Perceived Social Support as a Predictor of Mortality in Coronary Patients: Effects of Smoking, Sedentary Behavior, and Depressive Symptoms

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Objective: Numerous studies have shown network assessments of social contact predict mortality in patients with coronary artery disease (CAD). Fewer studies have demonstrated an association between perceived social support and longevity in patient samples. It has been suggested that 1 of the mechanisms linking social support with elevated risk for mortality is the association between social support and other risk factors associated with decreased longevity such as smoking, failure to exercise, and depressive symptoms. The present study examined an assessment of perceived support as a predictor of all-cause and CAD mortality and examined the hypothesis that smoking, sedentary behavior, and depressive symptoms may mediate and/or moderate this association. **Methods:** Ratings of social support and the risk factors of smoking, sedentary behavior, and depressive symptoms were examined as predictors of survival in 2711 patients with CAD, and associations between support and these risk factors were assessed. Smoking, sedentary behavior, and depressive symptoms were examined as mediators and/or moderators of the association between social support and mortality. **Results:** Social support, smoking, sedentary behavior, and depressive symptoms were predictors of mortality (p's <.01). Results also indicated that sedentary behavior, but not smoking status or depressive symptoms, may substantially mediate the relationship between support and mortality. No evidence for moderation was found. **Conclusions:** The relation between social support and longevity may be partially accounted for by the association between support and sedentary behavior, health behaviors, mortality.

CAD = coronary artery disease; MOSS = Mediators Of Social Support; ISEL = Interpersonal Support Evaluation List; CHF = congestive heart failure; HR = hazard ratios.

INTRODUCTION

Research examining the association between social support and longevity has focused primarily on 2 methods of assessing support. One method concerns the size of an individual's social network and frequency of social contact, and another focuses on perceptions of the quality of support available and received. A number of studies have demonstrated an association between network support and mortality in patients with coronary artery disease (CAD) (1–5), and 2 studies have shown that a 1- or 2-item assessment of perceived support predicts longevity (6,7). Only 1 study has reported an association between perceptions of support and mortality in patients with CAD using a validated instrument designed to assess social support (8).

Smoking behavior (9), a sedentary lifestyle (10), and the presence of depressive symptoms (11) are psychosocial factors that also predict longevity in patients with CAD. Individuals with lower levels of social support are more likely to smoke (12,13), are less likely to engage in exercise (14), and are more likely to be depressed (15). Thus, it has been suggested that the relationship between social support and these risk factors may help account for the association between social support and mortality (16). To our knowledge, however, there are no prospective studies that have examined these risk factors as mediators of the relation between mortality and

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support, perhaps in part as a result of the fact that large samples are required to explore such effects, and many studies in this area have been conducted on small samples with a limited number of events.

Although somewhat more speculative, it is also possible that these risk factors may moderate the association between support and mortality. For individuals who smoke heavily, and or engage in little or no physical activity, social support may have little influence on mortality. Whereas individuals who are moderate or light smokers, or who maintain a moderate level of physical activity, it is possible that social influences may be more capable of having a positive influence.

Similarly, regarding the interaction between depression and social support, it is plausible that for high levels of depression, social support may offer little in the way of help regarding depressive symptoms and thus mortality. However, moderate to milder levels of depression may be amenable to the positive influences of support. Conversely, it is also quite possible that social support may offer little benefit to people who are not depressed.

The present study examined perceptions of social support as a predictor of mortality in a sample of 2711 patients with CAD. Social support ratings, repeated assessments of smoking and sedentary behavior, and depressive symptoms were initially examined separately as predictors of survival. Moreover, the present study used repeated measures of the behavioral risk factors of smoking and sedentary behavior to take into account the duration of the behaviors and changes that may have transpired after diagnosis. Support ratings were then used to predict patterns of smoking and sedentary behavior, and symptoms of depression. Finally, these risk factors were examined as mediators and/or moderators of the association between social support and mortality.

METHODS Patient Population

The data were gathered as part of a prospective cohort study, Mediators Of Social Support (MOSS) (17,18). MOSS was designed to explore the effect of

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SOCIAL SUPPORT AND MORTALITY

social support on mortality and other health outcomes (eg, activities of daily living, psychosocial well-being) in cardiac patients and to examine potential mediators of those relationships. Patients with CAD without a history of revascularization who were referred to the Duke University Cardiac Catheterization Laboratory between July 1992 and January 1996 were approached regarding potential participation in the MOSS study. Eligible patients were those who were referred to Duke University Medical Center for diagnostic cardiac catheterization and found to have significant CAD (\geq 75% stenosis of at least 1 coronary artery).

Patients were excluded from the study if they had any of the following: prior angioplasty, congenital heart disease, primary valvular heart disease, substance dependence, history of impairing psychologic disorder, or an inability to give informed consent.

A member of the MOSS research group obtained informed consent from all qualified patients who agreed to participate. At baseline, before discharge, patients enrolled in MOSS were given a questionnaire battery designed to assess social support and psychosocial risk factors related to CAD. Patients were contacted by phone at 3 months, 6 months, and 1 year after enrollment for interviews assessing the risk factor behaviors of smoking and exercise.

At baseline, 2911 patients had complete data for social support, smoking, exercise, and medical covariates. Of these patients, 2711 had 1 or more follow-up assessments of smoking and/or exercise behavior, and these participants comprise the present sample. The 200 patients with missing follow-up data were significantly more likely to have lower social support ratings and to be sedentary at baseline but were not more likely to smoke. In addition, to reduce participant burden, exercise behavior at 1 year was assessed in only a random half of the sample. This afforded the opportunity to assess different psychosocial constructs in the remaining participants. The random half who did not receive assessment of exercise behavior at 1 year were not significantly different from those that did with respect to social support, exercise, and smoking behavior at baseline, and they were no different with respect to their exercise or smoking behavior at the 3- and 6-month follow ups. Assessment of depressive symptoms was incomplete for 92 patients. These patients did not differ from the full sample with respect to social support ratings or smoking behavior, but they were somewhat less likely to engage in exercise.

Patients were followed for survival status up to 11.1 years, with a mean of 7.3 years. There were 964 total deaths (35.6%), of which approximately half (511) were classified as cardiac related. Table 1 presents the characteristics of the sample.

Measures Social Support

The Interpersonal Support Evaluation List (ISEL) (19) was used to assess perceptions of social support. The ISEL consists of 40 items that assess the following dimensions of support: appraisal, self-esteem, belonging, and tangible. Internal reliability (alpha coefficient) has been reported to range between 0.88 and 0.90; 6-month test-retest reliability has been found to be 0.74 (19). A shortened 16-item version of the ISEL was used to limit patient burden (17,20). Items were rated on a 4-point scale, with a potential range of 0 to 48 for total ISEL scores. Higher scores reflect greater perceived support.

Smoking and Exercise

Current smoking status (yes/no) was assessed at baseline and follow up. Similarly, at baseline and follow up, patients were asked about their exercise behavior, and for the present study, patients who reported no weekly exercise were coded as sedentary.

Patterns of behavior for smoking and exercise over baseline and follow up were coded such that higher values reflect the type of activity that places an individual at risk (ie, continuing to smoke and remaining sedentary). Thus, smoking behavior patterns were coded as follows: 2 = patient reported smoking at baseline and every available follow up; 1 = patient reported a mixed pattern of behavior, eg, reported smoking at baseline, reported not smoking at 1e follow up and reported smoking again at another follow up, and; 0 = did not report smoking at baseline nor at any follow up. Similar coding rules were used for sedentary behavior. The numbers of patients in each smoking pattern group were 2 = 220 (8.1%), 1 = 497 (18.3%), and 0 = 1994 (73.6%). Similarly, for exercise groups, the numbers of patients were 2 = 688 (25.4%), 1 = 1344 (49.6%), and 0 = 679 (25.0%).

Repeated assessment of smoking was available on the following percentages of patients at successive follow ups: 81.4%, 93.8%, and 87.1%. Similarly, the follow-up percentages for exercise were: 80.2%, 92.5%, and 92.3% (1-year rate based on random-half selected to receive exercise follow up).

Symptoms of Depression

Depressive symptoms were assessed at baseline using the Center for Epidemiologic Studies Depression Scale (CESD) (21). The CESD is a 20item self-report scale designed to measure depressive symptomatology in a general population. Higher scores represent depressive responses, and a score of 16 or greater is generally considered suggestive of a depressive disorder. Measures of internal consistency for the CES-D are acceptable, with alpha coefficients of 0.85 in a general population and 0.90 in a patient sample. Test–retest correlations range between 0.45 and 0.70 (21).

Disease Severity

Disease severity was controlled with measures representing the number of diseased vessels, left ventricular ejection fraction, and the presence or absence of congestive heart failure (CHF; coded 0, 1). Age in years was also controlled in all analyses.

Characteristic	
Age, mean (years) (SD)	62.4 (10.9)
Male/female	1862 (68.7%)/849 (31.3%)
ISEL Social Support, mean (SD)	38.6 (7.2)
Smoker	553 (20.4)
Sedentary	1624 (59.9%)
CESD depressive symptoms, mean (SD)	14.9 (11.1)
Congestive heart failure	556 (20.5%)
No. of narrowed coronary arteries (75% stenosis)	
1	863 (31.8%)
2	719 (26.5%)
3	1129 (41.7%)
Ejection fraction, mean (SD)	53.0 (15.0)

TABLE 1. Baseline Patient Characteristics (n = 2711)

Note: n for CESD ratings = 2619.

SD = standard deviation; ISEL = Interpersonal Support Evaluation List; CESD = Center for Epidemiologic Studies Depression Scale.

Statistical Analysis

Cox proportional hazards survival analyses were conducted to examine social support, smoking, exercise, and depressive symptoms as predictors of mortality. Next, associations among social support, smoking, exercise measures, depressive symptoms, and mortality were examined to confirm their tenability as mediators (22). ISEL scores were examined as predictors of behavior patterns for smoking and exercise with ordinal logistic regression, controlling for gender, the disease severity constructs listed here, and age. Similarly, ISEL scores were examined as predictors of depressive symptoms using multiple linear regression.

Cox models were then conducted to examine whether smoking, exercise, or depressive symptoms might mediate (or moderate) the relation between social support and longevity. Dummy variables were constructed for the smoking and exercise variables, with the lowest risk group as the referent.

There is an extensive literature on mediation and moderation, although the definitions and criteria or meeting those definitions vary somewhat (22-24). The most typical definition of mediation is that of a variable that represents an intervening or explanatory mechanism in the causal chain between a predictor and outcome. Cast in this light, the prerequisites for mediation are 1) a nonzero relation between the predictor and the outcome; 2) a nonzero relation between the putative mediator and the outcome; and 3) a nonzero relation between the predictor and the mediator. Given these prerequisites, the hypothesis of mediation is tested by entering both the mediator and predictor into a regression-type equation simultaneously. If the relation between the predictor and outcome is diminished substantially with the addition of the mediator to the model, and if the relation between the mediator and outcome remains substantial and statistically significant, the mediation hypothesis is supported. Moderation, in contrast, is generally defined as the effect of a predictor depending on the level or value of a second predictor, in other words, statistical interaction. Moderation is tested by including a productinteraction in a model that already contains the corresponding component main effects. For example, if smoking moderates the relation between social support and survival, we would expect the relation of social support and survival to be different between smokers and nonsmokers.

ISEL scores were modeled as predictors of mortality, and the mediation hypothesis was tested by including measures for smoking (or exercise, or depressive symptoms) in models that included ISEL scores. In the present study, support for mediation would be demonstrated if the regression coefficient for social support was substantially weakened after introduction of the smoking (or exercise, or depressive symptoms) variables into the model. Moderation was tested by evaluating the inclusion of interaction terms between the ISEL scores and the smoking (or exercise, or depressive symptoms) items. Both all-cause and CAD mortality were examined. The following covariates were included in all models: gender, age, CHF, number of diseased vessels, and left ventricular ejection fraction. Hazard ratios (HR) were calculated to reflect a 2-standard deviation difference for all continuous variables.

RESULTS

Associations Among Social Support, Smoking, Exercise, Depressive Symptoms, and Mortality

Social support, smoking, sedentary behavior, and depressive symptoms were all significant predictors of all-cause mortality in separate models, following adjustment for medical and demographic covariates (Table 2). Similarly, support (HR, 0.80; range, 0.68–0.95; p <.01), smoking (moderaterisk group HR, 1.13; 0.88–1.45; *p* <.33); high-risk group HR, 1.47; 1.04–2.08; p < .02), and sedentary behavior (moderaterisk group HR, 1.48; 1.13–1.94; *p* <.01); high-risk group HR, 2.86; 2.16–3.78; p < .01) were also predictors of CAD mortality, adjusted for covariates. The association between depressive symptoms and CAD mortality was marginally significant (HR, 1.20; 0.98–1.43; p < .06). In addition having CHF, an increased number of diseased vessels, a decreased ejection fraction, and being older were each associated with decreased survival (Table 2). These results were similar for CAD mortality (all p's <.01).

Ordinal logistic models showed that higher social support ratings at baseline were associated with a decreased likelihood of a high risk pattern of behavior for smoking, with a difference of 2 standard deviations associated with an odds ratio (OR) of 0.69 (p < .01). Similarly, higher baseline support scores were associated with a pattern of less sedentary behavior (OR, 0.54; p < .01). Linear regression analyses showed

Predictor	(Model 1) Social Support	(Model 2) Smoking	(Model 3) Sedentary Behavior	(Model 4) Depressive Symptoms
Social support Smoking	0.81 (0.72–0.92)†	_	—	_
Moderate-risk group	_	1.42 (1.20–1.69)†	_	_
High-risk group	_	1.77 (1.39–2.25)†	_	_
Sedentary behavior				
Moderate-risk group	_	_	1.49 (1.23–1.81)†	_
High-risk group	_	_	2.96 (2.42-3.62)†	_
Depressive symptoms	_	_	_	1.23 (1.08–1.41)†
Age	1.63 (1.52–1.76)†	1.74 (1.61–1.87)†	1.56 (1.45–1.68)†	1.63 (1.51–1.76)†
Gender	0.91 (0.79–1.04)	0.90 (0.78-1.03)	1.07 (0.93–1.23)	0.91 (0.79–1.05)
No. of diseased vessels	1.26 (1.16–1.37)†	1.28 (1.18–1.39)†	1.23 (1.13–1.33)†	1.27 (1.17–1.38)†
Congestive heart failure	1.77 (1.53–2.05)†	1.82 (1.57–2.11)†	1.63 (1.41–1.89)†	1.77 (1.52–2.06)†
Ejection fraction	0.69 (0.65-0.74)†	0.69 (0.65–0.74)†	0.70 (0.66–0.75)†	0.69 (0.65–0.74)†

TABLE 2. Psychosocial and Behavioral Risk Factors as Predictors of All-Cause Mortality

Note: Values reported are hazard ratio (95% confidence interval) and were standardized to reflect a 2-standard deviation difference for all continuous variables; dummy variables were constructed for follow-up smoking, with the group that did not report smoking at baseline, nor at any follow up, used as the referent group. Moderate-risk group = patient reported a mixed pattern of behavior, eg, reported smoking at baseline, reported not smoking at 1 follow up and reported smoking again at another follow up. High-risk group = patient reported smoking at baseline and all reported follow ups. Gender coded as male = 0, female = 1. * p < .05.

 $[\]dagger p < .01.$

SOCIAL SUPPORT AND MORTALITY

that social support ratings were inversely associated with depressive symptoms (regression weight = -6.9; p < .01).

Gender and all medical covariates were related to follow-up sedentary behavior (all p's <.02). Sedentary patients tended to be female, older, and were more likely to have CHF, a larger number of diseased vessels, and a lower ejection fraction. Age, ejection fraction, and number of diseased vessels were related to smoking behavior during follow up (all p's <.01) such that smokers tended to be younger, have lower ejection fraction, and a larger number of diseased vessels.

Smoking, Exercise, and Depressive Symptoms as Mediators/Moderators

When smoking pattern scores were added to a model that already included ISEL scores, social support remained a significant predictor of all-cause mortality (Table 3). The reduction in the parameter estimate for ISEL scores with smoking patterns included in the model was 0.01 (5%), suggesting that smoking does not mediate the relation between social support and survival. Similar results were found for mortality resulting from CAD, ie, the reduction in the parameter estimate for ISEL scores when smoking patterns were added was 0.01 (5%). Tests for moderation were not significant, suggesting that the effect of social support on all-cause and CAD mortality did not depend on the level of smoking behavior.

Unlike smoking, the inclusion of sedentary behavior substantially reduced the association between ISEL scores and all-cause mortality (Table 3). The reduction in the parameter estimate for social support after adding sedentary behavior to the model was 0.12 (60%), suggesting mediation. Similar results were found for CAD mortality, ie, the reduction in the parameter estimate was 0.11 (50%). Tests for moderation were not significant, suggesting that the effect of social support on mortality did not depend on the level of sedentary behavior. Finally, it should be noted that the effects of the 2 risk behaviors were not substantially altered with the inclusion of the ISEL scores.

When depressive symptoms were included in a model that contained ISEL scores, social support remained a significant predictor of mortality (Table 3). The reduction in the parameter estimate for ISEL scores with depressive symptoms added to the model was 0.04 (19%), suggesting only a modest effect of mediation. Results were consistent for mortality resulting from CAD, ie, the reduction in the parameter estimate for ISEL scores when depressive symptoms were added was 0.03 (14%). Tests for moderation were not significant, suggesting that the effect of social support on all-cause and CAD mortality did not depend on the level of depressive symptoms.

In attempt to assess potential bias resulting from differing patterns of missing data at follow up for smoking and exercise patterns, analyses were repeated in the following subgroups: 1) 2511 patients who had data available for at least 2 of the 3 follow-ups, 2) 2644 patients who survived the final 1-year follow up, and 3) the 1266 random half of patients who did not receive the exercise questionnaire at 1 year. The results were not substantially different from those in the original models. Specifically, smoking did not mediate the association between ISEL scores and mortality, whereas sedentary behavior continued to do so.

DISCUSSION

The current findings suggest that perceptions of social support predict longevity in patients with CAD. In addition, the present results may provide information regarding 1 of the mechanisms underlying this association. Specifically, our findings indicate that sedentary behavior may partially account for the relation between social support and mortality. Moreover, the current findings offer no support for the hy-

	(Model 1)	(Model 2)	(Model 3)
Predictor:	Social Support and Smoking	Social Support and Sedentary Behavior	Social Support and Depressive Symptoms
Social support	0.83 (0.73–0.94)†	0.92 (0.81–1.04)	0.84 (0.74–0.97)†
Smoking			
Moderate-risk group	1.42 (1.19–1.70)†	_	—
High-risk group	1.74 (1.36–2.21)†	_	—
Sedentary behavior			
Moderate-risk group	—	1.49 (1.23–1.80)†	—
High-risk group	_	2.90 (2.37–3.55)†	—
Depressive symptoms	_	_	1.20 (1.02–1.35)*
Age	1.75 (1.62–1.89)†	1.57 (1.46–1.69)†	1.64 (1.52–1.76)†
Gender	0.91 (0.79–1.04)	1.07 (0.93–1.23)	0.92 (0.79–1.06)
No. of diseased vessels	1.27 (1.18–1.39)†	1.23 (1.14–1.34)†	1.27 (1.17–1.38)†
Congestive heart failure	1.79 (1.55–2.08)†	1.62 (1.40–1.88)†	1.76 (1.51–2.04)†
Ejection fraction	0.70 (0.65–0.75)†	0.70 (0.65–0.75)†	0.69 (0.65–0.74)†

TABLE 3. Psychosocial and Behavioral Risk Factors as Mediators of the Relationship Between Social Support and All-Cause Mortality

Note: Values reported are hazard ratio (95% confidence interval) and were standardized to reflect a 2-standard deviation difference for all continuous variables; Dummy variables were constructed for follow-up smoking, with the group that did not report smoking at baseline, nor at any follow up, used as the referent group. Moderate-risk group = patient reported a mixed pattern of behavior, eg, reported smoking at baseline, reported not smoking at 1 follow up, and reported smoking again at another follow up. High-risk group = patient reported smoking at baseline and all reported follow ups. Gender coded as male = 0, female = 1. *p < .05.

 $[\]dagger p < .01.$

potheses that smoking or depressive symptoms account for this association.

Results from two analyses in the present sample support the theory that social support is an important aspect of successful recovery from a cardiac event. First, examination of the association between support and exercise behavior in the present sample indicates that individuals with positive perceptions of their social support are less likely to be sedentary. For example, patients who were active at baseline and all follow ups had social support scores that were over half a standard deviation higher than patients who were sedentary at baseline and remained so throughout the study. Thus, it might be concluded that positive support leads to maintaining an exercise program and/or discordant relationships may deter adherence to exercise regimes. Related research in postcoronary patients has also shown that social support is positively associated with maintenance of weight loss (25). The second, and perhaps more noteworthy finding from the present results suggests that this association between support and physical activity may be 1 of the mechanisms through which social support may increase longevity in cardiac patients. However, it should also be noted that our conclusions regarding the directionality of this finding can only be tentative, that is, lower levels of physical activity may lead to decreases in social support that in turn may negatively affect mortality. Lastly, as in any observational study, other unmeasured variables may account for these findings. For example, social support may be acting as a proxy for a number of variables related to negative affect such as hostility.

As with sedentary behavior, smoking was negatively associated with social support in the present sample. Patients who continued to smoke had social support scores that were approximately two standard deviations lower than those of nonsmokers. However, it should be noted that other studies that have examined the association between support and smoking behavior have yielded negative or opposite findings (25,26). Thus, the association between smoking and support may be more complex than we are able to capture with the present data. For example, the relation between smoking cessation and social support is likely to be partially determined by the smoking status of the individuals who comprise one's support network.

Unlike sedentary behavior, smoking behavior did not substantially alter the relation between social support and mortality in the present sample. Related research inpatient samples has shown that social support is associated with CHD after controlling for smoking (6,27). Finally, we have shown in another sample of patients with CAD that patterns of smoking behavior during hospitalization and recovery account for only a modest amount of the association between depression and mortality (28). Taken as a whole, such results suggest that the association between mortality and psychosocial constructs such as social support and depression are not strongly confounded by smoking.

Smoking was significantly associated with mortality in the present sample and research has shown that smoking cessation

improves the prognosis of cardiac patients (9). Despite these findings, many patients with CAD continue to smoke (29), a fact supported by the present findings, ie, 26% of the sample reported smoking at baseline and/or follow up. Thus, it is important to develop smoking cessation programs tailored toward these patients (30) and to continue investigation of potential psychosocial deterrents to smoking (31).

As demonstrated in prior research (11), symptoms of depression were significantly associated with longevity. Moreover, depressive symptoms only modestly reduced the association between social support and mortality in the present sample. Likewise, depressive symptoms remained an independent predictor of mortality when support was included. That these effects are independent and additive may have important clinical implications, possibly indicating that these psychosocial facets may need to be treated specifically.

Results of the present study also add support to the literature linking sedentary behavior and cardiovascular disease. In the current sample, repeated self-reported patterns of physical inactivity were associated with a nearly three-fold increase in mortality. Although the benefits of exercise with respect to CAD prevention and rehabilitation are currently widely accepted (32), the present results add to this literature by demonstrating a prospective relationship between exercise and mortality that is independent of disease severity and social support.

Apart from smoking, sedentary behavior, and depressive symptoms, other factors associated with social support such as immunologic and neuroendocrine functioning, healthcare utilization, and alcohol consumption have been postulated as potential direct and/or indirect mechanisms that could account for the relation between support and mortality (33). Continued research is necessary to better understand the mechanisms that may underlie the risk associated with lack of social support.

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SOCIAL SUPPORT AND MORTALITY

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