Emotional Style and Susceptibility to the Common Cold

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Objective: It has been hypothesized that people who typically report experiencing negative emotions are at greater risk for disease and those who typically report positive emotions are at less risk. We tested these hypotheses for host resistance to the common cold. **Methods:** Three hundred thirty-four healthy volunteers aged 18 to 54 years were assessed for their tendency to experience positive emotions such as happy, pleased, and relaxed; and for negative emotions such as anxious, hostile, and depressed. Subsequently, they were given nasal drops containing one of two rhinoviruses and monitored in quarantine for the development of a common cold (illness in the presence of verified infection). **Results:** For both viruses, increased positive emotional style (PES) was associated (in a dose-response manner) with lower risk of developing a cold. This relationship was maintained after controlling for prechallenge virus-specific antibody, virus-type, age, sex, education, race, body mass, and season (adjusted relative risk comparing lowest-to-highest tertile = 2.9). Negative emotional style (NES) was not associated with colds and the association of positive style and colds was independent of negative style. Although PES was associated with lower levels of endocrine hormones and better health practices, these differences could not account for different risks for illness. In separate analyses, NES was associated with reporting more unfounded (independent of objective markers of disease) symptoms, and PES with reporting fewer. **Conclusions:** The tendency to experience positive emotions was associated with greater resistance to objectively verifiable colds. PES was also associated with reporting fewer unfounded symptoms and NES with reporting more. **Key words:** emotions, immunity, disease susceptibility, common cold, psychological, rhinovirus, affect, personality assessment.

CI = confidence interval; **NES** = negative emotional style; **PES** = positive emotional style

INTRODUCTION

The hypothesis that the extent to which an individual typically experience an array of negative emotions contributes to their risk for morbidity has received considerable attention (1, 2). This negative emotional style (NES; also termed neuroticism or negative affectivity) can include a broad range of aversive moods such as anxiety, hostility, and depression (1). Current evidence suggests that a more extreme NES is associated with more health complaints, but not with a greater risk for verified diseases (3), including colds and flu (4, 5). In contrast, there has been little interest in the disease risk for persons with a PES characterized by moods such as happy, pleased, relaxed, and lively.

Positive and negative emotions are not opposite ends of a continuum (6). They are only modestly correlated (1) and the experience of positive and negative emotions are associated with activation of different areas of the brain (7). PES may promote health by encouraging health enhancing behaviors (1, 8), building resources to cope with stress (9) and enhancing regulation of emotion-sensitive biological systems (10). Consequently, we hypothesized that PES is associated with positive health practices, lower levels of stress hormones and resistance to infectious illness (11, 12).

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METHODS

Design

After assessment of emotional styles, demographics, virus-specific antibody levels, endocrine hormone levels, and health practices, volunteers were quarantined in separate rooms, exposed to one of two rhinoviruses, and followed for 5 days to assess infection and signs and symptoms of illness.

Subjects

Data were collected between 1997 and 2001. The subjects were 159 men and 175 women aged 18 to 54 years (means = 28.8, SD = ±10.4) who responded to advertisements and were judged to be in good health. They were studied in 10 groups and were paid $800 for their participation. The Carnegie Mellon University and University of Pittsburgh institutional review boards approved the study and informed consent was obtained from each subject.

Experimental Plan

Volunteers underwent medical screenings and were excluded if they had a history of psychiatric illness, major nasal or otologic surgery, asthma or cardiovascular disorders, or abnormal urinalysis, complete blood count (CBC), or blood enzymes, were pregnant or currently lactating, seropositive for HIV, or on regular medication. Specific serum antibody titer to the challenge virus, demographics and weight and height were assessed at screening. Baseline emotional styles, urine catecholamines, saliva cortisol, and health practices were assessed during the 6 weeks between screening and virus exposure.

During the first 24 hours of quarantine (before viral exposure), volunteers had a nasal examination and a nasal lavage. Baseline symptoms, nasal mucociliary clearance, and nasal mucus production were assessed. Volunteers were excluded at this point if they had signs or symptoms of a cold or if a viral pathogen was isolated from the nasal lavage.

Then, subjects were given nasal drops containing 100 to 300 $TCID_{50}$ of one of two types of rhinovirus (RV39 [N = 228] or RV23 [N = 106]). We used two rhinovirus types to establish the generalizability of any observed associations, at least across rhinoviruses. The quarantine continued for 5 additional days. On each day, volunteers reported their respiratory symptoms, were assessed for nasal mucociliary clearance and nasal mucus production, and nasal lavage samples were collected for virus culture. Approximately 28 days after virus exposure, blood was collected for serological testing. The investigators were blinded to all psychological and biological measures.

Emotional Styles

Volunteers were interviewed by phone on three evenings per week for 2 weeks during the month before quarantine and on the first evening (before viral challenge) of quarantine. They were asked how accurately (0 = not at all accurate to 4 = extremely accurate) each of nine positive and nine negative

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adjectives described how they felt during the last day. The positive adjectives represented three subcategories of positive emotion: vigor (lively, full-of-pep, and energetic), well-being (happy, pleased, and cheerful), and calm (at ease, calm, and relaxed) (13, 14). The nine negative adjectives represented three subcategories of negative emotion: depression (sad, depressed, and unhappy), anxiety (on edge, nervous, and tense), and hostility (hostile, resentful, and angry) (13, 14). Daily positive and negative mood scores were calculated by summing the ratings of the nine respective adjectives. The internal reliabilities (Cronbach's α) for the seven assessments ranged from 0.89 to 0.93 for positive and from 0.87 to 0.92 for negative scores. To form summary measures of emotional style, daily mood scores were averaged (separately for positive and negative) across the 7 days.

A short questionnaire measure of PES was also administered at screening, 4 to 6 weeks before quarantine. The positive emotions in this questionnaire were the same nine adjectives used in the multiple measurement approach. (Unfortunately, the complete set of negative emotions was not included in this scale.) In this case, instead of rating their emotions during the last day as in the interviews, the subjects rated the extent to which each adjective described how they "usually" are. Items were summed as above to create a PES questionnaire score. The internal reliability (Cronbach's α) of the scale was 0.81. This measure correlated $r = .49$, $p < .001$ with the PES measure based on multiple interviews.

Standard Control Variables

In the analyses, we controlled for prechallenge antibody titer (>4 or <8), age (18–21, 22–32, and 33–54 years), body mass index (weight [kg]/height $[m]^2$), race [white, other], sex, virus type (RV23 or RV39), month of exposure (March, May, July, September, or December), and education (less than high school graduate, more than high school but less than 2 years college, and more than 2 years college).

Pathways Linking Emotional Style to Susceptibility

Health practices and markers of endocrine function were assessed before virus exposure. Smoking rate was defined as the number of cigarettes smoked in a day. In calculating the average number of alcoholic drinks per day, a bottle or can of beer, glass of wine and shot of whiskey were each treated as a single drink (12, 15). Exercise was 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 (16) 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 (percent of time in bed sleeping), and duration (17). Dietary intake of vitamin C and zinc were assessed by standard questionnaire (18).

Epinephrine and norepinephrine were assessed in a 24-hour urine sample collected within 2 weeks before entering quarantine and assayed with the use of high-performance liquid chromatography with electrochemical detection (19). To assess cortisol release 12 saliva samples were collected via salivettes during the first day of quarantine (one sample per hour between 5:45 AM and 4 PM with others collected at 6:30 and 10:30 PM) and assayed using time-resolved immunoassay with fluorometric end point detection (20). The area under the curve was calculated to measure total free-cortisol release.

Viral Cultures and Antibody Response

The virus-specific neutralizing antibody titer was measured in serum that was collected before and 28 days after virus exposure (21) and the results were expressed as reciprocals of the final dilution of serum. Nasal lavage samples from each day were frozen at −80°C and later cultured for rhinovirus with the use of standard techniques (21).

Signs and Symptoms

On each day of quarantine, subjects were asked if they had a cold, and rated on a scale of 0 (none) to 4 (very severe) the severity of eight respiratory symptoms (congestion, runny nose, sneezing, cough, sore throat, malaise, headache, and chills) during the previous 24 hours (22).

Daily mucus production was assessed by collecting used tissues in sealed plastic bags (23). The bags were weighed and the weight of the tissues and bags subtracted. Nasal mucociliary clearance function was assessed as the time required for dye administered into the anterior nose to reach the nasopharynx (23).

Baseline-adjusted daily scores for each measure were calculated by subtracting the appropriate baseline score from each of the five postexposure daily scores. Negative adjusted scores were reassigned a value of 0. Total scores for symptoms, mucus weight, and nasal clearance were calculated by summing the respective adjusted daily scores over the 5 days.

Infections and Colds

Volunteers were considered to have a clinical cold if they were *both* infected and met illness criteria. Infection was defined as recovery of the challenge virus on any of the 5 postchallenge days or a ≥4-fold rise in virus-specific serum neutralizing antibody titer (preexposure to 28 days postexposure) (11, 15). Two illness criteria were used. The objective criterion for illness required a total adjusted mucus weight of at least 10 g *or* a total adjusted mucociliary nasal clearance time of at least 35 minutes (11). For those with clinical colds by this criterion, the mean total adjusted respiratory symptom score was 20.87 (SD = ±18.21) vs. 7.5 (SD = ±9.96) for those without colds ($t(332) = -8.46$, $p < .001$). The subjective criterion required a total adjusted symptom score ≥6 in addition to either reporting a cold or reporting rhinorrhea on 3 or more days of the trial (24).

Statistical Analyses

Scores for PES and NES, body mass, total symptoms, mucus weight, mucociliary clearance, cortisol, epinephrine, norepinephrine, cigarettes/d, alcoholic drinks/d, zinc, and vitamin C intake were log transformed (base 10) to better approximate a normal distribution. Stepwise logistic regression was used to predict the binary outcome, presence/absence of a cold (25) and multiple linear regressions to predict continuous outcomes (26). Initially, PES and NES were treated as continuous variables. There, the regression coefficient, its standard error and probability level are reported. To provide an estimate of relative risk, we also present odds ratios (OR) and 95% confidence intervals (CI) for emotional style scales categorized by tertile.

To determine whether the association between emotional style measures (entered *alone* in the second step) and susceptibility to colds was substantially modified after controlling for the other variables, these were added sequentially to the first step of the regression analyses. Interaction terms were entered in a third step.

RESULTS

In the primary analyses reported here, we used the multiple interview measures of PES and NES. We tested the associations of colds with PES and NES in separate equations that included standard controls. Increases in PES were associated in a dose-response manner with decreases in the rate of clinical colds whether defined as infection and objective ($b = -.48$ [±.19] for the continuous variable, $p < .02$, $N = 334$; adjusted OR = 2.9 [CI 1.41–5.91], 2.1 [CI 1.06 to 4.33], and 1; Figure 1) or infection and subjective ($b = -.39$ [± .18], $p < .04$, $N = 334$; adjusted OR = 2.4 [CI 1.24–4.73], 1.8 [CI .92 to 3.38] and 1; Figure 1) illness criteria. NES was not associated with the incidence of clinical colds defined by either criterion (Figure 2).

There were no interactions between standard control variables and PES in predicting clinical colds defined by either criterion. Hence, reported associations were similar across preexposure antibody levels, age, race, sex, education, body mass, season, and virus type (see Table 1 for breakdown by virus and antibody level).

To assess whether the association of PES and cold incidence was independent of any association with NES, we reran

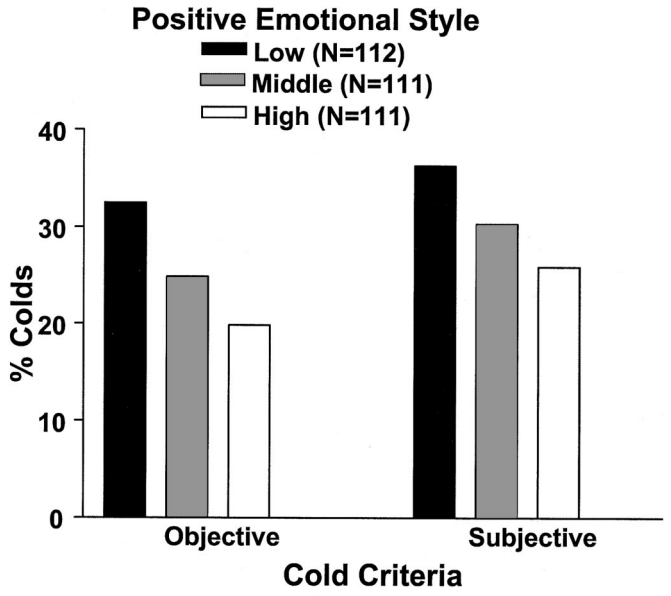

Fig. 1. Positive emotional style (by interviews) and incidence of clinical (infection + illness) colds using objective and subjective criteria for illness.

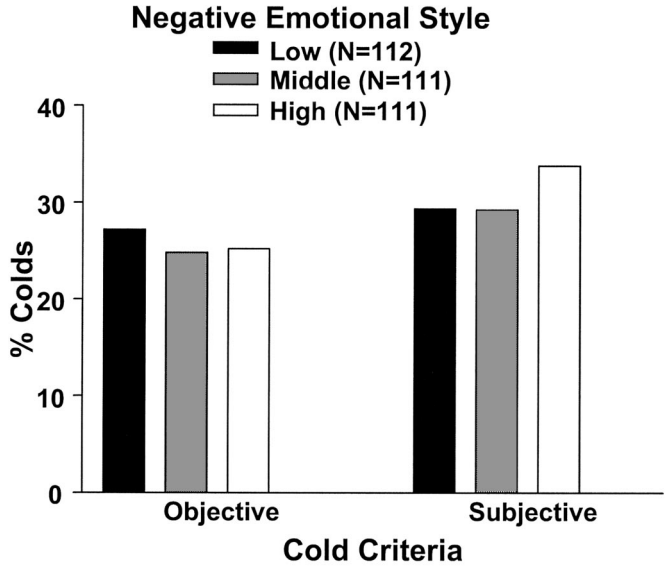

Fig. 2. Negative emotional style (by interviews) and incidence of clinical (infection + illness) colds using objective and subjective criteria for illness.

the equations reported above entering both PES and NES into the same equation. In all cases, adding NES did not alter the associations between PES and colds (all P values $\leq$ .03). Finally, we added the interaction between these styles to the equations. In no case did the interaction term approach significance.

To test whether our results were attributable to single components of emotional style (vs. the multidimensional representation tested above), we calculated the average response over the 7 interviews for each of the adjective subscales (vigor, well being, and calm for PES; depression, anxiety, and hostility for NES). Greater vigor ($p < .02$) and well-being ($p < .01$) were associated with fewer objective colds. There was a similar but nonsignificant ($p < .14$) association with calm. None of the NES subscales was associated with colds (P values $> .50$).

Because our definition of clinical colds combines infection with illness, the observed association between PES and clinical colds could have resulted from a decreased risk for infection and/or a decreased expression of illness among infected persons. However, PES was not associated with infection rates, but was associated with decreased rates of clinical colds among infected subjects (for objective criterion $b = -.69$ [$\pm.21$], $p < .001$, $N = 234$; OR = 4.1 [CI 1.9 to 9.0], 2.6 [CI 1.2 to 5.4], 1; for subjective criterion $b = -.60$ [$\pm.21$], $p < .004$, $N = 234$; OR = 3.5 [CI 1.6 to 7.4], 2.1 [CI 1.0 to 4.2], 1). Hence, the relation between PES and colds is attributable to infected people with lower PES expressing more signs and symptoms of illness. Separate analyses of the association of PES with the continuous measures of cold signs and symptoms in infected subjects was consistent with this association (mucus weights ($b = -.12$ [$\pm.05$], $p < .02$); mucociliary clearance function ($b = -.15$[$\pm.03$], $p < .001$); symptoms ($b = -.17$ [$\pm.05$], $p < .001$).

Health practices and endocrine measures were assessed as possible pathways linking PES to illness. Here, we report only analyses using the objective criterion but results are similar for the subjective criterion. PES was associated with better sleep quality ($p < .001$), sleep efficiency ($p < .02$), more dietary zinc ($p < .01$), more exercise ($p < .01$), and lower levels of epinephrine ($p < .04$), norepinephrine ($p < .05$) and cortisol ($p < .06$). Of these, none was significantly associated with colds, but there were marginal ($p < .15$) associations of greater sleep efficiency, greater zinc intake and greater cortisol concentrations with fewer colds. However, adding all eight health practice measures and the three endocrine measures to the equation (including standard controls) did not decrease the relation between PES and colds ($b = -0.66$ [$\pm.22$], $p < .003$, $N = 318$ (N reduced because of missing data); tertiles OR = 3.6 [CI 1.6–8.28], 2.4 [CI 1.1–5.36], 1). Hence, none of these hypothetical pathways was responsible for the reported relationship.

We also tested whether the simple questionnaire measure of PES administered at screening was associated with later susceptibility to illness. Although the effect sizes were attenuated, like the multiple interview measure, the questionnaire-based measure of PES was associated with greater risk for both objectively ($b = -0.48$ [$\pm.23$], $p < .04$; tertiles OR = 1.6 [CI = .85–3.06], 0.91 [CI = .46–1.78], 1) and subjectively defined clinical illness ($b = -0.57$ [+.23], $p < .02$; tertiles OR = 2.3 [CI = 1.21–4.30], and 1.3 [CI = 0.69–2.55], 1).

Finally, although NES was not associated with verified illness, we wanted to assess whether it would be associated with more "unfounded" health complaints. To do this, we predicted total adjusted self-reported symptoms from NES. This equation also included standard control variables, as well

TABLE 1. Rates of Clinical Colds for Objective and Subjective Illness Criteria Presented by PES Assessed by Interviews, Antibody Titers, and Virus Type

Prechallenge Ab Titer	Virus Type	Positive Emotional Style		
		Low	Middle	High
		Objective criteria		
≤4	RV23	23.0 (n = 22)	16.6 (n = 26)	14.3 (n = 21)
	RV39	42.8 (n = 52)	35.2 (n = 45)	29.2 (n = 43)
	Total	36.9 (n = 74)	28.4 (n = 71)	24.3 (n = 64)
≥8	RV23	18.4 (n = 9)	11.7 (n = 15)	9.4 (n = 13)
	RV39	25.7 (n = 29)	22.7 (n = 25)	15.4 (n = 34)
	Total	23.9 (n = 38)	18.6 (n = 40)	13.7 (n = 47)
		Subjective criteria		
≤4	RV23	28.0 (n = 22)	22.6 (n = 26)	21.0 (n = 21)
	RV39	48.6 (n = 52)	43.2 (n = 45)	36.9 (n = 43)
	Total	42.5 (n = 74)	35.6 (n = 71)	31.7 (n = 64)
≥8	RV23	19.4 (n = 9)	14.4 (n = 15)	12.8 (n = 13)
	RV39	25.7 (n = 29)	24.7 (n = 25)	20.0 (n = 34)
	Total	24.2 (n = 38)	20.9 (n = 40)	18.0 (n = 47)

Rates of clinical colds have been adjusted for standard control variables. The categorization of low, middle, and high scores on positive emotional style (PES) is based on whether the subject's PES score fell in the lowest (0–17.91), middle (17.92–23.49), or highest (23.50–36.00) tertile.

as the objective markers of disease–infection status, total adjusted mucus weights, and total adjusted mucociliary clearance. NES was associated with more reported symptoms of respiratory illness after removing possible contributions of objectively defined disease (see Figure 3; $b = 0.12$ [±.06], $p < .05$). Although PES was associated with verified illness in this study, it was possible that it might be associated with the extent of "unfounded" health complaints as well. Using the same statistical model (replacing NES with PES), we found that PES was associated with reporting fewer symptoms (see Figure 4; $b = .07$ [.03], $p < .04$) that were not explicable in terms of objective disease. When both PES and NES were entered into the same equation, neither was significant indicating overlapping variance in predicting symptoms.

DISCUSSION

Increases in PES were associated linearly with decreasing verified illness rates. This was true for both illness criteria and after controlling for prechallenge antibody level, demographics, body mass, season, and virus type. The similarity of the

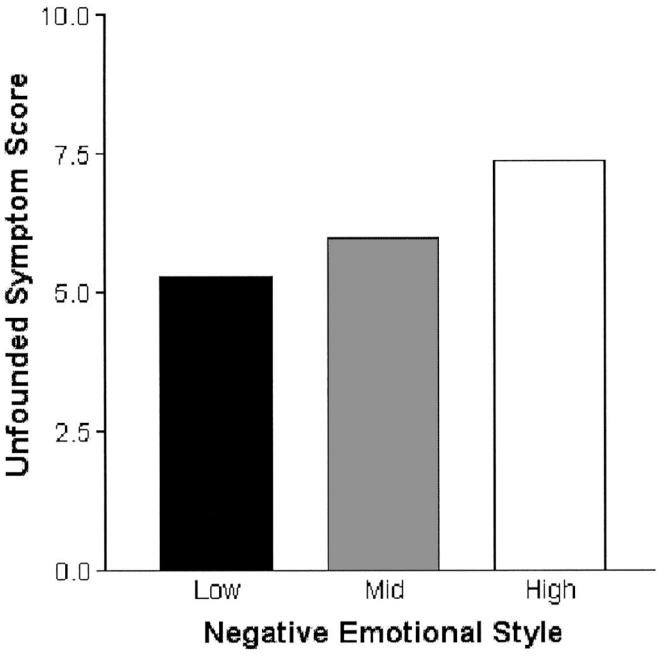

Fig. 3. Negative emotional style (by interviews) and unfounded symptoms (symptom scores adjusted for standard controls, infection status, total mucuciliary clearance, and mucus weights).

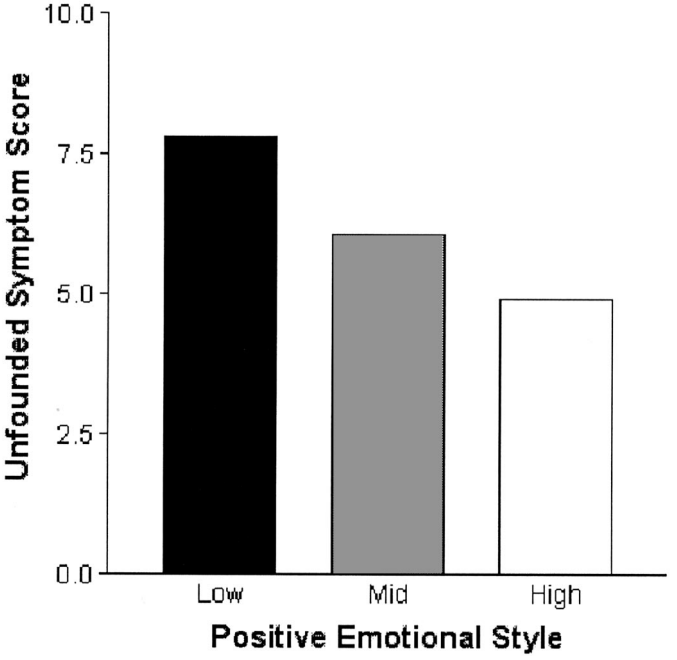

Fig. 4. Positive emotional style (by interviews) and unfounded symptoms (symptom scores adjusted for standard controls, infection status, total mucuciliary clearance, and mucus weights).

association across the two viruses suggests a biological generality for the effect. In contrast, our observation that NES did not predict verified colds but instead predicted greater reporting of unfounded symptoms is consistent with the existing literature (2–5). Interestingly, we also found that PES predicted less reporting of symptoms than predicted by objective markers of disease.

Emotional Style and Verified Disease

Each of the PES component subscales (vigor, calm, and well-being) showed the same associations verified disease as the total PES, while none of the NES subscales (anxiety, depression, and anger) was associated with colds. This suggests that the association of PES and colds reflects the role of positive emotionality as opposed to that of any specific positive emotion. Importantly, both the PES and NES measures included subscales of activated (anxiety and anger for NES; vigor and well being for PES) and unactivated (depression for NES and calm for PES) emotions (27).

Even though PES and NES were correlated ($r = -48$, $p < .001$), the association of PES with susceptibility to colds was independent of NES. While PES may be beneficial because it lessens or ameliorates the effects of negative moods, we found no support (no interaction between NES and PES) for that hypothesis.

Why can we not explain the association of PES and verified colds in terms of health practices or endocrine variables? PES was associated with better health practices and lower levels of catecholamines and cortisol, but none of these (alone or together) accounted for the PES association with colds. Few of the proposed mediators themselves predicted susceptibility to colds in this study. This is puzzling because several were predictive in previous studies (11). However, a lower rate of illness than we expected (usually 37% for objective criterion) 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 PES. Improved measurement reliability and greater sensitivity to mediator complexity and dynamics may be the solution. Multiple daily assessments of mediators (many which fluctuate in response to acute situations) would provide more reliable assessments. Moreover, assessments representing multiple views of the relevant endocrine systems (eg, shape of diurnal rhythms, stress reactivity, and receptor status) may tap important aspects of the regulatory response not represented by hormone concentrations alone.

The fact that PES was not associated with infection but was associated with the expression of signs and symptoms of illness among infected people suggests that the release or synthesis of inflammatory mediators such as proinflammatory cytokines, histamine, or bradykinins responsible for the signs and symptoms of illness may mediate the relation between PES and colds. The release of proinflammatory cytokines in response to infection is thought to be modulated by glucocorticoids, thus providing a hypothetical pathway by which psychosocial factors (via their influence on cortisol production) could control cytokine release (28). In addition, proinflammatory cytokines trigger further release of glucocorticoids, possibly exacerbating responses to psychosocial factors by positive feedback modulation. Our finding that PES was associated with cortisol levels is consistent with the possibility that proinflammatory cytokines mediate the PES-cold relation. However, this argument is weakened by the fact that cortisol levels themselves do not operate as mediating pathways.

There are two other interpretations for our failure to identify mediating factors. First, perhaps the prediction that health behaviors and endocrine responses are primary mediators is wrong. For example, positive emotions might have their influence through biological processes (eg, release of oxytocin) (29) that are different than those associated with negative emotions and stress. Second, although we controlled for demographics and other explanatory factors, it is possible that a factor(s) not assessed (eg, genetic factor, other personality characteristics) could account for both higher PES and host resistance.

It has been estimated that common colds occur at rates of 2 to 5 per person per year in adults, although in school children the incidence is more likely to be in the range of 7 to 10 colds per year (30). The data reported here suggest the possibility of developing risk profiles as well as considering the efficacy of implementing programs to enhance positive emotion to reduce risk. Depression screening instruments are commonly used in risk profiles administered in many health care settings. In evaluating these profiles, clinicians might pay attention to the positive items used in these screens (eg, "how happy are you?") in addition to the profile total score. In fact, the positive items on the Center for Epidemiological Studies of Depression Scale were already shown to predict incidence of stroke (31). Simple screening measures of PES (such as the ratings of how one "usually is" that were employed in our study) might also be useful. The accuracy of any of these screening techniques will increase if the same instrument is given at two or more office visits and the scores averaged across time.

Emotional Styles and Unfounded Symptoms

Although NES was not associated with risk for illness, it was associated with reporting symptoms after controlling for objective indicators of illness. There are several theories about why NES is related to unverified health complaints (4, 5), but a likely explanation is that those experiencing negative emotions interpret ambiguous physical sensations in a negative light, ie, as symptoms (32). This tendency would be exacerbated in a context in which subjects already know they will be receiving a cold virus. That the affective tone is the key to interpreting physical sensations is supported by the new finding here that PES is associated with reporting fewer symptoms than expected given objective markers of disease. In this case, higher PES might result in more positive interpretations of ambiguous sensations. The fact that NES and PES associations with symptoms overlap raises the question of whether

the existing literature on NES and symptoms reporting might actually be explicable in terms of PES.

CONCLUSIONS

In summary, we found that the tendency to experience positive emotions was associated with greater resistance to developing a common cold. However, this association was not mediated by either the assessed health practices or stress-related hormones. Interestingly, PES was associated with better health practices and lower basal levels of epinephrine, norepinephrine and cortisol. To the extent that these proposed pathways are risk factors for other diseases, it is possible that PES will be associated with lower risk for those diseases as well. Finally, we found that PES was also associated with reporting more and NES associated with reporting fewer self-reported symptoms after removing the possible contributions of the actual disease. This indicates that both of these emotional styles are associated with biases in interpreting our physical sensations.

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REFERENCES

1. Watson D. Intraindividual and interindividual analyses of positive and negative affect: their relation to health complaints, perceived stress, and daily activities. J Pers Soc Psychol 1988;54:1020–30.
2. Watson D, Pennebaker JW. Health complaints, stress and distress: exploring the central role of negative affectivity. Psychol Rev 1989;96: 234–54.
3. Costa PT, McCrae RR. Hypochondriasis, neuroticism, and aging: when are somatic complaints unfounded? Am Psychol 1985;40:19–28.
4. Cohen S, Doyle WJ, Skoner DP, Fireman P, Gwaltney JM Jr, Newsom JT. State and trait negative affect as predictors of objective and subjective symptoms of respiratory viral infections. J Pers Soc Psychol 1995;68: 159–69.
5. Feldman P, Cohen S, Doyle WJ, Skoner DP, Gwaltney JM Jr. The impact of personality on the reporting of unfounded symptoms and illness. J Pers Soc Psychol 1999;77:370–8.
6. Diener E, Emmons RA. The independence of positive and negative affect. J Pers Soc Psychol 1985;47:71–5.
7. Davidson R. Affective style, psychopathology, and resilience: brain mechanisms and plasticity. Am Psychol 2000;55:1196–214.
8. Myers DG, Diener E. Who is happy? Psychol Sci 1995;6:10–19.
9. Fredrickson BL. The role of positive emotions in psychology: the broaden-and-build theory of positive emotions. Am Psychol 2001;56:218–26.
10. Stone AA, Cox DS, Valdimarsdottir H, Jandorf L, Neale JM. Evidence that secretory IgA antibody is associated with daily mood. J Pers Soc Psychol 1987;52:988–93.
11. Cohen S, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM Jr. Social ties and susceptibility to the common cold. J Am Med Assoc 1997;277: 1940–4.
12. Cohen S, Tyrrell DAJ, Russell MAH, Jarvis MJ, Smith AP. Smoking, alcohol consumption and susceptibility to the common cold. Am J Public Health 1993;83:1277–83.
13. Usala PD, Hertzog C. Measurement of affective states in adults: evaluation of an adjective scale instrument. Res Aging 1989;11:403–26.
14. Benyamini Y, Leventhal EA, Leventhal H. Gender differences in processing information for making self-assessments of health. Psychosom Med 2000;62:354–64.
15. Cohen S, Tyrrell DAJ, Smith AP. Psychological stress and susceptibility to the common cold. N Engl J Med 1991;325:606–12.
16. Paffenbarger RS Jr, Blair SN, Lee I, Hyde RT. Measurement of physical activity to assess health effects in free-living populations. Med Sci Sports Exerc 1993;25:60–70.
17. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index. Psychiatry Res 1989;28:193–213.
18. Block G, Hartman AM, Naughton D. A reduced dietary questionnaire: development and validation. Epidemiology 1990;1:58–64.
19. Baum A, Grunberg N. Measurement of stress hormones. In: Cohen S, Kessler RC, Underwood GL, editors. Measuring stress. New York: Oxford University Press; 1995. p. 75–92.
20. Kirschbaum C, Hellhammer DH. Salivary cortisol in psychobiological research: an overview. Neuropsychobiology 1989;22:150–69.
21. Gwaltney JM Jr, Colonno RJ, Hamparian VV, Turner RB. Rhinovirus. In: Schmidt NJ, Emmons RW, editors. Diagnostic procedures for viral, rickettsial and chlamydial infections. 6th ed. Washington DC: American Public Health Association; 1989. p. 579–614.
22. Farr BM, Gwaltney JM Jr, Hendley JO, Hayden FG, Naclerio RM, McBride T, Doyle WJ, Sorrentino JV, Riker DK, Proud D. A randomized controlled trial of glucocorticoid prophylaxis against experimental rhinovirus infection. J Infect Dis 1990;162:1173–7.
23. Doyle WJ, McBride TP, Swarts JD, Hayden FG, Gwaltney JM Jr. The response of the nasal airway, middle ear and Eustachian tube to provocative rhinovirus challenge. Am J Rhinology 1988;2:149–54.
24. Jackson GC, Dowling HF, Anderson TO, Riff L, Saporta MS, Turck M. Susceptibility and immunity to common upper respiratory viral infections–the common cold. Ann Intern Med 1960;53:719–38.
25. Hosmer DW Jr, Lemeshow S. Applied logistic regression. New York: Wiley, 1989.
26. Cohen J, Cohen P. Applied multiple regression/correlation analysis for the behavioral sciences. Hillsdale, NJ: Erlbaum Associates; 1975.
27. Larsen RJ, Diener E. Promises and problems with the circumplex model of emotions. In: Clark MS, editior. Emotion. London: Sage Publications; 1992. p. 2–59.
28. Dobbs CM, Feg N, Beck FM, Sheridan JF. Neuroendocrine regulation of cytokine production during experimental influenza viral infection. J Imm 1996;157:1870–1877.
29. Taylor SE, Cousino Klein L, Lewis BP, Gruenewal TL, Gururn RAR, Updegraff JA. Biobehavioral responses to stress in females. Tend-and-befriend, not fight-or-flight. Psych Rev 2000;107:411–429.
30. Johnston S, Holgate S. Epidemiology of viral respiratory infection. In: Myint S., Taylor-Robinson D, editors. Viral and other infections of the human respiratory tract. London: Chapman and Hall, 1996; p. 1–28.
31. Oster GV, Markides KS, Peek MK, Goodwin JS. The association between emotional well-being and the incidence of stroke in older adults. Psychosom Med 2001;63:210–215.
32. Cohen S, Williamson G. Stress and infectious disease in humans. Psych Bull 1991;109:5–24.