Published in final edited form as:

J Appl Soc Psychol. 2006 July 31; 31(6): 1111–1132. doi:10.1111/j.1559-1816.2001.tb02665.x.

Social Support Mediates Loneliness and Human Herpesvirus Type 6 (HHV-6) Antibody Titers

Denise Dixon, Stacy Cruess, Kristin Kilbourn, Nancy Klimas, Mary Ann Fletcher, and Gail Ironson

University of Miami

Andrew Baum

University of Pittsburgh Medical Center

Neil Schneiderman and Michael H. Antoni¹

University of Miami

Abstract

The current study investigated the impact of a severe environmental stressor and the role that declining social integration played in mediating its effect on loneliness and immune status. Increased loneliness and decreased social support in the months following the stressor (storm) were significantly associated with increased HHV-6 antibody titers, reflecting poorer control over the virus. Poorer social integration mediated the relationship between loneliness and HHV-6, even after controlling for nonspecific polyclonal B-cell activation, disease status (CD3+CD4+ cell counts), living arrangements, acute social losses (bereavement), and potential disruptions in social-support resources. These findings suggest that specific elements of social support may explain the oft-noted negative effects of loneliness on the immune system, and generalized to a medically vulnerable population.

Currently, HIV infection is perceived as a chronic, debilitating, life-threatening illness with inherent challenges to physical and psychological health. Persons with HIV frequently confront challenges related to feeling stigmatized by the general public, alienated from friends and family, and fearful of potential threats to their health and lives (Antoni et al., 1990,1991;Antoni & Schneiderman, 1998; Leserman et al., 1995,1999;Leserman, Perkins, & Evans, 1992;Miller, Kemeny, Taylor, Cole, & Vissher, 1997;Zuckerman & Antoni, 1995).

These chronic stressors, in addition to other psychosocial factors, such as social isolation, poor social support, and loneliness, have been associated with poorer immune functioning, including reactivation of latent herpesviruses in both healthy and medically vulnerable populations (Glaser & Kiecolt-Glaser, 1987; Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Glaser et al., 1987; Kiecolt-Glaser, Dura, Speicher, Trask, & Glaser, 1991; Kiecolt-Glaser et al., 1984a, 1984b, 1987, 1988; McLamon & Kaloupek, 1988). Reactivation of certain latent herpesviruses, such as human herpesvirus Type 6 (HHV-6), have been implicated in morbidity and mortality in persons infected with HIV (Ablashi, Bembau, & DiPaolo, 1995; Ablashi, Chatlynne, & Whitman, 1997; Blasquez, Madueno, Jurado, Fernandez-Arcas, & Munoz, 1995; Dolcetti et al., 1996; Knox & Carrigan, 1994, 1996;

Luppi & Torelli, 1996; Lusso & Gallo, 1995; Lusso, Garzino-Demo, Crowley, & Malnati, 1991). Stress has been implicated as having a greater impact on immune-compromised individuals (Antoni & Schneiderman, 1998; Glaser & Kiecolt-Glaser, 1987). For example, prior research has demonstrated age dependence in cellular immunity among depressed individuals (Guidi et al., 1998; Irwin et al., 1998; Schleifer, Keller, & Bartlett, 1999; Schleifer, Keller, Bond, Cohen, & Stein, 1989). Also, well-trained athletes appear to be more susceptible to infection in the hours or days following an event as a result, at least in part, of the effects of diminished cellular immunity following intensive training (Mackinnon, 1997).

HHV-6, described as the first T-lymphotropic human herpesvirus, was originally isolated from patients with AIDS and lymphoproliferative disorders in 1986 (Ablashi et al., 1995; Knox & Carrigan, 1994; Lusso, 1991). Although both the primary reservoir and mode of transmission remain poorly understood, HHV-6 has been implicated in the pathogenesis of HIV (Ablashi et al., 1995; Blasquez et al., 1995; Lusso & Gallo, 1995; Lusso et al., 1991). Like HIV, HHV-6 demonstrates a primary tropism for T-helper-inducer (CD3+CD4+) cells, and therefore may interact directly with HIV in individual cells and accelerate the kinetics of cell death (Lusso & Gallo, 1995).

Research indicates that HHV-6 induces HIV expression and enhances HIV replication through transactivation of the HIV long terminal repeat (LTR) (Lusso & Gallo, 1995; Lusso et al., 1995). While HHV-6 infects CD3+CD4+ cells primarily, it can also infect and replicate in natural killer (NK) and T-suppressor-cytotoxic (CD3+ CD8+) cells (Lusso & Gallo, 1995; Lusso et al., 1991), with implications for the abatement of anti-HIV cellular immunity. Also, HHV-6 has been isolated in fibroblasts, epithelial cells, megakaryocytes, neural cells, and rarely in B cells (Lusso & Gallo, 1995). Moreover, HHV-6 up-regulates and induces *de novo* expression of the CD4+ receptor, thereby broadening the cellular host range of HIV (Lusso & Gallo, 1995; Lusso et al., 1991). Following primary infection, HHV-6 remains latent in CD3+CD4+ cells until reactivated (Lusso & Gallo, 1995). While the precise mechanism remains unclear, transient or sustained immune suppression of the host has been implicated in HHV-6 reactivation (Ablashi, Chatlynne, & Whitman, 1997; Lusso & Gallo, 1995).

As mentioned, psychosocial factors such as social support and loneliness have been associated with the reactivation of human herpesvirus infections, as indicated in elevated antibody titers to HHV-6 (Cruess et al., 2000; Dixon et al., 1998, 1999; Glaser et al., 1985, 1987; Glaser & Kiecolt-Glaser, 1987; Kiecolt-Glaser et al., 1988; McLamon & Kaloupek, 1988). Also, research to date has determined the importance of social-support networks in maintaining overall psychological and physical health (Broadhead et al., 1983; Cohen, 1988; Cohen & McKay, 1984; Cohen & Syme, 1985; Cohen & Wills, 1985; Leserman et al., 1999; Penninx et al., 1998; Wortman, 1984). In particular, social support impacts both immediate and longer-term health of individuals infected with HIV (Antoni et al., 1990, 1991; Antoni & Schneiderman, 1998; Leserman et al., 1999; Turner, Hays, & Coates, 1993; Zuckerman & Antoni, 1995). Past research has established that social support buffers the effects of acute or chronic stress on psychological and physical health (Cohen & Wills, 1985; Dixon et al., 1998, 1999; Penninx et al., 1998). Both direct and indirect mechanisms for immediate and longer-term health outcomes have been postulated (Antoni et al., 1990; Antoni & Schneiderman, 1998; Cohen & Wills, 1985; Leserman et al., 1999; Penninx et al., 1998).

Studies examining components of social support have suggested that both total and individual components of perceived social support are associated with lower levels of depression, hopelessness, anxiety, and loneliness (Antoni et al., 1990; Antoni & Schneiderman, 1998; Hays, Chauncey, & Tobey, 1990; Hays, Turner, & Coates, 1992;

Kiecolt-Glaser et al., 1988; Miller et al., 1997; Namir, Alumbaugh, Fawzy, & Wolcott, 1989; Penninx et al., 1998; Turner et al., 1993). Further, loneliness has been implicated in short- and long-term morbidity and mortality in healthy and medically vulnerable populations (Herlitz et al., 1998), although this has not been a consistent finding in HIV-infected persons (Miller et al., 1997). Miller et al. attributed these inconsistent findings to potential differences in mode of transmission, to the disease status of the individual, or to uni-identified mediators of this relationship. However, they found that factors such as medication use, sexual risk behaviors, social withdrawal, bereavement, AIDS-related symptoms, repressive coping, finding meaning and personal growth, or the presence of a primary romantic partner did not mediate the relationship between loneliness and immune status in HIV-positive (HIV+) gay men. Therefore, they stated a need to explore further other potential mediators of the relationship between loneliness and immune-system status.

Psychological distress reactions are well documented in persons experiencing extreme environmental stressors, including natural disasters such as hurricanes and earthquakes (Adams & Adams, 1984; Bland, O'Leary, Farinero, Jossa, & Trevisan, 1996; Carr et al., 1995, 1997; David et al., 1996; Davidson, Hughes, Blazer, & George, 1991; Ironson et al., 1997; Lima, Pai, Santacruz, Lozano, & Luna, 1987; Lima et al., 1993; McDonnell, Troiano, Barker, Noji, & Hlady, 1995; Rubonis & Bickman, 1991; Schnurr, 1996). For example, among hurricane survivors, Ironson et al. found that greater reported levels of loss, negative (intrusive) thoughts, and post-traumatic stress disorder (PTSD) were associated with decreased natural-killer cell cytotoxicity (NKCC), while higher white-blood cell (WBC) counts were significantly related with greater amounts of loss and PTSD experienced. Further, among earthquake victims, Solomon, Segerstrom, Grohr, Kemeny, and Fahey (1997) found that subjects reporting high distress had lower numbers of CD3+ and CD3+CD8+ cells and a lower proliferative response to PHA. Also, lower levels of social support have been associated with higher postdisaster psychological distress in these populations (Carr et al., 1995; Dixon et al., 1998, 1999). Since prior work indicates that social support may directly influence both mental and physical well-being in HIV-infected persons (Miller & Cole, 1998), it is important to evaluate how social-support losses after major environmental stressors may be associated with both psychological distress and health in these immune-compromised individuals.

We tested whether increased feelings of loneliness following the hurricane were associated with increased HHV-6 antibody titers, reflecting poorer immune status. We also determined if perceived social support was associated with HHV-6 antibody titers. Finally, we investigated the role of perceived social-support losses as a mediator of the association between loneliness and HHV-6 titers in HIV+ gay men dealing with the aftermath of the storm.

Method

Participants

Forty-four HIV-infected gay or bisexual men agreed to participate in a study investigating psychological and physiological consequences of Hurricane Andrew. Subjects were recruited through flyers, HIV/AIDS service organizations, and special immunology clinics at two major hospitals in the Southeast. Only those individuals residing in Dade County, Florida, both at the time of Hurricane Andrew as well as at the time of entry into the study (within 6 months following the storm) were included as subjects. Two men were excluded from the study because they were residents of Dade County during the hurricane's land-fall. All but one of the subjects were asymptomatic or mildly symptomatic at the time of the first assessments. Two subjects were dropped from the study because they did not complete the assessments, leaving a total of 40 subjects.

This sample has been described in detail elsewhere (Benight et al., 1997). In brief, our sample ranged in demographic and psychosocial characteristics as indicated in Table 1. Forty-two participants completed psychosocial packets.² The mean age of participants was 37.21 years ($SD = 7.89$). The majority of these men (50%) were well educated, with 63.1 % of them reporting at least a college degree. Approximately half of the men indicated that they received an income of more than \$20,000 per annum. The ethnic composition of the sample reflected that of the greater Miami area, with the majority of individuals identifying with non-Hispanic White (44.7%) and Hispanic (39.6%) ethnic groups. The majority of men (62%) reported that they lived with someone at the time of the storm.

Procedure

Subjects were assessed within 6 months of the landfall of Hurricane Andrew (August 24, 1992), beginning on October 1, 1992, and continuing through February 24, 1993. Subjects were first assessed for anxiety and depression using the Structured Interview Guide for Anxiety and Depression (SIGH-AD; Hamilton, 1960) and for cognitive deficits using the Mini Mental Status Exam (MMSE; Folstein, Folstein, & McHugh, 1975). All potential subjects were required to meet specific cutoff scores on these instruments (<25 on the MMSE, and a depression rating of <15 on the SIGH-AD). Subjects with depression scores greater than or equal to 15 were referred to a mental-health practitioner for care. The primary purpose of this screening was to exclude individuals with any major cognitive impairments or major psychopathology, and to create a relatively homogeneous sample of mentally healthy individuals. All of the potential subjects who met these criteria were given a psychosocial assessment packet with instructions and were scheduled for a second appointment 1 week later, at which time they returned their questionnaires, had their blood drawn, and were paid \$25 for their participation.

Psychological Measures

Revised UCLA Loneliness scale—This scale (Russell, Peplau, & Cutrona, 1980; Russell, Peplau, & Ferguson, 1978) is a 20-item questionnaire used to measure one's subjective satisfaction with interpersonal relationships. Subjects responded to questions along a 4-point Likert scale ranging from 1 (*I have never felt this way*) to 4 (*I have felt this way often*). Negative items were reversed and then summed with positive items in order to provide an overall loneliness score. The scale has demonstrated adequate reliability and validity (Knight, Chisholm, Marsh, & Godfrey, 1988; Russell et al., 1978, 1980).

Social support—Social support was measured using subscales from the Social Provisions Scale (SPS; Cutrona & Russell, 1987), a 24-item self-report scale that measures perceived social support across several areas, and from the Interpersonal Support Evaluation List (ISEL; Cohen & Hoberman, 1983). Together, these subscales assessed subjects' perceptions of social support, including the dimensions of attachment, belonging, guidance, reliable alliance, reassurance of worth, social integration, tangible social support, and total social support. Subjects rated items along a 4-point Likert scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*), based on the degree to which each question represented the provisions received from one's current social-support network. The total score was derived by summing all of the subscales. Both the total scale and the sub-scales have demonstrated adequate reliability and validity (Cohen & Hoberman, 1983; Cutrona & Russell, 1987).

²Only 2 subjects that had been recruited initially were excluded from the study, after we discovered that they had not been living in the Miami area during the hurricane.

Immune Measures

The immune measures for this study included T-helper/inducer (CD3+CD4+) cell counts, Immunoglobulin (IgG) antibody titers to HHV-6, and total IgG levels. Total IgG levels were determined to control for nonspecific polyclonal B-cell activation. Rate nephelometry (ICS Analyzer II; Beckman Instrument, Brea, CA) was used to quantitate IgG levels following a rate kinetic method outlined by Fritsche and DeLeon (1975). Sera were diluted with a nonionic detergent buffer. Diluted samples were aliquoted into cuvettes containing polyethylene glycol (PEG) reactant buffer to increase reaction rate and decreased complex solubility. Monoclonal antibody (Mab) to IgG was determined from the standard curve for each Mab using the maximum rate.

Lymphocyte Phenotyping

A phlebotomist collected peripheral venous blood at ambient temperature between the hours of 8:00 a.m. and 12:00 noon using sterile evacuated tubes containing sodium heparin (Vacutainer, cat# 6489, Becton-Dickinson, Rutherford, NJ). Single laser flow cytometry (EPICS C, Coulter Instruments Laboratories, Hialeah, FL) using whole-blood samples and three-color immunofluorescence procedures determined the number and percentage of T-cell lymphocyte phenotypes, with fluorescein isothiocyanate (FITC), rhodamine (RDI), or energy-coupled dye (ECD) as the directly conjugated Mabs. Samples were prepared with a Coulter MultiQPrep System (Coulter, Hialeah, FL), and lymphocyte counts were obtained from the CBC with a Coulter MaxM. Whole blood (100 μ L) was incubated with Mabs for 10 min at 23°C with shaking. A whole-blood lysis method was used to eliminate erythrocytes, and the specimens were washed once prior to analysis. Stained specimens were analyzed with the Epics Elite Analyser flow cytometer utilizing the 488 nm laser line for quantification of percentage of positive cells by direct immunofluorescence. Bit maps were set on the lymphocyte population of the forward-angle light scatter versus 90-degree light scatter histogram. Percentage of positively stained cells for each marker pair, as well as doubly stained cells, was determined with Prism software (Beckman-Coulter, Hialeah, FL). The percentage values were converted to absolute counts by multiplying the lymphocyte counts obtained from the MaxM hematology counter (Beckman-Coulter, Hialeah, FL).

HHV-6

Detection of IgG antibodies to HHV-6 was measured with Indirect Fluorescence Assay (IFA; Stellar Bio System, Columbia, MD). Subject sera were applied to cultured cells containing inactivated viral antigens in wells on glass microscope slides. Specimens were incubated for 30 min to allow for the formation of antigen/antibody complexes with the HHV-6 antigens in infected cells. A brief washing step removed nonspecific antibody and other unreacted serum proteins. Cells were incubated for 30 min with fluorescein-conjugated goat anti-human IgG, then washed to remove unreacted conjugate before viewing with fluorescence microscopy. Antibody values were determined with the highest dilution factor at which HHV-6 antibodies were detected. Values were then log-10 transformed to normalize the distribution. Only subjects with sufficient samples for the IFA and with positive titers to HHV-6 were included in the statistical analyses.

Control Variables

A number of self-reported hurricane-related variables were investigated in this study as potential control variables. Subjects were asked if they either moved out or were forced to evacuate their homes as a consequence of Hurricane Andrew. Estimated material damage as a result of the storm was assessed on a 4-point Likert scale across six areas. Specifically, subjects were asked to rate the amount of hurricane-related damage to their homes, including their roofs, windows, and possessions, and to their automobiles. In addition, they

were asked to assess the amount of hurricane-related water damage, as well as other miscellaneous damage. These responses were rated on a 4-point scale ranging from 0 (*no damage*) to 3 (*major-damages*). These scores were summed to yield a *total damage score*, which ranged from 0 to 18. This measure of damage was consistent with the measure of damage used in the Community Hurricane investigation (Ironson et al., 1997), with which the present men were compared. Also, we evaluated the health status of subjects through the determination of CD3+CD4+ counts and HIV-related symptoms.

Results

Social Support, Loneliness, and HHV-6

Means and standard deviations for the SPS (Cutrona & Russell, 1987), ISEL (Cohen & Hoberman, 1983), and Revised UCLA Loneliness scale (Russell, Peplau, & Cutrona, 1980; Russell, Peplau, & Ferguson, 1978) are reported in Table 2. The SPS and ISEL values are similar to those that we have documented in other samples of HIV-infected gay men (Lutgendorf et al., 1998).

The relationships among the control variables, psychosocial variables, and CD3+CD4+ cell counts are presented in Table 3. There were significant intercorrelations among the social-support subscales and loneliness. However, there were no significant relationships between the control variables (living arrangements, moving out or forced evacuation, or estimated damage) and either social support or loneliness. In addition, there were no significant relationships between the control or psychosocial variables (social support or loneliness) and CD3+CD4+ cell counts.

Zero-order correlations, standardized beta weights, and semipartial correlations are presented in Table 4 for each of the separate hierarchical regression analyses, with social support and loneliness as predictor variables, regressed against HHV-6 antibody titers as the criterion variable. First, we performed a loglinear base 10 ($\log_{10}$) transformation of HHV-6 antibody titers in order to obtain an approximately normal distribution for this measure. For each equation, we entered CD3+CD4+ cell counts (as an index of disease status) and total IgG (as an index of nonspecific polyclonal B-cell activation), followed by the dichotomous variable of living arrangements (living alone vs. living with someone else) in separate blocks. Next, we entered either loneliness, or subscale or total scores for social support as predictor variables, each regressed against the $\log_{10}$ HHV-6 antibody titers. Attachment, social integration, reassurance of worth, guidance, total social support, and loneliness were each significantly associated with HHV-6 antibody titers after controlling for disease status, nonspecific polyclonal B-cell activation, and living arrangements.

Exploration of Mediators of Social-Support Relationships

Next, we explored possible mediators of the significant relationship between loneliness and HHV-6. We considered both total social support and subscale scores as potential mediators between increased overall loneliness (predictor) and increased HHV-6 antibody titers (criterion). All of the social-support measures met our first criterion, that all three variables (predictor, outcome, and mediator) demonstrated significant correlations with one another. We then tested the mediator effect by utilizing multiple regression to perform a path analysis. While controlling for nonspecific polyclonal B-cell activation (IgG antibody titers) and CD3+CD4+ cell counts (as an index of disease status), we entered each of the social-support variables (prospective mediators) into the equation, followed by the UCLA Loneliness measure (predictor; Russell, Peplau, & Cutrona, 1980; Russell, Peplau, & Ferguson, 1978), all regressed against HHV-6 antibody titers (outcome). A variable was considered a potential mediator if the beta weight of the previously significant Loneliness $\times$

HHV-6 association became nonsignificant ($p > .05$) after including the mediator variable into the overall regression equation.

Because of the high degree of association among social-support subscales, we focus here on the one subscale that was most strongly associated with HHV-6 titers: social integration. Here we found that social-integration scores mediated the association between increased loneliness and HHV-6 during the recovery period following the hurricane. The path diagram for this relationship is presented in Figure 1. As can be seen, the beta weight between loneliness and HHV-6 was significant ($\beta = 0.54, p < .01$), but became nonsignificant ($\beta = -0.02, ns$) when social integration was entered into the regression. The beta weight between social integration and HHV-6 was significant before ($\beta = -0.60, p < .01$) and retained significance after loneliness was entered into the regression ($\beta = -0.48, p < .05$). The equation was significant, $F_{\text{Change}}(2, 30) = 4.18, p < .05$, and the incremental variance contributed by social-integration scores ($sr^2 = .12, p < .05$) indicates that this measure of perceived social support accounted for a significant proportion of unique variance in HHV-6.

Discussion

This study investigated the impact of a severe environmental stressor, Hurricane Andrew, and the role of social support and loneliness in determining immune status in HIV+ gay men in the period of early recovery after the storm. The findings from our study support our first two hypotheses: Lower social support and feelings of loneliness in the months after the hurricane were associated with higher HHV-6 antibody titers. Specifically, perceptions of less attachment, social integration, reassurance of worth, guidance, and total social support, and more loneliness were associated with higher HHV-6 antibody titers, reflecting poorer control over this herpesvirus.

Reactivation of HHV-6 has been related to poorer immune status in HIV-infected individuals, in that active infection with HHV-6 has been implicated in the pathogenesis of HIV. For example, HHV-6 induces HIV expression and enhances HIV replication in CD3+CD4+ cells, potentially interacting indirectly with HIV in individual cells, and perhaps accelerating cell death as a result (Ablashi, Bembbaum, and DiPaolo, 1995; Blasquez et al., 1995; Lusso & Gallo, 1995; Lusso et al., 1991, 1995). Other research has shown that increased psychological stress has been associated with diminished host cellular immunity (Glaser & Kiecolt-Glaser, 1997; Glaser, Kiecolt-Glaser, Malarkey, & Sheridan, 1998; Hou, Coe, & Erickson, 1996; Stefanski & Engler, 1998, 1999), which is essential in controlling the expression or replication of latent herpesviruses since viral-infected cells are killed primarily by natural-killer and cytotoxic T cells. It is important to note that measuring antibody titers represents an indirect way of exploring these relationships. The presence of antibodies to a virus reflects a history of viral exposure, and increases in antibody titers are indicative of viral expression and may reflect inadequate cellular immune response to viral antigen (Glaser & Kiecolt-Glaser, 1997).

The effects from our investigation held even after controlling for disease status, nonspecific polyclonal B-cell activation, and current living arrangements. In addition, associations could not be accounted for by either sociodemographic or other hurricane-related variables.³ Our results are consistent with prior research, demonstrating associations between immune-system decrements and measures of diminished social support or increased loneliness

³We considered the potential impact of the number of bereavements (past 6 months) on the relationship between loneliness and social support. While significantly correlated with both loneliness and social integration, bereavement failed to mediate the relationship between these two variables.

(Baron, Cutrona, Russell, Hicklin, & Lubaroff, 1990; Esterling, Antoni, Fletcher, Margulies, & Schneiderman, 1994; Glaser & Kiecolt-Glaser, 1987; Glaser et al., 1985, 1987; Kiecolt-Glaser et al., 1984a, 1984b, 1987, 1988, 1991; Leserman et al., 1999; McLamon & Kaloupek, 1988).

Moreover, the present study went on to determine that one form of perceived support, perceptions of social integration, mediated the relationship between feelings of loneliness and higher HHV-6 antibody titers. Thus, our results provide further evidence that social-support resources in the early recovery period of environmental stressors may influence immune status in medically vulnerable populations, such as HIV-infected gay men. These findings are consistent with past research that has determined associations between diminished social support and immune-system status in populations with HIV (Leserman et al., 1999), but further establishes social integration as an important mediator of the relationship between loneliness and immune status.

Social integration relates to a perception of belonging to a group that shares similar interests, concerns, and recreational activities, which in turn provides a sense of comfort, security, pleasure, and a sense of identity for the individual (Cutrona & Russell, 1987; Weiss, 1974). This social resource is acquired most often from friends (rather than family: Cutrona & Russell, 1987; Weiss, 1974). Therefore it may be particularly salient for HIV+ gay men, who may rely on gay culture to provide this important social resource, which they may find lacking within heterosexually based culture. Therefore, the lonelier gay men in our sample—those who perceived important deficits related to meaningful close relationships and intimacy—evidenced immune decrements, possibly because they saw themselves as alienated from their social ties, even perhaps the larger gay community. We determined that these findings held even after we controlled for hurricane-related variables, such as unforced or forced evacuation, or estimated damage as a result of the storm, suggesting that the phenomenon occurred across the range of hurricane victims.

Prior research has determined that other factors, such as time since HIV diagnosis; bereavement; health-compromising behaviors, such as alcohol or other substance use or abuse; caffeine consumption; and smoking relate to immune status in HIV-infected persons and thereby represent potential mitigating factors in the relationships between stressors, social support, and immune status (Antoni et al., 1990, 1991; Antoni & Schneiderman, 1998; Cruess et al., 2000; Ironson et al., 1990, 1994; Martin, 1988; Miller & Cole, 1998). When we conducted separate extensive analyses relating these other factors to immune status, our findings remained unchanged. In addition, our findings were independent of current living arrangements, suggesting that the source of social integration was likely outside the home.

Prior work with HIV+ persons measured social integration by determining the number of peers within the social network and failed to find consistent associations with immune status (Miller et al., 1997). We were able to detect the role of social integration's role as a protective factor, both independently and in mediating the impact of loneliness and poorer immune status. Our success may have been a result of our operationalization of social integration as the perception of adequate social needs, independent of actual quantity of social contacts (Cutrona & Russell, 1987; Weiss, 1974). These findings seem to enhance or complement prior research by identifying a link between a specific element of social support, loneliness, and immune-system status (Glaser & Kiecolt-Glaser, 1987; Glaser et al., 1985, 1987; Kiecolt-Glaser et al., 1984a, 1984b, 1987, 1988, 1991; Leserman et al., 1999; McLamon & Kaloupek, 1988). For example, our findings shed additional light on the research by Leserman et al., which demonstrated that cumulative stressful life events and depressive symptoms, combined with lower cumulative social support, was associated with

faster progression to AIDS in HIV-infected gay men. While all of the men in our sample suffered a severe environmental stressor, those men who described intense negative feelings related to lack of intimacy and meaningfulness and who felt less integrated within their social spheres demonstrated poorer immune-system status. Therefore, these men may present a risk for faster progression to AIDS in the years following the storm and could be identified as candidates for psychosocial interventions designed to enhance social support and interpersonal coping strategies (Lutgendorf et al., 1998).

A few caveats are in order. Given the small sample size, the results of this study should be considered preliminary in nature and, therefore, should be interpreted with caution. This study derived results from a population of highly educated gay men with more than adequate levels of income. Therefore, one cannot generalize these findings to other HIV+ populations, such as elderly persons, heterosexuals, women of color, poor or drug-involved individuals, persons with cognitive impairments, or clinically depressed or anxious individuals as determined by clinical interview. Also, we were limited in our ability to generalize the effects of Hurricane Andrew to other major disasters or types of severe stressors. Moreover, we were prevented from determining HIV/AIDS staging data, as stipulated by the Centers for Disease Control and Prevention, which limited the interpretation of our findings in terms of clinical disease status. However, based on CD3+CD4+ cell counts and raw number of symptoms, we did not find relevant associations between social support, loneliness, and disease status or progression.

In summary, this study provides important preliminary information regarding associations between diminished social integration and feelings of loneliness on the one hand, and HHV-6 antibody titer elevations in HIV+ persons during the early recovery period after a severe environmental stressor on the other. These medically vulnerable individuals, in great need of maximizing their immune-system functioning, were adversely affected by perceptions of loneliness and not feeling connected with their own peer networks. These findings suggest that social integration may represent the domain from which loneliness emanates in HIV-infected gay men during stressful events and through which loneliness relates to poorer immune-system status (i.e., increased HHV-6 antibody titers). Moreover, these findings suggest a need to carefully assess the experience of loneliness from the point of view of disrupted social integration in HIV+ individuals, and perhaps other socially marginalized populations undergoing challenging environmental stressors. Future research should use larger sample sizes and longitudinal designs to replicate the findings of this investigation with HIV-infected and other vulnerable populations exposed to severe stressors in order to develop appropriate intervention strategies designed to preserve and enhance social-support resources.

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Figure 1.

Path diagram for model testing social integration as a mediator of the association between loneliness and human herpesvirus Type 6 (HHV-6). All equations control for total Immunoglobulin G (IgG) and CD4 cell counts. Standardized beta weights for factors entered in model individually are within parentheses, and standardized beta weights with all factors in the diagram entered simultaneously are outside parentheses. Significant relationships are indicated by single (* $p < .05$), double(** $p < .01$), or triple(***) $p < .001$) asterisks. HHV-6_(ln) = loglinear (base 10) transformation of HHV-6 antibody titers, IgG=immunoglobulin G, CD4 cell counts=CD3+CD4+ cell counts.

Table 1

Description of the Sample

Variable	M	SD	Percentage
Age (in years)	37.21	7.89	
Income ($\geq$ \$20K/year)			50.0
Education			
Less than 8th grade			2.6
High school graduate			5.3
Trade school			2.6
Some college			26.3
College degree			36.8
Graduate degree			26.4
Ethnicity			
American Indian			2.6
Non-Hispanic White			44.7
Hispanic			39.6
African American			2.6
Haitian			2.6
Other or unknown			7.9
Living arrangements			
Live alone			34.5
Live with lover/partner			37.9
Live with roommate			10.3
Live with parents			13.8
Other			3.4
Hurricane-related variables			
Moved out			16.2
Evacuated			39.5
Material damage			68.4
Number of bereavements in past 6 months			
0			47.4
1			39.5
2			5.3
3			2.6
4			5.3

Table 2

Means and Standard Deviations of Social Provisions Scale (SPS), Interpersonal Support Evaluation (ISEL), and Revised UCLA Loneliness Scale

Variable	M	SD
SPS–Attachment	11.97	2.89
SPS–Social integration	12.92	2.76
SPS–Reassurance of worth	12.59	2.53
SPS–Reliable alliance	13.57	2.55
SPS–Guidance	13.51	2.36
ISEL–Belonging	6.53	1.30
ISEL–Tangible	9.81	1.98
SPS + ISEL–Total social support	88.73	14.30
Revised UCLA loneliness scale	42.10	12.22

Table 3

Correlations Among Control, Psychosocial Variables, and CD4 Cell Counts

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. SPS-Attachment ^a	—	.59***	.76***	.68***	.77***	.73***	.61***	.85***	-.80***	.23	-.06	.06	-.11	-.13
2. SPS-Social integration ^a		—	.63***	.75***	.73***	.74***	.70***	.84***	-.73***	-.10	-.19	-.11	-.04	-.16
3. SPS-Reassurance of worth ^a			—	.83***	.83***	.62***	.61***	.88***	-.81***	-.19	-.22	.06	-.30	-.14
4. SPS-Reliable alliance ^a				—	.89***	.64***	.73***	.91***	-.85***	-.21	-.29	.01	-.18	-.05
5. SPS-Guidance ^a					—	.72***	.77***	.94***	-.87***	-.13	-.23	-.18	-.19	-.04
6. ISEL-Belonging ^a						—	.74***	.83***	-.77***	.15	.05	.00	-.10	-.07
7. ISEL-Tangible ^a							—	.83***	-.64***	.01	-.21	-.12	-.07	-.26
8. SPS-ISEL total ^a								—	-.90***	-.06	-.20	-.05	-.17	-.14
9. Revised UCLA Loneliness scale									—	.10	.07	.03	.28	.01
10. LIVARRANGE ^a										—	.21	.11	.44*	.11
11. MOVEOUT ^a											—	.10	.10	.10
12. EVACUAT ^a												—	-.29	.04
13. ESTDAMAG ^a													—	.04
14. CD4 cell counts														—

ISEL total = total social support, LIVARRANGE = living arrangements, MOVEOUT = material damage.

^aSPS = Social Provisions Scale, ISEL = Interpersonal Support Evaluation List, SPS-moved out of home, EVACUAT = forced evacuation, ESTDAMAG = estimated* $p < .05$, two-tailed.*** $p < .001$, two-tailed.

Table 4

Summary of Hierarchical Regression Analyses for Variables Predicting HHV-6 Antibody Titers

Variable	HHV-6		
	Zero-order correlation	Standardized β	Semipartial correlation (sr^2)
Attachment	-.29 [*]	-.32 [*]	.10 [*]
Social integration	-.44 ^{**}	-.40 ^{**}	.17 ^{**}
Reassurance of worth	-.44 ^{**}	-.40 ^{**}	.17 ^{**}
Reliable alliance	-.30 [*]	-.26	.07
Guidance	-.36 ^{**}	-.34 [*]	.12 [*]
Belonging	-.21 [†]	-.23	.06
Tangible support	-.20	-.16	.03
Total social support	-.38 ^{**}	-.35 [*]	.13 [*]
UCLA Loneliness scale	.30 [*]	.29 [†]	.09 [†]

Note. Each regression equation controlled for CD4 cell counts, Total Immunoglobulin G (IgG), and living arrangements (living alone vs. with someone else). HHV-6=human herpesvirus Type 6, IgG = immunoglobulin G.

[†] $p < .10$, two-tailed.

^{*} $p < .05$, two-tailed.

^{**} $p < .01$, two-tailed.

^{***} $p < .001$, two-tailed.