CHAPTER 13

SOCIAL SUPPORT

CAUSE OR CONSEQUENCE OF POOR HEALTH OUTCOMES IN MEN WITH HIV INFECTION?

ROBERT M. KAPLAN, THOMAS L. PATTERSON, DAVID KERNER, IGOR GRANT, AND THE HIV NEUROBEHAVIORAL RESEARCH CENTER

A substantial literature argues that people who have smaller social networks experience increased risk of death and other negative health outcomes. Early sociological research, for instance, found a link between social support and suicide risk (Durkheim, 1951). Since that early observation, researchers have probed the link between social support and health in a variety of ways. The literature, on the relationship between social support and a variety of different causes of death has evolved over a course of 20

The San Diego HIV Neurobehavioral Research Center (HNRC) group is affiliated with the University of California, San Diego; the Naval Hospital, San Diego, and the San Diego VA Medical Center, and includes Igor Grant, M.D., Director; J. Hampton Atkinson, M.D., Codirector; Robert A Velin, Ph.D., Center Manager; Edward C. Oldfield III, M.D., James L. Chandler, M.D., Mark R. Wallace, M.D., and Joseph Malone, M.D., Coinvestigators Naval Hospital San Diego; J. Allen McCutchan, M.D., P.I. Medical Core; Stephen A. Spector, M.D., P.I. Virology Core; Leon Thal, M.D., P.I. Neurology Core; Robert K. Heaton, Ph.D., P.I. Neuropsychology Core; John Hesselink, M.D. and Terry Jernigan, Ph.D., Co-P.I.s Imaging Core; J. Hampton Atkinson, M.D., P.I. Psychiatry Core; Clayton A. Wiley, M.D., Ph.D., P.I. Neuropathology Core; Richard Olshen, Ph.D. and Ian Abramson, Ph.D., Co-P.I.s Biostatistics Core; Nelson Butters, Ph.D., P.I. Memory Project; Renée Dupont, M.D., P.I. SPECT Project; Dilip Jeste, M.D., P.I. Psychosis Project; Hans Sieburg, Ph.D., P.I. Dynamical Systems Project; and James D. Weinrich, Ph.D., P.I. Sexology Project.

ROBERT M. KAPLAN • Department of Family and Preventive Medicine, University of California, San Diego, La Jolla, California 92093-0622. THOMAS L. PATTERSON AND IGOR GRANT • Department of Psychiatry, University of California, San Diego, La Jolla, California 92092-0622. DAVID KERNER • SDSU/UCSD Joiny Doctoral Program in Clinical Psychology, University of California, San Diego, La Jolla, California 92092-0622.

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years (Berkman, 1995). However, the literature on social support and health outcome leaves many questions unanswered. For example, most studies relating social support to mortality outcomes define social support in terms of network size. Current definitions of social support include qualitative (emotional), quantitative (adequacy), as well as measures of social network size.

One of the difficulties in separating explanations for the relationship between social support and health outcome is that most of the studies are cross-sectional. In this chapter, we present longitudinal data in which health-outcome data and social support are measured at multiple points in time. Longitudinal studies are needed, because social support may be determined by health variables, as opposed to being a determinant of health outcomes. This chapter focuses on outcomes for men with HIV disease. After a brief introduction to epidemiology, we will review the HIV epidemic. Then, we will briefly review the literature linking stress, social support, and immune status in HIV patients. The remainder of the chapter presents original data evaluating the causal direction of social support and immune status in HIV-infected patients. We are interested in separating two causal pathways. In particular, we consider whether poor social support causes reductions in immune status against the alternative explanation that declining health causes reductions in social networks. Most studies begin with the assumption that social support is a predictor of health outcome and fail to test the alternative directional hypothesis. We suggest that the causal pathway between social support and health outcome may be bidirectional.

EPIDEMIOLOGY OF SOCIAL SUPPORT AND HEALTH OUTCOMES

Epidemiology is the study of the determinants and distribution of disease. The hallmark of epidemiological methodology is the prospective/longitudinal cohort study. Major investigations, such as the Framingham Heart Study, attempted to establish prospective predictors of mortality in a random sample from the general population. For example, the Framingham Study began with 5,127 participants who had no visible signs of heart disease. Each participant was given a physical examination and a detailed interview that included lifestyle and demographic characteristics. Then, each participant was followed every other year (Kannel, 1987). Other major epidemiological investigations have used similar methodologies.

Most epidemiological studies were started some years ago, before formal measures of social support had been developed. Nevertheless, simple measures of social network, often extracted *post hoc* from a database, appeared to be predictive of health outcomes in a variety of studies. The Alameda County Population Monitoring Study demonstrated that a simple measure of social network was a significant predictor of longevity. The measures of social support included marital status, number of close family and friends, church membership, and group membership. Men with weak social networks were nearly 2.5 times as likely to die within a defined time period as men with extensive networks. Women benefited even more from established social networks (Berkman & Breslow, 1983).

Similar results were obtained in Tecumseh, Michigan, where 2,754 men and women were studied. In this investigation, men who were married, who attended

church, and who participated in voluntary organizations and community activities were significantly less likely to die within a 10-year period than were men who were disconnected. However, the Tecumseh Study did not show similar relationships for women (House, Robbins, & Metzner, 1982). In contrast to the findings of Berkman and Breslow (1983) and House et al., in the Durham County, North Carolina study (Blazer, 1982), no consistent pattern of increased mortality rates were associated with a progressive decrease in social support. Rather, in this data set, there appeared to be a threshold effect in which only those individuals, either male or female, who were at the extreme end of the continuum in terms of the least amount of social support, had increased mortality rates. In a study of residents of Evans County, Georgia, those with the fewest ties were at increased risk for mortality. The findings reported were significant for older white males only and not for black individuals and white females (Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986).

Several studies have suggested that the combination of high stress and low social support is a particularly strong predictor of negative outcome. For example, 142 women in the Framingham Heart Study had more cardiovascular disease if they worked in clerical roles and had nonsupportive spouses (Haynes & Feinleib, 1980). A study of Swedish workers revealed that risk for cardiovascular disease was excessive among workers who had low social support, perceived their jobs to be stressful, and felt they had little control over their work environment (Welin et al., 1985). In one study of survivors of myocardial infarction, survivors were classified according to social isolation and stress, and then followed prospectively. Those who experienced low stress and were socially connected had one-fourth the rate of mortality in comparison to those who were under high stress and were isolated (Ruberman, Weinblatt, Goldberg, & Chaudhary, 1984). Despite these strong results, some studies have also failed to show a relationship between social support, stress, and health outcomes (Cohen & Syme, 1985). Many studies focus on the impact of social support on only one disease state or outcome variable. Aneshensel, Rutter, and Lachenbruch (1991) argued that the impact of stress and social support on health may be underestimated if the range of outcomes is limited. An alternative is to go beyond mortality to consider disease stage and measures of disability and health-related quality of life.

It is difficult to make comparisons across studies that have different conceptualization of health outcome. Populations varied greatly from study to study, as did definitions of social support. The measures of social support were usually crude. Some studies merely recorded the presence of a spouse or participation in group activities. In addition, the degree of satisfaction associated with these relationships in specific types of support received were often not considered. Nevertheless, these studies generally show a relationship between social relationships and longevity (Berkman, 1995; Davidson & Shumaker, 1987). These findings have intrigued epidemiologists and have supported the notion that friends and family are health assets.

Although the relationship between social support and mortality may seem impressive, these relationships are primarily correlational. There are at least three rival explanations for the association between the presence of social relationships and health. First, there is the assumed explanation that the correlation between social support and disease is causal—high support protects against illness. The second explanation is that individuals who are sick drive away their social support system.

This suggests that early illness causes changes in social support. A third explanation is that a third variable, such as social class, personality, and so on, causes both poor social support and poor health outcomes.

It is important to emphasize that epidemiological studies use different measures than do psychological investigations. The psychological construct *support* is usually defined as the number of social contacts, and *health* is most often defined as survival or mortality. Most support for the argument that social support protects against illness comes from epidemiological studies. However, these observational studies do not establish the causal relationship between network size and mortality. Furthermore, the epidemiology studies have rarely considered satisfaction with social support. As a result, many of the inferences about the relationship between support satisfaction and mortality remain highly speculative. The epidemiological studies have stimulated interest in the relationship between social support and health, but provide only a very small piece of the puzzle. To follow up on the suggestive evidence from epidemiological studies, we need to focus on a wider array of health outcomes and learn more about the nature of social interactions among those with serious illness. This chapter focuses on social support among men infected with the human immunodeficiency virus (HIV). HIV disease will be reviewed briefly in the next section.

HIV DISEASE

The acquired immune deficiency syndrome (AIDS) has directly affected millions of people worldwide and indirectly affects virtually every one of us. HIV, a retrovirus of the T-cell leukemia/lymphoma line, reduces the immune system's ability to recognize and destroy infectious agents. For healthy individuals, the immune system is capable of overcoming many common infections. As HIV disease progresses, an overall loss of immune competence leaves HIV-infected individuals susceptible to a variety of opportunistic infections.

As of June 1995, 470,228 cases of HIV infection had been reported in the United States. Among these, approximately half (52%) were among men who have sex with other men. Another 25% were reported among those who inject nonprescription drugs. About 7% of the cases were men who have sex with other men and also inject drugs. Most of the other cases were among those who have received blood products because of hemophilia (1%) or transfusion (2%). A growing number of cases were associated with heterosexual contact (8%), especially among intravenous (IV) drug users (data from Centers for Disease Control HIV/AIDS Surveillance Report 1995, 1997, No. 1). In addition to the medical issues, HIV infection has serious social consequences. Infected individuals are told to change the most central aspects of their lifestyles, and fear of infection may disrupt many social relationships. The stigma associated with the illness may cause disruptions in employment, housing, and friendships. In the next section, the impact of the illness on the immune system will be briefly reviewed.

Psychoneuroimmunology and HIV Infection

In the 1980s, medical and social science researchers from the field of psychoneuroimmunology recognized HIV disease as an appropriate model for testing the

effects of psychosocial variables on physical illness (Glaser & Kiecolt-Glaser, 1987). Both the immune and neural systems appeared to be central to HIV progression, and the course of HIV illness varied substantially among infected individuals, suggesting that a variety of factors were related to disease progression. The psychoneuroimmunological hypothesis in HIV has been tested empirically, using a variety of markers of HIV disease progression. HIV infection is detected by observing antibodies to the virus in serum, and is characterized by depletion and infection of CD4+ T-cells, which leaves the immune system compromised and prone to contracting opportunistic infections (signaling disease progression).

The basic premise of the psychoneuroimmunological hypothesis is that the immune systems of individuals who experience major stress in their lives are compromised. The way this works in HIV is as follows. When someone contracts the HIV virus, the virus attaches and enters cells in the host's body, usually CD4+ helper/inducer T-cell lymphocytes. The CD4+ lymphocytes are white blood cells that activate the immune system in order to combat foreign invaders, known as foreign *antigens*, in the body. HIV multiplies in the body when these CD4+ cells replicate in response to contact with an antigen, such as a cold virus. Thus, the life cycle of HIV consists of a series of steps in which the virus uses the host's own cells to reproduce enormous numbers of new viruses. As more and more CD4+ cells become infected, the immune system becomes unable to respond to infectious agents. Individuals die, therefore, from other infections that the body is no longer able to fight off, not from the HIV virus itself. In addition to destroying the CD4+ lymphocytes, HIV infects a number of other cells, including macrophages, skin, lymph nodes, and endothelial cells of the brain. Thus, the picture of the infection is more complicated than can be presented here. A central premise underlying the observed association between life adversity and health status is that stress exerts a suppressive effect on immune functioning. Studies from several laboratories have shown that a variety of stressors affect the immune response in animals as well as humans (Borysenko & Borysenko, 1982; Palmblad, 1981). Because HIV disease is by nature an immunosuppressive disorder, it has been an especially fertile ground for the study of the impact of psychoneuroimmunological factors in health.

Does the Health of HIV-Positive Individuals Who Experience Greater Stress in Their Lives Decline More Rapidly Compared to Less Stressed Individuals?

Currently, the best prognostic indicators of increased HIV-disease symptomatology are absolute CD4+ cell number (Fahey et al., 1990; Pederson et al., 1990) and presence of circulating P_{24} antigen (among advanced HIV disease stages), although elevated β_2-microglobulin levels have also been noted to rise with disease progression (Volberding & McCutchan, 1989). Psychoneuroimmunological studies have used a number of outcomes including CD4+ lymphocyte count (Perry, Fishman, Jacobsberg, & Frances, 1992; Rabkin, Remien, Katoff, & Williams, 1993), percent CD4+ lymphocytes (Patterson et al., 1995), CD4+/CD8+ ratio (Antoni et al., 1990; Goodkin et al., 1992), natural killer (NK) cell cytotoxicity (Antoni et al., 1990; Goodkin et al., 1992), absolute NK cell count (Sahs et al., 1994), time from seroconversion to AIDS diagnosis (Goodkin et al., 1992), survival time (Greco & Stazi, 1987), symptoms of HIV illness

(Kessler et al., 1991; Rabkin et al., 1991), degree of physical impairment (Rabkin et al., 1993), as well as a host of other biological markers for HIV-disease progression. Of these outcome measures, the most frequently used is CD4+ cell count, the immune marker most closely linked to the clinical consequences of HIV infection (Moss et al., 1988). Enumeration of CD4+ cells and other lymphocyte subsets have served as laboratory outcome measures in recent studies of HIV, life adversity, psychosocial moderators, and physical and mental health outcome. Preliminary analysis of data from an intensive psychoimmunological study of men with AIDS indicated significant negative correlations between absolute number of CD4+ helper cells and less tension-anxiety, depression-dejection, fatigue-inertia, and anger-hostility on the Profile of Mood States (POMS; Solomon, Temoshok, O'Leary, & Zich, 1987). Furthermore, Solomon and colleagues found self-rated "ability to say 'no' to unwanted favors" to be positively related to absolute number of NK cells, which are important in fighting neoplastic disease, and this ability was the best predictor of "positive" immune parameters overall. However, the results from other studies have been mixed. Rabkin et al. (1991) found no relationship between immune status as measured by CD4+ and CD8+ cell subsets and depression, distress, and negative life events, consistent with Kessler et al.'s (1991) report of no relationship between stress and percentage decrease of CD4+ cells or development of thrush and/or fever.

Previously Rabkin, Williams, Neugebauer, Remien, and Goetz (1990) reported a relationship between high levels of hopelessness and low levels of social support and more depressive symptoms, but no relationship was found between hopelessness, HIV symptoms, and negative life events. In contrast, Blaney et al. (1991) reported that both negative life events and social support were significant predictors of psychological distress (main effects only, no interaction effects), but immune and physical health outcomes were not reported. Similarly, Dew, Ragni, and Nimorwicz (1990) found depressive symptoms in HIV seropositive men to be related to recent loss events and current unemployment, and history of personal or family psychiatric treatment, but detected no interaction effects between "vulnerability factors" and symptoms.

In summary, there is a lot of excitement about psychoneuroimmunology. Some evidence suggests that HIV patients who experience serious life stress have poorer prognoses than those who experience less stress. However, studies investigating these issues have produced inconsistent results. Currently, we are unable to say with confidence that life stress is a significant factor in the progression of HIV disease.

HIV and Social Support

Social support is another psychosocial variable that has been examined as a moderator of HIV disease progression. Although social support is consistently related in an inverse direction to psychological distress among asymptomatic, HIV-positive gay males (Blaney et al., 1991; Patterson et al., 1993), associations with physical symptoms are less supported. It is believed that social support may act indirectly on physical health by easing the emotional or tangible burden of increased physical symptoms (Hays, Turner, & Coates, 1992); however, this stress-buffering hypothesis has not been tested prospectively among HIV-positive individuals. We previously studied 414 HIV-positive males using survival analysis (Patterson et al., 1996). Our

analysis was based on the existing empirical evidence that psychosocial variables may predict the course of HIV-illness disease progression (described by advance in symptoms, decline in CD4+ cell count, and mortality). We found that depressive symptoms predicted shorter longevity after controlling for symptoms and CD4+ cell count. Large social network sizes predicted longevity among those with AIDS-defining symptoms at baseline, but not among other subjects. Therefore, psychosocial variables and affective states may be related to disease outcome only during later stages of HIV disease. Although the results provide support for psychoneuroimmunological effects in HIV, other confounding explanations may still apply. More longitudinal research is needed to assess the impact stressful life adversity, social support, and affective feeling states may have on HIV-disease progression above and beyond that which is determined by the natural course of HIV diseases and demographic or background characteristics that may influence health status, such as age and socioeconomic status.

Although social support is likely to play a similar role with the context of HIV-related disease as it does with other health problems, only a handful of studies have reported beneficial effects of social support, and longitudinal data are even more scarce. In a cross-sectional study, Wolf et al. (1991) found that less perceived available social support was associated with more use of avoidant coping and greater mood disturbance, including higher levels of self-reported depression and anxiety, and lower levels of vigor among HIV-infected men (half medically asymptomatic, half symptomatic with AIDS or AIDS-related conditions). Similarly, Namir, Wolcott, and Fawzy (1989) found social support to be related to physical and mental health within their sample of 50 men with AIDS. Specifically, instrumental or tangible support was the only variable to significantly predict physical health scores, and it was significantly associated with mood disturbance as well.

It is possible that the type of social support most associated with physical illness varies according to disease stage or level of disability. Thus, in Namir et al.'s (1989) sample of men with full AIDS diagnoses, tangible support was most associated with physical health, perhaps reflecting more progressed disease and increased disability. Furthermore, Zich and Temoshok (1987) found that less available social support was associated with more physical symptoms for men with AIDS, but not for men with AIDS-related conditions (ARC) or HIV-positive asymptomatics. However, less available social support was related to higher levels of hopelessness and depression for all HIV-infected men in their sample. Among asymptomatic HIV-infected men, Blaney et al. (1991) found main effects for negative life events and emotional support. Together, these findings suggest that the domains of social support most closely associated with physical symptomatology may depend on disease status or level of physical dysfunction. Although satisfaction with emotional support appears to be a good predictor of mood and possibly physical symptoms, specific characteristics of emotional support, such as availability of a close companion and reciprocity of social support, warrant further study.

Summary

In summary, studies consistently show that smaller social support network size is associated with poor health outcomes. Fewer studies show that instrumental social

support protects against failing health. However, most current studies have used cross-sectional designs. Most authors believe that social support either directly causes poor health outcomes or provides an inadequate buffer against life stresses. An alternative explanation is that low social support is a consequence rather than a cause of poor health status. The remainder of this chapter evaluates these alternative explanations, using a longitudinal cohort of men with HIV infection. The male participants in this study were evaluated at 6-month intervals over the course of 18 months. At each evaluation, measures of social support and immune status were gathered.

METHOD

Subjects and Procedure

For the present analyses, the sample comprised 397 HIV-positive heterosexual and gay men who were participating in a longitudinal cohort study at the HIV Neurobehavioral Research Center (HNRC), University of California, San Diego. The sample was recruited from military personnel seen at the Naval Medical Center, San Diego, as well as from the civilian community in the greater San Diego area. Inclusionary criteria of the HNRC required that participants be male residents of San Diego County with at least 10 years of formal education, and willing to undergo extensive evaluations semiannually for up to 5 years. Individuals were excluded if their medical history might confound interpretation of neurological or neuropsychological findings, such as a history of IV drug use, head injury with loss of consciousness exceeding 30 minutes, or a primary diagnosis of thought disorder. The present sample was selected from the HNRC main cohort, based on two additional criteria: (1) availability of psychological variables from four consecutive measures, 6 months apart; and (2) participation in the study for at least 1 year.

Demographic characteristics for the sample are shown in Table 13.1. The average participant was 32.7 years of age ($SD = 7.1$), had 14 years of formal education, and had known of his HIV-positive status for 2 years. Reflecting the epidemiological characteristics of the epidemic present when the study began, the sample was largely Caucasian (76%), and of lower middle-class socioeconomic (SES) background (a Hollingshead SES rating of 4). Participants with more severe HIV symptoms were older, had known of their HIV-positive status longer, but they were not different in background, education, ethnicity, and SES. As expected, differences in CD4+ cell count and serum β_2-microglobulin levels differed significantly between baseline and 1-year follow-up (see Table 13.1).

Stage of HIV infection was determined at baseline using the 1993 Revised Classification System for HIV Infection (Centers for Disease Control, 1993). As shown in Table 13.1, the majority of participants (73%) were asymptomatic at baseline (symptom category A). Using the bidimensional definition for an actual AIDS diagnosis (CD4+ less than 200/mL and/or AIDS-indicator opportunistic infections), 40 participants (10%) had reached these criteria prior to or concurrent with their baseline assessment. Of the total sample, 16 (4%) had CD4+ cell counts less than 200/mL. The distribution of HIV severity in the sample might be described as somewhat bimodal: a large cluster of mostly asymptomatic cases with varying levels of immune function, and a smaller

TABLE 13.1. Demographic and Background Characteristics of Sample (n = 397) at Baseline and 1-Year Following

	Baseline	One year
Age	32.7 (7.1)	—
Years of education	14.0 (2.1)	—
Hollingshead SES (1–5)	3.7	—
Years HIV+	2.0 (1.4)	—
CD4+ count	473.5 (260.7)	365.4 (278.4)*
Beta-2 Microglobulin	2.98 (1.44)	3.36 (1.55)*
Ethnicity		
Caucasian	76%	—
African-American	15%	—
Latino	7%	—
Other	2%	—
CDC		
A	73%	45%
B	23%	38%
C	4%	17%

*$p < .05$

cluster of cases with an AIDS diagnosis and severely compromised immune function. All participants completed a 2-hour psychosocial battery, including measures of life stress, coping, social support, and depressive symptoms.

Measures

Depressive Symptoms

The clinician-rated Hamilton Rating Scale for Depression (HRSD) is a 21-item clinical instrument for assessing depressive symptoms based on 3- or 5-point Likert-type scale responses. The HRSD has well-documented validity (Endicott et al., 1981), and it has been used extensively in clinical trials of antidepressant drugs. Interrater reliability has been found to be in the range of 0.80 to 0.91 (Hamilton, 1969; 1974).

The Beck Depression Inventory (BDI) was also given to all participants. The BDI is a self-administered questionnaire consisting of 21 items, each having four graded statements pertaining to how the subject has been feeling during the past week (Beck, 1967). The statements within a question are ordered (0 to 3) to show increasing depressive symptomatology. Summary scores are calculated (range, 0 to 63). The items of the BDI are clinically derived and have undergone extensive reliability and validation studies (Beck, 1976). Internal consistency assessments of reliability have been high (> .90) in most evaluations.

Social Support

Social support was assessed using the Social Support Questionnaire, a 5-item self-report measure developed by Schaefer, Coyne, and Lazarus (1981). Participants identify

individuals who provide support and rate them on a 5-point Likert-type scale for the degree of useful information, reliable help, emotional uplift, caring, and trust they receive from them. Mean ratings across relationship categories yielded summary scores for emotional and informational support. Social network size was calculated as the total number of all social support contacts listed. Test-retest reliability is .66 and internal consistency (alpha) is .95.

HIV-Disease Classifications

The 1993 Centers for Disease Control (CDC) classification system for HIV infection was used to classify subjects within the 3×3 matrix of CDC classifications ranging from A1 (asymptomatic, with CD4+ cell count greater than or equal to 500 mL) to C3 (AIDS-indicated symptoms, with CD4+ cell count less than 200/mL).

RESULTS

Several analyses were conducted to address the causal relationship between social support and outcomes in HIV disease. These analyses used four waves of HNRC data. These data were collected at baseline, 6 months, 12 months, and 18 months.

The initial analyses involved simple bivariate correlations between CD4+ cells and size of social network. At each evaluation, there was a significant (or marginally significant) correlation between network size and immune status (i.e., CD4+ cell number). At baseline, the relationship was $r = .15, p = .008$; at 6 months, it was $r = .10$, $p = .08$; at 1 year, it was $r = .25, p = .001$; and at 18 months, the association was $r = .34$, $p = .001$. These findings confirm that social support and immune status are correlated at each point in time.

In order to investigate the causal pathway, a cross-lagged correlational model was employed. The cross-lagged correlation model attempts to determine the direction of causation. The model evaluates causation by examining asymmetries in the correlations between two variables over time. The basic question is whether network size is a strong predictor of CD4+ cells, or the alternative explanation, that CD4+ cells are a strong predictor of network size. The model considers the temporal sequence (Does the measurement of the predictor precede the measurement of the outcome?), the synchronous correlations, and the stability of the measures over time.

The model is shown in Figure 13.1. According to the logic of the cross-lagged model (Kenny, 1975), lagged correlations can suggest direction of causation. The lagged correlations are shown as diagonals on the figure. Figure 13.1 shows that baseline social support network size is not predictive of network size 18 months later ($r = .08, p = $ ns). On the other hand, CD4+ cell counts at baseline do predict these values 18 months later. Of particular importance are the diagonals in the figure. Baseline social support network size is uncorrelated with CD4+ cell number 18 months later. Conversely, baseline CD4+ cell level is a strong, significant predictor of network size 18 months following the initial evaluation ($r = .26, p = .002$). The temporal relationship favors the explanation that illness causes decline in social support over the alternative that low support causes progression of illness.

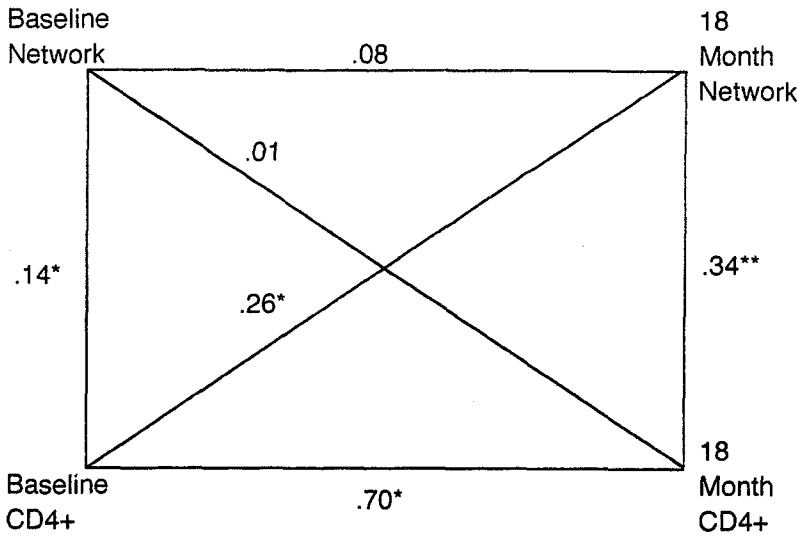

FIGURE 1. Cross-lagged panel model for network size and disease severity.

A variety of analyses were performed in order to evaluate this issue in greater detail. One approach involved breaking the subjects into smaller groups, based on their change in immune status over the 18-month evaluation. Since the study is still in progress, this allowed us to use only 78 subjects for whom data were complete. The four groups were those whose immune status was relatively high and stable (stable/high group, $n = 14$), those whose immune status was relatively low but stable (stable/low group, $n = 35$), those who began the study with relatively low immune status and declined slowly (slow-decline group, $n = 21$), and those who began the study with relatively high immune status and declined rapidly (rapid-decline group, $n = 8$). Figure 13.2 shows changes in CD4+ cells for these four groups. The lines for the two stable groups are relatively flat over the 18 months of follow-up. The rapid-decline groups shows a sharp fall over time, whereas the slow-decline group starts at a lower level and declines to levels reflecting more serious illness.

Figure 13.3 describes changes in social support network size for these four groups. The most important feature of Figure 13.3 is the line for the rapid-decline group. As demonstrated, those who have rapidly declining levels of immune status also show a significant decline in social support network size. As might be predicted, those with stable, higher levels of immune status maintain a relatively stable social support network size. Those with stable, low immune status begin with lower levels of social support, but show some nonsignificant increase over the course of time. The slow-decline group reflects the expected pattern through the first year of evaluation. However, this group does show an unexpected increase in network size (although not statistically significant) at the final follow-up. Overall, this analysis suggests that progression of illness, particularly for those with rapid changes in immune status, is followed by reductions in network size. Those with stable illness tend to have more stable social networks.

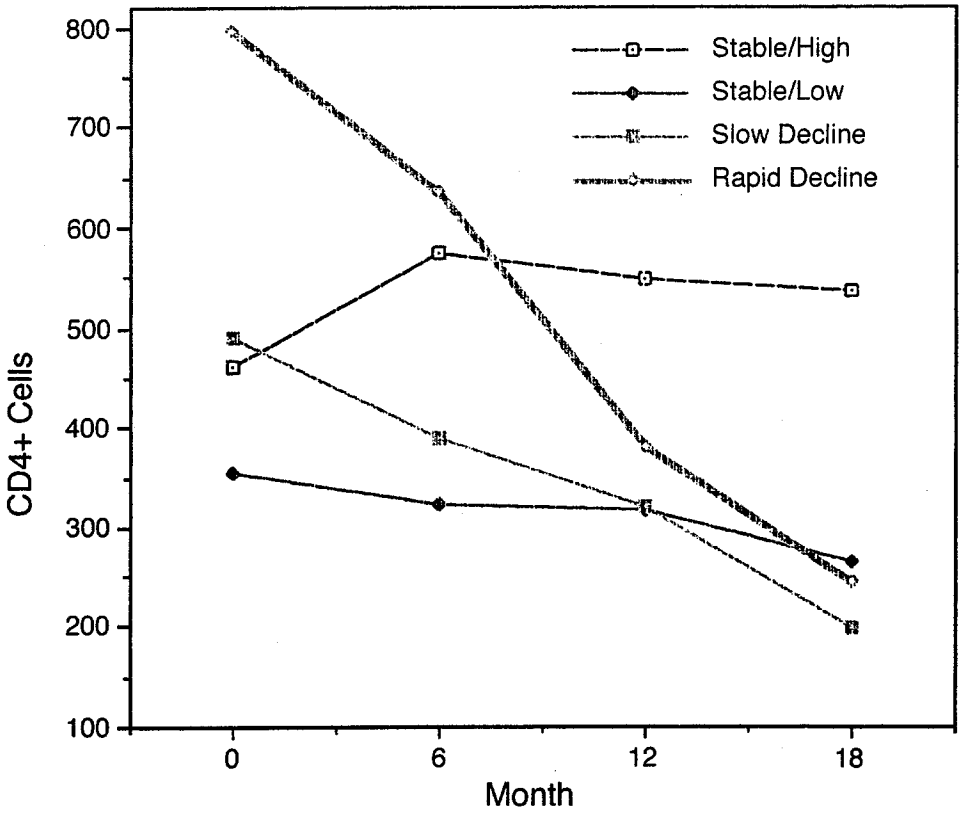

FIGURE 2. Changes in CD4+ cells for four CD4+ groups: Stable/High, Stable/Low, Rapid Decline, Slow Decline.

Detailed evaluations for measures of instrumental and emotional-social support did not reveal consistent patterns. Similarly, all evaluations for the HRSD were nonsignificant. Figure 13.4 shows the outcomes for the BDI. Interestingly, at all time periods, the rapidly declining group had the lowest BDI scores, and the slowly declining group had the highest BDI scores. These differences were not statistically significant at baseline or at 6 months. However, the differences between these two groups were statistically significant (as evaluated by analysis of variance) at the 12-month ($p < .02$) and the 18-month ($p < .05$) evaluations. The finding suggests that depression is related to the stability groupings. Furthermore, those with rapidly declining immune status started and ended the study with greater levels of depression than those whose illness progressed slowly. These data may suggest that depression is related to poorer prognosis. However, it is not possible to infer direction of causation from these results.

In order to investigate the relationship between advancing illness and social support further, we performed more detailed analyses on 52 subjects who had less than 200 CD4+ cells at the third visit. The threshold of 200 CD4+ cells was chosen, because this represents entry into the terminal stage of the illness. This stage, which may

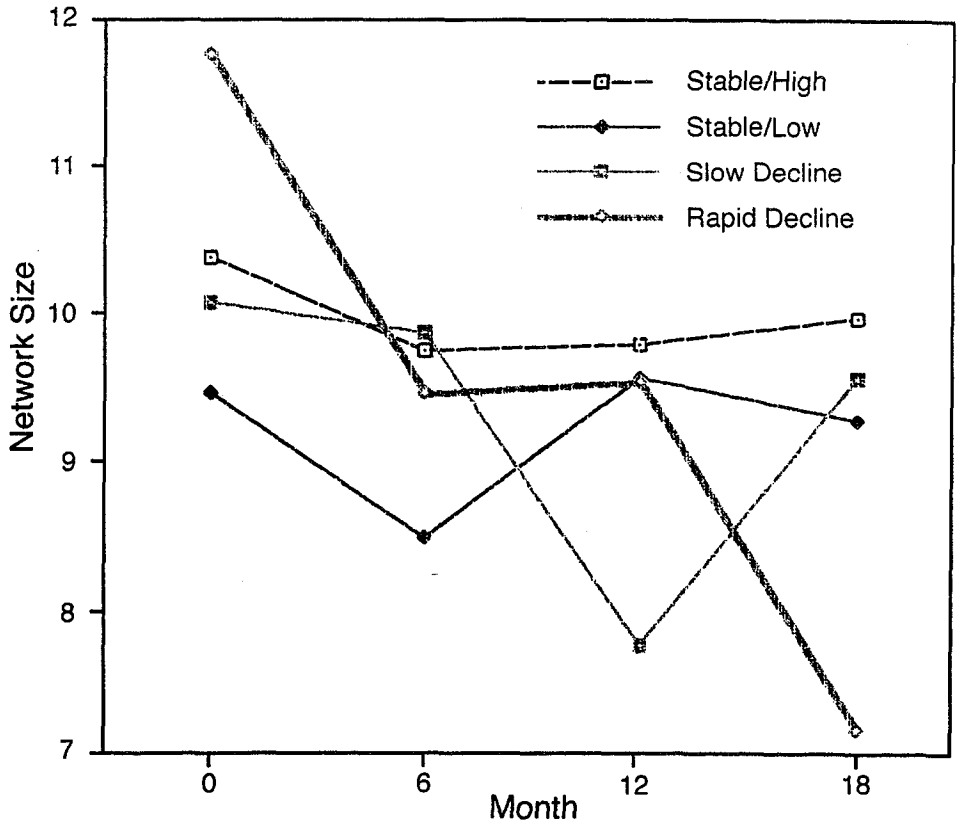

FIGURE 3. Changes in social network size for four CD4+ groups: Stable/High, Stable/Low, Rapid Decline, Slow Decline.

last for some years, is typically referred to as AIDS. These analyses focused on 31 subjects who made the transition from 200-500 CD4+ cells to the AIDS (less than 200 cells) category between the first and third visits. We refer to these subjects as the Transition group. For comparison, we considered 21 subjects who were in the AIDS category at both the initial and third visit (AIDS group).

Figure 13.5 shows the network size for the men at the initial and third visits. The figure demonstrates that, at the initial visit, the AIDS patients had smaller social networks than their peers, who, at the time, had not reached the diagnosis of AIDS ($t = 2.01$, $df = 51$, $p = .05$). At the third visit, when both groups were in the AIDS category, the network sizes were nearly identical ($t = -.11$, $df = 51$, $p = .91$). These findings provide more evidence that worsening illness causes network size to decline.

The findings for instrumental support are summarized in Figure 13.6. In contrast to network size, AIDS patients appear to receive more instrumental support than their peers in earlier stages of the illness. At the initial visit, instrumental support scores were significantly higher for AIDS patients ($t = -2.35$, $df = 51$, $p = .025$). However, at the follow-up, the instrumental support for those subjects making the transition from

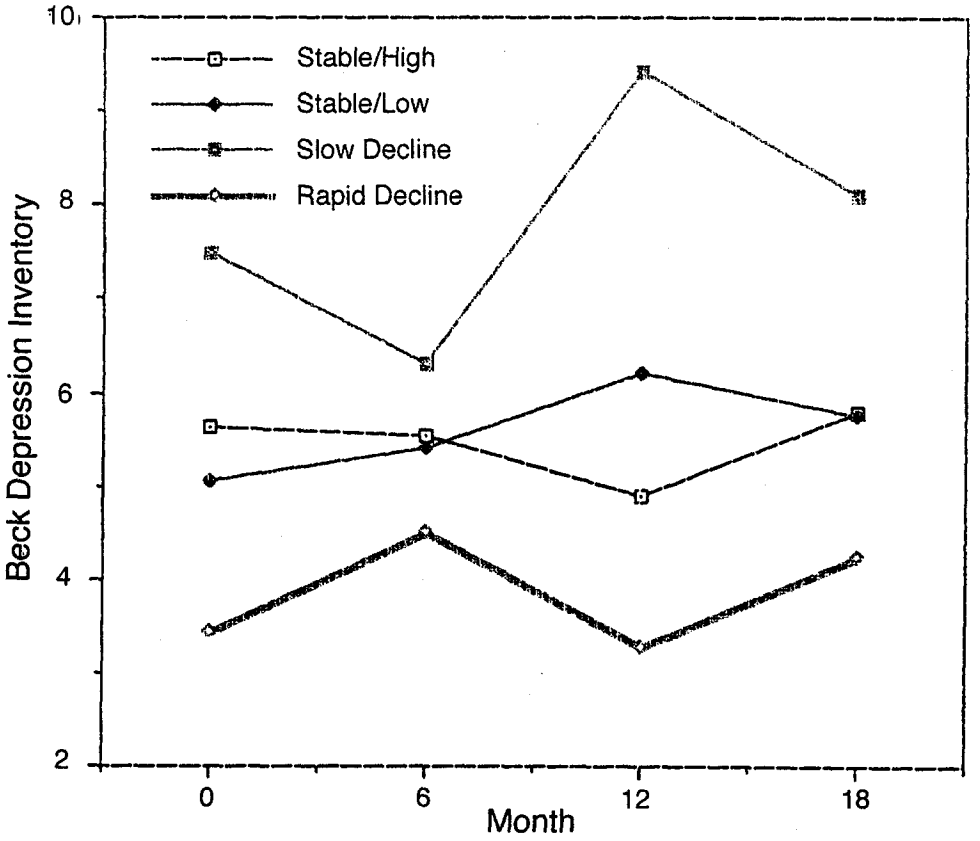

FIGURE 4. Changes in Beck Depression Inventory scores for four CD4+ groups: Stable/High, Stable/Low, Rapid Decline, Slow Decline.

HIV-positive to AIDS increased, whereas it remained steady for those who remained in the AIDS category. At the third visit, differences in instrumental support between these two groups were nonsignificant ($t = -.54$, $df = 51$, $p = .59$).

These differences could not be explained by differences in depression. Table 13.2 summarizes the depression outcomes. As the table shows, scores on both HRSD and BDI measures were comparable between these groups at each assessment. Furthermore, there were no differences in emotional support.

Summary of Results

In summary, we conducted a series of different analyses designed to investigate the relationship between social support and immune status. The cross-lagged correlation model attempted to evaluate the strength of two alternative causal pathways. The first pathway was that low social support caused reductions in immune status. Little evidence was provided to support this hypothesis. Alternatively, the model provided more evidence for the hypothesis that declining immune status causes reductions in

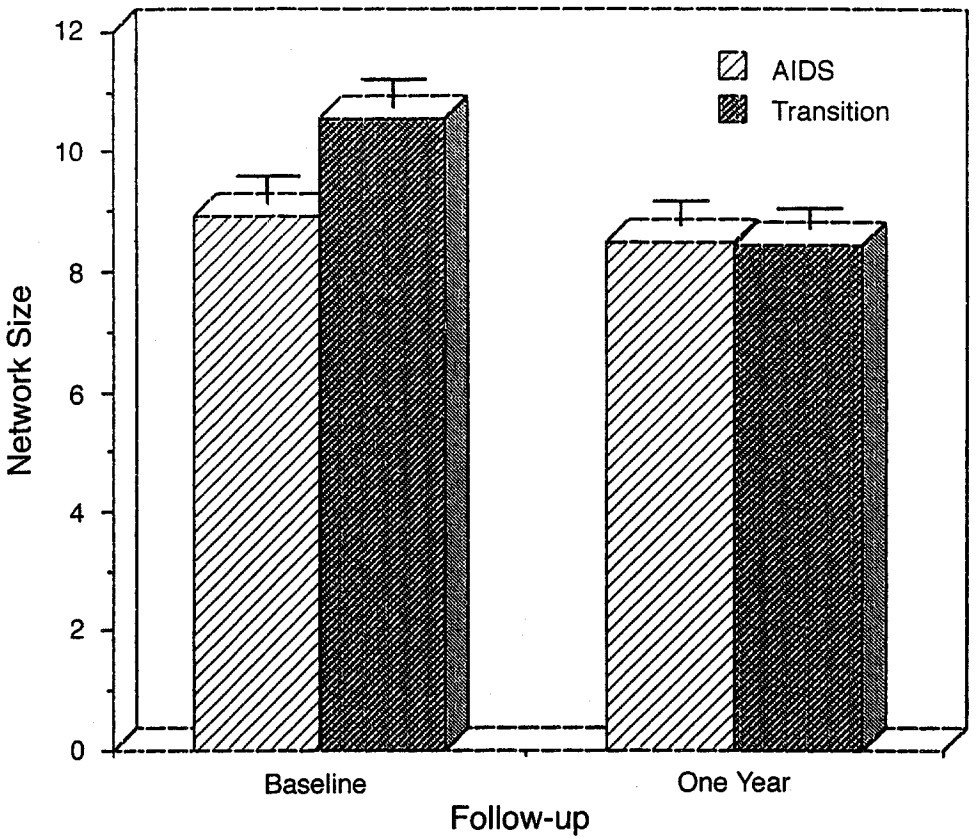

FIGURE 5. Network size for the men who had AIDS at the initial and third visit, and for those who made the transition to AIDS between the first and third visit.

social support network size. There are many problems with the crossed-lagged correlational model. One problem is that network size was not stable over the course of time. The low correlation between network size at baseline and at follow-up may indicate that network is measured with error. This problem could hinder the interpretation of the model.

A series of other analyses tended to support the hypothesis that declining health status causes reductions in social network size. For example, the group of patients with rapidly declining immune status also experienced systematic reductions in network size over the course of four evaluations that were spread over 18 months. Among patients who made the transition to AIDS status during the study, network size declined significantly. Although patients with rapid decline in immune status tended to be depressed throughout the study, the depression remained relatively stable. Thus, changes in depression could not explain changes in network size. Overall, these data appear to support the hypothesis that reductions in immune status and progression of HIV infection cause reductions in social support network size.

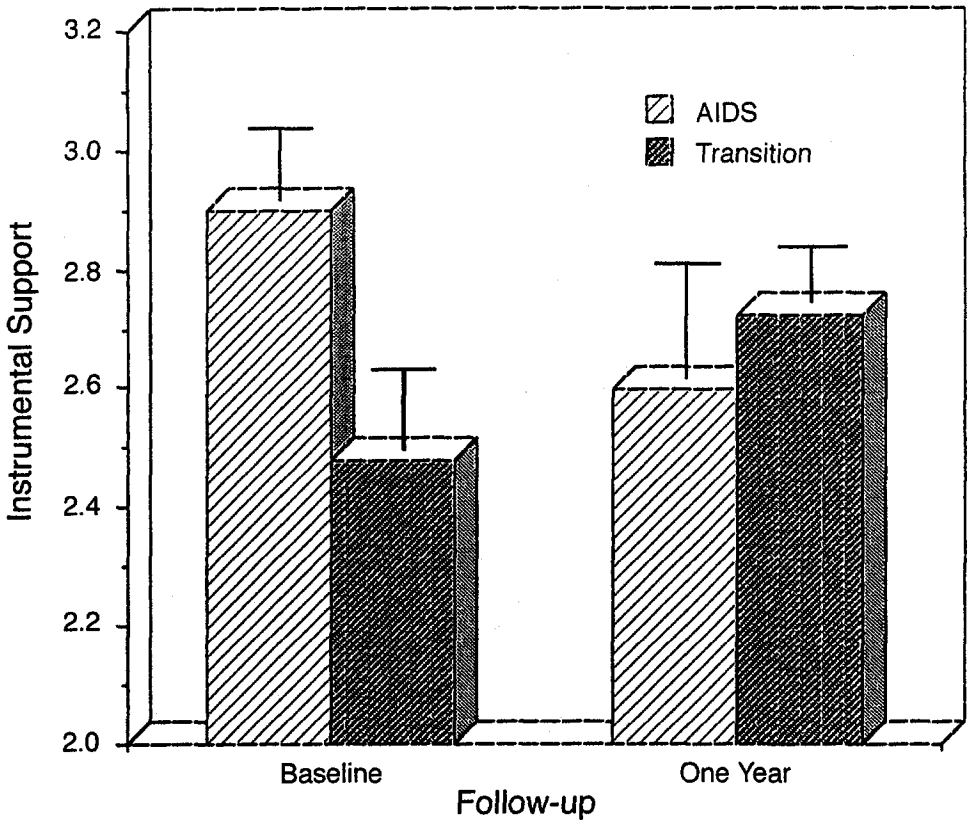

FIGURE 6. Instrumental support for the men who had AIDS at the initial and third visit, and for those who made the transition to AIDS between the first and third visit.

DISCUSSION

A substantial literature argues that people with small social networks are at risk for poor health outcomes (Berkman, 1995). In this chapter, we suggest an alternative explanation for the relationship between social support and health outcome. Specifically, we suggest that illness can cause modifications in the social environment. Studies of asthma, cancer, and other diseases are common stimuli for alterations in family

TABLE 13.2. Depression Changes for AIDS and Transition to AIDS Subjects

	Baseline				One year			
Measure	AIDS	Transition	*t*	*p*	AIDS	Transition	*t*	*p*
Beck	9.88 (8.66)	8.65 (9.55)	−.45	.66	10.25 (8.67)	9.83 (7.22)	−.18	.85
Hamilton	5.85 (5.92)	6.35 (6.60)	.28	.78	7.52 (6.48)	5.52 (4.14)	−1.36	.18

environment. For example, AIDS patients may be victimized by family members and friends. These potential supporters may feel uncomfortable interacting with someone who has an infectious disease. Disturbance in marital relationships often follow diagnosis and treatment of serious conditions such as heart disease. In diseases such as HIV infection, the illness almost certainly will interfere with intimate relationships (Semple et al., 1993).

Our findings are limited to HIV-infected men, and we are uncertain how well they generalize to other chronic disease groups. Nevertheless, we do feel they have some important implications. HIV-infected individuals represent an interesting population for the study of social support and health. They share much in common with other disease groups (e.g., deterioration of health status, reduced quality of life, increased contact with the healthcare system). However, HIV disease differs in interesting ways from other chronic disease groups, such as heart disease and cancer. Unlike other diseases, HIV is primary seen in young people who have not typically begun to come to terms with their own mortality. In addition, HIV carries a stigma that other diseases do not, due to its association with homosexuality and drug use. HIV-infected individuals are also likely to be poor and are often members of minority groups. Thus, HIV may be associated with a unique set of stressors not seen in other chronic disease groups. For many HIV-infected individuals, certain important aspects of social support may be less available then in other chronic diseases. This is especially true for the homosexual men, such as those who participated in this study. Marriage, for instance, one of the primary sources of structural support, is typically not available for this group (although significant partnership may be). In addition, many HIV-positive individuals lose many friends to the epidemic, further reducing their ability to receive social support.

Epidemiological studies usually attempt to control for disease severity. However, measures of disease severity are often inadequate. When controls for health status are entered into multivariate equations, it is likely that the analyses will underadjust, because the measures of health status simply do not reliably capture the construct. Another issue is that early phases of illness may interfere with social relationships. As people progress through illness, they may not be interested in walking, dancing, or leisure activities, and these may be the early symptoms of an undiagnosed condition.

CRITERIA FOR CAUSAL DIRECTION

Epidemiologists consider five criteria for establishing causal relationships. These are temporality, strength, consistency, gradient, and plausibility. We will address each of these briefly.

Temporality

Temporality occurs when the cause precedes the outcome. The crossed-lagged correlations and the analyses of specific groups suggest that reduced CD4+ cells occurs prior to reductions in social support network. Thus, the temporality data appear to support the alternative hypothesis that illness causes low support.

STRENGTH

The strength of association between social support network size and immune status is relatively weak. However, the magnitude of the relationship is approximately equal to that of other epidemiological associations.

CONSISTENCY

Epidemiologists argue that causal relationships must be established consistently across studies. Our data are preliminary. We are unaware of other studies that have reported this same relationship. Thus, the consistency criterion is not met by our observations. We encourage other investigators to evaluate this hypothesis using longitudinal data.

GRADIENT

For some, but not all, biological outcomes, there is a gradient between the causal factor and the outcome. For example, there is a systematic relationship between the number of cigarettes smoked and mortality. The data we present here suggest there is a systematic relationship between network size and immune status. Furthermore, this relationship becomes stronger over the lagged observations. Over the course of time, network size declines substantially, corresponding to reductions in CD4+ cells. This observation tends to argue in favor of the hypothesis that increasing illness causes reductions in social support network size.

PLAUSIBILITY

Epidemiologist argue that there must be some reasonable hypothesis that explains the relationship between a causal factor and an outcome. Many authors have argued that social support buffers stress, thus resulting in reduced disease impact. The alternative explanation suggests that illness limits social activity. For example, health outcomes are largely behavioral (Kaplan, 1990). As illness progresses, capabilities for performing activities of daily living decline. For example, men with advanced HIV infection may not have enough energy to attend parties, gather with friends, or perform usual role activities. Furthermore, the illness and the associated stigma may result in avoidance by friends and peers. Thus, the alternative hypothesis seems logically plausible. Indeed, it requires fewer assumptions than the stress-buffering model.

RELATED STUDIES

It would appear that our results are in contrast to the majority of published studies. However, upon reexamination, many studies could be reinterpreted within this framework. For example, in a recent study, we administered the social support questionnaire to adults with advanced lung disease. These patients were then followed prospectively over the course of 6 years. Using a Cox Proportional Hazard Model, social support

satisfaction significantly predicted survival up to 6 years. However, when the data were analyzed in more detail, it was noted that network size was related to disease severity at baseline. It is possible that increased severity of illness may interfere with an individual's ability to elicit, make use of, or be satisfied with the support that is obtained from others. In turn, this may deepen the sense of social isolation (Grodner et al., 1996).

A related study evaluated predictors of survival following cardiac surgery in the elderly. In this study, older adults who did not participate in community groups or feel comfort from religion were more likely to die within 6 months of surgery than those who were more socially active. The authors adjusted for biological variables and presurgical activity. However, these typically serve as an underadjustment in multivariate analysis. Nonparticipation in social activities may be a sensitive measure of severity of illness. Thus, we would expect that those who fail to participate in community groups may be more severely ill than those who are more active. Furthermore, those with declining health status may also feel less comfortable with religion (Oxman, Freeman, & Manheimer, 1995).

There have been relatively few studies of the progression of HIV illness in relation to social support. A report by Theorell and colleagues (1995) may, at first, appear inconsistent with our findings. These investigators studied a group of hemophiliacs in Sweden who were infected with the HIV virus. The subjects were divided into those with high or low availability of attachment. Subjects with high availability of attachment progressed more slowly than their peers with low availability of attachment. When considering these results, it is worth noting that availability of attachment is more similar to our instrumental social support variable than to network size. Indeed, our data show that instrumental support actually improves with severity of illness. In other words, availability of instrumental support may also be a consequence rather than a cause of HIV progression. Had we measured availability of attachment, we might have expected it to increase with advancing disease. It is also possible that social support functions differently among Swedish hemophiliacs than among American homosexual men.

SUMMARY

An extensive literature argues that high levels of social support provide protection against disease progression. An alternative explanation is that decreased social activity is a consequence rather than a cause of illness. Cohort data from HIV-infected men suggest that social network size declines as men become progressively more ill. Furthermore, our data indicate that illness progression precedes rather than antecedes declines in social network. Most published studies are based on cross-sectional observations that are unable to disentangle cause and effect. Our data suggest that social support should not always be conceptualized and investigated as a predictor of health outcome. Indeed, changes in social support may also be a consequence of illness. Furthermore, there may be reciprocal influences. The relationship between social support and outcome may be bidirectional.

We offer these observations to stimulate discussion. The study has many significant limitations. First, this study is based on a relatively small group of HIV-infected

men. We are uncertain as to whether these observations have any relevance for women or will be generalizable to larger groups. Another concern is that these data are based exclusively on HIV-infected men. HIV disease may represent a unique illness that is not like other chronic diseases. There is a serious social stigma associated with this illness that may not generalize to other diseases. Furthermore, HIV may be unique, in that it will almost certainly disrupt intimate relationships. Another problem is that HIV-infected men tend to associate with others with the same condition. Some of the reduction in social support network size may reflect losses in friends and lovers due to death. Unfortunately, we cannot determine this from the data available to us in the HNRC project.

Finally, it is important to emphasize that our findings apply only to social network size. Some of our evidence suggests that instrumental social support actually improves with advancing infection. In other words, as disease progresses, HIV-infected men may have fewer people in their support network. However, those who remain in the network can be counted on more than those who are lost.

In conclusion, there is sufficient evidence to believe that there is an association between social support and health outcomes. However, determination of the causal direction is deserving of further study.

DIRECTIONS FOR FUTURE RESEARCH

The relationship between social support and health outcome has fascinated investigators for several decades. However, few studies have performed longitudinal evaluations. Longitudinal studies are necessary in order to separate the causal pathways between support and health outcome. Most investigators believe that low social support causes poor health outcomes by providing an inadequate buffer against life stress. Data from patients with HIV infection suggest that the progression of illness may cause reductions in social support network size. We believe that the direction of causation may be even more complex. For example, life stress may inhibit immune status, allowing the disease to progress. Once the disease progresses, social support may be reduced. In turn, this may lead to an even poorer buffer against life stresses. Additional research is necessary in order to investigate these questions. We encourage investigators to include measures of social support in ongoing studies. Measurement of social process in epidemiological studies is typically inadequate. Few studies have considered satisfaction with support, and even fewer have attempted to build models that examine causal pathways.

In conclusion, few data are available to address the relationship between social support and health outcome. We believe this is a rich field for future investigation, and we encourage continuing explorations of these problems.

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