Social Support and Its Relationship to Morbidity and Mortality After Acute Myocardial Infarction

Systematic Overview

Farouk Mookadam, MD, FRCPC, MSc(HRM); Heather M. Arthur, PhD, NFESC

Among the commonly understood socioeconomic determinants of health, social change, disorganization, and poverty have been associated with an increased risk of morbidity and mortality. One of the postulated mechanisms through which these determinants have been linked to health and illness is their relationship to social support. The health determinant, social isolation or lack of a social support network (SSN), and its effects on premature mortality after acute myocardial infarction mandate further scrutiny by the cardiovascular community for several reasons. First, as a predictor of 1-year mortality, low SSN is equivalent to many of the classic risk factors, such as elevated cholesterol level, tobacco use, and hypertension. Second, treatment of acute myocardial infarction is costly. Because low social support is associated with an increased 1-year mortality, neglecting the role of the SSN may diminish the possible gains accrued during acute-phase treatment. Therefore, lack of an SSN should be considered a risk factor for subsequent morbidity and mortality after a myocardial infarction. Finally, cardiac rehabilitation programs and other extant prevention strategies can be better used to reduce mortality after myocardial infarction. This article systematically reviews recent evidence related to SSNs to provide an update on the role of social support in cardiovascular disease–related outcomes.

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Cardiovascular disease (CVD) is a major global health problem, accounting for 40% to 50% of all deaths in industrialized countries and associated with significant morbidity and escalating health care costs. Prevention of CVD, and death due to CVD, has been achieved through the modification of known risk factors, such as hyperlipidemia, diabetes mellitus, smoking, and elevated blood pressure. However, several complex biological, sociological, psychological, and environmental processes may play an important role in the development and progression of atherosclerosis and its associated morbidity and mortality.

Among the commonly understood socioeconomic determinants of health, social change, disorganization, and poverty have been associated with an increased risk of morbidity and mortality. One of the postulated mechanisms through which these determinants have been linked to health and illness is their relationship to social support. At a basic level, social support has been thought to mediate the relationship to illness through its influence on behavioral patterns that either increase or decrease risk for disease or through effects on biological responses. Research has shown that social support may offer protection against the negative health consequences associated with stressful events. For example, the relationship between widowhood and excess mortality is striking, especially in the first year of living alone, and the size of one's social network has been inversely related to mortality, independent of risk factors for heart disease. In addition, depression prevalence among persons with heart disease is as high as 35%, and this group reportedly has poor social support networks (SSNs).
An indication of the association between psychosocial factors and mortality after myocardial infarction (MI) was elucidated 25 years ago in the β-Blocker Heart Attack Trial. The β-Blocker Heart Attack Trial completed psychosocial interviews with 2320 male survivors of acute MI. Results showed that social isolation and high levels of stress were associated with a 4-fold increase in mortality at 3 years when compared with men with low stress levels and low social isolation scores. The excess mortality was noted for all-cause mortality and sudden cardiac death. High levels of social stress and social isolation were independent contributors to excess mortality. While the β-Blocker Heart Attack Trial is salutary in its approach, methodologic concerns preclude generalizability. Patients eligible for β-blockade therapy only were studied; the trial was restricted to men, and the psychological interviews started at 6 weeks, precluding data on patients dying in the first 6 weeks after MI. In this study, depression showed no influence on mortality when other important clinical variables were controlled.

There is disagreement in the scientific community about a precise definition of social support. Indeed, the meaning and significance of social support may vary throughout one’s life. Generally speaking, 3 broad categories are commonly described: social networks, social relationships, and social supports. Social networks refer to several features of a person’s everyday contacts, including size, density, reciprocity, durability, intensity, and frequency. Social relationships refer to the existence, quantity, and type of relationships. Social support refers to the resources provided by others (emotional, functional, and informational) and the quality of those resources. More recently, social support has been conceptualized in 2 domains: social integration and social isolation.

The health determinant, social isolation or lack of an SSN, and its effects on premature mortality after acute MI mandate further scrutiny by the cardiovascular community for several reasons. First, as a predictor of 1-year mortality, a low SSN is equivalent to many of the classic risk factors, such as elevated cholesterol level, tobacco use, and hypertension. Therefore, the absence of social supports should be considered a risk factor for complications after MI. In fact, many of the biochemical markers that mediate the effects of elevated cholesterol level or tobacco use are also elevated in the depressed individual. Second, treatment of acute MI is costly. Because low social support is associated with increased 1-year mortality, neglecting the role of the SSN may diminish the possible gains accrued during acute-phase treatment. Finally, cardiac rehabilitation programs and other extant prevention strategies can be better used to reduce mortality post-MI.

This systematic overview appraises recent evidence related to SSNs to provide an update on the role of social support in CVD-related outcomes. The operational definition of social support is provided by Cohen and includes structural support measures (marital status and number of relationships) and functional support measures that assess the functions of interpersonal relationships (provides affection, having a feeling of belonging, or material aid). The structural index of social ties is termed social integration or the SSN and includes marital status, close family friends, and participation in formal and/or informal group activities.

**SEARCH STRATEGY**

A computerized MEDLINE search (from January 1, 1966, to March 30, 2002) was undertaken to identify English-language studies using the search terms “social isolation” and “social support” combined with epidemiological terms, such as “incidence” and “prognosis,” and the key word for outcome, “myocardial infarction or cardiovascular mortality.” These terms were sought in abstracts, titles, and the text (if they occurred). Forty-five articles were identified. Six were deemed relevant (ie, they were randomized controlled trials or cohort studies showing an association between the determinant and outcome). Of these 6 studies, 5 were analyzed; the sixth had to do with congestive heart failure–related mortality and depression. A search of the CINAHL (Cumulative Index for Nursing and Applied Health Literature) database, between January 1, 1966, and March 30, 2002, was also conducted to review the social science literature. Nine articles were identified, with no new relevant information noted. Emphasis in the CINAHL database seemed to be on the relationship between workplace environment and social support and the role of workplace stress in contributing to the incidence and prevalence of cardiovascular-related morbidity and mortality. The 5 selected articles are summarized.

Depressive symptoms were measured by the Center for Epidemiological Studies Depression Scale. Functional status was measured using the 36-Item Short-Form Health Survey; the Perceived Stress Scale was used to measure stress, and the Cook-Medley Hostility Scale was used to assess cynicism and hostile affect.

Brummet et al conducted a prospective cohort study of a population of patients undergoing coronary angiography; the patients had documented coronary artery disease. A total of 430 subjects were followed up at 3 and 6 months, at 1 year, and annually thereafter for a mean of 47.3 months. By using the Mannheim approach, a comprehensive assessment of social support was undertaken by interview. This study showed that an SSN of fewer than 3 persons was associated with a 2.4 relative risk of excess mortality from cardiac death. Non–cardiac-related mortality showed similar results, with a relative risk of 2.11. There was no sex difference. In general, fewer social supports resulted in social isolation. Socially isolated individuals lacked a person they could identify for psychological support or someone they could speak with in a crisis. They were unmarried, had no confidante, or experienced less satisfaction with their few contacts when they were present. Paradoxically, these patients reported being satisfied with the amount of their social contact despite the fact that their SSN was small. Income and smoking did not predict survival in this group, nor did disease severity index or functional level. The relationship between SSN and mortality was nonlinear, and exhibited a threshold effect.

Berkman and Syme conducted the Alameda County study, which used a stratified systematic sample of 698 adults who were followed up prospectively for 9 years.
Mortality data were collected for 692 individuals. Social contact information was collected from 4 sources, including marriage, friends and relatives, church membership, and formal and informal group association. The main finding was that socially isolated individuals were more likely to die than those with more extensive contacts, with a relative risk of 2.3 for men and 2.8 for women. This was independent of factors such as self-reported health, year of death, social economic status, tobacco consumption, alcohol consumption, obesity, physical activity, and use of preventative services. Furthermore, a dose-response relationship was noted.

Frasure-Smith et al \(^{17}\) conducted a study to examine the interrelationship between baseline depression, as measured by the Beck Depression Inventory (BDI) \(^{18}\) and social support, using the Perceived Social Support Scale. \(^{19}\) This was a prospective cohort study of 877 consecutive patients post-MI. 32% of whom had mild to moderate depression. Depressed patients showed an excess of 1-year cardiac mortality, with an odds ratio of 3.4 \((P = .006)\) when compared with nondepressed patients, even after controlling for 1-year predictors of cardiac mortality, such as age, Killip class, sex, non-Q-wave MI, left ventricular ejection fraction, and tobacco use. No measure of social support showed any relation with cardiac mortality; however, there was a significant inverse interaction between the Perceived Social Support Scale score and depression (ie, at low levels of perceived social support, the impact of depression on mortality was marked). At moderate and high levels of perceived social support, there was no depression-related excess cardiac mortality. Furthermore, a regression analysis showed the impact of social support on 1-year BDI change scores in a dose-response manner.

Berkman et al \(^{20}\) conducted a prospective community-based cohort study of 194 patients older than 65 years who were hospitalized for MI. Detailed information on the Social Network Index, encompassing the SSN and emotional support, was obtained. Depression was measured using the Center for Epidemiological Studies Depression Scale; the severity of MI was measured using clinical criteria and a comorbidity index for concurrent medical conditions. Results showed excess 6-month mortality in patients who lacked emotional and social support, with an odds ratio of 2.9 (95% confidence interval, 1.2-6.9) after controlling for MI severity, comorbid illness, hypertension, smoking, and social demographic factors. Social support assessment preceded hospitalization for MI, and subjects were part of the Epidemiological Study of Elderly Program in New Haven. \(^{21}\) Patients with many social network ties had a lower risk of death.

Case et al \(^{22}\) conducted a prospective, cohort, multicenter Canadian-US study that followed up 1234 early post-MI patients for 1 to 4 years (mean, 2.1 years). Two psychosocial variables, living alone and disrupted marriage, were part of the risk model. Living alone independently predicted mortality, with an odds ratio of 1.5 (95% confidence interval, 1.0-2.9), but disrupted marriage was not a risk factor. The excess mortality associated with living alone showed a graded response, with incidence of cardiac death at 12.4%, 6.6%, and 4.4% for those living alone, those living with 1 person, and those living with more than 2 persons, respectively. The usual risk predictors of morbidity and mortality post-MI (function class, ejection fraction <40%, lower level of education, &-blocker nonuse, premature ventricular contractions, and a history of MI) remained essentially unchanged when living alone was factored into the model or left out, identifying it as an independent hazard of 1.58. This risk hazard is similar to 4 of the 6 risk predictors previously listed. This study did not identify the features of living alone that may be responsible for the excess hazard. It also showed that living with 1 vs 2 people does not provide an incremental mortality reduction. Marriage disruption was not a risk as long as cohabitation continued.

Additional information regarding these 5 articles is available from the authors.

**SUMMARY OF LITERATURE REVIEWED**

Social isolation or lack of an SSN is associated with increased mortality and morbidity, with an odds ratio of 2.0 to 3.0. This excess morbidity and mortality is independent of known predictors of cardiac mortality in the short-term (≤6 months) and long-term (≥6 years) post-MI periods. The usual predictors of premature mortality, such as hypertension, poor cardiac function, cardiac arrhythmia, tobacco consumption, previous MI, age, and female sex, were accounted for by regression analysis in all of the studies. Lack of social support and depression are interrelated in a complex manner. In the 20% to 30% of patients post-MI who are mildly to moderately depressed (BDI score, >10), a strong SSN ameliorates the effects of depression on cardiac mortality. A striking consistency of lack of SSN and its effect on CVD-related mortality lends credibility to its role. The risk apportioned by a lack of SSN is similar to the known risk factors for premature mortality post-MI.

**COMMENT**

Despite 30 years of research related to the role of social support in health and illness, the mechanism/mediator role of social isolation is not clearly identified and more research in this area is still needed. Clearly, there is a complex interaction of determinants that influence biological, social, psychosocial, and behavioral factors.

Social isolation can be anxiety provoking and stressful, thus activating neuroendocrine and physiologic pathways that are likely deleterious. Social support has salutary consequences via the buffering effect from the support itself and from the social control that may be exercised by the health-promoting behaviors of others and the discouraging of negative behavior by health professionals. Social support networks may also interact by tapping into other supportive resources, such as medical referral networks, group therapy, or informational opportunities relating to employment (eg, shopping).

Biological processes that are influenced by lack of social support include neuroendocrine responses, immune responses, and hemodynamic alterations via the renin-angiotensin-aldosterone system, which are known to be injurious to arterial walls and the myocardium itself. The hemodynamic alteration and resultant shear
stress promote coronary artery disease and CVD in general (Figure).

A plausible model of the complexity of the social support disease connection has been proposed by Cohen.9 Further research focusing on behavioral, biological, neuroendocrine, and immunologic mechanisms needs to be undertaken. Research investigating these, if done separately, will be costly. However, an opportunity exists in CVD clinical trials to do such research at only marginal incremental cost if social support questions are built in a priori or conducted as sub-studies.

While there is interdependence between high BDI scores and social isolation by regression analysis, each has been shown to have an independent negative impact on survival.23,24 Based on the BDI, depressed patients were more likely to undergo coronary intervention and had higher rates of complications, including higher re-infarction, heart failure, and recurrent ischemia rates. These findings continue to fuel the controversy between depression at the time of acute MI and mortality. Similarly, Mayou and colleagues25 found that depression did not predict 1-year mortality among 347 patients post-MI.

**RECOMMENDATIONS BASED ON CURRENT EVIDENCE**

Although the literature is conflicting, on balance it seems that the relationship between social isolation and CVD-related mortality is non-linear, with a 2- to 3-fold excess mortality in the most isolated groups and little or no variation in those with moderate to high levels of social support. This implies that deficiency beyond a certain threshold is deleterious to health. Incremental gains in social networks do not enhance health or well-being measurably. Therefore, it is important to assess and, if possible, satisfy the minimal threshold in the most vulnerable isolated group.

To our knowledge, there are no robust data to support the use of a psychosocial intervention for depression in the post-MI period. One frequently cited randomized trial26 of a home-based psychological nursing intervention in distressed post-MI patients failed to show benefit in men and had the propensity for harm among women. This study, however, was underpowered because the anticipated 10% mortality post-MI did not materialize and the actual mortality was only 2.5%. Furthermore, nurses who were involved in the intervention may not have had the requisite training and the frequency of the intervention, which was monthly, may have been insufficient.

There are challenges to devising social arrangements that engender social interaction and promote development of SSNs to reduce morbidity and mortality post-MI. Lavis and Sullivan27 have gone so far as to suggest that national or provincial resources can be manipulated to promote the development of SSNs: they propose the use of income tax to provide infrastructure for social organizations and the use of personal taxation to provide incentives for involvement in social organizations. Alternatively, promotion of social support structures can also be accomplished by using public school property after hours as a venue for social programs to bolster the SSN among isolated individuals. Introducing a buddy system to socially isolated post-MI patients through volunteer and/or publicly funded or subsidized programs may also be beneficial.

Peer support has been a useful adjunct to the multifactorial rehabilitation interventions that are typically offered to patients with cardiac disease. Stewart et al28 tested the effect of a 12-week support intervention for post-MI patients and their spouses. Three types of support were provided: emotional, informational, and affiliational. Participants reported positive effects of the intervention on coping, outlook, confidence, and spousal relationship. Hildingh and Fridlund29 found that the type of patient who attended peer support groups after a cardiac event reported more health problems than nonattenders and scored higher on several dimensions of social support. Arthur et al30 found that a facilitated peer support group for women with heart disease provided an environment that enhanced recovery following discharge from the hospital. Women described the recovery period as terrible. They felt isolated, vulnerable, and confused, and suppressed their emotions within their families and with friends. Family and friends seemed less likely to understand their feelings when compared with their support group peers. The group setting seemed to be more suitable for expressing feelings, helped relieve anxiety, and helped bring suppressed emotions to the surface. The findings from these 2 latter studies suggest that existing social networks may be a necessary, but insufficient, ingredient for long-term adaptation and positive outcomes. In addition, based on the work of Hildingh and Fridlund, a prerequisite for benefit from peer support groups may be the availability, and prior use, of SSNs in other aspects of life.

Rehabilitation provides an opportunity to offer preventive strategies to patients who have a low SSN. One suggestion would be channeling rehabilitation into a more social activity, with a club/membership feature and a buddy system of partnering individuals with a lack of social support with those volunteers who have well-established SSNs.

The fact that an SSN is not routinely integrated into patient assessments by general practitioners implies even less detection by cardiovascular specialists. Therefore, screening for a lack of an SSN with a self-measurement scale may be a useful tool, with implications for mortality reduction that are similar to predischarge stress testing, laboratory variables, and other routinely performed measures. Likewise, screening for depression, which may have an association with lack of an SSN, would require a simple screening tool, such as the BDI. Screening for depres-
tion is a recommendation that was incorporated into the 1995 Canadian Cardiovascular Society Guidelines for acute MI management.31

Several decades of research related to social support have shown a consistent relationship between SSNs and CVD. Recent literature has continued to show this relationship. The challenge remains to plan and evaluate interventions that target either the contributing factors (social network size) or possible mediators (social isolation and social network function) of the relationship. Although scientists have consistently documented the association between CVD and low social support, much more research is required to elucidate strategies for modifying the negative effects of this relationship, particularly at the public health level.

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Correspondence: Heather M. Arthur, PhD, NFESC, McMaster University, Faculty of Health Sciences, 1200 Main St W, Room HSC 2J29, Hamilton, Ontario, Canada L8N 3Z5 (arthurhm@mcmaster.ca).

REFERENCES


