SOCIAL CONTACTS AND ISCHAEMIC HEART DISEASE

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ABSTRACT

The association between measures of social connections and mortality from ischemic heart disease was studied using data from the Alameda County Study in California and the North Karelia Study in Eastern Finland. In both studies, there is a significant association between the extent of social connections and mortality from ischemic heart disease. Those who are socially isolated are at 2 to 3-fold increased risk of death over 5 to 9 years when compared to those most connected. These results are found when there is extensive adjustment for traditional cardiovascular risk factors. Analyses using a variety of techniques provide no evidence that this association is due to the impact of prevalent disease on the extent of social contacts. Furthermore, changes in social connections during one 9-year period are prospectively associated with increased risk of death from ischemic heart disease in a subsequent 9-year period. Finally, evidence is presented indicating that the level of social connections modifies the association between diastolic blood pressure and risk of death from ischemic heart disease.

KEY WORDS: ISCHEMIC HEART DISEASE; PREVENTION

INTRODUCTION

Over the last decade, there has been a growing recognition that psychosocial factors are critically involved in the development and progression of cardiovascular disease (CVD). These influences affect the entire trajectory of CVD, ranging from the adoption, maintenance, and cessation of cardiovascular risk factors, to the interpretation and presentation of symptoms, utilization of medical care, survival post-cardiovascular event, to quality of life post-event.

Although many psychosocial factors have been studied, increasing attention is being given to those related to social support and social network participation. A recent search of the U.S. MEDLINE system indicates that there has been an almost exponential increase in the number of scientific articles which used social support or social networks as a descriptor [1]. In the period 1966—1970, there were only 8 articles so described. From 1981 to 1986, there were, however, over 2,000!

Probably the most influential of these studies is that reported by Berkman and Syme [2]. Their analyses examined 4,700 persons, age 39—59, who were part of the Alameda County Study’s 1965 cohort [3] and assessed the association between a simple measure of social connections and 9-year mortality from all causes. This measure, called the social network index, combines information concerning marital status, number of close friends and relatives, how often these friends and relatives are seen, and membership in church groups and other kinds of organizations. There was a clear gradient of risk associated with different levels of this index. Generally speaking, men and women in the bottom category were at 2—2.5 times the risk of death from all causes than those in the top category, even when a number of possible confounders were taken into account. Similar findings have been reported from studies in Michigan, Georgia, and North Carolina [4–6] in the United States, as well as from Sweden and Finland [7, 8]. Although there are some differences between the results of these studies, the similarities are sufficiently strong as to be quite impressive.

SOCIAL CONTACTS AND ISCHAEMIC HEART DISEASE MORTALITY

Unfortunately, few of these studies have looked specifically at cardiovascular outcomes. What evidence there is, however, is encour-
aging. In further analyses of the Alameda County cohort, a clear association was seen for deaths from ischaemic heart disease (IHD) during the 9-year follow-up period. Figure 1 presents the results of these analyses, based on 9 years of follow-up of 2,352 persons 50 years of age or older at the beginning of the study (9). There is a clear gradient of risk associated with different levels of the social network index, and this gradient is found for both men and women and in both age groups. A number of potential confounding factors were examined (Table 1). Health status at baseline; health habits such as smoking, physical activity, and relative weight; socioeconomic status; psychological measures; and age were all examined as possible confounders. With adjustment for all of these, those who were most isolated on the social network index were at almost 3 times the risk of death from IHD compared to those who were least isolated.

**CHANGES IN SOCIAL CONTACTS AND IHD MORTALITY**

In order to have more confidence in the association between the extent of social contacts and IHD mortality, a series of analyses was devoted to the impact of changes in social contacts and subsequent risk of death. A number of previous analyses have indicated that events such as loss of a spouse or an important change in social contacts might be associated with increased risk of cardiovascular outcomes (10, 11). However, such studies generally begin analyses after the change in social contacts has occurred and are not able to take into account other factors such as those related to changes in health occurring prior to the loss, which might themselves be associated with increased risk of death. It was possible to avoid these problems in further analyses utilizing the Alameda County Study 1965 cohort (12). Study participants in 1965 were reinterviewed in 1974, and their mortality was ascertained for the period 1974–1983. Thus it was possible to examine the association between changes in social contacts during the period 1965–1974, with risk of death from IHD during the subsequent 9-year period. In addition, information on health status in 1965 and changes in health status between 1965 and 1974 were included in the analyses. Table 2 indicates the results of these analyses. Decreases in the number of close friends, the number of close friends and relatives seen at least once per month, or the total number of contacts per month were

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**TABLE 1**

Relative risk of ischemic heart disease death (1965—1974) (9)
> 50 years, Alameda County Study.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Relative Risk</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social network (low/high)</td>
<td>2.86</td>
<td>1.65, 4.96</td>
</tr>
<tr>
<td>Health practices [0/5]</td>
<td>3.11</td>
<td>2.68, 3.61</td>
</tr>
<tr>
<td>Perceived health (poor/excellent)</td>
<td>1.48</td>
<td>1.14, 1.93</td>
</tr>
<tr>
<td>Helplessness (+/-)</td>
<td>1.53</td>
<td>1.06, 2.18</td>
</tr>
<tr>
<td>Life satisfaction (+/-)</td>
<td>1.07</td>
<td>0.64, 1.80</td>
</tr>
<tr>
<td>Depression (+/-)</td>
<td>0.95</td>
<td>0.67, 1.35</td>
</tr>
<tr>
<td>Socioeconomic status [low/other]</td>
<td>1.03</td>
<td>0.83, 1.26</td>
</tr>
</tbody>
</table>

Plus: age, sex, physical health status
TABLE 2
Association between changes in social contacts (1965—1974) and ischemic heart disease mortality (1974—1983), Alameda County Study.

<table>
<thead>
<tr>
<th>1965—1974 Change</th>
<th>Males</th>
<th></th>
<th>Females</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Relative risk</td>
<td>p (</td>
<td></td>
<td>Relative risk</td>
</tr>
<tr>
<td>Friends (−10)</td>
<td>1.65</td>
<td>(0.02)</td>
<td>1.41</td>
<td>(0.17)</td>
</tr>
<tr>
<td>Relatives (−10)</td>
<td>1.47</td>
<td>(0.12)</td>
<td>1.53</td>
<td>(0.12)</td>
</tr>
<tr>
<td>Number see ≥1/month (−10)</td>
<td>1.57</td>
<td>(0.06)</td>
<td>1.75</td>
<td>(0.04)</td>
</tr>
<tr>
<td>Total contacts/month (−60)</td>
<td>1.19</td>
<td>(0.02)</td>
<td>1.15</td>
<td>(0.04)</td>
</tr>
</tbody>
</table>

1 Compared to no change from logistic model with adjustment for age, prevalent chronic conditions and symptoms, incident chronic conditions and symptoms, and 1965 level of social connections.

all associated with increased risk of death from IHD. Those who reported 10 fewer close friends in 1974 than in 1965 were at substantially increased risk compared to those who reported the same number (OR_{men} = 1.65; OR_{women} = 1.41). Decreases in the number of close friends and relatives seen at least once a month were also associated with increased risk of death from IHD (OR_{men} = 1.57; OR_{women} = 1.75). Interestingly, decreases in the number of close relatives were not associated with increased risk. These results of the association between changes in social contacts and IHD mortality are particularly striking given that there was adjustment for various chronic conditions and symptoms at baseline as well as for newly occurring conditions and symptoms in 1974.

FURTHER EXAMINATION OF POTENTIAL CONFOUNDERING BY PREVALENT DISEASE

It was possible to further examine the role of prevalent disease in the association between social connections and IHD mortality using data collected as part of the North Karelia Study [8]. This is an important issue because it is certainly possible that being ill could influence the nature and extent of one’s social contacts. These analyses utilized a study population of 13,301 men and women, age 39—59, who were respondents in either the 1972 or 1977 surveys in North Karelia and Kuopio counties (13). In this cohort, there were a total of 598 deaths from all causes during the first 5 years of follow-up. Of these, 297 were attributed to CVD (ICD 390—458) and 223 from IHD (ICD 410—414). A simple index of social contacts was constructed from 5 items concerning the extent and frequency of social connections (Table 3).

The age-adjusted risk of death during the 5-year follow-up for each quintile of the social connections score is presented in Fig. 2. For men, there was 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 who are in the lowest quintile compared to those in the highest are 2.42 for deaths from all causes, 2.42

![Mortality by social connections](image)

Fig. 2. 5-year risk of death by social connections quintile, men and women age 39—59 in North Karelia Study.
TABLE 3
Items used in social connection index.

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. Married = 1, else = 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. 1–2 = 2, 3–4 = 3, 5–6 = 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, 2–3 times a month = 5, once a week or more = 6

for deaths coded CVD, and 1.92 for deaths coded IHD. 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 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 males [p = 0.0002] but not for females [p = 0.46]. In exploratory analyses in which other risk variables were included in multivariate models, there continued to be no differences in risk associated with social connection 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 these analyses. Because of these findings, subsequent analyses were restricted to males.

The role of prevalent illness was examined in three ways. In the first strategy, there was adjustment for prevalent conditions. Table 4 presents the results of multiple logistic analyses in which there was adjustment for a large number of variables, including reports of the following individual conditions diagnosed and/or treated by a doctor during the last 12 months: myocardial infarction, angina pectoris, emphysema or chronic bronchitis, asthma, cardiac insufficiency, and diabetes. Respondents also indicated if they were currently taking any medication for cardiovascular conditions. Information was also available on diagnosis of hypertension and use of antihypertensive medications. All of these measures were coded as dichotomous variables. Information was also available on total serum cholesterol level (mg/dl), smoking (number of cigarettes smoked per day), blood pressure (1/3 SBP + 2/3 DBP), body mass index (weight in kg/height in m²), and family history of CVD. Those in the most isolated quintile were again at elevated risk for death from all causes, CVD, and IHD. There is no evidence that the association between the social contacts score and mortality from all causes, CVD, or IHD is influenced by adjustment for prevalent illness or, for that matter, the major risk factors for IHD mortality.

In a second strategy, all respondents who reported prevalent illness at baseline were excluded from the analyses. Again, there was a gradient of risk associated with the level of social contacts, and the risk estimates were very close to those estimated in the previous models. In the third strategy, evidence for effect modification was examined, e.g., if the association between social contacts and the mortality outcomes varied as a function of prevalent illness. In no case was there evidence for even a marginally significant or otherwise important interaction.

Thus, using three different analytic strategies, we find no evidence, whatsoever, for the speculation [14] 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 exam-
ine this association using adjustment for prevalent illness, exclusion of those with prevalent illness, or allowing for interaction with prevalent illness are all consistent. Those in the lower two quintiles have 1.5—2 times greater risk of death compared to those in the most connected quintile. This result is found for deaths from all causes, CVD, and IHD.

THE ROLE OF CARDIOVASCULAR RISK FACTORS

It would simplify our explanatory efforts considerably if the elevated risk associated with social isolation was attributable to different levels of conventional risk factors such as smoking, physical activity, or lipid levels. However, the distributions for these factors are not remarkably different as a function of level of social contacts, and there is no evidence for confounding due to these factors in multivariate analyses. However, it is possible that psychosocial factors such as social connections may modify the influence of conventional risk factors. This possibility was examined in a series of logistic models in which the interaction between the social connections score, in these analyses dichotomized, and each major risk factor for IHD was examined. There was no evidence for effect modification when the interaction with levels of serum cholesterol, body mass index, smoking, and family history was considered. However, most interestingly, there was a significant interaction between level of social contacts and weighted mean blood pressure level. To eliminate any problems resulting from the inclusion of those who were under treatment for hypertension, they were excluded. Upon further examination, this interaction was found to be restricted to the diastolic component, and the relationship was best described by a log-linear-quadratic model [15]. As can be seen in Fig. 3, the risk of death from all causes rises much faster in the isolated than in the non-isolated. In fact, it almost appears as if being non-isolated is dampening the relationship between diastolic blood pressure and risk of death. Fig. 4 shows that similar findings were seen for IHD mortality, although, of course, the overall level of risk is much lower.

These results are in accord with findings which suggest that relative weight and physical activity may modify the risk associated with a given level of blood pressure. It is not clear what pathways are associated with this modification of the relationship between level of diastolic pressure and risk of death. Current efforts are being devoted to examine whether this effect reflects exogenous pathways involving behavioral differences or if it reflects a psychosocial modifier of the complex set of pathophysiological changes which link heightened blood pressure and cardiovascular events.

CONCLUSIONS

Evidence from the Alameda County and North Karelia Studies indicates that there is a substantial association between the extent of one’s social contacts and risk of death from cardiovascular causes. This effect does not appear to
be due to the impact of prevalent disease on level of social contacts, nor is it attributable to higher levels of conventional cardiovascular risk factors among those with lower levels of social contacts. Furthermore, the extent of social connection seems to have an influence on pathophysiological processes associated with blood pressure regulation.

There are, however, a number of questions which must be answered before we can reach any understanding of these effects. First, the nature of the social contacts association must be clarified. For example, are there particular aspects of social contacts which are most important, are there individual differences or personality aspects which must be taken into account, and are other aspects of social functioning, such as social support, important? Secondly, basic epidemiologic knowledge is lacking which could clarify the relationship between social connections and CVD. For example, are social connections associated with the incidence of ischemic events, are there different patterns of association for fatal and non-fatal events, are levels of social connections associated with progression of disease, and are they associated with other pathways such as those related to prostaglandins?

Many of these questions are currently being addressed in the Kuopio Ischemic Heart Disease Study currently in progress. The answers from this investigation and others being conducted elsewhere will help to clarify the pathways by which the hemodynamic environment and the social environment produce cardiovascular events.

REFERENCES


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