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The Role of Social Relations in Health Promotion

LISA F. BERKMAN, PHD

In considering new paradigms for the prevention and treatment of disease and disability, we need to incorporate ways to promote social support and develop family and community strengths and abilities into our interventions. There is now a substantial body of evidence that indicates that the extent to which social relationships are strong and supportive is related to the health of individuals who live within such social contexts. A review of population-based research on mortality risk over the last 20 years indicates that people who are isolated are at increased mortality risk from a number of causes. More recent studies indicate that social support is particularly related to survival postmyocardial infarction. The pathways that lead from such socioenvironmental exposures to poor health outcomes are likely to be multiple and include behavioral mechanisms and more direct physiologic pathways related to neuroendocrine or immunologic function. For social support to be health promoting, it must provide both a sense of belonging and intimacy and must help people to be more competent and self-efficacious. Acknowledging that health promotion rests on the shoulders not only of individuals but also of their families and communities means that we must commit resources over the next decade to designing, testing, and implementing interventions in this area.

Key words: Social relationships, social support, cardiovascular disease, mortality.

INTRODUCTION

As our nation confronts a health care crisis and as disease, disability, and violence become centered more and more in the poorest, most isolated, and marginal segments of our populations, it is time to consider new paradigms for the prevention and treatment of disease and disability. In considering new preventive efforts, it is important to keep in mind that individuals do not live in a vacuum, rather they are enmeshed in a social environment and in a series of social relationships. There is now a substantial body of evidence that indicates that the extent to which these relationships are strong and supportive and individuals are integrated in their communities is related to the health of the individuals who live within such social contexts.

Almost 20 years ago, epidemiologists who were interested in how social conditions might influence health status began to develop the idea that one of the most important factors that protected people from what seemed to be overwhelming insults, both natural and humanmade, was the extent to which people maintained close personal relationships with others, i.e., the degree to which they were socially integrated into their communities and had deep and abiding social and psychological resources (1–4).

Unlike most epidemiologic research, the hallmark of much of this work from the start was its focus, i.e., not on any specific disease but rather on the degree to which these social conditions influence what was rather loosely termed "host resistance." In other words, in contrast to a host of other conditions and exposures, including psychological stressors, that result in classes of *specific* psychosomatic diseases, this class of experiences seemed to make people more vulnerable to a broad range of diseases and disabilities, which ranged from pregnancy complications and infant health in the early parts of life to disability in old age (1). This idea was defended on the basis of principles articulated in the early part of this century by such scientists as Wade Hampton Frost (5), who in speaking about declining rates of tuberculosis occurring in the first half of the century wrote

"One of the most important factors in the decline of tuberculosis has been progressively increasing human resistance due to environmental improvements such as better nutrition and relief from physical stress tending to raise what may be called *nonspecific resistance*" (5).

To give you an idea of the novelty of the proposition that, first, social factors might be determinants of disease and, second, they might influence *a broad range of disease* outcomes, I would like to recount to you a small part of an exchange that took place during my doctoral comprehensive examinations in 1975. During these tests, candidates were asked to defend dissertation plans and underlying epidemio-

From the Yale University, New Haven, Connecticut.

Address reprint requests to: Dr. Lisa Berkman, Yale University School of Medicine, Department of Epidemiology and Public Health, 60 College Street, New Haven, CT 06520.

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logic theory. The committee came quickly to a discussion of how social networks might influence health status. I offered the opinion that social support or networks influence many disease outcomes because these social conditions influence susceptibility to disease in general. I went on to add some hypothetical pathways involving neuroendocrine regulation and potential immune responses, which in turn might influence both diseases of an infectious nature and cancer and heart disease. At that point, the senior member of this committee, a man distinguished by his identification of viruses associated with major illnesses, proclaimed, "Over the last 150 years of medical research from Pasteur, Koch onward, research has proceeded *successfully* along the lines of identifying one cause of one disease with the theory of disease specificity being one of the major advances in our thinking over the last century." In fact, he correctly recounted, we often *name* diseases after specific etiologic agents once they are identified. He went on to question whether we should ignore this vast body of evidence and return to an era in which we invoke such vague concepts as "social forces" or perhaps "fevers," "miasma," and "consumption" and that furthermore we permit such forces to rule our current models of disease causation.

With more evidence than was available 20 years ago, I would like to respond to this issue today the way I responded to it then with a "yes."

Between 1979 with the Alameda County Study and 1994, there have been eight community-based prospective studies that reveal an association between what we have now come to call "social integration" and mortality rates, usually death from all causes (6–13). Although there are substantial variations among these studies in measurement of social relationships, in types of communities under investigation, and in length of follow-up, they show remarkably consistent results. In almost all cases, those who are most socially isolated and "disconnected" are at increased risk.

In the first of these studies from Alameda County (6), men and women who lacked ties to others (in this case, based on an index assessing contacts with friends and relatives, marital status, and church and group membership) were 1.9 to 3.1 times more likely to die in a 9-year follow-up period from 1965 to 1974 than those who had many more contacts.

The relative risks associated with social isolation were *not* centered in one cause of death; rather, those who lacked social ties were at increased risk of dying from ischemic heart disease (IHD), cerebrovascular and circulatory disease, cancer, and a final category including respiratory, gastrointestinal, and all other causes of death. Clearly, this social condition is not associated exclusively with increased risks from, say, coronary heart disease (CHD).

Although this study opened the field to much work in this area, there were some methodologic problems with it. Perhaps the most important of which was that the Alameda County Study had no clinical assessment of health status and relied exclusively on subject's self-reports of conditions. Thus, it is open to the criticism that prevalent disease might influence the extent to which people could maintain active social relationships and this association might be the underlying reason why isolation is related to mortality risk. Although this is a viable hypothesis, a study of men and women in the North Karelia CHD Study supports the importance of this new social risk factor independently of prevalent CHD or other standard cardiovascular risk factors (9). In this study, men and women were enrolled in a study of cardiovascular diseases. Prevalent cases of CHD were excluded; traditional CHD risk factors, blood pressure, cholesterol level, and other behavioral risk factors were assessed. The percentage of subjects who died from IHD and other causes was significantly associated with the level of social connectedness, a measure similar to the one used in Alameda County. Among men, those who were disconnected were at increased risk of dying of CHD and had an increased total mortality rate. There was no association between social ties and mortality rates among women, but it is important to note that women had very low mortality rates from any cause in this study of middle-aged men and women. The low prevalence of the outcome renders interpretation of risk difficult.

Data from these two studies of what I have called first-generation studies because they often used crude or post hoc measures of social networks are illustrative of the others in Table 1. Overall, they consistently show that people who are isolated are at increased mortality risk from a number of causes. Furthermore, they have several important methodologic strengths, which give us have confidence in the findings, as follows.

1. These studies are *prospective cohort* studies in which ascertainment of social ties is not biased by differential recall.

2. The studies are population based. They are not composed of cohorts of volunteers or small, nonrandom segments of the population.

3. The studies have remarkably few losses to follow-up. In Alameda County, less than 4% were lost to follow-up over a decade. In a more recent study of elderly men and women in the Established

TABLE 1. Social Network/Support Studies of All-Cause or CHD Mortality Rate

Study	Outcome	Sample Size	Age Range	Number of Outcome Events[a]	Average Follow-Up
Community-Based					
Alameda County, Berkman and Syme (6)	All-cause mortality	4725	30–69	371	9 years
Tecumseh, Michigan, House et al. (8)	All-cause mortality	2754	30–69	259	9–12 years
Duke, Blazer (7)	All-cause mortality	331	65	50	30 months
Swedish men, Welin et al. (12)	All-cause mortality	989	50, 60	151	9 years
Evans County, Georgia, Schoenbach et al. (11)	All-cause mortality	2059	15	530	12 years
Swedish population, Orth-Gomer and Johnson (10)	All-cause mortality	17,433	29–74	841	6 years
North Karelia, Finland, Kaplan et al. (9)	All-cause mortality	13,301	39–59	598	5 years
EPESE, Seeman et al. (13)					
East Boston	All-cause mortality	3807	65+	892	5 years
Iowa		3097		583	
New Haven		2788		803	
Survival Post-MI					
B-HAT, Ruberman et al. (14)	All-cause mortality	2320	30–69	184	3 years
Swedish men, Orth-Gomer et al. (15)	All-cause mortality	150	40–65	34	10 years
Diltiazem Post-Infarction Trial, Case et al. (17)	Recurrent nonfatal MI or cardiac death	1234	25–75	226	25 months
Angiography and coronary artery disease, Williams et al. (16)	Cardiovascular death	1368	46–58	237	4–11 years
EPESE, Berkman et al. (18)	All-cause mortality	194	65+	76	6 months

[a] Number of events = actual number of outcome events in the cohort described in outcome column (e.g., deaths from all causes, CHD, or recurrent nonfatal MIs).

Populations for the Epidemiologic Study of the Elderly (EPESE), less than 1% were lost to follow-up (13).

4. This population-based sampling strategy coupled with small loss to follow-up leads to minimal selection bias.

5. Finally, when the studies are examined en masse, most relevant covariates have been included in most analyses, although certainly not all have been examined.

Thus, we can be confident that, based on these findings, risks are meaningful, consistent, and of sufficient magnitude that we are observing an important phenomenon.

In spite of this strong evidence, there is still much that is not known and will need to be known to improve health by modifying social conditions. For instance, first, we have little information on where along the spectrum of disease development these social factors have their greatest impact. Do they influence the development of risk factors or health behaviors, subclinical disease, incidence of clinical events, or case fatality? Second, we need a much clearer understanding of the mechanisms or pathways that link socioenvironmental conditions to morbidity and mortality rates. Finally, of the utmost importance is the need to learn whether we can modify or intervene on these conditions to improve health status.

In the remainder of this discussion, I focus some attention on each of these issues.

STUDIES OF RECOVERY FROM MYOCARDIAL INFARCTION (MI)

Although the previously mentioned community-based studies showed social isolation or lack of social ties to be related to mortality risk, these studies did not clarify where along the spectrum of disease social factors might have their greatest impact. For instance, do they influence the mortality rate by influencing the onset or progression of clinical disease or survival after an event?

Recently, researchers have began to address this question by examining the influence of social ties on survival in patients post-MI. In five studies, patients who lacked social support, lived alone, or had not been married had an elevated mortality risk post-MI (Table 1) (14–18). In the first of these, Ruberman et al. (14) explored 2320 male survivors of acute MI who were participants in the Beta-Blocker Heart Attack Trial. Patients who were socially isolated were more than twice as likely to die over a 3-year period than those who were less socially isolated. When this measure of social isolation was combined with a general measure of life stress, which included items related to occupational status, divorce, expo-

sure to violent events, retirement, or financial difficulty, the risks associated with high-risk psychosocial status were even greater. Those in the high-risk psychosocial categories were four to five times as likely to die as those in the lowest risk categories. This psychosocial characteristic was associated with death from all causes and sudden deaths. It made large contributions to mortality risk in both the high-arrhythmia and low-arrhythmia groups. In this study (and most of the studies in which subjects are recruited postevent), the investigators were not able to determine the temporal association between the assessment of psychosocial resources and the severity of disease. Nonetheless, it serves as a powerful model for future studies.

In a second Swedish study of 150 cardiac patients and patients with high-risk factor levels for CHD, the finding that lack of support predicts death was further confirmed (15). Patients who were socially isolated had a three times higher 10-year mortality rate than did those who were socially active and integrated. Because these patients were examined extensively for cardiological prognostic factors at study entry, it was possible to disentangle effects of psychosocial and clinical characteristics.

In a third study, Williams et al. (16) enrolled 1368 patients who were undergoing cardiac catheterization from 1974 through 1980 and found to have significant coronary artery disease. They examined survival time until cardiovascular death through 1989. Men constituted 82% of the sample. In this study, men and women who were unmarried or without a confidant were over three times as likely to die within 5 years compared with those who had a close confidant or who were married (odds ratio (OR), 3.34; confidence interval (CI), 1.8–6.2). This association was independent of other clinical prognostic indicators and sociodemographic factors, including socioeconomic status.

In another study, Case et al. (17) examined the association between marital status and recurrent major cardiac events among patients post-MI who were enrolled in the placebo arm of a clinical trial, the Multicenter Diltiazem Post-Infarction Trial. These investigators reported that living alone was an independent risk factor with a hazard ratio of 1.54 (CI, 1.04–2.29) for recurrent major cardiac event, including both nonfatal infarctions or cardiac deaths.

In a fifth study, we explored the relationship between social networks and support and mortality rate among men and women hospitalized for a MI between 1982 and 1988 who are participants in the population-based New Haven EPESE (18). Over the study period, 100 men and 94 women were hospitalized for an MI. Thirty-four per cent of women and 44% of men died in the 6-month period after MI (Figure 1).

Among both men and women, emotional support, measured prospectively, that is, before the MI was related to both early in-hospital death and later death over a 1-year period. Among those admitted to the hospital, almost 38% of those who reported no source of emotional support died in the hospital compared with 11.5% of those with two or more sources of support. The patterns remained steady throughout the follow-up period. At 6 months, the major end point of the study, 52.8% of those with no source of support had died compared with 36.0% of those with one source and 23.1% of those with two or more sources of support. These figures did not change substantially at 1 year. As Figure 1 shows, the patterns were remarkably consistent for both men and women, younger and older people, those with greater or lesser comorbidity, and those with more or less severe cardiovascular disease, as assessed by a Killip classification system. In multivariate models that control for sociodemographic factors, psychosocial factors, including living arrangements, depressive symptoms, and clinical prognostic indicators, men and women who reported no emotional support had almost three times the mortality risk compared with subjects who reported at least one source of support (OR, 2.9; 95% CI, 1.2–6.9).

The findings from these studies are strong and consistent, although the measures of support and the populations vary dramatically. Whereas they in no way preclude the possibility that these social factors influence the onset of CHD, they strongly suggest that they influence survival post-MI.

This large body of research focusing on mortality rates, and more recently case-fatality rates, specifically, is complemented by a more limited but growing body of literature on disease progression (19, 20) incidence and functional disability (21–24), especially related to CHD and cerebrovascular disease. In considering ways in which we can maximize opportunities to make substantial progress in this area over the next years, we should investigate the influence of social relationships on the full spectrum of disease development to understand where interventions will be most effective.

PATHWAYS LEADING TO DISEASE: THE SEARCH FOR A BIOLOGIC LINK

Social networks and the degree to which individuals are embedded in supportive social relationships

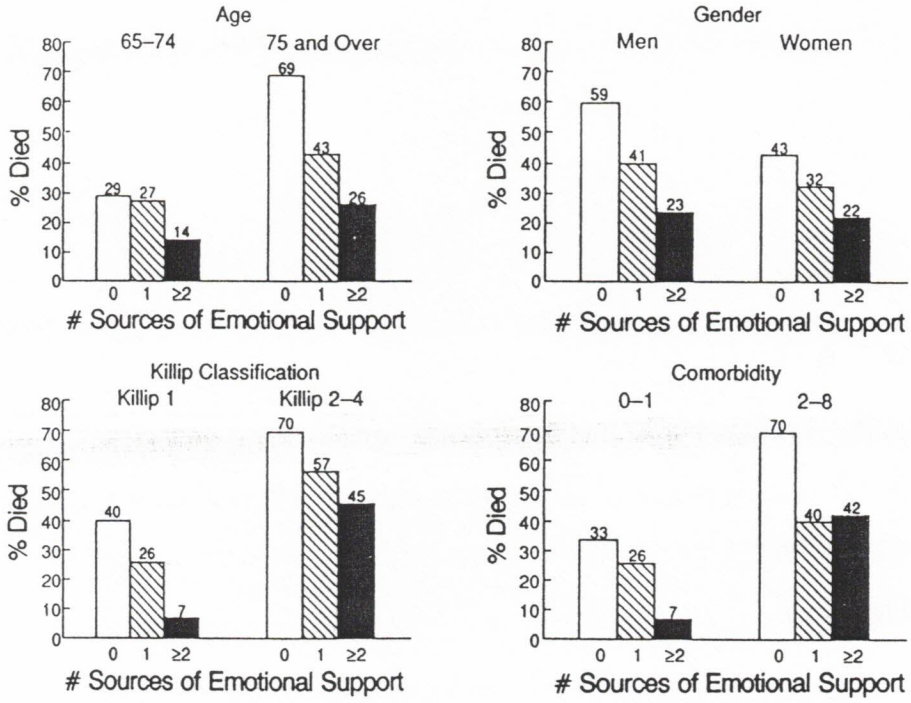

Fig. 1. Percentage of patients with MIs who died within 6 months by level of social support. Adjustments were made for age (*top left*); gender (*top right*); severity of MI, as defined by Killip class (*bottom left*); and comorbidity (*bottom right*). (From Berkman et al. (18) with permission.)

are related to many different outcomes, probably for many different reasons. For example, social networks are related to a broad array of health behaviors that range from the likelihood that women will engage in cancer screening (25–27), dialysis (28, 29), and smoking (30) and alcohol consumption (31). They may influence health outcomes through these behavioral mechanisms or by more direct physiologic pathways. Although a thorough review of this physiologic literature is beyond the scope of this article, several recent studies are noteworthy and have provided important pieces to the puzzle of how such a fundamentally social experience could get "inside the body." These findings suggest that there are likely to be multiple biologic pathways involved in this linkage and, furthermore, that the pathways invoked are likely to cascade and influence a number of disease outcomes. By examining these mechanisms, we can begin to understand how several basic physiologic regulatory mechanisms, themselves intertwined, might be responsive to socioenvironmental stressors (32–34). The notion of "generalized resistance" or "susceptibility to disease" need not be interpreted literally as a single mechanism or pathway but, rather, as a more general route, referring to complex physiologic systems with feedback loops and potentials to cascade and influence an even broader array of end points.

The two physiologic systems that are the focus of most recent attention are the immunological and neuroendocrine systems, with the neuroendocrine systems being closely tied to cardiovascular reactivity. There are several excellent reviews in this field (35–38), and the reader may refer to them for more extensive and detailed discussions. I take this opportunity to discuss several individual studies that, in themselves, are illustrative of the excellent work done in this area.

Early work on the link between immunological parameters and social relations was often focused on the effects of severe social losses and death of the spouse. These early landmark studies supported the idea that bereavement and loss were associated with suppressed immune function, particularly cellular immunity (39, 40).

This work was followed by the study of caregivers living with chronically ill spouses (41, 42). The findings of these more recent studies are generally consistent with those focused on bereaved men and women and support the general hypothesis that immune function, especially cellular immunity and natural killer cell activity, is influenced by aspects of social relationships. However, because these losses or impending losses are so severe and involve so many other physical and economic stresses and psychological threats, it is hard to interpret these

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associations as specifically linking social support to changes in immune function. Only examination of specific findings within these studies gives stronger support for the direct importance of the influence of social relationships per se. For instance, in the study by Kiecolt-Glaser et al. (42) on family caregivers of patients with Alzheimer's disease (AD), it was reported that caregivers whose relatives were institutionalized did not differ significantly in most health behavioral or psychological parameters from those who lived with patients with AD. These data suggest that it was the emotional loss of the spouse or parent that was more critical to changes in immune function rather than the physical stresses associated with providing ongoing care to the patient with AD. Moritz et al. (43) reported similar findings with regard to depression among elderly couples in which one spouse is cognitively impaired.

Finally, other studies in which investigators examined a) the quality of marital relationships and loneliness (44–47) in less than tragic circumstances in humans and b) affiliation in nonhuman primates (48, 49) consistently point out the importance of the quality of social relationships in influencing cellular immune response.

The work in the area of neuroendocrine function and social relations is small, although there is a much larger body of work on social factors and cardiovascular reactivity and physiological arousal, which is closely linked to neuroendocrine function (50). Animal studies consistently show that how animals are grouped and the degree to which they are isolated from mothers or any animal or touched influences neuroendocrine function, cardiovascular reactivity, lipid metabolism, and the development of atherogenesis (51–56). In humans, much less is known specifically on the association between support and neuroendocrine function or reactivity, although several studies (57–59) have illustrated that touching reduces cardiovascular reactivity. Seeman et al. (60) have just completed an analysis of neuroendocrine functioning and social relationships among older high-functioning men and women. In this study, social relationships, especially the quality of emotional support, was related to urinary levels of epinephrine, norepinephrine, and cortisol among older men and women independent of other behaviors or levels of comorbidity. Emotional support was more strongly and consistently associated with these neuroendocrine parameters than were measures of social ties (e.g., integration), instrumental support, or negative aspects of social relationships. This is one of the only studies to test the hypothesis that social support is directly associated with neuroendocrine function.

In regard to cardiovascular reactivity, Kamarck et al. (61) completed a recent well-designed experimental study that builds on much of the earlier work on the effects of touching or companionship on blood pressure and cardiovascular function (62). In their study, subjects completed two laboratory tasks. One-half of the subjects worked alone. The other half had a friend accompany them. Subjects who had a supportive partner in the room during tasks showed significantly reduced heart rate activity and tended to have lower systolic and diastolic blood pressures. In addition, there was an interesting interaction between Type A behavior and social support, whereby support was associated with attenuated systolic blood pressure only for those individuals classified as being Type A. Among the potential mechanisms that might explain the association between having a supportive partner and decreased cardiovascular arousal, the authors suggest that the "friend's presence may have acted as a conditioned stimulus or a 'safety signal,' altering neural input to the heart during challenge" (p. 54).

The evidence linking social relationships and support to health outcomes by way of physiologic responses that involve neuroendocrine regulation and immunological function is plausible, yet progress in this area remains a major challenge, necessitating serious interdisciplinary collaboration and great sensitivity to measurement of both psychosocial and physiologic conditions.

A CALL FOR THE DEVELOPMENT OF PSYCHOSOCIAL INTERVENTIONS

Although research based on observational studies will undoubtedly continue to yield productive insights into the role that social conditions play as etiologic factors and in the prognosis of disease, it now seems clear that we should take the next step forward to develop psychosocial interventions whereby we might attempt to alter or modify social networks and support to improve health outcomes. The development of such interventions is critical from both a scientific and a public health standpoint. From a scientific perspective, clinical trials and experimental work have important methodologic strengths that complement observational studies. Clinical trials allow us to test the principle that, by modifying exposure to risk, we change the disease outcome. Also, randomization provides us with confidence (although not assurance)

that both measured and unmeasured covariates are equally distributed in the intervention and control groups. From a public health perspective, if we are unable to translate scientific discoveries into practices that will improve the health and well-being of the public, we have failed in our most fundamental mission.

There are several key theories that have guided our group at Yale in the development of interventions. A social network or support intervention is based on the premise that the individual who is ill or at risk and his or her network represent an interdependent and dynamic system. Thus, improvement or even maintenance of health in one individual is influenced not only by the individual's behavior but also by the behaviors of others in the network and their abilities to communicate optimally. These types of interventions build on social support group and family therapy theories in which the family or network is seen in its entirety as a functioning unit (63–67).

Social learning theory also provides a useful model for understanding the acquisition of specific behaviors within social support interventions (68). A central tenet in this theory is the role of self-efficacy or the belief that one can successfully perform behaviors to produce a desired outcome. Self-efficacy is believed to be shaped by past and present behavior and by the social environment through observation of behaviors in others and verbal support and persuasion. Thus, according to this theory, for social support to be health promoting, it must provide not only a sense of belonging and intimacy, but it must also help people to be more competent and self-efficacious. Support that encourages dependence may not be health promoting (69, 70). Social learning and support group theories have been used successfully in the development of self-management programs aimed at minimizing the impact of chronic illness on functional capacity (71) and have been designed to help people manage a chronic condition (72–76). Many programs aimed at altering specific behaviors have used support groups of patients to induce behavioral and psychological changes (73, 77, 78). Finally, some programs have specifically been aimed at providing support and/or supportive counseling to improve health outcomes in patients with MIs (79), cancer (80, 81), and stroke (82). These programs have an excellent potential to generalize to other areas with the use of intervention strategies that range in focus from patient support groups to individualized professional intervention with the patient and/or family. Our own bias in this regard is that interventions that aim at restructuring naturally occurring networks and resources for support will be more effective than those that rely on short-term constructed support groups. We recognize, however, that support groups often have the advantage of bringing people together with shared concerns or of providing opportunities for interaction among people who are socially isolated.

Because naturally occurring networks form the basis for long-term behaviors, we found an excellent intervention model that was developed at the Center for Family Research at George Washington University School of Medicine, which combines both natural and "constructed" supports (83). This model brings both patients and families with different chronic illnesses together in small groups for a number of structured sessions. The multifamily discussion group as envisioned by the George Washington group has three components: a) educational, b) family issues, and c) affective. The model is designed to mix families with different chronic illnesses so that biomedical aspects of any single disease do not overwhelm the discussion of general psychosocial stressors. Such a multifamily intervention also has the advantages of not isolating the "sick" patient from other family members nor of focusing all intervention efforts on a single family or network so that they find it difficult to identify their situation as common or shared with others.

Over the next years, as more interventions are developed, we will undoubtedly see variations on this theme work with equal efficacy. Similarly, as other clinical trial methodologists have come to appreciate, individually tailored, stepped-care approaches will become better defined. It will also be important to remember that large-scale macrosocial, economic, and welfare policies influence the ability of families and communities to maintain high levels of social integration and support. In this vein, larger scale interventions aimed not at individuals but at communities or work sites may also make significant contributions to strengthening social ties.

In this era of health care reform, it is important to support efforts to promote financial access to medical care for all Americans; however, we cannot expect that such access will erase social inequalities in health. To improve health among vulnerable and high-risk populations, we will need to focus on preventive efforts that, at their core, promote social support and develop family and community strengths. Acknowledging that health promotion rests on the shoulders not only of individuals but also of their families and communities means that we must commit resources over the next decade to

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designing, testing, and implementing interventions in this area. This is a critical next step. For, as an African saying goes, "It takes an entire village to raise one child."

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REFERENCES

1. Cassel J: The contribution of the social environment to host resistance. Am J Epidemiol 104:107–123, 1976
2. Cobb S: Social support as a moderator of life stress. Psychosom Med 38:300–314, 1976
3. Bruhn J, Wolf S: An Anatomy of Health: The Roseto Story. Oklahoma City, OK, University of Oklahoma Press, 1979
4. Matsumoto YS: Social stress and coronary heart disease in Japan: A hypothesis. Milbank Q 48:9–36, 1979
5. Frost WH: How much control of tuberculosis? Am J Public Health 27:759–766, 1937
6. Berkman LF, Syme SL: Social networks, host resistance and mortality: A nine year follow-up study of Alameda County residents. Am J Epidemiol 109:186–204, 1979
7. Blazer D: Social support and mortality in an elderly community population. Am J Epidemiol 115:684–694, 1982
8. House JS, Robbins C, Metzner HL: The association of social relationships and activities with mortality: Prospective evidence from the Tecumseh community health study. Am J Epidemiol 116:123–140, 1982
9. Kaplan JR, Salonen JT, Cohen RD, et al: Social connections and mortality from all causes and cardiovascular disease: Prospective evidence from Eastern Finland. Am J Epidemiol 128:370–380, 1988
10. Orth-Gomer K, Johnson J: Social network interaction and mortality: A six year follow-up of a random sample of the Swedish population. J Chronic Dis 40:949–957, 1987
11. Schoenbach VJ, Kaplan BG, Freedman L, et al: Social ties and mortality in Evans County, Georgia. Am J Epidemiol 123:577–591, 1986
12. Welin L, Tibblin G, Svardsudd K, et al: Prospective study of social influences on mortality: The study of men born in 1913 and 1923. Lancet 1:915–918, 1985
13. Seeman TE, Berkman LF, Kohout F, et al: Intercommunity variations in the association between social ties and mortality in the elderly: A comparative analysis of three communities. Ann Epidemiol 3:325–335, 1993
14. Ruberman W, Weinblatt E, Goldberg JD, et al: Psychosocial influences on mortality after myocardial infarction. N Engl J Med 311:552–559, 1984
15. Orth-Gomer K, Unden AL, Edwards ME: Social isolation and mortality in ischemic heart disease. Acta Med Scand 224: 205–215, 1988
16. Williams RB, Barefoot JC, Califf RM, et al: Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease. JAMA 267:520–524, 1992
17. Case RB, Moss AJ, Case N, et al: Living alone after myocardial infarction. JAMA 267:515–519, 1992
18. Berkman LF, Leo-Summers L, Horwitz RI: Emotional support and survival following myocardial infarction: A prospective population-based study of the elderly. Ann Intern Med 117: 1003–1009, 1992
19. Seeman TE, Syme SL: Social networks and coronary artery disease: A comparison of the structure and function of social relationships as predictors of disease. Psychosom Med 49: 340–353, 1987
20. Kaplan JR, Manuck SB, Clarkson TB, et al: Social status, environment, and atherosclerosis in cynomolgus monkeys. Arteriosclerosis 2:359–368, 1982
21. Orth-Gomer K, Rosengren A, Wilhelmsen L: Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. Psychosom Med 55:37–43, 1993
22. Glass TA, Matchar DB, Belyed M, et al: The impact of social support on outcome in first stroke. Stroke 24:67–70, 1993
23. Evans RL, Bishop DS, Matlock AL, et al: Prestroke family interaction as a predictor of stroke outcome. Arch Phys Rehab Med 68:508–512, 1987
24. Colantonio A, Kasl S, Ostfeld A, et al: Psychosocial predictors of stroke outcomes in an elderly population. J Gerontol 48:S261–S268, 1993
25. Suarez L, Lloyd L, Weiss N, et al: Effect of social networks on cancer-screening behavior of older Mexican American women. J Natl Cancer Inst 86:775–779, 1994
26. Calnan M: Patterns in preventive behavior: A study of women in middle age. Soc Sci Med 20:263–268, 1985
27. Kang SH, Bloom JR: Social support and cancer screening among older black Americans. J Natl Cancer Inst 85:737–742, 1993
28. Steidl JH, Finkelstein FO, Wexler JP, et al: Medical condition, adherence to treatment regimes and family functioning: Their interactions in patients receiving long-term dialysis treatment. Arch Gen Psychiatry 37:1025–1027, 1980
29. Kaplan De-Nour A: Dialysis within the context of the family. Int J Fam Psychiatry 1:61–75, 1980
30. Mermelstein R, Cohen S, Lichtenstein F, et al: Social support and smoking cessation maintenance. J Consult Clin Psychol 54:447–453, 1986
31. Berkman LF, Breslow L: Health and Ways of Living. New York, Oxford University Press, 1983
32. Rabin BS, Cohen S, Ganguli R, et al: Bidirectional interaction between the central nervous system and immune system. Crit Rev Immunol 9:279–312, 1989
33. Brindley DN, Rolland Y: Possible connections between stress, diabetes, obesity, hypertension and altered lipoprotein metabolism that may result in atherosclerosis. Clin Sci 77:453–461, 1989
34. Selye H, Tuchweber B: Stress in relation to aging and disease. In Everitt A, Burgess J (eds), Hypothalamus, Pituitary and Aging. Springfield, IL, Charles C. Thomas, 1975, 553–569
35. Seeman TE, Robbins RJ: Aging and hypothalamic-pituitary-adrenal response to challenge in humans. Endocr Rev 15:233–260, 1994
36. Cohen S: Psychosocial models of the role of social support in the etiology of physical disease. Health Psychol 7:265–297, 1988
37. Kennedy S, Kiecolt-Glaser JK, Glaser R: Social support, stress, and the immune system. In Sarason BR, Sarason IG, Pierce GR

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(eds), Social Support: An Interactional View. New York, Wiley & Sons, 1990, 253–266
38. Ader R, Felten DL, Cohen N: Psychoneuroimmunology, 2nd Edition. New York, Academic Press, 1991
39. Schleifer SJ, Keller SE, Camerino M, et al: Suppression of lymphocyte stimulation following bereavement. JAMA 250: 374–377, 1983
40. Bartrop RW, Luckhurst E, Lazarus L, et al: Depressed lymphocyte function after bereavement. Lancet 1:834–836, 1977
41. Baron RS, Cutrona CE, Hicklin D, et al: Social support and immune function among spouses of cancer patients. J Pers Soc Psychol 59:344–352, 1990
42. Kiecolt-Glaser JK, Glaser R, Shuttleworth EC, et al: Chronic stress and immunity in family caregivers of Alzheimer's disease victims. Psychosom Med 49:523–535, 1987
43. Moritz DJ, Kasl SV, Berkman LF: The health impact of living with a cognitively impaired spouse: Depressive symptoms and social functioning. J Gerontol 44:S17–S27, 1989
44. Kiecolt-Glaser JK, Fisher LD, Ogrocki P, et al: Marital quality, marital disruption and immune function. Psychosom Med 49:13–34, 1987
45. Thomas PD, Goodwin JM, Goodwin JS: Effect of social support on stress-related changes in cholesterol level, uric acid level, and immune function in an elderly sample. Am J Psychiatry 142:735–737, 1985
46. Glaser R, Kiecolt-Glaser JK, Speicher CE, et al: Stress, loneliness and changes in herpes virus latency. J Behav Med 8:249–260, 1985
47. Kiecolt-Glaser JK, Garner W, Speicher CE, et al: Psychosocial modifiers of immunocompetence in medical students. Psychosom Med 46:7–14, 1984
48. Cohen S, Kaplan JR, Cunnick J, et al: Chronic social stress, affiliation and cellular immune response in nonhuman primates. Psychol Sci 4:301–310, 1992
49. Kaplan JR, Manuck SB, Heise EB, et al: The relationship of agonistic and affiliative behavior patterns to cellular-mediated immune function among cynomolgus monkeys *Macaca fascicularis* living in unstable social groups. Am J Primatology 25:157–173, 1991
50. Krantz DS, Manuck SB: Acute psycho-physiologic reactivity and risk of cardiovascular disease. A review and methodologic critique. Psychol Bull 96:435–464, 1984
51. Clarkson TB, Kaplan JR, Adams MR, et al: Psychosocial influences in the pathogenesis of atherosclerosis among nonhuman primates. Circulation 76(Suppl 1):I29–40, 1987
52. Stanton ME, Patterson JM: Social influences on conditioned cortisol secretion in the squirrel monkey. Psychoneuroendocrinology 10:125–134, 1985
53. Meaney MJ, Aitken DH, Van Berkel C, et al: Effects of neonatal handling on age-related impairments associated with the hippocampus. Science 239:766–768, 1988
54. Shively CA, Clarkson TB, Kaplan JR: Social deprivation and coronary artery atherosclerosis in female cynomolgus monkeys. Atherosclerosis 77:69–76, 1989
55. Shively CA, Adams MR, Kaplan JR, et al: Social stress, ovarian function and coronary artery atherosclerosis in primates. In Zajkowski SM, Hill D, Clarkson TB (eds), Women, Behavior and Cardiovascular Disease (Publication No. NIH, 94–3309). Bethesda, MD, National Institutes of Health, 1994, 127–144
56. Sapolsky RM: Endocrine aspects of social instability in the olive baboon. Am J Primatology 5:365–379, 1983
57. Drescher VM, Whitehead WE, Morril-Corbin ED, et al: Physiological and subjective reactions to being touched. Psychophysiology 22:96–100, 1985
58. Lynch JJ, Thomas SA, Paskewitz DA, et al: Human contact and cardiac arrhythmia in a coronary care unit. Psychosom Med 39:188–192, 1977
59. Whitcher ST, Fisher JD: Multidimensional reaction to therapeutic touch in a hospital setting. J Pers Soc Psychol 37:87–96, 1979
60. Seeman TE, Berkman LF, Blazer D, et al: Social ties and support and neuroendocrine function: The MacArthur Studies of Successful Aging. Ann Behav Med 16:95–106, 1994
61. Kamarck T, Manuck SB, Jennings JR: Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. Psychosom Med 52:42–58, 1991
62. Lynch JL: The Broken Heart: The Medical Consequences of Loneliness. New York, Basic Books, 1979
63. Speck R, Attneave C: Family Networks. New York, Vintage, 1973
64. Vaux A: Social Support: Theory Research and Intervention. New York, Praeger, 1988
65. Spiegal D, Spira J: Supportive Expressive Group Therapy: A Treatment Manual of Psychosocial Intervention for Women with Recurrent Breast Cancer. Stanford, CA, Psychosocial Treatment Laboratory, 1991
66. Minuchin S: Families and Family Therapy. Cambridge, MA, Harvard University Press, 1974
67. Rolland JS: Families, Illness and Disability: An Integrative Treatment Model. New York, Basic Books, 1994
68. Bandura A: Social Foundations of Thought and Action. A Social Cognitive Theory. Englewood Cliffs, NJ, Prentice Hall, 1986
69. Wortman C, Lehman D: Reactions to victims of life crisis: Support that doesn't help. In Sarason IG, Sarason BR (eds), Social Support: Theory, Research and Application. Dordrecht, Martinus Nijhoff, 1985
70. McLeroy KR, DeVillis R, DeVellis B, et al: Social support and physical recovery in a stroke population. J Soc Pers Relations 1:395–413, 1984
71. Tobin DL, Reynolds RVC, Hoylroyd KA, et al: Self-management and social learning theory. In Hoylroyd KA, Creer DL (eds), Self-Management of Chronic Disease: Handbook of Clinical Intervention and Research. Orlando, FL, Academic Press, 1985, 29–55
72. Lorig K, Cauvin J, Holman H: Arthritis self-management: A study of the effectiveness of patient education for the elderly. Gerontologist 24:455–457, 1984
73. Lorig K, Mazonson PD, Holman HR: Evidence suggesting that health education for self-management in patients with chronic arthritis has sustained health benefits while reducing health care costs. Arthritis Rheum 36:439–446, 1993
74. Wilson W, Pratt C: Impact of diabetes education and peer support upon weight and glycemic control of elderly persons with noninsulin dependent diabetes mellitus (NIDDM). Am J Public Health 77:634–635, 1987
75. Clark NM, Rakowski W, Ostrander L, et al: Development of self-management for elderly heart patients. Gerontologist 28: 491–494, 1988
76. Clark NM, Becker MH, Janz NK, et al: Self-management of chronic disease by older adults. J Aging Health 3:3–27, 1991
77. Ornish D, Brown SE, Scherwitz LW, et al: Can lifestyle changes reverse coronary heart disease? Lancet 336:129–133, 1990
78. Friedman M, Thoresen CE, Gill JJ, et al: Alteration of type A behavior and its effect on cardiac recurrences in post-

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myocardial infarction patients: Summary results of the recurrent coronary prevention project. Am Heart J 112:653–665, 1986
79. Frasure-Smith N, Prince R: Long-term follow-up of the ischemic heart disease life stress monitoring program. Psychosom Med 51:485–513, 1989
80. Spiegal D, Bloom JR, Yalom ID: Group support for metastatic cancer patients: A randomized prospective outcome study. Arch Gen Psychiatry 38:527–533, 1981
81. Fawzy FI, Kemeny ME, Fawzy NW, et al: A structured psychiatric intervention for cancer patients. II. Changes over time in immunologic measures. Arch Gen Psychiatry 47:729–735, 1990
82. Evans RL, Matlock AL, Bishop DS, et al: Family intervention after stroke: Does counseling or education help? Stroke 19: 1243–1249, 1988
83. Gonzalez S, Steinglass P, Reiss D: Putting the illness in its place: Discussion groups for families with chronic medical illnesses. Fam Process 28:69–87, 1987