

9-2003

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Published In

Psychological Science, 14, 5.

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Research Article

SOCIABILITY AND SUSCEPTIBILITY TO THE COMMON COLD

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Abstract—*There is considerable evidence that social relationships can influence health, but only limited evidence on the health effects of the personality characteristics that are thought to mold people's social lives. We asked whether sociability predicts resistance to infectious disease and whether this relationship is attributable to the quality and quantity of social interactions and relationships. Three hundred thirty-four volunteers completed questionnaires assessing their sociability, social networks, and social supports, and six evening interviews assessing daily interactions. They were subsequently exposed to a virus that causes a common cold and monitored to see who developed verifiable illness. Increased sociability was associated in a linear fashion with a decreased probability of developing a cold. Although sociability was associated with more and higher-quality social interactions, it predicted disease susceptibility independently of these variables. The association between sociability and disease was also independent of baseline immunity (virus-specific antibody), demographics, emotional styles, stress hormones, and health practices.*

There has been much recent emphasis on the role of social relationships in health (e.g., Cohen, Gottlieb, & Underwood, 2000; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). The structure of people's social networks (Brissette, Cohen, & Seeman, 2000), the support they receive from others (Helgeson & Gottlieb, 2000; Wills & Shinar, 2000), and the quality and quantity of their social interactions (Kiecolt-Glaser & Newton, 2001; Reis & Collins, 2000) have all been identified as potential predictors of their health and well-being. Although these various indicators of people's social lives are to some extent molded by their personalities, there has been much less interest in the role of socially relevant dispositions in health.

This article focuses on sociability, a disposition that is generally recognized as a determinant of quality and quantity of social interaction. We define sociability as the quality of seeking others and being agreeable (Liebert & Spiegler, 1994; Reber, 1985). We assume that sociability plays a role in the development and maintenance of social networks, intimate relationships, and social supports. If our assumption is correct, one would expect that more sociable people would be healthier than less sociable people. This could occur because better and closer relationships might increase positive and decrease negative affect, promote positive health practices, help regulate health-relevant biological systems, or provide social support in the face of stressful events (Cohen, 1988; Cohen et al., 2000; Uchino et al., 1996).

The first question we raise in this article is whether those individuals who seek out interactions and are generally agreeable and genial in

company are somehow protected from illness. Two factors of the Big Five personality factors can be viewed as combining to represent the central components of sociability: extraversion, the personality dimension that reflects an individual's preferences for social settings, and agreeableness, the dimension of personality that underlies geniality (Costa & McCrae, 1992). Of these, only extraversion has been seriously considered in terms of its implications for health. Eysenck (1967) proposed that extraversion is characterized by low resting levels of electrocortical and sympathetic nervous system (SNS) activity (Geen, 1997). Because activation of these systems is related to suppression of immune function, low activation would be expected to reduce risk for developing disease when exposed to infectious agents. Three studies of susceptibility to infection by common cold viruses found that extraversion is related to reduced susceptibility (Broadbent, Broadbent, Phillpotts, & Wallace, 1984; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997; Totman, Kiff, Reed, & Craig, 1980). None of these studies addressed the potential psychological or biological pathways through which extraversion might influence disease susceptibility.

The second question we address is how sociability might get inside the body. On the psychological side, we were interested in whether associations of sociability and health are mediated through interpersonal behavior. Are sociable people healthier than others because they interact more often or with more people, have fewer conflicts or more satisfactory interactions, and have more social support? More and better interactions could facilitate the regulation of emotions and provide the motivation and opportunity to take better care of oneself. In turn, helping regulate emotional response contributes to the regulation of emotion-related biological systems that have implications for immune competence. These systems include the SNS and the hypothalamic-pituitary-adrenal-cortical (HPA) axis. In the study we report here, we tested whether sociability is associated with the ability to resist infectious illness and examined plausible explanations for how such an association might occur. Social, psychological, and biological data were collected from volunteers who were subsequently exposed to one of two rhinoviruses that cause common colds. The major outcome was whether or not volunteers developed verifiable disease.

METHOD

Participants

Data were collected between 1997 and 2001. The participants were 159 men and 175 women, ages 18 to 54 years, who responded to newspaper advertisements and were judged to be in good health after a medical examination. They were paid \$800 for their participation.

Procedure

Table 1 summarizes the sequence of the study.

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Table 1. *Temporal sequence of a trial*

<u>2 months before quarantine</u>
Eligibility screening:
Physical exam
Blood for preexisting antibody to virus
Extraversion and agreeableness questionnaires (first administration)
Social-network questionnaire
Demographics
<u>2 to 4 weeks before quarantine</u>
Daily interviews:
6 daily assessments of social interactions and affect
<u>Quarantine Day 0</u>
Extraversion and agreeableness questionnaires (second administration)
Positive Relationship With Others Scale
Social-support questionnaire
Health-practice questionnaires
Saliva cortisol and urine epinephrine samples
Nasal secretions for virus culture
Baseline signs and symptoms of respiratory illness
Daily affect assessment
<u>End of Day 0</u>
Inoculation with virus
<u>Quarantine Days 1 through 5</u>
Nasal secretions for virus culture
Signs and symptoms of respiratory illness
<u>4 weeks after virus challenge</u>
Blood for antibody to virus

Eligibility screenings

At the onset of the study, all volunteers underwent medical-eligibility screenings. They were excluded from the study if they had a history of any psychiatric or chronic physical disease, had abnormal blood or urine profiles, were pregnant or currently lactating, had antibody for HIV, or were on a regular medication regimen. In addition, during the first 24 hr of quarantine (Day 0, before virus exposure), volunteers had a nasal examination. They were excluded from the study at this point if they had symptoms of a cold.

Data collected before exposure to the virus

Demographics, all psychological data, immunity to the experimental virus (levels of preexisting antibody), weight and height, SNS and HPA hormones, and health practices were assessed during the 8-week period before exposure to the virus. Baseline symptoms and objective signs of illness were assessed during the day (Day 0) before virus exposure.

Virus exposure and assessments of illness

Volunteers were quarantined in separate rooms and exposed (after 24 hr) to one of two types of rhinovirus, RV39 ($n = 228$) or RV23 ($n = 106$). On each of the 5 days after exposure, they reported their respiratory symptoms and were assessed for objective indicators of infection (virus culture of nasal secretions) and illness. Four weeks after virus exposure, a blood sample was collected to test for an additional marker of infection—increases (from baseline) in level of antibody to

the virus. Investigators were blinded to all psychological and biological measures.

Psychological Measures

Sociability

We used three measures to assess sociability: extraversion, agreeableness, and positive relationship style. Extraversion and agreeableness were assessed twice (8-week interval) before virus exposure; positive relationship style was assessed on the day before exposure (Day 0). Extraversion and agreeableness were each measured with an eight-item subscale from a short version of the Goldberg Big Five Questionnaire (Cohen et al., 1997; Goldberg, 1992). Each item on these subscales is a trait (e.g., extraversion: talkative, bashful; agreeableness: generous, unsympathetic), and respondents indicated how accurately the trait described how they “typically are,” on a scale ranging from 0 (*not at all accurate*) to 4 (*extremely accurate*). The nine-item Positive Relationship With Others Scale (Ryff, 1989) is intended to more broadly tap sociability. Participants indicated their agreement that each item described them, using a 6-point scale ranging from 1, *strongly disagree*, to 6, *strongly agree*. An example of an item from this scale is “Most people see me as loving and affectionate.” For all three scales, the appropriate items were reversed, and the scale scores were summed. The test-retest correlations were .79 for extraversion and .69 for agreeableness (all $ps < .001$). The internal reliabilities were .83 to .85 for extraversion, .76 to .79 for agreeableness, and .78 for positive relationships. To obtain final scale scores for extraversion and agreeableness, we averaged the scores from the two assessments.

We then entered the three final scale scores into a principal component factor analysis. All three scales loaded on the first principal component (.69, .64, and .80, respectively). To create a single sociability scale, we transformed the final scale scores into z scores and added the three scores together.

Social interactions

We used telephone interviews to assess daily social interactions. Volunteers were interviewed on 3 evenings a week (2 weekdays and 1 weekend day) for 2 weeks during the month before quarantine. The interview included a review of the interpersonal interactions participants had over the day and was modeled after the Rochester Interaction Record (Reis & Wheeler, 1991). An interaction was defined as spending time with one or more persons for 10 consecutive minutes or longer. For each one-on-one or group interaction, participants indicated with whom they interacted, when the interaction started and ended, how pleasant it was (from 1, *unpleasant*, to 7, *pleasant*), and the level of disagreement or conflict (none, mild, moderate, severe).

We derived a number of scores from the 6 days of interaction interviews, including total number of interactions; average number of people interacted with per day; average pleasantness of interactions; and percentage and number of interactions that were pleasant (≥ 5 on the 7-point scale), were unpleasant (≤ 3), or involved moderate to severe conflict.

Social network and social support

We administered two standardized questionnaires. The Social Network Index (SNI; Cohen et al., 1997) assessed the number of social roles regularly engaged in (e.g., spouse, friend, family member,

worker) and the number of people talked to (in person or on the phone) within these roles in a 2-week period. Marital status was also recorded. The 12-item version of the Interpersonal Support Evaluation List (ISEL; Cohen, Mermelstein, Kamarck, & Hoberman, 1985) assessed participants' perception that others would provide them with support in the face of stressful events. Internal reliability for the ISEL was .87.

Emotional styles

Data on emotional style were collected during the six interviews described earlier, as well as on the evening of the Day 0 (before virus exposure) of quarantine. Each evening, participants were asked how accurately (from 0, *not at all accurate*, to 4, *extremely accurate*) each of nine positive and nine negative mood adjectives described how they felt during the last day (Cohen, Doyle, Turner, Alper, & Skoner, in press). The positive adjectives included *lively*, *happy*, and *relaxed*. The negative adjectives included *sad*, *on edge*, and *angry*. Daily positive-mood scores were calculated by summing the ratings of the nine positive adjectives, and daily negative-mood scores were calculated by summing the ratings of the nine negative adjectives. The internal reliabilities (alphas) for the seven interviews ranged from .89 to .93 for the positive-mood scale and .87 to .92 for the negative-mood scale. To form measures of emotional style, we averaged daily mood scores (separately for positive and negative) across the seven interviews.

Control Variables

We examined eight control variables that might provide alternative explanations for the relation between sociability and illness. These included levels of antibody to the experimental virus before challenge (titer of ≤ 4 or ≥ 8), age (18–21, 22–32, 33–54), body mass index (weight in kilograms/height in meters²), race (Caucasian, other), gender, and virus type (RV23 or RV39). Also included were month of exposure (March, May, July, September, or December) and education level (high school graduate, high school graduate with less than 2 years of college, and high school graduate with 2 or more years of college).

Pathways Linking Sociability to Susceptibility

Health practices

Smoking rate was defined as the number of cigarettes smoked a day. In calculating the average number of alcoholic drinks per day, we treated a bottle or can of beer, glass of wine, or shot of whiskey as a single drink. Exercise was measured by the number of days per week engaged in an activity long enough to work up a sweat, get the heart thumping, or get out of breath (Paffenbarger, Blair, Lee, & Hyde, 1993) multiplied by a rating, from 0 (*no effort*) to 10 (*maximum effort*), of the associated level of exertion. Assessments of sleep quality included subjective quality, efficiency (percentage of time in bed sleeping), and duration (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Dietary intake of vitamin C and zinc was assessed by standard questionnaire (Block, Hartman, & Naughton, 1990).

Endocrine hormones

Samples for hormone assessments were collected on the 1st day of quarantine. The SNS hormones epinephrine and norepinephrine were

assessed in a 24-hr urine sample and assayed using high-performance liquid chromatography with electrochemical detection. To assess the release of the HPA hormone cortisol, we collected 12 saliva samples via salivettes (cotton rolls). Approximately 1 sample was collected per hour between 5:45 a.m. and 4:00 p.m., with others collected at 6:30 and 10:30 p.m. Levels of salivary cortisol were determined via time-resolved immunoassay with fluorometric end-point detection. Area under the curve was calculated to measure total free-cortisol release.

Infections and Colds

Infectious diseases result from the growth and action of microorganisms or parasites in the body. Infection is the multiplication of an invading microorganism. Clinical disease occurs when infection is followed by the development of signs and symptoms characteristic of the disease.

Infection

The presence of an infectious agent can be established directly through the use of culturing techniques (in this case, finding the virus in nasal secretions). Nasal secretion samples collected daily in a saline wash of the nose were frozen and later cultured for rhinovirus using standard techniques (Gwaltney, Colonno, Hamparian, & Turner, 1989). Infection can also be detected indirectly by examining changes in specific antibody to the infectious agent. When exposed to foreign agents, the immune system produces protein molecules (antibodies) that help mark and destroy invading microorganisms. The production of antibodies to a specific infectious agent is evidence for the presence of that agent. Hence, we compared virus-specific antibody levels measured in serum collected before and 28 days after exposure (Gwaltney et al., 1989).

Signs and symptoms

At the end of each day of quarantine, participants rated the severity of eight respiratory symptoms (congestion, runny nose, sneezing, cough, sore throat, malaise, headache, and chills) during the previous 24 hr (Jackson et al., 1960). Ratings ranged from 0 (*none*) to 4 (*very severe*) for each symptom. Ratings of the eight symptoms were summed to create daily symptom scores. Participants were also asked each day if they had a cold.

We assessed daily mucus production by collecting used facial tissues in sealed plastic bags (Doyle, McBride, Swarts, Hayden, & Gwaltney, 1988). The bags were weighed and the weight of the tissues and bags subtracted. Nasal mucociliary clearance function is an objective measure of what is experienced as congestion. Specifically, it refers to the effectiveness of nasal cilia in clearing mucus from the nasal passage toward the throat. Clearance function was assessed as the time required for a dye administered in the nostrils to reach the throat (Doyle et al., 1988).

To create baseline-adjusted daily scores for each measure, we subtracted the appropriate baseline score (day before challenge) from each of the five postchallenge daily scores. Adjusted daily scores that were negative were rescored as 0. We then summed the appropriate adjusted daily scores across the 5 days to create total adjusted symptoms, mucus weight, and mucociliary clearance scores.

Table 2. Control variables associated with risk of common cold

Control measure	Illness criterion	
	Objective	Subjective
Preexisting antibody	$b = -0.65 \pm 0.28, p < .02$	$b = -0.80 \pm 0.26, p < .002$
Virus type (RV23 or 39)	$b = -0.82 \pm 0.30, p < .006$	$b = -0.74 \pm 0.28, p < .007$
Age	n.s.	$b = -0.66 \pm 0.29, p < .05$

Note. Each result is from a separate equation in which the individual control variable was the only predictor.

Definition of a cold

Volunteers were considered to have a clinical cold if they both were infected and met illness criteria. They were classified as infected if the challenge virus was isolated on any of the 5 postchallenge study days or there was at least a 4-fold rise in virus-specific serum antibody titer from before exposure to 28 days after exposure. We used two alternative illness criteria. The objective criterion required a total adjusted mucus weight of at least 10 g or total adjusted mucociliary nasal clearance time of at least 35 min (Cohen et al., 1997). The subjective criterion (modified Jackson criterion) required a total adjusted symptom score of 6 or higher, in addition to either reporting having a cold or reporting runny nose on 3 or more days (e.g., Cohen et al., 1997).

Statistical Analyses

Body mass index, total symptom scores, mucus weight, mucociliary clearance scores, cortisol level, epinephrine and norepinephrine levels, number of cigarettes per day, number of alcoholic drinks per day, and zinc and vitamin C intake were all log-transformed (base 10) to better approximate a normal distribution. We used stepwise logistic regression to predict the binary outcome presence/absence of a cold. Sociability measures were treated as continuous variables, and we report the regression coefficients, with standard errors and probability levels. In several cases, we also provide an estimate of relative risk—the ratio of risk (odds ratio and 95% confidence interval, CI) of participants with lower levels of sociability (each of the bottom 4 quintiles) relative to participants with the highest sociability (top quintile). We sequentially added variables to the first step of regression analyses in

order to determine whether the association between sociability (entered alone in the second step) and susceptibility to colds is substantially reduced after controlling for the contribution of other variables. All analyses we report included the eight control variables. Interaction terms were entered together in a third step of the equation.

RESULTS

Table 2 presents the significant associations between control variables and frequency of colds. Having previous antibody and being exposed to RV23 rather than RV39 were both associated with fewer colds by both the objective and the subjective illness criteria. For the subjective criterion only, being 18 to 21 years old was associated with fewer colds than being older. None of these variables, however, were associated with sociability.

We examined the association of each of the components of sociability with frequency of colds. As is apparent from Table 3, higher scores for extraversion, agreeableness, and positive relationships were all associated with decreased risk for colds, irrespective of the illness criterion. To simplify presentation, for the remaining analyses we use the sociability index. This index provides a broader conceptual scope and better reliability than the three individual measures.

As is apparent from Figure 1, increases in sociability were associated in an approximately linear manner with decreases in the rate of illness defined by both criteria (statistics for sociability treated as a continuous variable are in Table 3). The adjusted odds ratios were 2.9 (CI = 1.12, 7.37), 3.0 (CI = 1.22, 7.47), 2.2 (CI = 0.89, 5.34), 1.4 (CI = 0.52, 3.66), and 1 (reference group) for objectively defined colds and 4.4 (CI = 1.76, 11.16), 4.8 (CI = 2.00, 11.74), 2.3 (CI = 0.96, 5.58),

Table 3. Associations (adjusted for controls) between continuous sociability measures and risk of common cold

Sociability measure	Illness criterion	
	Objective	Subjective
Extraversion	$b = -0.06 \pm 0.03, p < .03$	$b = -0.10 \pm 0.03, p < .001$
Agreeableness	$b = -0.07 \pm 0.04, p < .06$	$b = -0.11 \pm 0.04, p < .005$
Positive relationships	$b = -0.04 \pm 0.02, p = .05$	$b = -0.05 \pm 0.02, p < .006$
Sociability index	$b = -0.19 \pm 0.07, p < .006$	$b = -0.30 \pm 0.07, p < .001$

Note. Each result is from a separate equation.

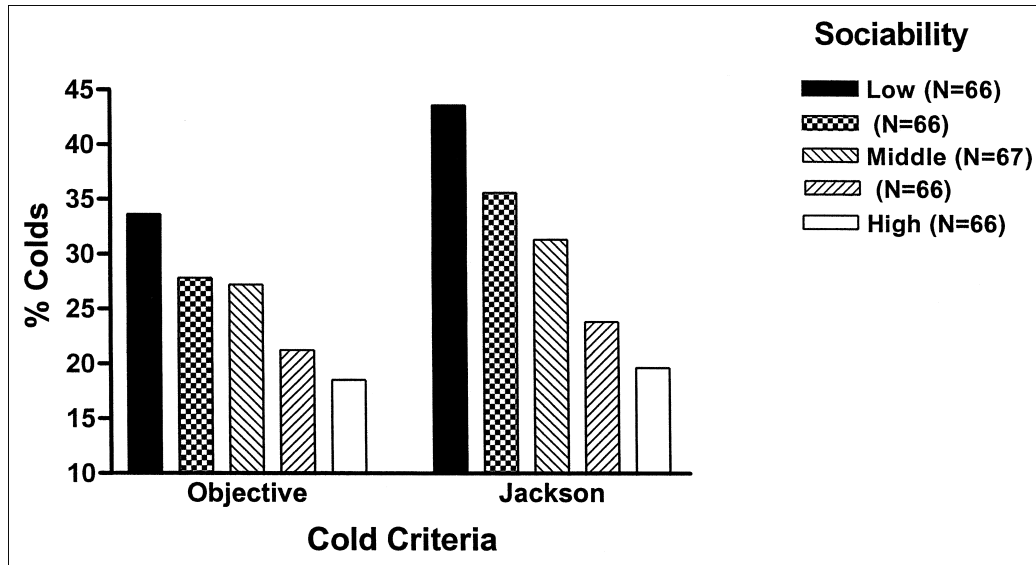


Fig. 1. Rate of developing colds (adjusted for controls) as a function of sociability quintile. Colds were defined either as infection plus objective signs of illness or as infection plus subjective symptoms of illness (modified Jackson criterion).

1.0 (CI = 0.38, 2.70), and 1 (reference group) for subjectively defined colds.

There were no statistically reliable interactions between control variables and sociability in predicting objective clinical colds. There was a sociability-by-sex interaction predicting subjective colds. Rates of colds decreased with sociability for both sexes, although low sociability was associated with a greater risk among women than men ($b = -0.34 \pm 0.14, p < .02$). Hence, the reported associations were similar across prechallenge antibody levels, age, race, sex (for the objective criterion), education, body mass index, month of exposure, and virus type.

We proposed that associations between sociability and risk for colds might be attributable to the ability of sociable people to develop and maintain relationships, particularly supportive ones. The correlations between sociability and the questionnaire and interview variables were consistent with this proposal (see Table 4). Marital status was not associated with sociability. However, none of the relationship or support variables were themselves associated with colds. Moreover, when we entered them all in the first step of the equation, they did not reduce the association between sociability and colds ($b = -0.19 \pm 0.09, p < .03$, for the objective criterion; $b = -0.35 \pm 0.09, p < .001$, for the subjective criterion). Hence they were not, alone or in combination, responsible for the sociability-illness link.

In an analysis reported elsewhere (Cohen et al., in press), we found that positive emotional style was associated with lower risk of developing a cold. In the present study, sociability was associated with increased positive emotional style ($r = .45, p < .001$) and decreased negative emotional style ($r = -.29, p < .001$). Adding positive and negative emotional style to the equation, however, also had only a minimal effect on the sociability-cold relation ($b = -0.17, \pm 0.08, p < .05$, for the objective criterion; $b = -0.33 \pm 0.09, p < .001$, for the subjective criterion).

Finally, health practices and endocrine measures were assessed as possible pathways linking sociability to illness. Correlations indicated that sociability was associated with better sleep quality (.20, $p < .001$)

and sleep efficiency (.15, $p < .006$), but not sleep duration (.02, n.s.), and with more vitamin C (.12, $p < .03$), but not dietary zinc (.06, n.s.) or exercise (.05, n.s.). Sociability was also related to lower cortisol levels ($-.15, p < .009$) and to lower levels of epinephrine ($-.11, p < .05$), but not to norepinephrine (.01, n.s.). Sociability was not associated with smoking or alcohol use. When the five variables associated with sociability were entered into an equation predicting colds, better sleep efficiency ($p < .04$) and higher cortisol levels ($p < .05$) were as-

Table 4. Correlations between sociability and questionnaire and interview measures of social interaction

Measure of social interaction	Correlation
Interview	
Total number of interactions	.29*
Average number of people interacted with per day	.21*
Average pleasantness of interactions	.29*
Percentage of interactions that were pleasant	.31*
Number of interactions that were pleasant	.39*
Percentage of interactions that were unpleasant	-.12*
Number of interactions that were unpleasant	-.05
Percentage of interactions that involved moderate to severe conflict	-.14*
Number of interactions that involved moderate to severe conflict	-.01
Questionnaire	
Number of social roles	.18*
Number of people interacted with within social roles	.23*
Perceived availability of social support	.55*

* $p < .001$

Sociability and the Common Cold

sociated with less risk. However, adding all five variables to the equation (including the control variables) did not decrease the relation between sociability and colds ($b = -0.22 \pm 0.08$, $p < .004$, for the objective criterion; $b = -0.30 \pm 0.08$, $p < .001$, for the subjective criterion; $n = 315$ because of missing data). Hence, none of these five variables were mediators.

DISCUSSION

We found that sociability was associated with greater resistance to developing colds when persons were experimentally exposed to a cold virus. Although this association was found individually for extraversion, agreeableness, and positive relationships, the largest association was found when these variables were combined to form a single sociability score. The relation between sociability and disease susceptibility was found irrespective of whether colds were defined as infection and self-reported symptoms or as infection and objective signs of illness. In both cases, the relation was approximately linear, with increases in sociability associated with decreases in disease susceptibility. That these associations were found after entering eight control variables is notable. In particular, by controlling for preexisting antibody (immunity) to the virus, we excluded as a possible explanation the idea that sociable people had more social contact and hence were more likely to have been infected in the past and have developed immunity to the virus. The association also was equal (no interactions) across prechallenge virus-specific antibody levels, age, race, sex (for the objective criterion), education, body mass index, season, and virus type. The consistency of associations for the two different viruses is especially important in that it indicates the biological generality of the association. We did find, however, that low levels of sociability produced a greater risk for women than men when the subjective definition of illness was employed.

How does sociability get inside the body? On the psychological side, we were interested in whether associations of sociability and health are mediated through interpersonal behavior. In fact, sociability was moderately associated with both increased rate and increased quality of interactions. Sociable people also had more diverse and larger networks, and perceived greater availability of social support. Sociability was similarly associated with better sleep and diet, and more positive and less negative emotions. However, our analyses failed to support any of these as potential mediators. The sociability index was associated with colds even after controlling for these alternatives.

On the biological side, we were interested in whether sociability might modulate emotion-related biological systems, like the SNS and the HPA axis, that are known to influence immune response. Sociability was associated with lower concentrations of the HPA hormone cortisol and the SNS hormone epinephrine. However, these hormones failed to meet the criteria for mediation of the sociability-cold relation.

How could perceived measures of sociability predict colds when behavioral measures of social interaction do not? One possibility is that perceived measures are partly determined by individual differences that bias people's estimates of their own sociability. In turn, it might be that it is these individual differences, not true (measured without error) social dispositions, that predict health. Alternatively, it is possible that our behavioral assessment was not optimal. We defined interactions in terms of minimal time (10 min). More meaningful ways of breaking up the stream of behavior (e.g., specific activities such as eating a meal or watching television together) might provide

an assessment more highly correlated with sociability questionnaires and with health.

Why can we not explain the association between sociability and colds in terms of health practices or endocrine variables? Few of these proposed mediators themselves predicted susceptibility to colds in this study. This is puzzling because several were predictive in previous studies (Cohen et al., 1997). However, a lower rate of illness than we expected (usually 37% for the objective criterion, but 25.7% in this study) may have resulted in insufficient power for detecting these effects. Even so, one would expect that the effect sizes of putative mediators would be at least as great as that of sociability. Reliability of measurement and sensitivity to the dynamics of mediators could be improved by measuring mediators multiple times. Moreover, broader views of the relevant biological systems (e.g., shape of diurnal rhythms, stress reactivity, and binding affinity) may tap important aspects of regulatory response not picked up by hormone concentrations.

If the proposed mediators are truly not pathways, how then could sociability be associated with resistance to colds? One explanation is that sociability, a highly heritable characteristic, is partly determined by a gene or genes that contribute to sociability but at the same time contribute to biological processes that play a role in the body's ability to fight off infection. This argument is consistent with evidence that first- and second-degree relatives of extremely shy children have a greater prevalence of hay fever than relatives of more sociable children (Kagan, Snidman, Julia-Sellers, & Johnson, 1991).

Our data indicate that sociability is an important predictor of illness that does not depend on more traditional interaction variables for its relation with disease outcomes. This may suggest that sociability is really what is behind associations of other social variables with health. However, this possibility is not supported by work that has controlled for sociability when predicting health from social-network diversity or from social conflicts (Cohen et al., 1997, 1998). More likely, sociability has its own independent associations that may be mediated through pathways that do not involve social conflicts, support, and social networks.

Finally, greater sociability was associated with more and better social interactions, performance of health-enhancing behaviors, and better regulation of emotions and stress-hormone levels. Although none of these were pathways linking sociability to resistance to colds, these results suggest that sociability may be linked to other disease processes as well.

Acknowledgments—This work was supported by a grant from the National Institute of Mental Health (MH50429), a Senior Scientist Award to Sheldon Cohen from the National Institute of Mental Health (MH00721), and a supplemental grant from the John D. and Catherine T. MacArthur Foundation Network on Socioeconomic Status and Health. The collaboration was facilitated by the Pittsburgh NIH Mind-Body Center (HL65111 and HL65112). We are indebted to Andrew Baum, Amber Baptiste, Janet Schlarb, James Seroky, Bill MacDonald, Clemens Kirschbaum, and the volunteers for their contributions to the research and to Michael Scheier for his comments on an earlier draft.

REFERENCES

- Block, G., Hartman, A.M., & Naughton, D. (1990). A reduced dietary questionnaire: Development and validation. *Epidemiology*, *1*, 58–64.
Brissette, I., Cohen, S., & Seeman, T.E. (2000). Measuring social integration and social

- networks. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 53–85). New York: Oxford University Press.
- Broadbent, D.E., Broadbent, M.H.P., Phillpotts, R.J., & Wallace, J. (1984). Some further studies on the prediction of experimental colds in volunteers by psychological factors. *Journal of Psychosomatic Research*, 28, 511–523.
- Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R., & Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index. *Psychiatry Research*, 28, 193–213.
- Cohen, S. (1988). Psychosocial models of social support in the etiology of physical disease. *Health Psychology*, 7, 269–297.
- Cohen, S., Doyle, W.J., Skoner, D.P., Rabin, B.S., & Gwaltney, J.M., Jr. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association*, 277, 1940–1944.
- Cohen, S., Doyle, W.J., Turner, R.B., Alper, C.M., & Skoner, D.P. (in press). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine*.
- Cohen, S., Frank, E., Doyle, W.J., Skoner, D.P., Rabin, B.S., & Gwaltney, J.M., Jr. (1998). Types of stressors that increase susceptibility to the common cold in adults. *Health Psychology*, 17, 214–223.
- Cohen, S., Gottlieb, B.H., & Underwood, L.G. (2000). Social relationships and health. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 3–25). New York: Oxford University Press.
- Cohen, S., Mermelstein, R., Kamarck, T., & Hoberman, H. (1985). Measuring the functional components of social support. In I.G. Sarason & B.R. Sarason (Eds.), *Social support: Theory, research and application* (pp. 73–94). The Hague, The Netherlands: Martinus Nijhoff.
- Costa, P.T., & McCrae, R.R. (1992). Four ways five factors are basic. *Personality and Individual Differences*, 13, 653–665.
- Doyle, W.J., McBride, T.P., Swartz, J.D., Hayden, F.G., & Gwaltney, J.M., Jr. (1988). The response of the nasal airway, middle ear and Eustachian tube to provocative rhinovirus challenge. *American Journal of Rhinology*, 2, 149–154.
- Eysenck, H.J. (1967). *The biological basis of personality*. Springfield, IL: Thomas.
- Geen, R.G. (1997). Psychophysiological approaches to personality. In R. Hogan, J. Johnson, & S. Briggs (Eds.), *Handbook of personality psychology* (pp. 387–414). New York: Academic Press.
- Goldberg, L.R. (1992). The development of markers for the Big-Five factor structure. *Psychological Assessment*, 4, 26–42.
- Gwaltney, J.M., Jr., Colonno, R.J., Hamparian, V.V., & Turner, R.B. (1989). Rhinovirus. In N.J. Schmidt & R.W. Emmons (Eds.), *Diagnostic procedures for viral, rickettsial and chlamydial infections* (6th ed., pp. 579–614). Washington, DC: American Public Health Association.
- Helgeson, V.S., & Gottlieb, B.H. (2000). Support groups. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 221–245). New York: Oxford University Press.
- Jackson, G.C., Dowling, H.F., Anderson, T.O., Riff, L., Saporta, M.S., & Turck, M. (1960). Susceptibility and immunity to common upper respiratory viral infections—the common cold. *Annals of Internal Medicine*, 53, 719–738.
- Kagan, J., Snidman, N., Julia-Sellers, M., & Johnson, M.O. (1991). Temperament and allergic symptoms. *Psychosomatic Medicine*, 53, 332–340.
- Kiecolt-Glaser, J.K., & Newton, T.L. (2001). Marriage and health: His and hers. *Psychological Bulletin*, 127, 475–503.
- Liebert, R.M., & Spiegel, M.D. (1994). *Personality: Strategies and issues*. Pacific Grove, CA: Brooks/Cole.
- Paffenbarger, R.S., Jr., Blair, S.N., Lee, I., & Hyde, R.T. (1993). Measurement of physical activity to assess health effects in free-living populations. *Medicine and Science in Sports and Exercise*, 25, 60–70.
- Reber, A.S. (1985). *The Penguin dictionary of psychology*. London: Penguin Books.
- Reis, H.T., & Collins, N. (2000). Measuring relationship properties and interactions relevant to social support. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 136–192). New York: Oxford University Press.
- Reis, H.T., & Wheeler, L. (1991). Studying social interaction with the Rochester Interaction Record. *Advances in Experimental Social Psychology*, 24, 269–318.
- Ryff, C.D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, 57, 1069–1081.
- Totman, R., Kiff, J., Reed, S.E., & Craig, J.W. (1980). Predicting experimental colds in volunteers from different measures of recent life stress. *Journal of Psychosomatic Research*, 24, 155–163.
- Uchino, B.N., Cacioppo, J.T., & Kiecolt-Glaser, J.K. (1996). The relationship between social support and physiological processes. *Psychological Bulletin*, 119, 488–531.
- Wills, T.A., & Shinar, O. (2000). Measuring perceived and received social support. In S. Cohen, L.G. Underwood, & B.H. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 86–135). New York: Oxford University Press.

(RECEIVED 7/18/02; REVISION ACCEPTED 10/25/02)