, but all-cause mortality.² To date, eight prospective epidemiological studies have reported an increased risk of total mortality in socially isolated individuals.³⁻¹⁰ Despite the wealth of evidence on social networks and total mortality, some important questions remain unanswered.

Firstly, it is not clear whether the increased risk of total mortality reflects a generalised susceptibility to illnesses among socially isolated individuals,¹¹ or whether such persons are at especially high risk of mortality from specific types of illness. There is some evidence that social isolation is associated with an increased risk of cardiovascular disease mortality^{6,12} and total cancer mortality.¹³ However, virtually all of the prospective studies to date have reported data on total, but not cause specific, mortality. Secondly, it is not known where along the spectrum of disease social networks exert their impact. The major prospective studies of social networks have examined mortality, but not disease incidence. Thus it is not clear whether social networks influence disease incidence, recovery, or case fatality.¹⁴

To address some of these unanswered questions about social networks, we prospectively studied the associations between social networks and subsequent disease incidence and cause specific mortality.

Methods

The health professionals follow-up study is a longitudinal study of risk factors for cardiovascular disease and cancer among 51 529 US men aged 40 to 75 years in 1986. The study population consists of 29 683 dentists, 10 098 veterinarians, 4185 pharmacists, 3745 optometrists, 2218 osteopathic physicians, and 1600 podiatrists. The study began in 1986, when cohort members completed a mailed questionnaire on heart disease and cancer risk factors, medical history, and diet. Follow up questionnaires were sent in 1990 and 1992 to update this information.

ASSESSMENT OF SOCIAL NETWORKS AND OTHER EXPOSURES

All participants in the study were asked to complete the Berkman-Syme social networks

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index as part of the 1988 mailed questionnaire. The social networks index was originally developed in the Alameda County study,³ and is a composite measure of four types of social connection: marital status (married versus not married); sociability (frequency and number of contacts with extended family and close friends, rated on a scale of 1 to 5, with high values associated with many contacts and low values with few); church group membership (yes versus no); and membership in other community organisations (yes versus none). Responses to the index are categorised into four levels of social connection: low networks (level I) – for example, characterised by individuals with low intimate contacts (not married, fewer than six friends or relatives), and no membership in either church or community groups; medium networks (level II); medium-high networks (level III); and high networks (level IV). Further details of the construction of the social networks index are described elsewhere.¹⁵

In addition to the assessment of social networks, we obtained information from the baseline and follow up questionnaires on the participants' medical history, current smoking habits, body mass index, levels of physical activity, alcohol intake, parental history of myocardial infarction (including the age of each parent at the first event), and participants' self reported history of diabetes mellitus, hypertension, and hypercholesterolaemia. The accuracy of a self reported diagnosis of hypertension was confirmed and reported in a validation study among 100 cohort members.¹⁶

ASSESSMENT OF TOTAL AND CAUSE SPECIFIC MORTALITY

The primary end points in our study comprised deaths from all causes that occurred between the return of the 1988 questionnaire and January 31, 1992. Most deaths were reported by next of kin, work associates, or postal authorities. In addition, the mortality surveillance included systematic searches of the vital records using the national death index to discover deaths among participants who were persistent non-responders to the questionnaire mailings. We estimate that more than 98% of the deaths are ascertained by these methods.¹⁷

Physicians reviewed death certificates and hospital or pathology reports to classify individual causes of death. As well as analysing total mortality, the deaths were grouped into four broad categories: cardiovascular diseases (coronary heart disease, ICD 9th revision codes 410 to 414, and 798; and stroke ICD codes 430 to 438); total cancers (ICD 9th revision codes 140 to 209); external causes of injury (all ICD "E" codes, which included deaths from accident, poisoning, suicide, and other trauma); and all remaining causes of death.

ASSESSMENT OF CARDIOVASCULAR DISEASE INCIDENCE

We also examined all incident cases of coronary heart disease and stroke occurring between the return of the 1988 questionnaire and January

31, 1992. We wrote to all participants reporting an incident myocardial infarction or stroke on the 1990 or 1992 questionnaires to request permission to review their medical records. A definite myocardial infarction was classified according to the criteria of the World Health Organization,¹⁸ and required symptoms plus either typical electrocardiographic changes or high levels of cardiac enzymes. A definite stroke was classified as confirmed if the criteria of the national survey of stroke were met.¹⁹ Incident cases of non-fatal myocardial infarction and non-fatal stroke were classified as "probable" if the hospital records could not be obtained but the event required admission to hospital and the diagnosis was corroborated by supplementary correspondence or telephone interview.

Fatal coronary heart disease was made up of cases of fatal myocardial infarction confirmed by hospital records or autopsy reports; cases of sudden cardiac death (defined below); plus other deaths from coronary heart disease as determined from death certificates, if evidence of *previous* coronary heart disease was available (either from hospital records or interviews with the next of kin) and this was the underlying and most plausible cause of death. In no instance did we use death certificate classification alone to categorise a death as due to coronary heart disease. Sudden cardiac death was defined as death occurring within 1 hour of the onset of symptoms in a man with no previous serious illness, for which no other plausible cause of death – other than coronary disease – was reported.

Death due to stroke was ascertained by physician review of hospital records and autopsy reports. In this study, 92% of the non-fatal myocardial infarction and fatal coronary heart disease, and 87% of the non-fatal and fatal strokes were classified as definite events.

STUDY POPULATION AND DATA ANALYSIS

Because the onset of major illness could itself influence an individual's social networks, we excluded from analyses all members of the cohort who reported a diagnosis of myocardial infarction, stroke, or cancer (except non-melanoma skin cancer) before the return of the 1988 questionnaire ($n = 6115$ men). We cannot exclude the possibility that some men had undiagnosed illnesses at baseline. On the other hand, there is no reason to believe that an undiagnosed illness would have affected an individual's social network. We additionally excluded 12 790 men who after three mailings did not complete the main 1988 questionnaire but instead answered a shorter questionnaire that did not include the Berkman-Syme social networks index. This left a total of 32 624 men available for follow up. After repeated mailings to non-respondents, including the use of certified mail, we obtained a 96% response rate to the 1990 questionnaire, and a 94% response rate to the 1992 questionnaire.

In the analyses, each participant accumulated person-months of follow up from the date of return of the 1988 questionnaire to

Table 1 Age standardised distribution of risk factors in relation to level of social network index

Risk factor	Berkman-Syme social network index			
	IV (high)	III	II	I (low)
No (%)	16 807 (51.5)	6 216 (19.1)	7 706 (23.6)	1 895 (5.8)
Smoking (%)	7.3	9.1	9.5	12.1
Hypertension (%)	19.1	20.4	20.5	20.1
Diabetes (%)	2.7	2.5	2.8	2.8
High cholesterol (%)	16.8	18.0	17.4	16.4
Body mass index (kg/m ²)	25.1	25.0	24.9	24.9
Parental history of MI before age 60 (%)	11.7	12.5	13.1	12.0
Alcohol intake >15 g/d (%)	11.4	16.0	17.0	19.6
Physical activity (MET-h/week)	20.0	19.5	19.5	20.1
Blood pressure check in in past 2 years (%)	86.2	83.2	82.7	79.3
Cholesterol check in past 2 years (%)	70.4	68.6	65.8	62.1
Screening physical exam in past 2 years (%)	66.8	64.0	60.3	57.1

Table 2 Age adjusted and multivariate relative risks of total and cause specific mortality according to level of social network index 1988-92

	Berkman-Syme social network index				Test for trend, p
	IV high	III	II	I (low)	
<i>Total mortality</i>					
Cases	234	101	133	43	
Age adjusted RR	1.00	1.11 (0.88, 1.41)	1.22 (0.99, 1.51)	1.57 (1.14, 2.17)	0.004
Multivariate RR*	1.00	1.06 (0.84, 1.35)	1.13 (0.91, 1.40)	1.38 (0.99, 1.93)	0.06
<i>Cardiovascular disease (stroke and coronary heart disease)</i>					
Cases	63	31	45	14	
Age adjusted RR	1.00	1.28 (0.83, 1.96)	1.53 (1.05, 2.24)	1.90 (1.07, 3.37)	0.006
Multivariate RR	1.00	1.21 (0.79, 1.87)	1.44 (0.98, 2.13)	1.76 (0.97, 3.16)	0.02
<i>Total cancer</i>					
Cases	100	41	39	12	
Age adjusted RR	1.00	1.05 (0.73, 1.52)	0.84 (0.58, 1.21)	1.02 (0.56, 1.84)	0.55
Multivariate RR	1.00	1.03 (0.71, 1.48)	0.78 (0.54, 1.13)	0.87 (0.47, 1.60)	0.25
<i>Accidents and suicides</i>					
Cases	16	10	18	4	
Age adjusted RR	1.00	1.68 (0.77, 3.66)	2.48 (1.29, 4.75)	2.22 (0.76, 6.47)	0.008
Multivariate RR	1.00	1.62 (0.73, 3.59)	2.35 (1.19, 4.62)	1.99 (0.66, 5.99)	0.02
<i>Other causes</i>					
Cases	55	19	31	13	
Age adjusted RR	1.00	0.88 (0.52, 1.48)	1.23 (0.79, 1.89)	1.97 (1.09, 3.58)	0.06
Multivariate RR	1.00	0.78 (0.46, 1.33)	1.09 (0.70, 1.70)	1.48 (0.79, 2.76)	0.32

* Multivariate relative risks adjusted for age (5 year age categories), time period (1988-90; 1990-92), smoking status (never, former, and current in categories of 1 to 14, 15 to 24, and 25 or more cigarettes per day), history of hypertension, diabetes mellitus, and hypercholesterolemia, diagnosis of angina pectoris, deciles of body mass index, parental history of myocardial infarction before age 60 (yes/no), daily alcohol intake (0, 0.41-4.9, 5.0-14.9, 15.0-24.9, 25.0-49.9, or 50 or more g/day), and tertiles of physical activity.

January 31, 1992 (or, for those who died or developed cardiovascular disease, up to the date of the event). Proportional hazards models were used to adjust for age (in 5 year age categories), cigarette smoking (categorized as current smokers of 1 to 14, 15 to 24, or 25+ cigarettes per day, past, or never smokers), alcohol intake (0.0 g/day, 0.01-4.9, 5.0-14.9, 15.0-24.9, 25.0-49.9, and 50.0 or more g/day), body mass index (deciles), history of hypertension, diabetes mellitus, hypercholesterolaemia, angina pectoris, family history of myocardial infarction before age 60, and physical activity (tertiles). When appropriate, we performed the Mantel test for linear trend across levels of social networks, and reported the two tailed p values.²⁰

Results

CHARACTERISTICS OF THE STUDY POPULATION

Altogether 5.8% of the study population were socially isolated (level I of the social networks index), while 23.6% were in level II (medium

networks), 19.1% were in level III (medium-high networks), and 51.5% were socially well integrated (level IV). We compared the age standardised distributions of health behaviours and risk factors across the levels of social networks (table 1). Socially isolated individuals were more likely to be current smokers and to drink more than 15 g/day alcohol (p for linear trend across categories of social networks <0.0001, for both smoking and drinking). Socially isolated men were also less likely to have undergone a blood pressure check, a serum cholesterol check, or a physical examination during the past two years for screening purposes. Other health related habits were distributed similarly across the groups.

TOTAL MORTALITY

During the four years of follow up, 511 deaths occurred in 122 911 person-years. One hundred and fifty three individuals died of cardiovascular diseases (135 coronary heart disease deaths and 18 stroke deaths); 192 of total

Table 3 Age adjusted and multivariate relative risks of total, non-fatal, and fatal stroke incidence according to level of social network index 1988–92

	Berkman-Syme social network index				Test for trend, <i>p</i>
	IV high	III	II	I (low)	
<i>Total stroke</i>					
Cases	40	21	33	10	
Age adjusted RR	1.00	1.39 (0.82, 2.37)	1.82 (1.15, 2.87)	2.21 (1.12, 4.35)	0.002
Multivariate RR*	1.00	1.31 (0.77, 2.23)	1.72 (1.08, 2.73)	2.02 (1.00, 4.08)	0.008
<i>Fatal stroke</i> †					
Cases	5	0	6	2	
Age adjusted RR	1.00	—	2.64 (0.84, 8.27)	3.64 (0.78, 16.9)	0.04
<i>Non fatal stroke</i>					
Cases	35	21	27	8	
Age adjusted RR	1.00	1.55 (0.90, 2.66)	1.65 (1.00, 2.71)	1.96 (0.92, 4.17)	0.02
Multivariate RR	1.00	1.50 (0.87, 2.60)	1.61 (0.97, 2.67)	1.86 (0.85, 4.06)	0.03

*Multivariate relative risks adjusted for age (5 year age categories), time period (1988–90; 1990–92), smoking status (never, former, and current in categories of 1 to 14, 15 to 24, and 25 or more cigarettes per day), history of hypertension, diabetes mellitus, and hypercholesterolemia, diagnosis of angina pectoris, deciles of body mass index, parental history of myocardial infarction before age 60 (yes/no), daily alcohol intake (0, 0.01–4.9, 5.0–14.9, 15.0–24.9, 25.0–49.9, or 50 or more g/day), and tertiles of physical activity.

† Too few cases to perform multivariate analysis.

cancer; 48 of accidents and suicide; and 118 of other causes.

Compared with the group with the most social connections (level IV), socially isolated individuals (level I) were at 1.5 times the age adjusted risk of total mortality (*p* for trend across categories of social networks index = 0.004) (table 2). Adjustment for a broad range of risk factors in multivariate analysis somewhat attenuated the association. The following components of the social networks index predicted total mortality (data not shown in tables): not being married (relative risk, 1.41; 95% CI 1.07, 1.87); not belonging to a church group (relative risk, 1.42; 95% CI 1.19, 1.70); and the absence of close relatives (relative risk, 1.34; 95% CI 1.01, 1.78).

CAUSE SPECIFIC MORTALITY

Socially isolated individuals were at increased risk of cardiovascular diseases (*p* for trend = 0.006), and accidents and suicides (*p* for trend = 0.008) though not total cancer (*p* for trend = 0.55). Of the individual components of the social networks index, being unmarried was the strongest predictor of cardiovascular disease mortality (relative risk, 1.44; 95% CI 0.88, 2.38). We analysed stroke mortality separately from coronary heart disease mortality (data not shown in tables). Compared with the group with the highest level of social ties (level IV), the age adjusted relative risk of stroke mortality was 1.31 (95% CI 0.24, 7.23) among men in the medium-high group (level III), 4.90 (95% CI 1.70, 14.16) among men in the medium group (level II), and 6.59 (95% CI 1.78, 24.38) among men in the low group (level I) (*p* for trend, 0.0008, based on 18 deaths). By contrast, the association between social networks and coronary mortality was weaker: relative to the group with the highest level of social ties (level IV), the age adjusted relative risk of coronary heart disease mortality was 1.28 (95% CI 0.82, 1.99) among men in the medium-high group (level III), 1.31 (95% CI 0.86, 1.98) among men in the medium group

(level II), and 1.59 (95% CI 0.84, 3.02) among men in the low group (level I) (*p* for trend, 0.09, based on 135 deaths).

Three components of the social networks index strongly predicted mortality from accidents and suicides: not being married (relative risk, 2.92; 95% CI 1.50, 5.67); not belonging to a church group (relative risk 1.99; 95% CI 1.14, 3.47); and the absence of close relatives (relative risk, 3.83; 95% CI 1.97, 7.47). Twenty cases of suicide occurred during the four year study period. The age adjusted relative risk of suicide among individuals in the most socially isolated group (level I) was 3.22 (95% CI 0.92, 11.22) compared with those in the most well connected group (*p* for trend = 0.12). Previous studies have reported an association between social isolation and smoking related cancers.¹³ However, we found no significant association between social isolation and lung cancer mortality (relative risk in men with fewest social connections = 1.48; 95% CI 0.43, 5.07; *p* for trend = 0.52, based on 39 cases).

CORONARY HEART DISEASE AND STROKE INCIDENCE

Altogether 104 incident cases of stroke (91 cases of non-fatal stroke, and 13 cases of fatal stroke) and 403 cases of incident coronary heart disease (275 cases of non-fatal myocardial infarction, and 128 cases of fatal coronary heart disease) occurred during the four year follow up. If a participant developed a new episode of stroke or heart attack and died within the two year interval between two successive follow up questionnaires, then this person was classified as having had an incident fatal event.

A strong dose-response gradient was found between the level of social networks and stroke incidence (*p* for trend, 0.008) (table 3). Increased risks were observed for both fatal and non-fatal stroke, although there were too few cases of fatal stroke to carry out multivariate analysis. In contrast to stroke, social isolation was not associated with the incidence of either total coronary heart disease or non-fatal myo-

Table 4 Age adjusted and multivariate relative risks of total coronary heart disease, non-fatal myocardial infarction, and fatal coronary heart disease incidence according to level of social network, 1988–92

	Berkman-Syme social network index				Test for trend, <i>p</i>
	IV high	III	II	I (low)	
<i>Total coronary heart disease</i>					
Cases	188	90	100	25	
Age adjusted RR	1.00	1.27 (0.99, 1.63)	1.16 (0.91, 1.47)	1.17 (0.77, 1.77)	0.17
Multivariate RR*	1.00	1.26 (0.98, 1.62)	1.13 (0.89, 1.45)	1.14 (0.74, 1.73)	0.25
<i>Fatal coronary heart disease</i>					
Cases	56	25	37	10	
Age adjusted RR	1.00	1.16 (0.73, 1.86)	1.41 (0.93, 2.14)	1.52 (0.78, 2.98)	0.06
Multivariate RR*	1.00	1.10 (0.68, 1.77)	1.34 (0.88, 2.04)	1.42 (0.72, 2.81)	0.13
<i>Non fatal myocardial infarction</i>					
Cases	132	65	63	15	
Age adjusted RR	1.00	1.32 (0.98, 1.77)	1.05 (0.78, 1.41)	1.01 (0.59, 1.72)	0.69
Multivariate RR*	1.00	1.32 (0.98, 1.79)	1.04 (0.76, 1.40)	1.00 (0.58, 1.71)	0.19

* Multivariate relative risks adjusted for age (5 year age categories), time period (1988–90; 1990–92), smoking status (never, former, and current in categories of 1 to 14, 15 to 24, and 25 or more cigarettes per day), history of hypertension, diabetes mellitus, and hypercholesterolemia, diagnosis of angina pectoris, deciles of body mass index, parental history of myocardial infarction before age 60 (yes/no), daily alcohol intake (0, 0.01–4.9, 5.0–14.9, 15.0–24.9, 25.0–49.9, or 50 or more g/day), and tertiles of physical activity.

Table 5 Age adjusted and multivariate relative risks of sudden cardiac death and non-sudden cardiac death, according to level of social network index 1988–92

	Berkman-Syme social network index				Test for trend, <i>p</i>
	IV high	III	II	I (low)	
<i>Sudden cardiac death</i>					
Cases	21	10	12	2	
Age adjusted RR	1.00	1.24 (0.58, 2.63)	1.22 (0.60, 2.49)	0.82 (0.19, 3.52)	0.78
Multivariate RR*	1.00	1.22 (0.57, 2.60)	1.10 (0.54, 2.26)	0.68 (0.16, 2.96)	0.94
<i>Non-sudden cardiac death</i>					
Cases	35	15	25	8	
Age adjusted RR	1.00	1.12 (0.61, 2.03)	1.52 (0.91, 2.54)	1.94 (0.91, 4.14)	0.04
Multivariate RR*	1.00	1.05 (0.57, 1.94)	1.48 (0.88, 2.49)	1.89 (0.87, 4.13)	0.05

cardiac infarction (table 4). There was a modest increase in risk of fatal coronary heart disease among individuals with the least social connections (multivariate relative risk, 1.42; 95% CI 0.72, 2.81). We examined this association in detail by separately analysing sudden cardiac death (45 cases) and non-sudden cardiac death (83 cases) (table 5). The rationale for performing this analysis was based on the theory that social relationships enhance disease survival through the provision of various types of support, for example, advice to stop smoking.²¹ If improved survival among individuals with already established disease is indeed the mechanism by which social relationships affect mortality, we would not expect to find a protective effect of social networks for sudden cardiac death, since by definition, death occurs within one hour of the onset of symptoms. Consistent with this hypothesis, we found no association between level of social ties and sudden cardiac death (*p* for trend, 0.94), but a dose-response gradient with risk of non-sudden cardiac death (*p* for linear trend = 0.05) (table 5).

Finally, we assessed potential effect modification by smoking status and diagnosis of hypertension. Within strata of smoking status (never, former, and current smokers), the strongest association of social networks with total mortality was found among current smokers. The relative risk of total mortality in the least socially connected group was 2.16

(95% CI 1.10, 4.23) among smokers, 1.73 (95% CI 1.09, 2.76) among former smokers, and 1.10 (95% CI 0.53, 2.28) among never smokers. The smoking χ social isolation interaction term was statistically significant ($p < 0.001$). The impact of poor social networks on cardiovascular mortality was stronger in men with a diagnosis of hypertension (relative risk, 2.62; 95% CI 1.07, 6.45) than in men without hypertension (relative risk, 1.57; 95% CI 0.74, 3.31). When we ran a model assessing interaction, the hypertension χ social isolation term was statistically significant ($p = 0.01$).

Discussion

Our findings are compatible with the notion that social networks reduce total mortality by lowering deaths from cardiovascular disease and accidents/suicides. In previous studies, the relative risks of total mortality among the most socially isolated individuals ranged from 1.8 (in the Evans County study) to 4.0 (in the Gothenburg study), whereas the present study found a multivariate relative risk of about 1.4. The smaller magnitude of risk found in the present study may be partly due to the high socioeconomic status and relative homogeneity of health professionals who continued our cohort. High socioeconomic status – associated with such resources as a high level of knowledge about health and disease, or access to health

care and other material goods – may reduce some of the disadvantages of social isolation.^{22,23} We were also able to adjust for a broader range of health behaviours and risk factors in this study compared with previous investigations.^{3,4} In terms of the magnitude of the association, an adjusted relative risk of total mortality of 1.4 is comparable to the effect of cigarette smoking on total mortality reported in some studies.^{24,25}

Four potential pathways have been proposed through which social relationships might affect health.¹⁴ Firstly they may have an effect through the provision of instrumental support, such as financial assistance or services in kind. Such tangible assistance could contribute to the recovery and survival of patients after major illness (such as stroke), independent of professional medical services. A second pathway is through the provision of information and advice that might assist individuals to seek medical care services, or to adopt more health promoting behaviours, such as smoking cessation. The existence of such a pathway is supported by two of our findings: that socially isolated men were less likely to undergo medical screening (table 1) and, secondly, that the effects of poor social networks were strongest among those who smoked or had hypertension. If risky health behaviours (such as smoking) are in the pathway through which poor social networks act, then control for such behaviours in multivariate analyses may constitute statistical over adjustment and attenuate the reported associations towards the null.

A third postulated pathway linking social networks to health is the provision of emotional support. A recent report²⁶ indicated that after an acute myocardial infarction, patients who lacked emotional support were nearly three times as likely to die during the first 6 months (odds ratio, 2.9; 95% CI 1.2, 6.9) compared with patients who received emotional support. A limitation of our study is that we did not collect data on such *functional* aspects of social ties. Thus, we are unable to distinguish the relative importance of different types of social support (eg, emotional, instrumental, informational) for the maintenance of health.

It has been suggested that social networks may directly influence host resistance and susceptibility to disease through alterations in neuroendocrine and immunological control systems.^{11,27,28} For example, stress induces a sustained increase in cortisol and insulin levels, which in turn may lead to hyperlipidaemia and accelerate atherogenesis.²⁹ However, few studies have actually demonstrated altered neuroendocrine control under conditions of stress.³⁰ Importantly, our data failed to corroborate the hypothesis of generalised disease susceptibility. Increased risks were found only for suicide (confirming Durkheim's original observation), and cardiovascular disease mortality.

Our analyses of coronary heart disease incidence suggest that social relationships affect *survival* following the onset of coronary heart disease, but not the incidence of new disease. A prospective study of social networks among

Japanese-Americans residing in Hawaii³¹ also found no association with incidence of non-fatal myocardial infarction. In our data, poor social ties were associated with an increased risk of fatal coronary heart disease, but only if sudden cardiac deaths were excluded as an end point. The finding that social networks have little or no influence on the risk of sudden cardiac death suggests that the protective mechanisms associated with social ties operate after the onset of illness. This interpretation is also consistent with several prospective studies among patients with *established* coronary heart disease,^{26,32-35} in which social relationships were found to predict survival after acute myocardial infarction.

The association of strong social ties with reduced stroke mortality was previously described in the Alameda County study.³ To our knowledge, no other study has reported a protective effect of social networks on stroke *incidence*. Several previous studies have reported a protective effect of social ties on cancer survival,^{13,35,36} although the evidence has been inconsistent^{37,38} depending on the stage of the cancer and the tumour site. We found no association of total cancer mortality with social ties, although our four year follow up period cannot exclude an effect on long term survival. We caution that some of our disease specific findings are based on limited duration of follow up with small numbers of cases. Nonetheless, our data suggest that social networks may improve the outcome of established disease, especially cardiovascular disease.

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