

SOCIAL CONNECTIONS AND MORTALITY FROM ALL CAUSES AND FROM CARDIOVASCULAR DISEASE: PROSPECTIVE EVIDENCE FROM EASTERN FINLAND

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Kaplan, G. A. (Human Population Laboratory, California Dept. of Health Services, Berkeley, CA 94704), J. T. Salonen, R. D. Cohen, R. J. Brand, S. L. Syme, and P. Puska. Social connections and mortality from all causes and from cardiovascular disease: prospective evidence from eastern Finland. *Am J Epidemiol* 1988;128:370-80.

The association between an a priori measure of social connections and five-year mortality from all causes, cardiovascular diseases (*International Classification of Diseases*, Eighth Revision (ICD-8) codes 390-458), and ischemic heart disease (ICD-8 codes 410-414) was studied in 13,301 men and women from eastern Finland who were first interviewed in 1972 or 1977. For men, there was a graded association between extent of social connections and mortality. In multivariate models with adjustment for age, smoking, serum cholesterol, mean weighted blood pressure, measures of prevalent illness, and other possible confounders, men who were in the two lowest quintiles of the social connections scale were at increased risk compared with those in the highest quintile (odds ratio (OR)_{all cause} = 1.54, 95% confidence interval (CI) = 1.21-1.95; OR_{cardiovascular disease} = 1.54, 95% CI = 1.11-2.13; OR_{ischemic heart disease} = 1.34, 95% CI = 0.94-1.90). No strong or consistent association was found for women. The association for men was modified by levels of blood pressure with the effect of low social connections greater at higher levels of blood pressure. In three separate analyses, there was no evidence for confounding or effect modification due to prevalent illness at baseline.

cardiovascular diseases; mortality; prospective studies; social isolation

In 1979, Berkman and Syme (1) presented evidence that an index of social net-

work participation based on reports of amount and frequency of contacts with friends and relatives, marital status, and group membership was strongly associated with nine-year mortality from all causes. This association was found for both men and women and was consistent across the age range 30-69 years. Their analyses, based on data collected as part of the Alameda County Study (2, 3), attracted considerable attention and have been quoted widely. Additional analyses of the Alameda County Study data using different subsamples or measures of social connections have confirmed the existence of this association (2, 3). However, other studies of the rela-

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tion between social connections and mortality, conducted in such diverse places as Michigan (4), Hawaii (5), Georgia (6), North Carolina (7), Connecticut (8), and Gothenberg, Sweden (9), have resulted in a series of findings which one observer has characterized as "inconsistent, confusing, and often contradictory" (10, p. 93).

For example, social activities were found to be associated with male mortality in the Tecumseh (4) and Gothenberg (9) studies, but not in the Hawaii (5) or Evans County (6) studies. Similarly, strong associations were found between social connections and female mortality in Alameda County, but the associations were weak or nonexistent in Tecumseh and Evans County. Furthermore, measures of contacts with friends and relatives were associated with mortality in the Alameda County, Gothenberg, and North Carolina studies, but not in the Tecumseh, Evans County, or Connecticut studies.

Because these studies have used different measures of social connections, populations, and outcomes in their analyses, it is difficult to disentangle the reasons for this inconsistent pattern of results (11, 12). However, this confusing array of results raises a number of issues which, if studied, might help to clarify the nature of the association between social connections and health.

By far the most important issue concerns the role of prevalent illness in the association between social connections and mortality. In the Hawaii study (5), an association was found between social connections and prevalence, but not incidence, of coronary heart disease. These findings raised the possibility that low levels of social connections were the result of disease and consequently were associated with mortality. To our knowledge, no study has, as yet, carefully examined the role of prevalent illness in the association between social connections and mortality.

Another issue concerns the role of marital status in the interpretation of the reported associations between social connec-

tions and mortality. Many indices of social connections, such as the social network index used by Berkman and Syme (1), have included marital status as one of the component items. There is sufficient evidence concerning the association between marital status and disease (13, 14), particularly in men, to raise the possibility that the association between social connections and mortality may be primarily due to the impact of marital status.

Little is also known about the potential role of factors which, on theoretical grounds, might modify the association between social connections and mortality. For example, we do not know if the association between social connections and mortality differs in urban and rural locations or as a function of level of education, marital status, or health status. Similarly, we know little about potential modifier effects associated with smoking, blood pressure, or other risk factors.

Another important issue concerns the specificity of the association between social connections and mortality from specific causes. Studies of the association between social connections and mortality outcomes have generally restricted their attention to deaths from all causes. Although suggestive findings have been reported for specific outcomes (1, 2), these results have not been presented in any detail.

Finally, additional information is needed concerning several other issues. There have been no careful analyses of the nature of the association between social connections and mortality that would allow conclusions about whether the association is a graded one, or if, instead, increased risk is primarily confined to those who are isolated (15). Additional information is also needed concerning sex differences in the association between social connections and mortality.

This report addresses these issues using data collected in eastern Finland (16, 17) on a large, representative sample for whom there were data available from laboratory analyses and physical exams, as well as self-reported information on social connections.

Follow-up through a national death registry enabled us to address a number of the important methodological and substantive issues raised earlier.

MATERIALS AND METHODS

Study population

In 1972 and 1977, a survey of a random 6.7 per cent sample of the population aged 25–59 (1972) and 30–64 (1977) years of two provinces of eastern Finland, Kuopio and North Karelia, was carried out (16, 17). Participation rates were high (93 per cent in 1972 and 90 per cent in 1977), resulting in a total sample of 11,839 participants in 1972 and 12,155 in 1977. Analyses in this report are restricted to the 13,301 persons who were aged 39–59 years at baseline examination.

Survey methods

The methods used in the surveys of these two cohorts have been reported elsewhere (16, 17). Essentially the same methods were used in 1972 and 1977. Examinations included a self-administered questionnaire, interviews, and physical measurements. Blood pressure was measured by 12 trained observers using a standard mercury sphygmomanometer. Participants rested in a sitting position for 15 minutes before measurement. Venous blood was drawn from participants for serum cholesterol determinations after they had fasted for at least four hours and avoided heavy meals on the preceding day. Serum cholesterol was determined from frozen samples in a central laboratory, standardized against World Health Organization reference material.

Prevalent illness

Prevalent illness was ascertained in several ways. Respondents indicated whether they had been diagnosed and/or treated by a doctor during the previous 12 months for any of the following conditions: myocardial infarction, angina pectoris, emphysema or chronic bronchitis, asthma, cardiac insufficiency, and diabetes. Respondents also

indicated whether they were currently taking any medication for cardiovascular conditions and if they had given up leisure time activities during the last year because of disease or disability. Information was also available on diagnosis of hypertension and use of antihypertensive medications. All of these measures were coded as dichotomous variables.

Risk factors

Standard cardiovascular risk factors measured were serum cholesterol (mg/dl), mean weighted blood pressure ($\frac{1}{3}$ systolic blood pressure + $\frac{2}{3}$ diastolic blood pressure), body mass index (weight (kg)/height (m^2)), and smoking (number of cigarettes smoked per day). These were all coded as continuous variables. Family history of cardiovascular disease was coded dichotomously based on either paternal or maternal death from cardiovascular disease before age 60 years. Mean weighted blood pressure was used in order to make use of information from both systolic and diastolic pressure readings.

Social connections index

Five questions were asked concerning the extent and frequency of social connections. The items, response categories, and coding are given in table 1. On an a priori basis, a social connections index was specified which simply summed the codes given in table 1. Since there was no basis for assigning unequal weights to the five questions, they were all equally weighted. Marital status, the only dichotomous variable, was given the codes of 1 (unmarried) and 4 (married) to equalize its contribution to the index. In addition, on an a priori basis, we decided to treat the index categorically by dividing the range of scores into quintiles. Quintiles were determined separately for men and women, although the differences in the distributions of the scores were small. The decision to categorize the score was based on a general lack of knowledge concerning the linearity of the association

TABLE 1
Items used in the social connections index

Questions asked	
1.	What is your current marital status?
2.	How often do you visit friends and relatives?
3.	On the average, how many different homes of friends or relatives do you visit per month?
4.	How many people usually come to see you or call you per day?
5.	How often do you go to meetings of clubs, associations, or societies?
Coding	
1.	Unmarried = 1, married = 4
2.	Never = 1; once a year or less = 2; a couple of times a year = 3; 1-3 times a month = 4; once a week = 5; several times a week = 6
3 and 4.	None = 1; 1 to 2 = 2; 3 to 4 = 3; 5 to 6 = 4; 7-10 = 5, more than 10 = 6
5.	Never = 1; once a year or less = 2; a couple of times a year = 3; once a month = 4; 2 to 3 times a month = 5; once a week or more = 6

between the index and risk of mortality. The use of a score based on quintiles allowed us to examine this issue in some detail and provided more flexibility in modeling the association.

Other potential confounders

Other variables were identified on both theoretical and empiric grounds, which might confound the association between the social connections index and outcomes. These included area of residence (Kuopio Province or North Karelia Province), cohort (1972 or 1977), education (number of years), and urban versus rural residence. The last variable refers to the division of each province into primarily rural areas and areas including and surrounding the major cities in each province. Approximately 60 per cent of the population lived in rural areas.

Ascertainment of outcomes

Mortality was ascertained via the Finnish National Death Certificate Registry, which records deaths of all Finnish citizens. In order to equalize follow-up periods for the two cohorts, we included only deaths occurring in a five-year period postbaseline.

The underlying cause of death was taken as that assigned by the Central Statistical Office of Finland after review of death certificate information, coded according to the *International Classification of Diseases, Eighth Revision (ICD-8)*. There were 598 deaths from all causes, of which 450 were men and 148 were women. Of this total, 297 deaths were from cardiovascular disease (ICD-8 codes 390-458), with 239 in men and 58 in women, and 223 were from ischemic heart disease (ICD-8 codes 410-414), with 192 in men and 31 in women.

Statistical methods

The 1972 and 1977 cohorts were combined into a single cohort. An indicator variable specifying to which cohort a respondent belonged was included in all multivariate analyses. Age-adjusted mortality rates were calculated for quintiles of the social connections score using the direct method, with the total cohort as the standard population. Multiple logistic regression analyses were carried out using the SAS LOGIST procedure (18). Separate analyses were carried out for men and women. The five quintiles of the social connections index were included in logistic models as four dummy variables, with the reference category as the most connected quintile.

RESULTS

The age-adjusted risk of death during the five-year follow-up for each quintile is presented in figure 1. For men, there is a strong and consistent gradient of risk, with the highest risk in the lowest quintile and the lowest risk in the highest quintile. The relative risks for those in the lowest quintile compared with those in the highest are 2.42 for deaths from all causes, 2.42 for deaths coded as cardiovascular disease, and 1.92 for deaths coded as ischemic heart disease. For women, the pattern is not as consistent. Those in the lowest quintile evidence the highest age-adjusted rates for all three outcomes; however, the gradients of risk are not as marked as they are for men. An

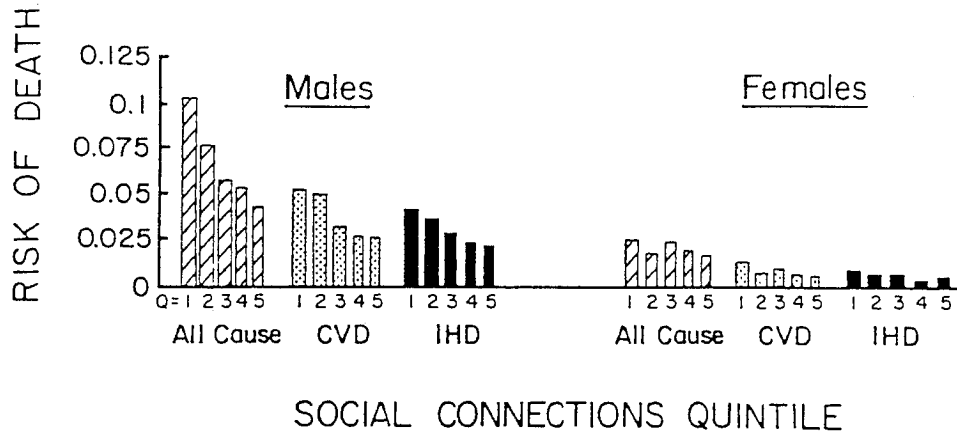


FIGURE 1. Five-year age-adjusted risk of death by quintile of social connections score, men and women in eastern Finland, first interviewed in 1972 and 1977.

overall test of the contribution of information on social connections to the prediction of all-cause mortality was calculated by comparing a model which contained age plus all other adjustment variables with one which also included the social connections dummy variables. This test indicated a significant contribution due to information on social connection for men ($p = 0.0002$), but not for women ($p = 0.46$). A global test of the social connections by sex interaction was consistent with an effect for men and no effect for women ($p = 0.096$). The difference in the patterns of risk for men and women is striking. In exploratory analyses in which other risk variables were included in multivariate models, there continued to be no differences in risk associated with social connections scores for women. This was also true for women when marital status was removed from the score. Although the apparent increased risk in the lowest quintile and weak gradient of risk for women is suggestive, the lack of a significant effect of social connections would seem to indicate that any increased risk, if present, is not sufficiently strong to be detected in this study. Because of these findings, subsequent analyses are restricted to men.

Multivariate analysis

Table 2 presents the results of multiple logistic regression analyses examining the association for men between quintiles on the social connections score and deaths

from all causes, cardiovascular disease, and ischemic heart disease.

For deaths from all causes, there is a clear gradient of risk associated with different social connections quintiles. The most isolated are at over twice the risk of those who are least isolated (odds ratio (OR) = 2.12, 95 per cent confidence interval (CI) = 1.44–3.12). Those in the second quintile also evidence significantly increased risk compared with the top quintile (OR = 1.78, 95 per cent CI = 1.20–2.66). Those who scored in the third and fourth quintiles evidence considerably less elevation in risk compared with the fifth quintile, indicating that for deaths from all causes, the major elevation in risk is associated with being in the lowest 40 per cent of the distribution on the social connections score.

A similar pattern of elevated risk is seen for deaths from cardiovascular disease and ischemic heart disease. In both cases, quintiles 1 and 2 show elevated odds ratios when compared with quintile 5. Respondents in quintiles 1 and 2 are at increased risk compared with those in quintiles 3 and 4 for deaths from all causes (OR = 1.54, 95 per cent CI = 1.21–1.96), cardiovascular disease (OR = 1.54, 95 per cent CI = 1.11–2.13), and ischemic heart disease (OR = 1.34, 95 per cent CI = 0.94–1.90).

The role of prevalent disease

In these analyses, we have included those with prevalent disease at baseline and adjusted for the effect of prevalent disease by

inclusion of indicator variables for a variety of conditions. Because of the controversy concerning the role of illness in the association between social connections and health, we examined this issue in two additional ways. First, we estimated the association between social contacts and deaths for those respondents who indicated no prevalent disease at baseline. Table 3 shows the results of this analysis, in which there was adjustment for age, cohort, prov-

ince, cholesterol, mean weighted blood pressure, body mass index, smoking, family history of cardiovascular disease, urban/rural residence, and education. Exclusion of those with prevalent illness resulted in a reduction of the number of persons at risk of 24, 23, and 23 percent for analyses of deaths from all causes, cardiovascular disease, and ischemic heart disease, respectively. The number of cases was reduced by 51, 55, and 57 per cent, respectively. This

TABLE 2

*Association between quintiles of social connections index and death from all causes, cardiovascular disease (CVD), and ischemic heart disease (IHD) in men from eastern Finland first interviewed in 1972 and 1977**

Variable	All causes		CVD		IHD	
	Odds ratio	<i>p</i>	Odds ratio	<i>p</i>	Odds ratio	<i>p</i>
Quintile 1† (lowest)	2.12	0.0001	1.63	0.058	1.67	0.069
Quintile 2†	1.78	0.005	1.98	0.008	1.73	0.057
Quintile 3†	1.28	ns‡	1.23	ns	1.36	ns
Quintile 4†	1.26	ns	1.06	ns	1.13	ns
Age (per year)	1.06	0.0001	1.05	0.001	1.05	0.004
Province	1.16	ns	1.37	0.037	1.26	ns
Urban/rural	0.82	ns	0.87	ns	0.87	ns
Serum cholesterol (10 mmol/liter)	1.02	0.05	1.06	0.0001	1.08	0.0001
Mean weighted blood pressure	1.02	0.0001	1.03	0.0001	1.02	0.0007
Cigarettes per day	1.03	0.0001	1.03	0.0001	1.03	0.0001
Body mass index	0.98	ns	0.98	ns	1.01	ns
Family history	0.99	ns	1.19	ns	1.12	ns
Prevalent myocardial infarction	2.75	0.0001	3.81	0.0001	3.58	0.0001
Prevalent angina	0.81	ns	1.04	ns	1.16	ns
Prevalent diabetes	1.24	ns	1.34	ns	1.02	ns
Prevalent cardiac insufficiency	1.14	ns	0.91	ns	0.91	ns
Prevalent emphysema	1.33	ns	0.89	ns	0.84	ns
Cardiovascular medication use	2.22	0.0001	2.55	0.0001	2.91	0.0001
Education (per year)	0.94	0.05	0.93	0.02	0.95	ns
Cohort (1977/1972)	0.97	ns	1.06	ns	0.88	ns

* Multiple logistic models with all variables included.

† Compared with quintile 5 (highest).

‡ ns, not significant.

TABLE 3

Association between quintiles of social connections index and deaths from all causes, cardiovascular disease (CVD), and ischemic heart disease (IHD) in men from eastern Finland without prevalent disease first interviewed in 1972 and 1977*

Quintile	All causes		CVD		IHD	
	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval	Odds ratio	95% confidence interval
1	2.00	1.18-3.39	1.80	0.87-3.72	1.72	0.77-3.84
2	1.65	0.96-2.84	1.78	0.86-3.70	1.50	0.66-3.43
3	1.45	0.84-2.51	1.27	0.59-2.74	1.41	0.61-3.23
4	1.02	0.55-1.91	0.81	0.33-1.99	0.88	0.33-2.33

* Multiple logistic model with adjustment for age, cohort, province, cholesterol, mean weighted blood pressure, body mass index, smoking, family history of cardiovascular disease, urban/rural residence, and education.

reduction in sample size and number of outcomes is presumably responsible for the increase in the size of the confidence intervals associated with the social connections quintiles shown in table 3. In all cases, there is a graded relation between the quintile of the social contacts index and the three outcomes. The magnitude and consistency of the associations with those found earlier suggest that the results observed previously are not due to the inclusion of prevalent cases in the analyses.

To further investigate the role of prevalent illness in the association between social connections and mortality, we examined a series of logistic models in which an interaction term between a dichotomized social contacts score and each measure of prevalent illness was included one at a time. The dichotomous social contacts score compared those in quintiles 1 and 2 (42.7 per cent) with those in quintiles 3-5. Results of a logistic analysis confirmed that this dichotomous social contact variable was associated with risk of death from all causes in a model containing all adjustment variables used in the analyses reported in table 3 (OR = 1.70, 95 per cent CI = 1.37-2.12). Subsequent models examined the interaction between this variable and, individually, current use of cardiovascular medications, report of angina pectoris, previous myocardial infarction, prevalent cardiac insufficiency, prevalent emphysema, and prevalent diabetes. In no case was there evidence for even a marginally significant or otherwise important interaction, indicating that prevalent illness does not appear to modify the association between social connection and all-cause mortality.

The role of marital status

Because known associations between marital status and mortality (13, 14) could account for the association between social connections and mortality, we generated a new social contacts score that did not include information on marital status. This score was entered into a logistic model as a dichotomous variable with those in the two

lowest quintiles of the score compared with all others. All adjustment variables previously considered plus marital status were included in this model. The approximately 38 per cent who were in the high-risk group were at increased risk of deaths from all causes (OR = 1.50, 95 per cent CI = 1.20-1.87). Thus, there is no evidence that for men marital status is a confounder of the association between social contacts and mortality from all causes. In order to examine the role of marital status as an effect modifier, we included an interaction term between marital status and the dichotomized contacts score in another model. There was no evidence for interaction (OR_{interaction term} = 1.06, 95 per cent CI = 0.61-1.83).

The role of risk factors

To examine potential effect modification between other significant risk factors and the social connections score, we carried out analyses in which an interaction term between the dichotomized social contacts score and a particular risk factor was added to a logistic model containing all other adjustment variables. Continuous variables were dichotomized in order to facilitate interpretation of interactions. The top quartile was compared with the lowest three quartiles for serum cholesterol (≥ 269 mg/dl versus other), body mass index (≥ 28.2 kg/m² versus other), and mean weighted blood pressure (≥ 119 mmHg versus other). Cut points based on dichotomization at the median values for these variables were also considered; however, the results were generally no different than for the above choices. Three methods of coding smoking were used: "ever" versus "never," "less" versus "more than one pack per day," and "never smokers" compared with "both light and heavy smokers." Evidence for effect modification was found only in the analysis of mean weighted blood pressure.

The association between the dichotomized social connections score and all-cause mortality was 1.47 times higher among those in the top quartile of blood

pressure compared with those in the lower three quartiles (95 per cent CI = 0.92–2.36). To further explore this relation, we modeled mean weighted blood pressure as a continuous variable, and a term for the interaction between the dichotomized social connections score and blood pressure was included in the model. The interaction coefficient was 0.016 (95 per cent CI = 0.001–0.031), indicating an increase of 1.6 per cent in the social connections odds ratio for each 1 mmHg increase in blood pressure. Thus, the odds ratio associated with the dichotomized social connections score was 1.41 at the 25th percentile of weighted mean blood pressure (102 mmHg) and 1.84 at the 75th percentile (119 mmHg).

This interaction is most strongly related to the diastolic component of mean weighted blood pressure. When the above models for all-cause mortality were fitted using diastolic and systolic pressures separately, the interaction coefficients were 0.02 (95 per cent CI = 0.003–0.037) and 0.008 (95 per cent CI = –0.002–0.018), respectively. The interaction with diastolic pressure was also found when cardiovascular disease ($\beta = 0.018$, 95 per cent CI = –0.004–0.040) and ischemic heart disease ($\beta = 0.018$, 95 per cent CI = 0.007–0.043) outcomes were considered.

To examine further this interaction between social connections and blood pressure, we carried out logistic analyses in which all respondents who reported a diagnosis of hypertension and/or the use of antihypertensive medication were excluded. The results of these analyses indicated an even stronger interaction between mean weighted blood pressure and social connections for all causes, cardiovascular disease, and ischemic heart disease outcomes. As in the previous analyses, this interaction was stronger for diastolic than for systolic pressure.

The role of other variables

Modification of the association between social connections and all-cause mortality was also examined with respect to a number

of other variables for which there is reason to believe such an effect might exist. Education, rural versus urban residence, and cohort were included individually in interaction with the dichotomized social contacts score. In no case was there any indication of any marginally significant or otherwise important interaction. The same results held when education was dichotomized in a number of alternative ways.

DISCUSSION

These analyses demonstrate a strong and consistent relation in men between an a priori measure of social connections and mortality from all causes, cardiovascular disease, and ischemic heart disease. To our knowledge, this is the strongest evidence to date that reduced social connections are related to mortality from cardiovascular disease. Risk varies in a graded fashion as a function of the social contacts score and is independent of a number of risk factors and other potential confounders or effect modifiers. In addition, there is the appearance of a threshold effect, with only those in the two lowest quintiles of the social connections index at substantially increased risk.

Using three different analytic strategies, we find no evidence whatsoever for the speculation (5) that the association between social contacts and mortality is an artifact of prevalent illness which results in low social contacts. The results of analyses which examine this association using adjustment for prevalent illness or exclusion of those with prevalent illness, or allowing for interaction with prevalent illness are all consistent. Those in the lowest two quintiles have one and one-half to two times greater risk of death compared with those in the most connected quintile. This result is found for deaths from all causes, cardiovascular disease, and ischemic heart disease.

Because these analyses of the role of prevalent illness are based on self-reports of diagnosed conditions, it is possible that there is some misclassification of prevalent

illness due to the limitations of such reports. However, the reports are strong predictors of mortality in these analyses. In logistic models, with adjustment for age, respondents who reported a prevalent condition (myocardial infarction, angina pectoris, cardiac insufficiency, or emphysema) or use of cardiovascular medications were at substantially increased risk of death. For example, those who reported a previous myocardial infarction were at increased risk for mortality from all causes (OR = 4.63, $p < 0.0001$), cardiovascular disease (OR = 7.09, $p < 0.0001$), and ischemic heart disease (OR = 7.46, $p < 0.0001$). Reports of angina pectoris (OR = 2.57, $p < 0.0001$), cardiac insufficiency (OR = 2.50, $p < 0.0001$), and use of cardiovascular medications (OR = 5.01, $p < 0.0001$) were also associated with ischemic heart disease death. Thus, it seems unlikely that the self-report of diagnosed conditions in this study results in any substantial bias.

It has been suggested that the variations in findings from studies of social connections and mortality might be due to differences in the communities and subgroups studied. For example, Berkman (19) and Syme (12) have both proposed that in communities or subgroups which are socially cohesive and well-integrated, social connections may have little relation to mortality. Thus, in this view, the weak associations between measure of social activities and risk for women in the Tecumseh Study analyses (4) and the absence of effects for men in the Honolulu Study (5) are due to high levels of social connections for Tecumseh women and Honolulu men. We would then expect to see associations between social connections and mortality only in groups or locations not characterized by high levels of social connections. This is an intriguing hypothesis which needs to be explicitly tested. Short of any such explicit a priori test, we can only rely on indirect sources of data.

One such source of data comes from a comparison of similar items in three of the

studies. We have compared items related to visits with friends and relatives in the current study ("How often do you visit friends and relatives?") with those in the Alameda County Study (1-3) ("How many of these close friends and relatives do you see at least once a month?") and the Tecumseh Study (4) ("How often did you visit with friends, neighbors? How often with relatives?"). Although the items are not identical, they presumably tap similar dimensions of social connections. Participants in this study reported 1.7 visits per month (men = 1.7, women = 1.7), compared with 4.8 in the Alameda County Study (men = 4.7, women = 4.9), and 2.8 in the Tecumseh Study (men = 2.6, women = 3.0).

To the extent that such questions measure integration and connectedness in subgroups or communities, these values are not consistent with the hypothesis that an association between social connections and mortality will not be found in communities or groups with high levels of connections. For example, there is little difference between the frequency of contacts for men and women in these three studies. Yet in the Alameda County Study, strong associations were found for both men and women, while in this study and the Tecumseh Study, strong associations were found only for men. When similar measures related to the prevalence of high levels of connections in men and women are calculated for these three studies, the pattern also does not support the above hypothesis.

It is, of course, possible that aspects of social connections which are critical for a protective effect are not being measured in the studies in which associations are weak, absent, or inconsistent. The questions used in studies of social connections and mortality certainly tap only limited aspects of social experience and interaction, and it is possible that they are particularly poor measures in groups which are highly connected or well-integrated (19). However, verification of such a conclusion will have to be based on careful and a priori specifi-

cation of critical dimensions of social interaction and how these might vary between communities or subgroups.

The interaction between blood pressure and risk for the isolated and the nonisolated is of some interest, particularly in view of the fact that no other significant effect modifiers were found. This interaction is present for both all-cause and cardiovascular outcomes and is not due to the inclusion of diagnosed hypertensives in the analyses. It is of interest to note that in studies of migration and acculturation to Western culture (20, 21), results indicated that there were increases in blood pressure associated with social change which were unexplainable by reference to factors known to influence blood pressure. In both studies, the social changes of migration and acculturation could be seen as movement away from more traditional societies with high levels of social connections.

It is not possible to specify which pathways account for this synergistic relation between social connections and blood pressure; both endogenous and exogenous pathways are possible. For example, neuroendocrine and other mechanisms involved in blood pressure regulation may also be involved in the increased risk associated with lower social connections. Variations in level of social connections may also be associated with poorer detection of hypertension or poorer blood pressure control or adherence to medication in those who are hypertensive. Since respondents in this study who were hypertensive but were previously undetected at baseline were referred to treatment as part of the study protocol, these latter possibilities are particularly intriguing. Further studies will be needed to examine these and other possibilities. In the meantime, it would be useful to collect, at the least, simple information on the social connections status of participants in studies that examine risk of all-cause or cardiovascular death in relation to blood pressure.

In summary, these analyses provide clear

evidence for men, but not for women, of a relation between social connections and risk of deaths from all causes and from cardiovascular causes. The increased risk associated with variations in social connections seems to be primarily present in those in the lowest 40 per cent of the distribution of social connections. The findings are not consistent with the hypothesis that the relation is an artifact of prevalent illness. Nor is there any evidence for variations in this association as a function of marital status, education, or urban versus rural residence, factors which would be expected to influence the quality, importance, and availability of social connections. The finding that there is an interaction between social connections and blood pressure, primarily diastolic pressure, raises a number of interesting hypotheses about the endogenous and exogenous pathways which might account for the observed association. However, any such explanation will also have to account for the observation that the association between low social connections and increased risk is strongest for deaths from all causes. Whether our ultimate understanding of the connections between social activities and health will require elaboration of the notion of "generalized susceptibility" (22) or discovery of both physiologic and behavioral cause-specific pathways remains an open question which can only be answered by further research.

REFERENCES

1. Berkman LF, Syme SL. Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. *Am J Epidemiol* 1979;109:186-204.
2. Kaplan GA. Psychosocial aspects of chronic illness: direct and indirect associations with ischemic heart disease mortality. In: Kaplan RM, Criqui MH, eds. *Behavioral epidemiology and disease prevention*. New York: Plenum Publishing Corporation, 1985:237-69.
3. Seeman TE, Kaplan GA, Knudsen L, et al. Social network ties and mortality among the elderly in the Alameda County Study. *Am J Epidemiol* 1987;126:714-23.
4. House JS, Robbins C, Metzner HL. The association of social relationships and activities with mortality: prospective evidence from the Tecum-

- seh Community Health Study. *Am J Epidemiol* 1982;116:123-40.
5. Reed D, McGee D, Yano K, et al. Social networks and coronary heart disease among Japanese men in Hawaii. *Am J Epidemiol* 1983;117:384-96.
 6. Schoenbach VJ, Kaplan BH, Fredman L, et al. Social ties and mortality in Evans County, Georgia. *Am J Epidemiol* 1986;123:577-91.
 7. Blazer DG. Social support and mortality in an elderly community population. *Am J Epidemiol* 1982;115:684-94.
 8. Zuckerman DM, Kasl SV, Ostfeld AM. Psychological predictors of mortality among the elderly poor. *Am J Epidemiol* 1984;119:410-23.
 9. Welin L, Tibblin G, Svardsudd K, et al. Prospective study of social influences on mortality. *Lancet* 1985;1:915-18.
 10. Zyzanski S. Social support and social networks: methodology discussion. In: Ostfeld AM, Eaker ED, eds. *Measuring psychosocial variables in epidemiologic studies of cardiovascular disease*. Bethesda, MD: National Institutes of Health, 1985:93-8. (NIH publication no. 85-2770).
 11. Berkman LF. Social networks, support, and health: taking the next step forward. *Am J Epidemiol* 1986;123:559-62.
 12. Syme SL. Social networks in relation to morbidity and mortality. Paper presented at Berzelius Symposium V: Social Support and Health. Malmo, Sweden, September 1985.
 13. Koskenvuo M, Kaprio J, Kesaniemi A, et al. Differences in mortality from ischemic heart disease by marital status and social class. *J Chronic Dis* 1980;33:95-106.
 14. Ortmeier CF. Variations in mortality, morbidity, and health care by marital status. In: Erhardt LL, Berlin JE, eds. *Mortality and morbidity in the United States*. Cambridge, MA: Harvard University Press, 1974.
 15. Cohen S, Syme SL. *Social support and health*. Orlando, FL: Academic Press, Inc., 1985.
 16. Puska P, Tuomilehto J, Salonen JT, et al. The North Karelia Project: evaluation of a comprehensive community program for control of cardiovascular diseases in 1972-1977 in North Karelia, Finland. Copenhagen: World Health Organization, 1981.
 17. Salonen JT, Puska P, Kottke TE, et al. Changes in smoking, serum cholesterol and blood pressure levels during a community-based cardiovascular disease prevention program—the North Karelia project. *Am J Epidemiol* 1981;114:81-94.
 18. SAS Institute. SAS supplemental library user's guide, 1980 edition. Cary, NC: SAS Institute, 1980:83-102.
 19. Berkman LF. The relationship of social networks and social support to morbidity and mortality. In: Cohen S, Syme SL, eds. *Social support and health*. Orlando, FL: Academic Press, Inc., 1985:241-62.
 20. Joseph JG, Prior IAM, Salmond CE, et al. Elevation of systolic and diastolic pressure associated with migration: the Tokelau Island Migrant Study. *J Chronic Dis* 1983;36:507-16.
 21. Page LB, Friedlaender J. Blood pressure, age, and cultural change: a longitudinal study of Solomon Islands populations. In: Horan MJ, Steinberg GM, Dunbar JB, et al., eds. *NIH blood pressure regulation and aging*. Proceedings from a symposium. New York: Biomedical Information Corporation, 1986:11-25.
 22. Cassel J. The contribution of the social environment to host resistance. The Fourth Wade Hampton Frost Lecture. *Am J Epidemiol* 1976;104:107-23.

