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BRIEF REPORTS

Being Popular Can Be Healthy or Unhealthy: Stress, Social Network Diversity, and Incidence of Upper Respiratory Infection

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Diverse social contacts are generally associated with better health. However, diverse contacts can increase exposure to infectious agents. This should increase risk for disease, particularly among those whose host resistance is otherwise compromised (e.g., stressed individuals). In this prospective study, healthy college students who completed questionnaires assessing social network diversity and stressful life events were subsequently interviewed weekly for 12 weeks to track incidence of upper respiratory infections (URIs). URI episodes were defined by a symptom criterion and by clinically verified self-reported illness. Stress and diversity of social contacts interacted; diversity was associated with more illnesses among those with more stressful life events and slightly fewer illnesses among those with fewer stressful life events. Associations remained after controlling for neuroticism.

Key words: stress, life events, social networks, upper respiratory infection

The hypothesis that multiple ties to friends, family, work, and community are beneficial in terms of physical health has gained substantial support recently. Particularly provocative is epidemiologic evidence that those who have more diversified social networks—that is, people who are married; people who interact with family members, friends, neighbors, and fellow workers; and people who belong to social and religious groups—live longer (House, Landis, & Umberson, 1988), are more likely to survive myocardial infarction (Berkman, 1995), and are less likely to suffer a recurrence of cancer (Helgeson, Cohen, & Fritz, 1998) than their counterparts with fewer types of social relationships. A study of host resistance to upper respiratory infections (URIs) found that persons with more diverse networks were less likely to develop a cold when they were experimentally exposed to a cold-causing virus (Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997). Even so, having a diverse social network may not always be a good thing. For example, it is likely that diverse social networks are associated with face-to-face contact with a broad range of people and hence an increased risk of exposure to a range of infectious agents.

Several studies provide support for increased numbers of URIs with increased exposure to others. Dingle, Badger, and Jordan (1964) found that individuals from larger families averaged more physician-verified colds per year than people from smaller families. Graham, Woodward, Ryan, and Douglas (1990) found that children prone to respiratory illness were more likely to attend day care, have siblings less than 14 years of age, and have mothers who regularly attended social or religious meetings. In a prospective study, Clover, Abell, Becker, Crawford, and Ramsey (1989) found that individuals from families reporting high levels of cohesion (emotional bonding) were more likely than their less-enmeshed counterparts to experience an incidence of laboratory-documented influenza B infection. The authors suggested that families reporting low cohesion levels may have touched less, kissed less, and shared eating and drinking utensils less, thus minimizing their viral exposure.

It is likely that the exposure effects of social contact occur primarily among vulnerable members of the population—those whose host resistance to infectious agents is otherwise compromised. Because psychological stress is associated with compromised host resistance to viral infectious illness (e.g., Cohen, Tyrrell, & Smith, 1991; Stone et al., 1992), those with elevated stress levels should be particularly susceptible to the increased risk of exposure associated with social diversity. Although there have
been no direct studies of the interaction of social diversity and stress in predicting URIs, two studies have investigated the interaction between stress and support network size. In the first (DeLongis, Folkman, & Lazarus, 1988) study, participants reported the number of people whom they perceived as sources of emotional support. Participants also rated their hassles and somatic symptoms (primarily URI symptoms) daily for 20 days. The greater the number of people in one’s support network, the greater was the within-subject association between same-day increases in both hassles and physical symptoms. In a second study, Turner Cobb and Steptoe (1996) assessed the number of people that participants could count on for social support and then monitored participants daily for 15 weeks to track and verify URI (by signs of illness). After the follow-up period, participants retrospectively reported distress from life events occurring during the follow-up. Under low stress, those reporting smaller support networks were more likely to experience a URI episode than those with larger networks. Under high stress, this advantage disappeared—illness risk for those with larger networks rose to the same level as those with smaller networks.

These studies provide a hint about the relationship between social networks and risk for URI among vulnerable people, but they are limited. This is so primarily because, although the number of supportive others provides a rough estimate of exposure, this type of social contact index is limited to supportive network members and usually confined to few close social domains (e.g., family, friends). People are often in close physical contact with many nonsupportive people and also interact in domains not tapped by support network questionnaires (e.g., church or sports). Whereas the Turner Cobb and Steptoe (1996) study had a clean URI assessment, the DeLongis et al. (1988) study included other psychosomatic symptoms such as backache and swollen ankles and may not have been addressing (they did not claim to) URI at all. Neither the DeLongis et al, study nor the Turner Cobb and Steptoe study assessed stressful events prospectively. This leads to the possibility that stressful events (or reports) might be affected by earlier illness. For example, those with larger networks who were ill might have been more likely to have interpersonal conflicts, or to perceive they did, clouding interpretation of the results.

The study reported here is prospective, with stress and network diversity assessed in healthy people before the 12-week URI-monitoring period. We used a network measure that assesses the number of different social domains an individual interacts in on a regular basis. We theorized that the greater the number of domains in which a person interacts, the greater their exposure to a range of infectious agents would be. We assessed illness with both self-report (symptom-based, after Turner Cobb & Steptoe, 1996) and objective (clinical examination) markers.

Relatedly, increased levels of neuroticism have been associated with both increased reported stress levels and symptom reports without physiological basis (e.g., Cohen & Williamson, 1991; Pennebaker, 1982). We relied on self-reports of stress, symptoms, and URI (verified only among those reporting sick). To be confident that neuroticism levels were not acting as a third variable influencing the associations found between stress and URI reports, we expanded on this work by statistically controlling for the effect of neuroticism in all analyses.

Consistent with previous stress research, we hypothesized that people reporting higher levels of stress would be more susceptible to URIs. Because network diversity has been associated with improved host resistance, we thought that those with more diverse networks who experienced few life events (and hence not particularly vulnerable) would have no more or possibly fewer URIs than their less diverse counterparts. In contrast, we thought that those with diverse networks who experienced more life events (and hence compromised host resistance) would have more URIs than their less diverse counterparts.

**Method**

**Participants**

Participants were solicited via electronic bulletin boards, school newspapers, and word-of-mouth to be part of a larger study on psychosocial predictors of URI. They were eligible to participate if they were 18–30 years of age; were students of either the University of Pittsburgh or Carnegie Mellon University; had no infectious illness 2 weeks prior to participation, no chronic illness, no personal history of cancer, no autoimmune disorders, and no current or history of psychological disorder; consumed no more than 12 alcoholic beverages on average per week; did not use street drugs; and were not currently pregnant or lactating. One hundred fifty-one people met these criteria. Of those potential participants, 115 (71%) participated. The main reason for nonparticipation was not appearing for scheduled appointments (85%); the other nonparticipants (15%) had difficulty with catheterization (blood taken as part of a reactivity protocol not reported here). Four cohorts of participants were run (fall 1996, n = 13; spring 1997, n = 44; fall 1997, n = 28; spring 1998, n = 30). The overall sample was 47% male and 53% female; 92% single; 76% Caucasian, 10% African American, 7% Asian, 2% Hispanic, and 5% “other race”; and had a mean age of 21.1 years (SD = 2.7 years). Participants received a total of $120 in compensation.

**Procedures**

On arriving at the lab, participants completed questionnaires assessing negative life events experienced over the past 12 months, social network diversity, neuroticism, and demographics. After completing a reactivity protocol for the main study (see Cohen et al., 2000), participants were then instructed about the 12-week follow-up period. (Neither life events nor network diversity was associated with any reactivity measures assessed for the larger study.) Weekly for 12 weeks, participants completed a diary to provide information concerning URI symptoms and whether they believed themselves to have had a cold or flu since last diary completion. At any time during the follow-up period, if participants felt they had a cold or flu, they were to contact the study coordinator to arrange an appointment with a student health center nurse practitioner to verify the URI’s presence. One participant was delinquent in returning the follow-up data and was terminated from the study, resulting in a total number of 114. Of the remaining participants, the majority (87%) completed and returned all 12 behavioral health diaries. Of the 1,368 potential weekly diaries (114 participants × 12 weeks), 1,343 (98%) complete diaries were received.

**Measures**

**Negative life events.** The Major Stressful Life Events Scale consists of questions about events in the life of the respondent (41 items) or close others (26 items; from Cohen et al., 1991). Participants indicated whether they had experienced each event during the past 12 months. They also indicated whether each reported event had a positive or negative impact on their lives. A few items, such as death of a family member, were assumed
to be consensually negative, and the respondent was not asked to provide an impact rating. A negative life-events score was derived for each participant by adding the number of events they reported that were negative (either consensual or participant rated). Because the distribution was substantially skewed, we coded events as below or above the median number of events (two events).

**Social network diversity.** The Social Network Index (Cohen et al., 1997) assesses participation in 12 types of social relationships. These include relationships with a spouse, parents, parents-in-law, children, other close family members, close neighbors, friends, workmates, schoolmates, fellow volunteers (e.g., charity or community work), members of groups without religious affiliations (e.g., social, recreational, or professional), and members of religious groups. One point is assigned for each relationship (possible score of 12) for which respondents indicate that they speak (in person or on the phone) to someone in that relationship at least once every 2 weeks.

**Neuroticism.** Neuroticism has been directly associated with reports of stress levels and reports of symptoms without a physiological basis (e.g., Cohen & Williamson, 1991; Pennebaker, 1982). Because we relied on participant reports of stress and participant judgment to determine when they were sick, we statistically controlled for the effect of neuroticism on reported URI occurrence. We assessed neuroticism during the laboratory visit with the 10-item Neuroticism subscale from the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1964). Responses were summed, yielding a neuroticism score ranging from 0–10. This scale was found to be moderately reliable ($\alpha = .67$).

**Standard demographic control variables.** Age was scored as a continuous variable. Sex was scored as a dichotomous variable. Race was categorized as Caucasian, African American, or other.

**Self-reported symptoms.** Weekly symptom scores were derived by summing answers to the following eight items, completed on a five-point Likert scale with ratings of zero (none) and one (mild) to four (very severe): nasal congestion, sneezing, runny nose, cough, headache, scratchy/sore throat, fever, and feeling under the weather. These symptoms resulted from an analysis of the self-reported symptoms that participants used to characterize their natural clinical colds after viral challenge (Jackson, Dowling, Spiesman, & Board, 1958). Following the example of Turner Cobb and Steptoe (1996), we derived a symptom-based cold incidence measure from participant report of two of the eight symptoms at the severe level. On the basis of this criterion, 38 participants were defined to have experienced a URI.

**Verified URI.** During the 12-week follow-up period, participants were asked to immediately contact the study coordinator if they felt they had developed a cold or flu. The coordinator then scheduled an appointment for a nurse practitioner examination. Participants were also sent for an examination if at the weekly interview they reported having a cold or flu. The nurse took a temperature reading and examined the participant’s eyes; ears; nose; throat; cervical, submandibular, and axillary lymph nodes; and chest/ lungs for the presence of abnormalities. On the basis of the information gathered above, the nurse determined whether the participant’s signs and symptoms were most consistent with a URI, some other disease process, or no apparent illness. Of the 51 people meeting criteria for verification, 30 actually went to the clinic. Reasons for participants not coming in to have their colds verified included the following: colds occurred over the weekend when the student health center was closed, participants failed to contact the study coordinator when sick (but reported a URI at the next interview), or participants missed student health center appointments and were better by the time an appointment was rescheduled. Of the 30 participants coming in for verification, 29 were diagnosed as having a URI. In our analyses, only those 29 were identified as having verified illness. Of the 38 people who had a URI defined by self-reported symptoms, 18 also had a medically verified URI, 14 reported having a URI but did not seek medical verification, and 6 neither self-reported a URI nor sought URI verification.

**Results**

We used logistic regression to examine whether stress, social network diversity levels, or their interaction was associated with verified and with self-reported symptom-based URI incidence during the 12-week follow-up period. We performed the analyses in the following manners: (a) entering the stress, social networks, and then their interaction into the regression; (b) forcing the standard control variables (age, sex, and race) into the regression before the main effects and interaction; and (c) forcing the standard controls and neuroticism into the regression and then the main effects and interaction. Because the results of these three analyses were virtually identical, we report the results of the last (most conservative). Because there were no effects of season (fall vs. spring), we collapsed data across seasons in our analyses.

Rates of URIs were higher among those having two or more negative life events, but the differences were not statistically significant for either verified ($b = 1.80, p = .06; \beta = 6.07$) or symptom-based ($b = 1.42, p = .11; \beta = 0.74$) URIs. More diversified social networks were marginally associated with a greater incidence of verified ($b = 0.35, p = .06; \beta = 1.42$) but not symptom-based ($b = -0.03, p > .80; \beta = 0.96$) URIs. More important, life events and network diversity interacted to predict the incidence of both symptom-based ($b = -0.29, p = .05; \Delta R^2 = .04, \beta = -0.75$) and verified ($b = -0.44, p = .03; \Delta R^2 = .04, \beta = -0.65$) URI episodes. To interpret these interactions, we performed a median split of network diversity (already completed for life events) and determined the percentage of URI occurrences for each of the four Stress × Network Diversity groups. Figure 1 contains the graphed results for symptom-based (A) and verified (B) URIs. These are observed rates and are not adjusted for covariates. In both cases, those with high network diversity and high levels of life events experienced the most URIs. In contrast, those with high network diversity and low levels of life events experienced the fewest URIs.

**Discussion**

Although having a diverse social network is generally beneficial to health, there are circumstances under which it would be detrimental. One such situation is when one’s host resistance to infectious agents is compromised. Under this condition, increased social contact and hence exposure to a greater range of infectious agents can lead to a greater incidence of URI communicated through social contact.

Our data are similar to two studies (DeLongis et al., 1988; Turner Cobb & Steptoe, 1996) previously reporting that measures tapping increased social contact are associated with greater incidence of URI under stress, but add to this literature in several key ways. First, the data held up when we controlled for neuroticism, a personality trait associated with both the occurrence (or reporting) of stress and the symptom reporting. Our results also held up when disease was verified using objective standards for disease incidence. Together, these techniques eliminate the possibility that the associations we report are attributable to psychosocial factors influencing presentation of symptoms. Our data are also prospective, eliminating the possibility that illness might have caused stress in those with more diverse networks. A final difference is
that earlier work focused on the size of support networks, whereas our analyses are based on a broader concept of network contact that includes supporters and nonsupporters alike. We find it interesting that, in theory, diverse networks represent a greater risk than more narrow (e.g., support) ones because they involve interaction with people in different subpopulations who might be carrying different viruses.

Forty-three percent of the 51 participants self-reporting a URI did not attempt to have their illnesses verified. Individuals with a verification attempt had more diverse social networks than those without, \( F(1, 50) = 5.92, p = .02 \). However, those with and without verification attempts did not differ in number of negative life events, \( F(1, 50) = 2.56, p = .12 \). The relation between social diversity and seeking verification suggests an alternative explanation for our results. It is possible that high-stress persons with symptoms were more likely to seek validation of those symptoms when they were in a more diverse network. Because they had more social roles (and hence obligations) they may have been motivated to play the sick role. This would be unnecessary for those without stress-related demands or for those with fewer social roles and hence social obligations. Because our results hold when using a definition of colds that did not even require participants to label themselves as ill (symptom-based URI), this alternative explanation seems unlikely.

Previous work from our lab (Cohen et al., 1997) suggests that having a more diverse social network is associated with being less likely to develop a cold when experimentally exposed to a cold-causing virus. The findings from the current study suggest that, in the naturalistic setting where exposure is left to vary, this protective effect of having a diverse social network is reserved only for times when one’s immune system can handle it (low stress).

The network diversity measure was not, of course, designed to directly assess potential for viral contact. However, it does measure the number of different groups of people with which one interacts. Because different pathogens may be represented in each group, it—better than just number of people—represents possible exposure to a range of infectious agents. The questionnaire includes people spoken to on the phone. However, it is not an on-line measure of exposure but rather a measure of the existence of relationships. People spoken to on the phone this week may well be people seen in person over the course of several months.

A particularly interesting implication of our results is the opening of the door to future work investigating the interactions between social diversity and other factors found to compromise host resistance in predicting incidence of infectious disease. Examples include age, immunosuppressive drugs or diseases, and depression. Because of this compromised host resistance, these populations may consequently pay a cost for being popular.

References


