

6

Psychosocial Stress, Social Networks, and Susceptibility to Infection

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During the past several decades, support has grown for the premise that psychological and social factors can influence physical health. This includes evidence that enduring stressful life events and prolonged negative moods (e.g., depression, anxiety, anger) can increase risk for physical illness and early death (e.g., reviews by Booth-Kewley & Friedman, 1987; Cohen & Williamson, 1991; Schneiderman et al., 1989). It also includes evidence that those who participate in diverse social networks that include family, friends, workmates, neighbors, and fellow members of social and religious groups live longer and healthier lives than their less socially adept counterparts (e.g., Berkman & Syme, 1979; House et al., 1988; Vogt et al., 1992).

My own interests in this area have focused on the potential impact of psychological and social factors on the immune system, and consequently on our ability to fight off infectious disease. Here I begin by providing a selective overview of the evidence for the effects of psychosocial factors on the functional capabilities of the immune system. I then discuss similar data linking these same factors to the onset and progression of infectious disease. I provide only cursory reviews and refer the reader to Cohen and Herbert (1996), Cohen, Miller, et al. (2001), and Herbert and Cohen

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(1993a, 1993b) for more detailed treatments. Finally, I suggest implications of this literature for understanding the role that religious participation might play in health.

Psychological Stress and Immunity

Over the last 15 years, dozens of studies have shown that stressful situations in people's lives are associated with alterations in immune function (see meta-analysis in Herbert & Cohen, 1993b). An example of this literature is a series of studies of the consequences of medical school examinations for cellular immune function reported by Glaser, Kiecolt-Glaser, and colleagues (e.g., Glaser, Pearson, Bonneau, et al., 1993; Glaser, Pearson, Jones, et al., 1991; Kiecolt-Glaser, Garner, et al., 1984). In these studies, medical students had their psychological stress responses and immune responses assessed during a low-stress baseline period (e.g., just following vacation) and again during a series of important exams. Students reported more stress during exams and showed a decrease in the function of cellular immune responses, including decreased NK activity, lymphocyte proliferation, and increased antibody to herpesviruses.

Chronic stressful events that last for a longer term (e.g., months or even years) have similar potential to influence the immune system. One example is the set of studies that assessed stress effects on residents of the area surrounding the Three Mile Island (TMI) nuclear power plant. TMI was the site of a serious accident in 1979 that released nuclear materials into the surrounding environment, and the level of distress among area residents remained quite high afterward (Baum et al., 1983). In fact, almost 10 years after the accident, McKinnon et al. (1989) found higher numbers of neutrophils and fewer B cells, suppressor/cytotoxic T cells, and NK cells in TMI residents than in demographically matched control subjects living in another area. They also found more antibody to herpesviruses in TMI residents, which suggests lower cellular immune competence.

Another chronic stressful event, providing care for Alzheimer's patients, has been associated with elevated levels of herpes antibodies (a marker of cellular immune suppression) (Kiecolt-Glaser & Glaser, 1987), and less production of antibody in response to an influenza immunization (Glaser, Kiecolt-Glaser, Malarkey, et al., 1998; Kiecolt-Glaser, Glaser, Gravenstein, et al., 1966).

Mood and Immunity

The effects of psychological stress on immunity are generally thought to be mediated by stress-elicited increases in negative mood. In turn, negative mood might trigger the release of immune-altering hormones, such as epinephrine, norepinephrine, and cortisol, or influence behaviors that might directly influence immune tissue, such as increased drinking and smoking and loss of sleep. Consequently, it is not surprising that the associations between negative moods and immunity generally parallel the psychological stress literature. For example, in nonclinical samples—that is, people who are not being treated for medical disorders—depressed mood is associated with decreased proliferative responses to mitogens (substances used to stimulate immune cell division) and decreased NK activity (see meta-analysis by Herbert & Cohen, 1993a). Moreover, similar (in quality) but more profound associations are found for clinically depressed patients.

Although it has received considerably less attention, anxiety appears to be associated with immune changes as well. For example, an anxious mood has been associated with decreased natural killer (NK) cell activity (Locke et al., 1984) and decreased proliferative response to both phytohemagglutinin (PHA) and concanavalin-A (Con-A), two commonly used laboratory mitogens (Linn, Linn, & Jensen, 1981).

Only a few studies have examined the association of positive mood states with immune outcomes. One daily diary study examined the relation between positive and negative mood states and antibody response to an orally ingested novel antigen over 8 weeks (Stone, Cox, et al., 1987). Antibody levels were higher on days when respondents reported high positive mood states and lower on days when they reported high negative mood states. These results were replicated in a subsequent study that monitored mood and antibody levels over a 12-week period (Stone, Neale, et al. 1994).

In two experimental studies, positive and negative affective states were induced in healthy subjects, and subsequent acute changes in immunity were documented. For example, Knapp et al. (1992) had subjects recall positive and negative experiences to induce "positive" and "negative" mood states. Both positive and negative moods were associated with decreased proliferative responses to PHA and increased numbers of neutrophils. Similar immune effects of positive and negative moods were attributed to

the fact that all subjects reported increased levels of excitement (arousal) during the mood inductions, regardless of the direction, or valence, of the mood.

Futterman et al. (1994) used actors reading written scenarios that depicted four different emotional states—(1) high arousal with positive mood, (2) high arousal with negative mood, (3) low arousal with positive mood, and (4) low arousal with negative—to induce these mood states in experimental subjects. Although NK activity was not associated with mood condition, lymphocyte proliferation did increase following positive moods and decreased following negative moods.

Thus, different moods may be associated with different immune responses. Clear interpretation of this work is impeded by lack of consensus on the dimensions on which mood should be classified. However, existing work suggests that the dimensions of valence and arousal may be important ones in relating moods to immune function.

Psychological States and Susceptibility to Infection

Invasion of the body by a disease-causing agent is not sufficient cause for disease. Rather, disease occurs when the host's defenses are compromised or unable to recognize the foreign material as posing a threat. Therefore, psychological variables that influence immunity have the potential to influence the onset and progression of immune system-mediated diseases. What is less clear is whether psychologically induced changes in immunity are of the magnitude or type that would alter the ability of the body to fight disease (Cohen & Williamson, 1991; Laudenslager, 1987; O'Leary, 1990).

The following discussion is limited to studies that address the role of psychological factors in the onset and progression of upper respiratory viruses and herpesvirus. Chapter 8 contains a detailed discussion of the role of stress and related psychological barriers in human immunodeficiency virus (HIV) infection.

Upper Respiratory Infections

Early prospective work by Meyer and Haggerty (1962) indicated that both disruptive daily events and chronic family stress were associated with greater risk for upper respiratory infections. Similar results were reported

by Graham et al. (1986), who collected measures of life stress from members of 94 families before and during a 6-month period in which diary data on subjects' respiratory symptoms also were collected daily. Illness episodes were validated by nose and throat cultures. Although high- and low-stress groups were almost identical with respect to demographics and health practices, the high-stress groups experienced more verified episodes of illness and more days with symptoms of respiratory illness.

In a study of susceptibility to influenza, Clover and colleagues (1989) assessed family relationships and individual stressful life events of 246 individuals in 58 families prior to the start of flu season. This study found that "stressed" families (i.e., those living in a rigid and chaotic environment) had a greater incidence of disease than nonstressed families (i.e., those living in a more "balanced," harmonious environment). Illness was not related to individual stressful life events in this study.

One might easily argue that the increased incidence of upper respiratory infections in individuals under stress in these studies may be attributable to stress-induced increases in exposure to infectious agents rather than to stress-induced immunosuppression. However, three recent viral-challenge trials suggest otherwise.

In a study of 394 volunteers, Cohen, Tyrrell, and Smith (1991, 1993) administered measures of stressful life events, perceived stress, and negative affect just before intentionally exposing subjects to one of five viruses that cause upper respiratory infections, better known as the common cold. We were interested in whether reports of stress could predict who would become ill. Only about 40% of exposed subjects develop colds, and all three stress measures predicted the probability of developing a cold, with greater stress linearly related to greater risk. Moreover, the relationships that we reported between increased stress and greater susceptibility to infection were found consistently across five different upper respiratory viruses. These results could not be explained by stress-elicited differences in health practices, such as smoking and alcohol consumption, or in the numbers of various white blood cell populations or total (nonspecific) antibody levels.

In another viral-challenge study, Stone, Bovbjerg, and colleagues (1992) replicated the relation between stressful life events and susceptibility to upper respiratory infections reported in Cohen, Tyrrell, and Smith (1991). Additionally, in a more recent study, Cohen, Frank, et al. (1998) demonstrated that although chronic stress predicted susceptibility to upper respiratory infections, acute stress did not. In this study, we administered a "life

stressor" interview and psychological questionnaires to 276 volunteers and also collected blood and urine samples from this group. We then inoculated the volunteers with common cold viruses and monitored them for the onset of disease. Although severe acute stressful life events (less than 1 month long) were not associated with developing colds with exposure to a virus, severe chronic stressors (1 month or longer) were associated with a substantial increase in risk of disease. This relation was attributable primarily to underemployment or unemployment and to enduring interpersonal difficulties with family or friends. Interestingly, this study found that the association between chronic stressors and susceptibility to colds could not be fully explained by differences among stressed and nonstressed persons in social network characteristics, personality, health practices, or prechallenge endocrine or immune measures.

In another set of studies, we focused on psychological predictors of disease severity (rather than episode onset). We found that state (but not trait) negative affect measured just prior to viral exposure was associated with more severe colds and influenza, as measured by the amount of mucus produced over the course of the illness (Cohen, Doyle, Skoner, Fireman, et al., 1995). Another study focusing on the acute stress reported at the time of viral inoculation produced similar results. Indeed, those reporting greater stress had more severe illnesses after infection by an influenza virus (Cohen, Doyle, & Skoner, 1999). Moreover, the expression of illness symptoms was highly interrelated with the release of too much of a chemical messenger (IL-6, a pro-inflammatory cytokine). IL-6 is released as part of the immune system response to infection (see chapter 3). A stress-induced failure to "turn off" the release of this cytokine might be responsible for the increased severity of illness for those reporting more stress.

In sum, both stressful life events and psychological stress (perceptions and negative affect) are associated with increased susceptibility to upper respiratory infection. These effects are not generally explicable in terms of stress-elicited changes in health behaviors. Moreover, initial evidence suggests that these associations might occur because stress is associated with a dysregulation of immune response.

Herpes Simplex Virus Infection

Herpesviruses are thought to be responsible for cold sores, genital lesions, infectious mononucleosis and mononucleosis syndrome, and deafness in

newborn infants (Kiecolt-Glaser & Glaser, 1987). Herpesviruses differ from most other known viruses in that after exposure, they are present in individuals all the time, although often in latent states. A robust cellular immune response plays a key role both in protection from initial herpesvirus infection and in keeping latent herpesviruses from becoming active (Glaser & Gottlieb-Stematsky, 1982).

Is stress associated with a recurrence of clinical disease (lesions) after a period of herpesvirus latency? A study of 125 college students provides an elegant test of the role of stress in herpes recurrence (Hoon et al., 1991). This work indicates that stress increases vulnerability to illness in general (nonherpes) and that it is this increase in nonspecific vulnerability that results in herpes recurrence. Although Hoon and colleagues did not address the physiological basis (e.g., cellular immunity) for this nonspecific vulnerability, they did find that the illness vulnerability measure was heavily influenced by highly prevalent infectious diseases (colds and influenza). Thus, an immune basis for this increased herpes recurrence is plausible.

The relationship between increased stress and increased susceptibility to herpes infection and recurrence is also supported by a series of studies of student nurses conducted in the 1970s. In these studies, negative moods at the beginning of the school year were generally associated with greater numbers of subsequent episodes of verified oral herpes (Friedmann et al., 1977, Katcher et al., 1973; Luborsky et al., 1976). Similar evidence for stress-induced recurrence is provided by both retrospective (e.g., Kemeny et al., 1989) and prospective studies of genital herpes (Goldmeier & Johnson, 1982; McLarnon & Kaloupek, 1988; see critiques in Cohen & Williamson, 1991).

In sum, herpes studies generally support a relation between negative emotional states, including stress, and disease recurrence. However, the evidence is not entirely consistent, and methodological limitations warrant cautious interpretation of these results. Moreover, existing work does not establish the extent to which such effects are mediated through immune or behavioral pathways.

Social Relationships and Immunity

Substantial evidence implicates interpersonal relationships in the maintenance of health (Cohen, 1988; House et al., 1988). This is a heterogeneous literature with studies of a wide range of relationship concepts. These

include whether or not one is involved in a relationship (e.g., marriage) or in multiple relationships (e.g., marriage, friendship, group member), whether they have a satisfactory marriage, whether they feel lonely, and whether they perceive that other people will provide them with social support when they need it. I provide only a short overview of social relationship research on immunity here, but refer the reader to Cohen, Underwood, and Gottlieb (2000) for detailed discussions of these concepts and the pathways through which they might influence health and to Cohen (1988), Cohen and Herbert (1996), Kiecolt-Glaser and Newton (in press), and Uchino et al. (1996) for reviews of this literature.

Probably the most provocative studies are a series of prospective community studies that show that people with diverse social networks—including spouse, friends, family members, neighbors, and fellow members of social and religious groups—suffer less illness and live longer than their more isolated counterparts (see reviews by Cohen, Gottlieb, & Underwood, 2000; House et al., 1988). The association between network diversity and mortality is extremely reliable among whites but is less clear among minority populations and more important for men than for women (House et al., 1988). There is no evidence at this time that the association between network diversity, morbidity, and mortality reported in the epidemiological literature can be explained in terms of the effects of social networks on the immune system.

One pathway through which the lack of social ties might influence health is through feelings of loneliness. In turn, the negative affective states triggered by loneliness, including depression and anxiety (Peplau & Perlman, 1982), might influence immunity. Several studies have examined the correlation between loneliness and immunity. For example, in their studies of first-year medical students, Kiecolt-Glaser and Glaser (Glaser, Kiecolt-Glaser, Speicher, et al., 1985; Kiecolt-Glaser, Garner, et al., 1984) found that persons higher in self-reported loneliness had lower NK activity and higher levels of herpesvirus antibody than those who described themselves as less lonely. In a related study, lonelier psychiatric inpatients had poorer NK cell function and lower proliferative responses to PHA than did patients who reported less loneliness (Kiecolt-Glaser, Ricker, et al., 1984b).

There is also substantial evidence that the loss of intimate social ties, especially marital disruptions (separation and divorce) are associated with poorer health (Verbrugge, 1979). In regard to marital discord and immunity, Kiecolt-Glaser, Fisher, and colleagues (1987) found that among 16

separated and divorced women there were higher levels of herpes antibody, a lower percentage of NK cells, and lower lymphocyte proliferative response to PHA and Con-A than among a comparison group of 16 married women. In a similar study, Kiecolt-Glaser, Kennedy, and colleagues (1988) found that among 32 separated and divorced men there were more infectious illnesses and higher levels of herpes antibody than among their 32 married counterparts. Social conflicts with spouses have also been found to influence immune response. For example, a study that categorized newlywed couples on the basis of observed interactions (Kiecolt-Glaser, Malarkey, et al., 1993) found that those who exhibited more negative or hostile behaviors showed greater decreases over 24 hours in NK activity and proliferative response to PHA and Con-A.

Perceived availability of social support has also been associated with immune function. In a study of 256 elderly adults, Thomas and colleagues (1985) found that persons who reported they had "confiding" relationships had greater proliferative responses to PHA than those without confiding relationships. Moreover, this relationship was unchanged after controlling for psychological distress and health practices. Similar results were found in a study by Baron and colleagues (1990) among 23 spouses of patients with cancer. In this study, six different provisions of social support (including emotional and instrumental forms of support) were associated with higher NK activity and better proliferative response to PHA (but not to Con-A). Better immune response among "supported" persons could not be explained by greater depression or more numerous stressful life events among those with less social support.

In addition, Glaser, Kiecolt-Glaser, Bonneau, and colleagues (1992) found that medical students who reported more available social support produced more antibody in response to a hepatitis B vaccination than those reporting less support. However, two studies of HIV-positive men were less successful in establishing relations between social support and immunity (Goodkin et al., 1992; Perry et al., 1992). However, HIV infection compromises the immune system to a degree so severe that the relatively small effects of social support on immune function might be undetectable.

It is likely that many of the beneficial effects of social support on health can be attributed to the receipt of or perceived availability of emotional support—that is, someone to talk to about problems (Cohen & Wills, 1985). Indeed, studies examining the potential health benefits associated with a person's disclosure of traumatic events have found a positive relationship between the opportunity to disclose and better health indices. For

example, Pennbaker and colleagues (1988) assigned 50 healthy undergraduates to write about either personal and traumatic events or trivial topics. They wrote for 20 minutes a day on 4 consecutive days. Immunologic data were collected before the study began (baseline), at the end of the intervention, and at 6-week and 4-month follow-ups.

This study found that blood drawn from subjects who wrote about traumatic events was more responsive to PHA (but not Con-A) than that from subjects who wrote about trivial events. There were no relations between disclosure and alcohol intake, caffeine intake, or exercise over the course of the study. In addition, subjects revealing traumatic events made fewer visits to the health center in the 6 weeks following the intervention than did members of the control group. However, the data do not support an immune pathway because the lymphocyte proliferation data were *not* correlated with health center visits. This absence of correlation also suggests that increases in health center visits may be driven by psychological influences on decision processes rather than by influences on actual illness (Cohen & Williamson, 1991). Another effect of the disclosure intervention on immunity was found by Petrie and colleagues (1995). In this study, subjects who disclosed produced more antibody in response to secondary immunization for hepatitis B than control subjects. Finally, a recent article has demonstrated associations between the disclosure manipulation and two diseases thought to be at least partly attributable to immune dysregulation (Smyth et al., 1999). Asthma patients who wrote about their problems had better lung function over the following 4 months than nontreatment controls. Rheumatoid arthritis patients who completed the intervention similarly demonstrated relative reduction in disease activity over the following 4 months in comparison with controls.

There have also been five studies of mortality in which cancer patients were randomly assigned either to social support groups in which they discussed their feelings with other patients or to standard care control groups. Two of these studies found that group participation was associated with better disease outcomes (Fawzy et al., 1993; Spiegel et al., 1989), and three found no differences (Cunningham et al., 1998; Illnycky et al., 1994; Linn, Linn, & Harris, 1982). (See chapter 5 for more details on this literature.) Although we have only a tentative understanding of the conditions under which such groups might improve health or how they work (Cohen, Gottlieb, & Underwood, 2000; Helgeson & Gottlieb, 2000), this research illustrates that under certain circumstances—that are not yet en-

tirely clear—providing both psychological and social support can beneficially affect the course of physical illness.

Social Relationships and Susceptibility to Infection

More direct evidence for the role of social relationships on immune functioning comes from studies of susceptibility to upper respiratory infections. Using the same paradigm as we used in our studies of psychological stress and susceptibility to infection, Cohen, Doyle, Skoner, Rabin, et al. (1997) recently assessed whether diverse ties to friends, family, work, and community are associated with increased host resistance to infection. In this study, a total of 276 healthy volunteers were assessed on the extent of their participation in 12 types of social ties (spouse, parent, friend, workmate, member of social group, and others) and were then given nasal drops containing one of two rhinoviruses and monitored for the development of a common cold.

This study found that participants with more types of, or more diverse, social ties were less susceptible to common colds (by either type of virus) and when they did catch colds, they produced less mucus, were more effective in ciliary clearance of their nasal passages, and shed less virus than those participants with fewer types of social ties. The participants' susceptibility to colds decreased in a dose-response manner with increased diversity of the social network. In fact, persons with the fewest social ties (1 to 3) had a 4.2 times greater adjusted relative risk of catching a cold than those with the most (6 or more) types of social ties.

Interestingly, in a recent animal study, Cohen, Line, et al. (1997) found that in Old World monkeys social status and not social stress was the factor most responsible for increasing susceptibility to viral infection. In this study, we randomly assigned 60 male cynomolgus monkeys to either a "stable" or "unstable" social (stress) condition for 15 months. Two markers of social status—social rank and percentage of behaviors that were submissive—also were assessed at independent observation periods over a 14-month period. Endocrine, immune, and behavioral responses were each assessed (at 3-month intervals) during the ninth through fourteenth months of the study. At the beginning of the fifteenth month, all animals were exposed to a virus (adenovirus) that causes a common cold-like illness.

Although the social instability manipulation was associated with increased agonistic behavior, as indicated by minor injuries and elevated noradrenaline responses to the unstable social condition, the manipulation did not influence the probability of being infected by the virus. However, low social status (as assessed by either marker) was associated with a substantially greater probability of being infected. It was also associated with less body weight, greater elevated cortisol responses to social reorganizations, and less aggressive behavior. None of these characteristics could account for the relation between social status and infection.

Religious Participation and Health

The most accepted association in the area of religion and health is that religious activities such as church attendance are associated with low morbidity and mortality (Ellison & Levin, 1998; McCullough et al., 2000; Oman & Reed, 1998). Specifically, prospective studies have found that greater frequency of attendance at religious services is associated with lower blood pressure, lower cause-specific mortality for coronary artery disease, lower incidence of physical disability, and lower all-cause mortality. An immune marker of ongoing inflammatory processes (the proinflammatory cytokine IL-6) has also been found to be lower among those who attend church more often (Koenig et al., 1997). Nevertheless, cautions have been raised in regard to methodological limitations of this literature and the fact that the effects often occur in some but not all subpopulations (Sloan et al., 1999).

The issue for this chapter is whether what we know about the role of stress and social relationships from research in health psychology, social epidemiology, and human psychoneuroimmunology can help us understand how and under what conditions religious participation might influence immune-related health. We suggest that there are two categories of pathways through which religious participation might influence health: (1) religious participation can contribute to the diversity of one's social network, enhancing beneficial cognitive and affective states, as well as promoting behaviors that benefit health, and (2) religious participation can provide the potential for reducing stress via the availability of coping resources.

We have discussed the provocative evidence that people with more diverse social networks live longer and healthier lives. In the realm of im-

mune response, having a more diverse network (e.g., a spouse, children, friends, workmates, and fellow social and religious group members) is associated with greater resistance to infectious agents. Several possible reasons explain why those who participate in a diverse social network are healthier (Cohen, 1988; Cohen, Underwood, & Gottlieb, 2000; Uchino et al., 1996), and four of them are discussed here.

1. Being subject to social controls and peer pressures may influence normative health behaviors. For example, people's social networks might influence whether they exercise, consume alcohol, eat low-fat diets, or smoke.
2. Being integrated in a social network may provide a source of generalized positive affect, senses of predictability and stability, a sense of purpose, a sense of belonging and security, and a recognition of self-worth because of demonstrated ability to meet normative role expectations (Cassel, 1976; Hammer, 1981; Thoits, 1983; Wills, 1985). These positive psychological states are thought to be beneficial because they reduce psychological despair (Thoits, 1985) and result in greater motivation to care for oneself (e.g., Cohen & Syme, 1985).
3. Having a wide range of network ties provides multiple sources of information and thereby increases the probability of having access to an appropriate information source. Information could influence health-relevant behaviors or help one avoid or minimize stressful or other high-risk situations.
4. A social network may provide tangible and economic services that result in better health and better health care for network members. For example, network members could provide food, clothing, and housing that operate to prevent disease and limit exposure to risk factors.

In the case of stress buffering, social support can operate by preventing responses to stressful events that are inimical to health (Cohen, 1988; Cohen, Underwood, & Gottlieb, 2000; Cohen & Wills, 1985). Support may play a role at two different points in the causal chain that links stressors to illness (cf. Cohen & McKay, 1984; House, 1981). First, the belief that others will provide necessary resources may redefine the potential for harm posed by a situation and bolster one's perceived ability to cope with imposed demands, thereby preventing a particular situation from being appraised as highly stressful (Thoits, 1986). Second, support beliefs may

reduce or eliminate the affective reaction to a stressful event, dampen physiological response to the event, or prevent or alter maladaptive behavior responses. The availability of persons to talk to about problems has also been found to reduce the intrusive thoughts that act to maintain chronic maladaptive responses to stressful events (Lepore et al., 1996).

The actual receipt of support in the face of stressful events could also play a role. Support may alleviate the impact of stress appraisal by providing a solution to the problem, by reducing the perceived importance of the problem, or by providing a distraction from the problem. It might also facilitate healthful behaviors such as exercise, personal hygiene, proper nutrition, rest, or adherence to medical regimens (cf. House, 1981; Cohen & Wills, 1985).

Participating in religious groups has the potential to influence all of these same mechanisms. In fact, in his summary of potential mechanisms that link religion to health, Pargament (1990) included reducing anxiety; enhancing the sense of meaning and purpose in life; and fostering personal growth, mastery, and control as possible explanations. He also included religious associations and beliefs as resources for coping with stress (e.g., Pargament, 1990, 1997; Pargament et al., 1990). Similarly, Ellison and Levin's (1998) list of mechanisms linking religion to health included regulation of health behaviors, promotion of positive self-perceptions, generation of positive emotions, and provision of social and coping resources.

One possibility is that religious participation is no different from other forms of social participation. In essence, being an active member of a church would be no different from being an active member of a bowling league or book club. In both cases, the other members of the group would be sources of recognition of self-worth, of information, of emotional and tangible resources, and so forth.

Alternatively, there could be something unique about religious participation. There are a number of possibilities here. One is that, for many, religious identification is a central social role with greater importance than others. It often involves greater commitment of time and a stronger identification with the beliefs and social norms of the group. For example, one would expect that, on the average, the health-relevant social norms of a religious group would have a greater impact on behavior than the consensual norms of fellow bowlers.

The breadth of social norms provided by religious identifications also may be major sources of structure, predictability, and purpose in life.

Moreover, religious groups often accept helping one another as a central assumption of their beliefs. In consequence, they might be more likely to provide support in time of need. Religious beliefs might play a role here as well. The stronger their beliefs, the greater their commitment would be to the group, and the greater the effects of the group on their behavior and affect. Finally, religious organizations may serve as an access point where individuals make contacts with other social domains, such as business, social, or recreational groups. Thus church attendance might have a synergistic effect on the size and diversity of a network and the resources it might provide (Ellison & George, 1994).

Evidence that religious participation enhances social resources is reported in data from large community samples. Bradley (1995) and Ellison and George (1994) report that frequent churchgoers have larger social networks, more contact with network members, more types of social support received, and more favorable perceptions of the quality of their social relationships than do nonmembers. These data are consistent with the hypothesis that churches may serve as an access point to other social domains but might also indicate self-selection of persons with interpersonal skills into church membership. In fact, Bradley (1995) found an overrepresentation of extroverted individuals and an underrepresentation of more neurotic persons among regular churchgoers.

In sum, belonging to a religious group should provide one with the same benefits of participating in any social domain. Moreover, certain characteristics of religious participation suggest that religious groups may be more effective at providing these benefits than other domains and that they may facilitate access to other social domains as well. However, the relative efficacy of religious participation in affecting health is not yet established, nor is it clear yet whether religious participation influences access to other social resources. Future research addressing these issues is imperative.

Moreover, this research should pay special attention to variables that might moderate the importance of church membership, such as individual commitment to the religious group and to its beliefs. The salience of norms for everyday living that contribute to feelings of self-esteem and self-control and of norms for a behavioral style that enhance health should be greatest among the more committed. Other potential important variables to consider include gender and physical well-being. Religious participation has been found to be more important for women than for men and

for people who are ill than for those who are well (cf. Sloan et al., 1999). This is probably because women are more committed to their participation, and religion gains a new importance for those who are ill. Again, assessment of individual commitment might help clarify these data.

Finally, new work should also pay special attention to the geographic area under study—its overall rate of religious participation and of religious belief and commitment. Certainly, the extent to which the religious groups' norms are also the community norms should enhance their effectiveness.

Conclusions

There is growing evidence that psychological stress and social factors including network diversity and perceived availability of social support influence immunity and immune system-mediated disease. There also is substantial evidence that these factors can influence both cellular and humoral indicators of immune status and function, and, at least in the case of the less serious infectious diseases (colds, influenza, herpes), there is consistent and convincing evidence of links between stress and disease onset and progression.

A noteworthy aspect of this literature is the importance of participation in social networks for maintaining health and preventing and recovering from disease. These effects occur either through direct psychological and biological influences of our social contacts or through the buffering effects of these contacts when we face stressful life events. We argue in this chapter that the same mechanisms that link our relationships in other social domains to health are likely to link participation in religious organizations to health. Moreover, we suggest the possibility that religious participation may be an especially important social domain in relation to health.

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