

Impact of social network on cardiovascular mortality in middle aged Danish men

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Abstract

Study objective—To estimate quantitatively (the aetiological fraction) the impact of poor social network on premature death from cardiovascular disease in middle aged, white men.

Design—The causality of the relationship has already been discussed in a large review, and it is assumed to be well documented. The numerical estimation of the impact was based on a review of all published cohort studies on the relationship between social network and mortality in white, middle aged men.

Results—The studies reviewed are all of high epidemiological quality and present a consistent and stable dose-response pattern. The aetiological fraction was estimated to be 30%, with a plausible range of 20-40%.

Conclusions—Social network was an important, independent, risk factor for cardiovascular disease in white, middle aged men. It had a strong impact on mortality, comparable to that of traditional risk factors. Social network should have a more central role in future epidemiological research into cardiovascular disease. The factors that result in a strong social network should be identified and strategies applicable in preventive work should be developed.

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The causal relationship between poor social network and health is now almost as well documented as that between smoking and health was in 1964 when the US Surgeon General established cigarette smoking as a cause for mortality and morbidity from a range of diseases.¹ House *et al*¹ reviewed the evidence from observational studies and interventional studies, biological understanding, and animal experiments (to the extent that these are possible). They concluded that there is "strong empirical evidence for a causal impact of social relationships on health"—a conclusion that has remained unchallenged. The present paper aims to provide a quantitative estimate of the size of the impact of social network on health in society and compare it with other causes of heart disease.

Cardiovascular or ischaemic heart disease is an obvious choice for further and more detailed study, as it is the main cause of premature death among middle aged Anglo-American and north-European men. Furthermore, its risk factors have been studied extensively, and the epidemiological studies on social network present a picture of an unusually consistent and stable "dose-response" relationship.

To assess the impact of a risk factor on mortality from cardiovascular disease in middle aged Danish men, an aetiological fraction is estimated. The aetiological fraction, a concept and measure of growing importance,² expresses the proportion of disease that would not have occurred had the risk factor not been present in the population.³ Unlike relative risk, the aetiological fraction depends not only on the strength of the causal relationship but also on the prevalence of the risk factor, putting as much emphasis on common, moderately hazardous risk factors as on rare extremely hazardous ones. The aetiological fraction has sometimes been defined by the simplistic formula:

$$(RR-1)^b / ((RR-1)^b + 1)$$

where RR is the relative risk and b is the fraction exposed. This is inappropriate, however, in many circumstances,⁴ and it is more correct to give a verbal definition, as several authors have done.³⁻⁷ Although the wording is slightly different, the meaning is as stated above.

Because the aetiological fraction depends on the distribution of the risk factor in the population, and on the distribution of other risk factors as well,³⁻⁶ the analysis has to be focused narrowly: in this study on middle aged Danish men. The result, however, is probably applicable to neighbouring countries and age groups and requires no or few analytical changes. No suitable studies have been carried out in Denmark, so empirical information was borrowed from other similar populations.

Methods

STUDY POPULATIONS

The material comprised all known prospective mortality studies on white (middle aged) men,⁸⁻¹⁵ as given in the review of House *et al*¹ together with a single study published later¹⁶ that corroborated House's findings. The studies and their characteristics are given in table I. Unfortunately, two of the reports give insufficient information for the calculation of any relevant aetiological fraction—the paper by Welin *et al* does not give information on the prevalence of the various levels of network index and that by Zuckerman *et al* lacks this information for white men.

Of the seven studies^{8-10 13-16} remaining for analysis, only two give the results of statistical analysis carried out specifically for cardiovascular or ischaemic heart mortality. Two reports mention that the analyses were carried out and give results similar to those for the reported total mortality. The three remaining studies were too small to

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perform separate analyses for various causes of death. No studies report results on morbidity.

Five of the seven studies cover middle aged men (45–55 years at entry). Two of these papers^{9 13} mention that no interaction between the effect of social network and age was found, so the reported results are applicable irrespective of age. The Finnish study covers middle aged men with a slightly wider age range. The last two studies give figures for three⁸ or four¹⁴ separate age groups, one or two covering middle age. The two studies of elderly men were included because they are the only studies that analyse a multidimensional social network score and report the results separately for each dimension.

Although social network is measured differently in all studies, the similarities are strong. Marriage and some sort of contact frequency with friends and family are always included. The social network is usually measured by means of a one dimensional score. Only two papers report results for more dimensions,^{10 16} and only these studies include the subjective experience (for example, satisfaction) of the social network. Participation in formal groups (church, etc) is covered in all but two studies.^{10 14}

STATISTICS

The aetiological fractions were calculated individually for each study in the following manner. When the dose-response relationship was not stable, the reference category was combined with the neighbouring categories to obtain a monotonous relation and a stable reference group before the aetiological fraction was calculated. Whenever possible the calculations are based on relative risks estimated in multivariate analyses; otherwise marginal or age adjusted relative risks were used. When the studies report only the analysis of a unidimensional index of social network, calculation of the aetiological fractions was performed according to principles outlined by Miettinen⁶ and Levin.⁵ Otherwise separate aetiological fractions were first calculated for each reported social network component and secondly combined into a single measure using Miettinen's formula 1⁶

$$(1-EF1)*(1-EF2)* \dots *(1-EFn)$$

where EF_n is the aetiological fraction for the nth component. Application of these methods of calculation make the following assumptions necessary: the social network has to be distributed independently of other risk factors (and of each other if more than one dimension is used) and there should not be any interactions between social network and other risk factors (and between

the various dimensions if more than one dimension is used).

The sampling error is not the only source of variability of the estimate. Following the recommendation of Greenland,¹⁷ no confidence intervals have been supplied. Instead, the sensitivity of the estimates as a result of differences in the choice of methodology and study base in the various studies is discussed. Based on this a point estimate with a set of plausible limits is given.

Results

The dose-response patterns for the five studies using a one dimensional score is given in the figure. The aetiological fractions are listed in table II together with the total aetiological fractions from the two studies that measure social network by means of a multidimensional score.

With few exceptions, the aetiological fractions lie in the range 25%–35%. The estimate based on the Swedish survey is probably biased strongly downward because of the application of an all-embracing summed score of 18 items that dilutes the true relationships.^{18 19} The questions cover all kinds of social contact: with parents, children, neighbours, coworkers, etc. For each age and sex group, only a few of the contact sources are of importance; the remaining items will mostly create "noise" in the summed score. The Evans County study is a study of a small town in a rural area and this may explain why the findings are not as strong as those in Alameda County: friends and relatives may be seen at work, while shopping, and when performing other routines of daily life. The result indicates that the applied measures of social network tend to differentiate less in a small rural community than in a metropolitan area. The study by Hanson *et al* yields the largest aetiological fraction. In this study the most elaborate measure of social network was used. Whether application of the method of Hanson *et al* in other studies would give equally high aetiological fractions or whether the comparatively high value is caused by chance in a small sample with many variables is unclear.

The interactions between social network and other cardiovascular risk factors were found to be negligible. None of the studies that examined this^{9 13 15} found any significant interactions. Nor did studies that applied a multidimensional score find any interactions between the social network dimensions. This finding agreed with the general experience that the multiplicative model is a good description of most data.²⁰ Deviation from the assumption of no interaction introduces only a minor bias into the estimates when the risk ratios are as small as those here.²¹

Table I Characteristics of nine follow up studies of social network

Year of publication	Authors and reference no	Place	Population size*	No of cases†	Age at baseline (y)	Follow up (y)	Mortality‡ T C I
1979	Berkman & Syme ⁸	Alameda County, USA	4724	211	30–69	9	x y y
1982	House <i>et al</i> ⁹	Tecumseh, USA	2754	172	35–69	9–12	x y
1982	Blazer ¹⁰	Durham County, USA	331	50 b	65–	2.5	x
1984	Zuckerman <i>et al</i> ¹¹	New Haven, USA	398	47 b	62–	2	x
1985	Welin <i>et al</i> ¹²	Gothenburg, Sweden	989	151	50,60	9	x
1986	Schoenbach <i>et al</i> ¹³	Evans County, USA	2059	665 ab	22–87	12	x
1987	Orth-Gomér & Johnson ¹⁴	Sweden	1 7443	841	29–74	6	x x
1988	Kaplan <i>et al</i> ¹⁵	Eastern Finland	1 3301	598	39–59	5	x x x
1989	Hanson <i>et al</i> ¹⁶	Malmö, Sweden	500	67	68	4–5	x

* Number of persons studied (both sexes). The drop out rate is below 30% in all studies, mostly in the range 10–20%.

† Number of men who died during follow up; (b) both sexes; (a) approximately.

‡ Outcome measure analysed: T=total mortality, C=cardiovascular disease mortality, I=ischaemic heart disease mortality; x=statistical analysis reported, y=analysis performed but not reported.

All traditional cardiovascular risk factors²² were controlled for in at least one (and most of them in most) of the studies, even though not all risk factors are simultaneously controlled for in any single study. It is not possible in any study to explain away the observed association between social network and mortality by means of the confounding factors measured. To the extent that some of the effect of social network is through one or more of the risk factors controlled for in the

multivariate analyses (smoking, blood pressure, serum cholesterol, physical activity, etc), the estimate of the aetiological fraction is biased downwards.

Based on the above results and considerations the aetiological fraction of social network in middle aged Danish men is estimated to be 30%. All the individual estimates lie in the range 10–50%. In line with the considerations of bias, source populations, and chance variations a narrower plausible interval of 20–40% is proposed.

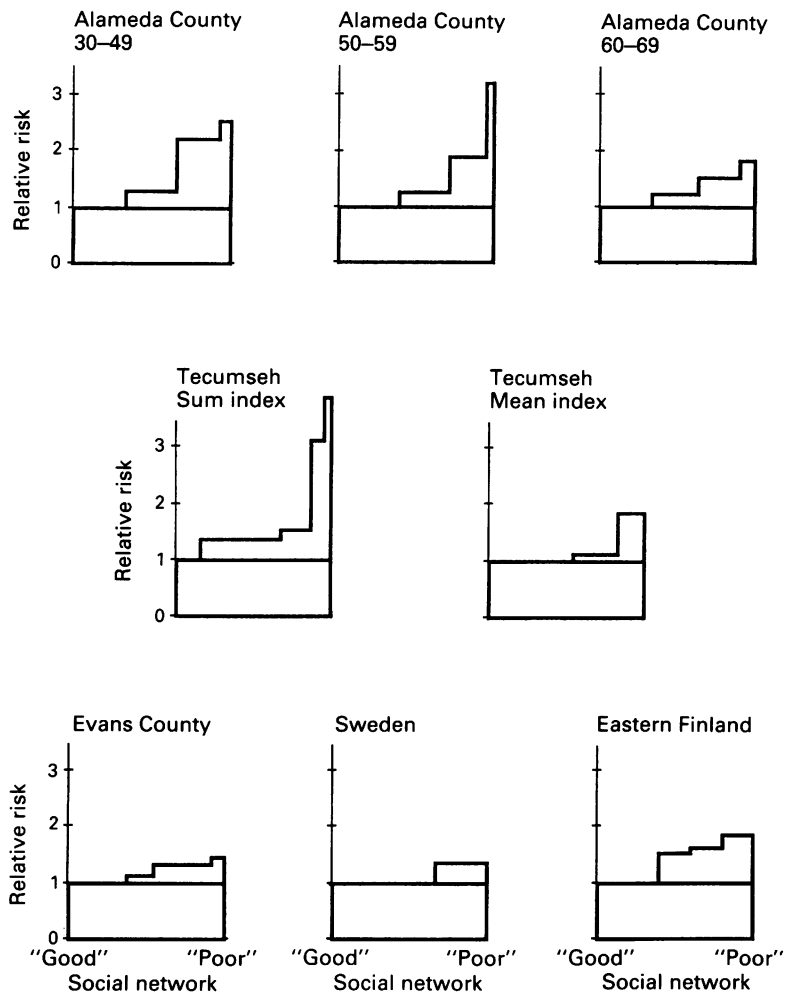


Figure Dose-response relationships between social network and mortality in five follow up studies.

The x axes are proportional to the entire populations, ordered according to the level of social network with the reference group to the left. The y axes display adjusted relative risks, and the horizontal line displays a relative risk of 1. (In the graphs "Tecumseh—mean index" and "Eastern Finland", three levels, respectively two quintiles, have been pooled to obtain a stable reference category.

Table II Aetiological fractions for social network in cardiovascular mortality in each of seven studies.

Study	Aetiological fraction (%)	Calculations based on	Relative risk estimates in paper
Alameda County ⁸	35, 29, 22*	Figure 1 (A), (p190)	Age adjusted
Tecumseh ⁹	38, 15†	Figure 2 (p137)	Age adjusted
Durham County ¹⁰	29	Table 7 (p687)	Multivariate
Evans County ¹³	15	Table 2 (p583)	Raw
Sweden ¹⁴	10, 11‡	Tables 3/4 (p953–4)	Multivariate
Eastern Finland ¹⁵	33, 28, 28 § π	Table 2 (p375)	Multivariate
Malmö ¹⁶	30, 32, 28 § **	Table 3	
	50	Table 3 (p102)	Multivariate

* Age 30–49, 50–59, 60–69 years respectively.

† Sum index and mean index respectively.

‡ Total and cardiovascular disease mortality respectively.

§ Total, cardiovascular, and ischaemic heart disease mortality respectively.

π All men.

** Men without prevalent disease at first examination.

Discussion

CAUSALITY

As causality is crucial in interpreting the aetiological fraction, some objections to the assumed causality should be examined.

The observed associations might as well be interpreted as being the result of a "reversed" causal relationship: that ill health leads to social isolation (supported by the Japanese data²³)

In theory this objection is correct, and in all studies on white, middle aged men an attempt has been made to measure and control for the "prevalent disease at baseline". In none of the studies has it been possible to nullify the observed association. This may be the result of insufficient or imprecise measurement of prevalent disease at baseline in each individual study, as some put emphasis on perceived health,¹² others on medical examination,¹³ etc. Because of the persistence of the controversy, Kaplan *et al* consider the question in detail using three different analytical strategies and conclude that "The findings are not consistent with the hypothesis that the relation is an artifact of prevalent disease".¹⁵

Many of the studies of social network and risk of coronary heart disease had poor measures of potential confounding factors, and could not adjust for them adequately in the analysis.

This is true with regard to the earliest studies. In the studies of Alameda county⁸ and Durham¹⁰ the cardiovascular risk factors, blood pressure and serum cholesterol, were not even measured and in the Tecumseh study⁹ smoking status was not determined. In later studies^{13 15 16} these variables were always included in the analyses. Furthermore, in two reports^{8 14} the potential confounding factors were controlled for one (or two) at a time using Mantel-Haentzel techniques, thereby not assuring full and simultaneous control of all potential confounding factors. In most studies, however, a proper multivariate analysis was performed including all the potential confounders measured. In the study by Kaplan *et al*¹⁵ more than 10 potential confounding factors are considered, and they conclude that, "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". Finally, intermediate risk factors should not be controlled for in the analyses.

Social network is not an "independent" risk factor for coronary heart disease, and "independent" action is not supported by any of the original studies.

As noted above the earliest studies did not have a design that allowed them to determine whether social network was an independent risk factor or not. This problem, however, has been solved in later papers.

Selecting studies of white men leads to the exclusion of an important negative study by Reed et al.²³

As the aim was to make an estimate relevant for Danish men, the most appropriate source of information is research from neighbouring countries or from countries similar in culture. The Scandinavian studies and the American results regarding men of Northwest European origin are consistently positive. The results are somewhat contradictory concerning black men, and the single Japanese study is negative. A socio-geographic trend seems to be present and does not nullify the observation among white men.

The hypothesis is not biologically plausible even though it is epidemiologically persistent.

This objection resembles that when the relationship between smoking and coronary heart disease was presented to the Surgeon General in 1964. A persistent epidemiological pattern was evident but no clear cut biological explanation could be given.

No commonly accepted rules exist that enable us to determine with certainty whether an observed relationship should be considered causal or not. How many possible confounders have to be included? How well have all the more or less well established risk factors to be measured to avoid any uncontrolled confounding? Is randomisation crucial? Are observational studies conducted under more realistic circumstances a *sine qua non*? All these are a matter of judgement. It is always possible to claim the existence of some unmeasured and unknown "third" variable, which may be the real cause and which accounts for the entire association observed, or that the confounders actually controlled are too poorly measured. On the other hand the simple, face value explanation sometimes may be much more likely than a complicated indirect one.

ESTIMATION PROCEDURE

Aetiological fractions are usually estimated by a formula that includes the exposure prevalence in the studied population and a relative risk. To achieve a sound estimate, this method precludes a measurement of "exposure" prevalence (here, prevalence of poor social network) in the target population that is in accordance with the measurements of the same variable in the published reports. No consensus has been reached regarding the proper measurement of social network, so the approach is applicable only to the populations within each individual study. As the operationalisation of social network varies among studies, the prevalences of "poor" social network are incomparable among studies and so are the related relative risks.

To avoid these problems, the following approach was followed. The levels of exposure within each study were placed in order, and a graph was constructed (the graphs in the fig) in which the x axis was proportional to the percentages of the population ordered from left to right, with those with the best social network ("no exposure") to the left and those with the poorest social network to the right. On the y axis the related relative risks were marked. This way of presenting the graphs enables us to compare the studies better. The figures confirm a slowly increasing risk from left to right, perhaps with a small high risk group to the extreme right.

Furthermore, the proportional construction of the x axis implies that the total area below the curve represents the total disease burden in the population, and the area between the curve and the horizontal line $RR=1$ (that is, the excess risk), represents the aetiological fraction. This kind of standardisation makes yet another visual comparison among the various studies possible. Is the main contribution to the aetiological fraction from a small subpopulation with a greatly increased risk or is it from a larger subpopulation with an only slightly increased risk? The graph does not give a consistent answer.

INTERPRETATION

If the causality is considered uncertain, the calculated aetiological fraction is an indicator of the possible size of the impact on public health. A large aetiological fraction motivates further research to clarify whether the observed associations are causal or not. If the causality is considered to be reasonably well established, then a large aetiological fraction motivates further clarification of the concept of social network, a translation into preventive action, and a discussion of its relation to other cardiovascular risk factors.

Although the social network is measured differently in all studies, the similarities with respect to both operationalisation and results are large. This hints at a robustness in the concept among white middle-aged men in spite of its status as an ill defined cultural variable.

The aetiological fraction is defined in terms of a hypothetical situation ("if the risk factor had not occurred"), so caution is needed in interpretation of the estimate and in translation into preventive potential. Almost no attention has been paid to social network as a dependent variable,¹ it is not obvious in which ways the optimal levels of the risk factor could be achieved, and whether this is possible at all in a population which is at present middle aged. Nevertheless the fraction is informative. It provides information on the aetiology of the disease in the actual population as well as on the best that can be achieved by a change of habit or living conditions in the next generation; but it does not indicate whether this change can be easily obtained.

Aetiological fractions for some of the traditional cardiovascular risk factors are listed for comparison. The estimates are based on samples which are not representative for the countries, but relate to middle aged Swedish men from Gothenburg²⁴ and US college students respectively.²⁵ Aetiological fractions were as follows: hypertension 17% and 9% respectively; smoking 39% and 25%; parental history 12% and 11%; and serum cholesterol (in the Swedish study only) 38%. The reported fractions are probably biased downwards, but a careful analysis of the risk factors from the viewpoint of this paper remains to be undertaken. In an earlier study we considered risk factors in the work place.³ From that report we cite the following aetiological fractions: passive smoking 2%, monotonous, high paced work 7%, and sedentary work 42%.

These three studies^{3 24 25} note that the sum of aetiological fractions may exceed 100%, and this is in fact the case in the Swedish study. In addition,

theoretical studies^{20 26} point out that the sum of the aetiological fractions may exceed 100%. Some conceptual, preventive, and scientific interpretations of this fact has been discussed previously.³

CONCLUSION

The original reports and the summary by House *et al* show that social network is an independent risk factor and the present study shows that it is an important, cardiovascular risk factor among white, middle aged men, with a large impact on mortality. The impact is comparable in size with that of the traditional cardiovascular risk factors. Social network should receive more attention in cardiovascular epidemiological research in the future. Its active components have to be determined and the sources of a strong social network need to be found. Strategies applicable to preventive work should be developed.

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