Social Networks Are Associated With Lower Mortality Rates Among Women With Suspected Coronary Disease: The National Heart, Lung, and Blood Institute-Sponsored Women's Ischemia Syndrome Evaluation Study

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Objective: To examine the association between social relationships measured by the Social Network Scale and coronary artery disease (CAD) risk and mortality among a sample of women with suspected CAD. **Methods:** Five hundred three women (mean age, 59 years) with suspected CAD warranting clinical investigation completed a diagnostic protocol including psychosocial testing, CAD risk factor assessment, and quantitative coronary angiography. Patients were subsequently followed for a mean of 2.3 years to track all-cause mortality. **Results:** Women reporting higher social network scores showed a consistent pattern of reduced coronary artery disease risk, including lower blood glucose levels ($r = -0.11$; $p = .03$), lower smoking rates (odds ratio [OR] = 0.81; 95% confidence interval [CI] = 0.71–0.93; $p = .002$), lower waist-hip ratios ($r = -0.18$; $p < .01$), and lower rates of hypertension (OR = 0.90; 95% CI = 0.81–0.99; $p = .04$) and diabetes (OR = 0.83; 95% CI = 0.73–0.94; $p = .004$). Based on quantitative angiogram findings, high social network scorers also had less severe CAD (mean angiogram stenosis value, 40.8 vs. 27.2 for low and high scoring social network groups, respectively; $p < .001$). Finally, mortality rates over follow-up showed a dose-response pattern in relation to quartile scorers on the Social Network Index, with low scorers showing more than twice the death rate of high scorers (relative risk = 2.4; $p = .03$). **Conclusions:** Among a cohort of women with suspected CAD, smaller social circles were associated with increased CAD risk factors and mortality, an effect that appeared to be explained largely by income level. The findings extend previous studies of social network effects on health by highlighting risk among women with suspected CAD, and suggest mechanisms for further study. **Key words:** coronary artery disease, mortality, women, social networks.

CAD = coronary artery disease; **WISE** = Women's Ischemia Syndrome Evaluation; **SES** = socioeconomic status; **SNI** = Social Network Index; **CI** = confidence interval; **RR** = relative risk.

INTRODUCTION

Measures of social connectedness—most often referred to as *social networks*—have established themselves among the most robust psychosocial predictors of mortality and morbidity (1–3). In total, more than a dozen prospective studies completed between 1970 and 2000 offer evidence in favor of relationships between social networks and the incidence of major health indicators including mortality, cardiovascular disease, dementia, and viral infections (4–7). Researchers speculate that the presence of larger social networks may moderate disease risk by reducing certain negative health behaviors such as smoking, by directly or indirectly improving immune system responses, by offering socioeconomic benefits, and by providing greater social support to reduce emotional distress, among other theories (8–10). At present, however, there is limited empirical evidence for these mechanism theories—particularly among women—and the identification of potential biological and behavioral pathways for social network benefits remains an important objective.

Although conceptually related, social networks are distinct from measures of social support (11,12). Items from social support questionnaires assess a largely qualitative impression of the practical and emotional support that a respondent perceives is available from others in their social circle. Social network items, in contrast, stress quantitative (ie, how many) aspects of social relationships: number of friends contacted, amount of contact with coworkers and family, frequency of social recreation, and marital status. Among these relationship domains, marriage generally proves to be the most powerful predictor of health outcomes (13), yet combined social network scores typically remain associated with disease and death even after controlling for the presence of a marital partner (5).

We attempted to expand on previous social networks research in the present study by using a validated measure of social networks to predict major coronary artery disease (CAD) risk factors, hospitalizations, and mortality over 2 to 4 years of follow-up in a population of women with suspected myocardial ischemia. Although several previous studies included gender-balanced samples (eg, 2), the proposed health benefits of social relationships for women are less consistent compared with men (3,5), and clinical samples including adequate women representation are virtually absent in this area. Our specific objective was to examine the relationship between social network scores and mortality over the initial follow-up period. Additionally, we hoped to identify behavioral, mood, socioeconomic, and biomedical factors that could

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explain—at least in part—the expected health benefits associated with larger social network standing.

METHODS

Participant Recruitment and Entrance Criteria

The Women's Ischemia Syndrome Evaluation (WISE) is a National Heart, Lung, and Blood Institute-sponsored multicenter study assessing cardiovascular function using state-of-the-art techniques in women referred for coronary angiography to evaluate chest pain or suspected CAD (14). Participants completed a battery of symptom and psychological questionnaires at baseline, along with quantitative coronary angiography, exercise stress testing, and other diagnostic procedures. Presented results are based on a total of 503 women with complete social network measures in addition to CAD risk factor and follow-up data.

Women were eligible for participation in WISE if they were older than 18 years and were referred for a coronary angiogram. Exclusion criteria included current pregnancy, cardiomyopathy, recent myocardial infarction or revascularization procedure (percutaneous transluminal coronary angioplasty [PTCA], coronary artery bypass surgery [CABG]), language barrier preventing questionnaire completion, and a history of congenital heart disease, among other criteria. Recruitment for WISE preceded psychological questionnaire development by approximately 3 months, accounting for the absence of questionnaire data among early participants. The WISE study received institutional review board approval from each participating site, and all participating women provided informed consent.

Measurement of Coronary Artery Disease, Coronary Artery Disease Risk Factors, and Mortality

Coronary disease severity was defined based on the maximum stenosis score identified from each participant's quantitative angiogram results. Each participant was also assigned a continuous CAD severity score based on a modified Gensini index (15).

Women were followed for the tracking of new cardiovascular events, rehospitalizations, and death over a mean 2.3 years of follow-up. Women without a minimum follow-up interval of 60 days were not included in the mortality analyses. For the purpose of the current article, cause of death was not differentiated because of insufficient numbers. Follow-up was conducted by telephone interview at 6 weeks and yearly thereafter. Follow-up consisted of a scripted interview by an experienced nurse or physician. Each patient was queried for the occurrence of hospitalizations and the reason for hospitalizations. Events were defined as hospitalization for unstable angina, myocardial infarction, congestive heart failure, stroke, other vascular events, and death. When a major cardiovascular event was identified, the referring physician was contacted for confirmation, dates, and documentation of the occurrence. In the event of death, a death certificate was obtained.

Major CAD risk factors in the WISE protocol included body mass index, smoking, diabetes history, and hypertension status. We focused on relationships between social network scores and major CAD risk factors measured as follows: a) smoking status, rated as current smoker/current nonsmoker; b) fasting high-density lipoprotein levels; c) fasting low-density lipoprotein levels; d) hypertension status (positive/negative diagnosis history); e) body mass index (less than/greater than 30.0) and waist-hip ratio scores; and f) diabetes status (positive/negative history) and fasting blood glucose values. Covariates included patient's age, menopausal status (a dichotomous yes/no variable), Beck Depression Inventory symptoms (16), and hormone replacement therapy use at study baseline. All testing was performed in accordance with institutional guidelines.

Socioeconomic Status Measures

As part of the baseline self-report battery, participants responded to questions indicating their current annual household income, employment status, and education history. Income and education history served as primary covariates in this article. For the purpose of statistical testing, we categorized each of the socioeconomic status (SES) markers into dichotomous variables reflecting a) high/low income, participants with an annual income below $20,000 versus those with an annual income exceeding this amount; and b) high/low education, those with less than a high school or general education development (GED) equivalency versus those reporting at least a high school education. We explored other categorization schemes with participant income data; however, the dichotomous ≥$20,000 variable provided the most discriminatory power and was used in all primary analyses.

Social Network Scores

We used participants' responses to the Social Network Index (SNI; 17) to estimate social network size. The SNI collects information on 12 types of social relationships, including friends, coworkers, marital status, close family and children, and participation in volunteer or organizational activities. Scoring of the SNI allows for two estimates: a) a measure of social network diversity based on the presence or absence of each of the 12 relationship domains over a 2-week period, with scores ranging from 0 to 12; and b) a measure of social contact computed by questions allowing participants to state the absolute number of contacts within each relationship domain in the previous 2-week interval. In the latter equation, scores can range from 0 to 168 (17). SNI scores reported herein represent the first scoring method, which most closely parallels the scoring algorithms used with other common social network scales. Where indicated, we used a median and quartile split transformations of the raw social network scores. In some cases, quartile splits did not produce equal sample sizes because of the distribution of scores falling precisely on the quartile values (N = 156, 105, 164, and 78 for quartiles 1–4, respectively). For the WISE sample, the median value fell at a score of 6 on the SNI.

Statistical Analyses

We used a series of hierarchical logistic (in the case of cross-sectional relationships) and Cox (used for prospective models) regression models to assess the following questions: a) Are social network scores associated with CAD risk factors in the WISE sample? b) Are social network scores associated with total mortality over follow-up after adjustment for covariates? c) Do measured CAD risk factors explain the relationship between social network scores and mortality outcomes among WISE participants? We performed simple correlations in the case of continuous CAD risk factor variables (eg, blood glucose values). Mediation and moderation test models were performed in accordance with methods reviewed by Baron and Kenny (18). In the case of observed mediator effects, we further completed regression models that assessed social network effects simultaneously with the covariate terms via forced entrance at the same step.

We also created a second outcome variable that combined mortality with stroke, myocardial infarction, congestive heart failure, or rehospitalization over follow-up. This combined variable produced a substantially larger number of total events and allowed us to examine social network effects on related categories of health outcomes.

Statistical Power

Power calculations for the level 1 regressions exploring associations between social network scores and CAD risk factors were completed based on the observed sample size of 503, with an estimated odds ratio of 2.0 and a two-tailed α set at 0.05. Statistical power for these tests was estimated at >0.90 based on these input values (19).

For the second level survival analyses, the low mortality rate in the sample as a whole tempered the power of these tests. Based on a projected risk ratio of 2.0 for risk of mortality among low vs. high social network participants, the total sample of 503, a total of 30 observed mortality events, and an α of 0.05, power was estimated at 0.71. All survival analyses were conducted as two-tailed tests.

RESULTS

Women's Ischemia Syndrome Evaluation Descriptive Summary

Table 1 summarizes the WISE sample in terms of CAD risk factors, CAD severity, and other demographic variables at baseline. Participants reporting smaller social networks dem-

TABLE 1. Baseline Demographic and Risk Factor Description, Mean (SD), of the Women's Ischemia Syndrome Evaluation Participants (N = 503)

	Low Social Network Group, N = 261	High Social Network Group, N = 242
Age	60.5 (11.3)	57.0 (10.9)*
Body mass index	29.6 (6.5)	28.8 (6.6)
Current smokers (%)	22.6	12.7*
Hypertension (%)	59	51.1*
Diabetic (%)	26.1	14.9**
Postmenopausal (%)	85.1	78.3
Completed high school (%)	72.8	92.8*
Annual income (% >$20,000)	45.5	82.4*
Beck Depression score	12.0 (8.5)	9.0 (7.1)*
Race (black, %/white, %)	17.2/82.8	13.1/86.9
Fasting blood glucose (mg/dl)	124.6 (64)	109.6 (43.3)*
Waist-hip ratio	0.88 (0.12)	0.84 (0.08)*
Mean coronary stenosis value	40.8 (35.3)	27.2 (33.9)*
Low-density lipoprotein cholesterol (mg/dl)	118.0 (42.8)	110.4 (35.2)
High-density lipoprotein cholesterol (mg/dl)	53.4 (12.6)	55.0 (11.7)

*Means differ at $p < .01$.

onstrated a consistent pattern of elevated disease risk factors, as evidenced by significantly higher rates of smoking, diabetes, hypertension, and depression symptoms, higher waist-hip ratios, and lower socioeconomic standing. The low social network group also showed statistically greater prevalence of underlying CAD based on angiogram findings (67% vs. 54% with at least one coronary stenosis value >50%; $p < .01$) relative to participants reporting higher social network scores. After adjusting for current smoking status, diabetes history, income, hypertension history, and waist-hip ratios, the highest quartile of social network scorers showed a 24% lower prevalence of angiographic CAD (defined as >50% stenosis; odds ratio = 0.76; 95% confidence interval [CI] = 0.61–0.95; $p < .02$) relative to the lowest quartile scorers.

Social Network Relationships With Mortality Risk Over Follow-up

We observed a total of 30 deaths (approximately 6% of sample) over an average 2.3 years of follow-up among WISE participants with complete social network scores. As indicated in Figure 1, the pattern of deaths decreased from the lower to upper quartile groups, with the most visually demonstrative differences appearing in quartiles 3 and 4. The lowest quartile scorers (N = 156; 12 events; mortality rate, 8%) showed a mortality rate exceeding 3 times that seen among the highest scoring social network quartile (N = 78; 2 events; mortality rate, 2.5%). Overall, each point increase on the SNI was associated with a 19% decrease in prospective mortality risk over follow-up (relative risk [RR] = 0.81; 95% CI = 0.66–0.99; two-tailed $p = .05$).

We examined mortality predictors after adjusting for age in separate models. Among these variables, baseline smoking status (RR = 2.0; 95% CI = 1.1–3.8, $p = .02$), self-reported income (RR for annual income >$20,000 = 0.48; 95% CI = 0.26–0.90; $p = .02$), depression symptoms (RR = 1.05; 95% CI = 1.0–1.1; $p = .02$), and social network scores (RR = 0.80; 95% CI = 0.65–0.98; $p = .04$) remained statistically reliable predictors of mortality after age adjustment. Low social network scores continued to be associated with an increased mortality risk after simultaneously controlling for differences in depression history, smoking status, and CAD severity in a single regression model, with covariate-adjusted results indicating a 2.4-fold increase in mortality relative for low vs. high scoring SNI participants (RR = 2.4; 95% CI = 1.04–5.4; $p = .03$). Marital status also predicted mortality among WISE participants (RR = 2.5 for unmarried relative to married participants; 95% CI = 1.2–5.2; $p = .01$). However,

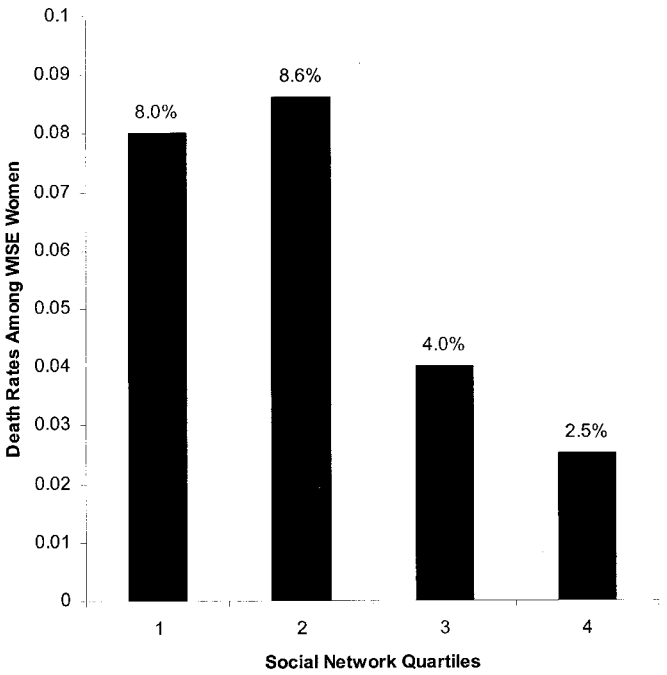

Figure 1. Mortality rates among Women's Ischemia Syndrome Evaluation subjects across quartiles of the Social Network Index (higher quartiles = higher social network scores).

social network scores remained predictive after controlling for the marital status item of the SNI (RR = 2.6; 95% CI = 1.1–6.0; $p = .02$).

Examining age-adjusted social network benefits across alternative health event categories was also suggestive of favorable outcomes. Higher social network scores were associated with lower total events (based on combined mortality, hospitalization, myocardial infarction, stroke, and congestive heart failure events over follow-up; RR = 0.85; 95% CI = 0.75–0.96; $p = .009$) and with lower rates of rehospitalization over follow-up (RR = 0.87; 95% CI = 0.77–0.99; $p = .03$).

Mediating or Moderating Effects of Covariates

A secondary objective of our analyses was to identify factors that explained, or mediated, the risk associated with smaller social networks. For a variable to function as a mediator, a) it must be related to the primary predictor (ie, social network scores), b) it must be related to the primary outcome variable (ie, mortality), and c) the presence of the mediator in the statistical model must eliminate or substantially reduce the effects of the primary predictor on the outcome variable (18). Based on these criteria, many of the demographic and CAD risk factors described in Table 1 met the initial assumption based on relationships with social network scores; however, only a handful (depression, smoking status, and income) demonstrated independent associations with mortality. Controlling for smoking status and depression severity did not change the magnitude of the social network relationship with mortality.

In contrast, Table 2 illustrates that inclusion of annual income eliminated the statistical significance of social networks as a predictor of mortality and, of equal importance, dramatically reduced the magnitude of effect (change in RR from 2.5 to 1.3), a result consistent with the interpretation of statistical mediation. Participants with smaller social networks were several times more likely to report an annual income below $20,000 ($r = 0.43$; $p < .001$ between social network scores and income level), and low income dramatically increased the risk of mortality over the 2.3-year follow-up interval. Although social network effects decreased after adjustment for income level, the reverse was not true. Low income level remained a reliable predictor of mortality with (RR = 2.6; 95% CI = 1.0–7.2; $p = .05$) or without (RR = 2.5; 95% CI = 1.1–6.0; $p = .03$) adjustment for social network scores.

The same pattern held true when assessing combined income and social network effects for the total events and rehospitalization statistics. After adjustment for income, social network scores no longer predicted either total events (RR = 0.95; 95% CI = 0.82–1.1; $p = .5$) or rehospitalization (RR = 0.96; 95% CI = 0.83–1.1; $p = .6$), whereas income status remained a reliable predictor of both outcome categories (RR = 0.68 and 0.72; 95% CI = 0.50–0.93 and 0.52–0.99; $p = .02$ and .04 for total events and rehospitalization, respectively).

Our examination of income as a potential moderator of social network effects was not as fruitful. Inclusion of a social network by income interaction term in the survival analysis models failed to provide further explanatory power in the prediction of mortality ($p > .5$), indicating that social network and income were better treated as separate risk factors. Although we did observe differences in the magnitude of social networks effects between low and high income participant groups (social network RR = 0.76 vs. 2.0 for low-income and high-income groups, respectively), results in these separate groups did not approach significance (p values $>.25$).

DISCUSSION

Although many previous studies have linked social isolation to an increased mortality risk (2,5,8–10), the current study is among the few to identify a relationship in a clinical sample of women presenting with suspected CAD (20,21). The unusual characteristics of women participating in WISE—signs and/or symptoms characteristic of CAD, elevated CAD risk factor status, and comparatively high levels of psychosocial distress—combined with the extensive battery of physical and psychological evaluations each participant completed, make the results distinct in many ways from previous efforts in this area (14).

TABLE 2. Results of Regression Models Exploring the Statistical Mediation of Social Network Effects

Level 1. Prediction of Mortality Outcomes Adjusted for Age, Depression, Smoking Status, and Atherosclerosis Severity			
Predictor	Relative Risk	95% CI	*p*
Social network (median split)	2.4	1.04–5.4	.03
Income (median split)	2.5	1.1–6.0	.03
Level 2. Association Between Social Network Standing and Income (<$20,000/yr)			
Predictor	Odds Ratio	95% CI	*p*
Social network (median split)	5.6	3.6–8.7	<.001
Level 3. Association Between Social Network Standing and Mortality After Adjusting for Age, Smoking Status, Atherosclerosis Severity, and Income Level			
Predictor	Relative Risk	95% CI	*p*
Social network (median split)	1.3	0.47–3.4	.6

CI = Confidence interval.

Despite these clinical and methodological differences, however, our primary findings were consistent with those from previous studies. Women reporting comparatively smaller networks of social relationships and comparatively higher levels of social isolation were at significantly increased risk of death at follow-up. Our findings suggested that socially isolated women were more than twice as likely to die over this interval relative to those reporting the largest social networks. This effect was robust to standard covariates such as age, depression, and angiographically defined CAD. Furthermore, the overall magnitude of effect rivaled or exceeded that of more commonly considered biomedical risk factors including smoking, diabetes, and hypertension histories. The results also held true on examination of social network scores with combined categories of health events and rehospitalization. We believe these results supplement the growing literature on social relationships and health by extending the risk associated with social isolation to a clinical sample of women among whom rates of verified heart disease and psychological distress were high compared with typical epidemiological samples.

Mediator Effects

We attempted to identify process variables for social network status in WISE using established procedures for testing statistical moderation and mediation (18). Mediators, by definition, represent explanatory variables, or factors that account for the effects of an independent variable on an associated outcome. Because proposed mediators must be independently related both to the predictor and outcome variables, we could consider only a handful of covariates—smoking, depression severity, and income—in this role. In addition, mediators are typically viewed as consequences of the predictor, in our case a product of social isolation.

From our list of candidates, both higher smoking rates and increased depression scores are conditions likely to result, or be promoted by, a limited network of social relationships. In contrast, lower income levels are at least as likely to precede social isolation as to function as a consequence. For this reason, we examined income in both a role as potential moderator (ie, interaction variable) of social network effects and a mediator role. Perhaps because of the loss of power in our moderator tests when assessing mortality rates across income subgroups, our results were inconsistent with the model that social network effects would be stronger at different levels of income. Instead, the best fitting interpretation was that social isolation effects in WISE were largely explained by income status. Women in the United States are at substantially greater risk of living in poverty relative to men but generally report larger and more diverse social networks. Although several previous mixed gender studies have found social isolation effects even after controlling for SES variables (5,20), it is arguable that our results can be partly explained by the greater prevalence and effect of low income in the exclusively female WISE sample. However, this account is in disagreement with at least one study reporting SES-robust social isolation effects in a similarly sized cohort of women—despite an income distribution paralleling that observed in WISE (21)—and warrants further inquiry.

The pathways linking social relationships and SES variables to physical health status are numerous and interrelated, with evidence supporting the involvement of factors across cultural, societal, interpersonal, and psychological levels (22,23). Disentangling this network of effects will likely require a series of large-scale prospective studies designed a priori to include measures of theory-identified mechanisms. Social network and SES measures were tertiary factors in the WISE protocol, included primarily to aid in the understanding of psychosocial factors affecting the CAD diagnosis process in women. For this reason, we do not have information concerning factors such as health care utilization, SES history, temporal changes in participants' social networks over the course of the study, or measures of qualitative relationship dimensions such as social support, which could improve our understanding of the reported relationships.

In spite of the statistical support for a mediating effect of income on social network effects in WISE, it would be unwarranted from these results to suggest that the solution to social isolation consists of financial handouts. The presence of either social isolation or low SES likely increases the risk of the other, and both conditions could arguably be a result of the symptoms and physical disability that initially supported their participation in WISE. Low SES and social network standing may also be at least temporarily produced by events such as injury, loss of spouse to divorce or death, and physical relocation. For this reason, interventions that improve quality of life or symptom severity may enable women to pursue vocational or social relationships to a greater degree, whereas community-level interventions that assist with job skills training or vocational placement with middle-aged and older women may offer carryover health benefits through improvements in income and social support. From any of these perspectives, our findings encourage the consideration by medical professionals of social and SES factors in the assessment and treatment of women with suspected CAD, because each may affect patients' subsequent health as powerfully as standard CAD risk factors, based on the results demonstrated in this sample.

Social Networks and Coronary Artery Disease Severity

Last, an unexpected finding in our analyses was that patients reporting smaller social networks demonstrated greater evidence of CAD on angiogram. A defining feature of WISE is that all participants had signs and symptoms suggestive of CAD and of sufficient severity to warrant an invasive coronary angiogram procedure. The objective of this methodology was to evaluate participants who were in almost all ways identical to the kinds of patients routinely evaluated by cardiologists, with the aim of improving the specificity and sensitivity of diagnostic examinations among symptomatic women (14). Typically, psychosocial factors are not consid-

ered by cardiologists in their evaluation of patients' symptom presentations, although previously published data from WISE indicate that psychological factors exert a powerful influence on physical symptom reporting and can assist in the prediction of CAD status (24). Perhaps the simplest interpretation of the association between social network scores and CAD severity is that greater disease—and, consequently, greater physical disability—was a contributing cause of social isolation. Combined with the observation that low social network participants showed greater CAD risk factor profiles and higher levels of distress in the form of depression symptoms, the reader may be disposed to the perspective that social isolation was merely a tertiary product of these more established predictors of morbidity and mortality. However, this perspective would ignore the result that, among the major CAD risk factors, only smoking was linked to mortality outcomes, and that social isolation was a stable predictor even after controlling for risk factors. A previous paper showed that SES effects remained after controlling for the availability of health insurance (25).

Unlike the mortality findings, social network relationships with CAD severity were robust to income adjustments. In interpreting these differences, it is important to stress that relationships with CAD severity were cross-sectional findings established at baseline testing, whereas associations with health events were prospective outcomes assessed over follow-up. Based on previously documented links between psychosocial characteristics, cardiac symptom presentation, and underlying disease severity among WISE participants at baseline testing (24), it is clear that psychological distress increased the reporting of physical and mental health symptoms in this sample. This relationship makes it more likely that distressed women would be referred for study participation and angiography testing, and increases the potential for positive ischemic tests results that are based on subjective angina reports. Although these selection and reporting biases are probably equally evident in routine cardiac evaluations with women outside WISE, they complicate comparisons between cross-sectional and longitudinal relationships because they likely have different effects depending on the variable and phase of the study. Although it appears inconsistent that social networks would be a robust predictor of CAD severity, but not mortality after income adjustments, it is also true that CAD severity itself was not a reliable predictor of survival outcomes in WISE. Further, social network scores were significant predictors of CAD severity, mortality, and rehospitalization after controlling for age and standard CAD risk factors, suggesting that this variable had widespread effects on the health status of WISE participants.

Study Limitations

The methodological and demographic characteristics of the WISE sample that distinguish our findings from many previous social network studies also make it difficult for us to generalize the results to groups of healthy women, or even to symptomatic male populations. Similarly, our follow-up interval (2.3 years) remains relatively brief at the time of this reporting, and the low death rate prevented us from examining outcomes beyond the global category of all-cause mortality. Although recruitment for WISE was intended to be representative of women undergoing CAD testing, the high levels of anxiety and depression symptoms reported in the sample suggest that the referral process may have been biased by psychological characteristics (eg, perhaps more isolated women were referred later or delayed seeking medical care, contributing to their greater mortality risk). The extent to which this affected the pattern of results is unclear.

Equally important, although we were able to explore connections among social networks, depression symptoms, and CAD-relevant health behaviors as possible mechanisms for increased mortality risk among socially isolated participants, we did not include measures of social support or assess possible stress-buffering properties of social relationships among WISE participants (26). Some of the most recent trends in social health research include studies attempting to identify factors that explain the apparent benefits of social relationships. Findings such as decreased pain perception in the presence of a supportive other (27), enhanced adrenergic receptor activity among participants reporting low social support (28), and smaller stress-induced cardiovascular reactivity patterns among more supported New York City traffic agents (29) are examples of current research offering insight into the processes by which social relationships may directly affect risk of disease and death. To date, unfortunately, no study has included such factors in the context of a prospective study tracking disease or mortality incidence.

Finally, the WISE protocol did not include follow-up assessments to track possible changes in women's social networks. As the length of follow-up increases, such changes become increasingly probable, and they could have a potential bearing on the predictive value of baseline measures. Efforts to track social network changes are likely most important in studies with follow-up intervals ranging several years or more and those dealing with older cohorts among whom loss of friends, spouses, and family to disease becomes more likely.

CONCLUSIONS

Among a sample of women with suspected CAD, smaller social networks were associated with higher rates of CAD risk factors, greater CAD severity based on quantitative angiogram findings, and an age-adjusted and disease-adjusted 2.4-fold risk of mortality over an average follow-up interval of 2.3 years, compared with those with the largest social circles. Although social network effects were robust to medical covariates and depression severity, secondary analyses suggested that the effects of social isolation could be explained, to a significant degree, on the basis of lower income levels. These results reinforce the importance of evaluating psychosocial characteristics among at-risk samples of women, and they improve our understanding of social network variables by highlighting factors that may be partially responsible for their deleterious associations with morbidity and mortality.

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