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State and Trait Negative Affect as Predictors of Objective and Subjective Symptoms of Respiratory Viral Infections

Sheldon Cohen
Carnegie Mellon University

William J. Doyle, David P. Skoner, and Philip Fireman
Children's Hospital, Pittsburgh, Pennsylvania

Jack M. Gwaltney, Jr.
University of Virginia School of Medicine

Jason T. Newsom
University of Pittsburgh

State and trait negative affect (NA) were measured in healthy people immediately before an illness was induced through exposure to a respiratory virus. State NA, disease-specific health complaints (e.g., runny nose, congestion, and sneezing), and an associated objective marker of disease severity (mucus secretion weights) were assessed daily during the illness. Baseline trait and state NA were both associated with increased numbers of subsequent complaints. Although greater numbers of complaints among people high in state NA were explicable in terms of greater disease severity, the association of trait NA and symptoms was independent of objective disease. The trait NA complaint association was also independent of state NA and hence not attributable to trait-elicited state affect. Greater trait NA was associated with biases in complaining during but not before illness. This suggested failure to discriminate between symptoms rather than increased sensitivity or hypochondriacal response.

Negative affect (NA) refers to undifferentiated subjective distress. It subsumes a broad range of aversive mood states (e.g., anxiety, hostility, and depression) that form a general distress factor (Watson, 1988). It can be measured either as a transient fluctuation in mood (state) or as a stable individual difference in affective level (trait). There is considerable interest in the role that NA plays in the onset and progression of physical illness (e.g., Costa & McCrae, 1985a; Watson & Pennebaker, 1989). The most common hypothesis is that NA is bad for one. Although myriad studies have been cited as providing support for this hypothesis (e.g., see review by Watson & Pennebaker, 1989), the literature is plagued by conceptual and methodological inconsistencies that make it difficult to provide a broad theoretical basis for understanding the nature of such a relation. Major problems include (a) a failure to clearly define health outcomes in terms of whether they represent biases in cognitive processes or underlying changes in disease progression, (b) a failure to separate the independent contributions of transient fluctuations in negative mood (state NA) and stable individual differences in negative affective level (trait NA), and (c) a failure to clarify whether NA-induced cognitive biases involve (over)-sensitivity to symptoms or (mis)interpretation of benign sensations as symptoms. This article addresses these and related issues by presenting data from a prospective study of symptom reporting among people experimentally infected with rhinovirus or influenza virus. State and trait NA were measured before respiratory illness was induced through viral exposure. After viral exposure, we assessed both disease-specific illness complaints and an associated objective indicator of disease severity.

Sheldon Cohen, Department of Psychology, Carnegie Mellon University; William J. Doyle, Department of Otolaryngology, Children's Hospital, Pittsburgh, Pennsylvania; David P. Skoner and Philip Fireman, Division of Allergy, Immunology, and Rheumatology, Children's Hospital, Pittsburgh, Pennsylvania; Jack M. Gwaltney, Jr., Department of Internal Medicine, University of Virginia School of Medicine; Jason T. Newsom, University Center for Social and Urban Research, University of Pittsburgh.

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Correspondence concerning this article should be addressed to Sheldon Cohen, Department of Psychology, Carnegie Mellon University, Pittsburgh, Pennsylvania 15213. Electronic mail may be sent to Cohen@psy.cmu.edu.

Is NA Associated With Complaining or With Poorer Health?

Both state and trait NA have been associated with elevated levels of somatic complaints (see reviews by Friedman & Booth-Kewley, 1987; Watson & Pennebaker, 1989). However, there is only scattered evidence that either state or trait NA predicts measures closely tied to verified pathology such as mortality rates and objective evidence of disease and dysfunction (see reviews by Costa & McCrae, 1985a; Watson & Pennebaker, 1989). Because there have been few well-designed studies addressing the relation of emotions and objective measures of physical ill-

ness, the status of this relation is still open to question (Cohen & Williamson, 1991). Of course, more health complaints among people high in NA could be attributable to biases in encoding or reporting symptoms, actual (biologically based) health problems, or both (Cohen & Williamson, 1991; Costa & McCrae, 1985a; Watson & Pennebaker, 1989). This ambiguity often leads to the same data being discussed in one context as support for NA influences on disease and in another context as support for NA influences on psychological processes involved in encoding and reporting symptoms and diseases. Given this confusion, it is unfortunate that only a few published studies have simultaneously addressed the relations among NA, symptom reporting, and associated objective measures of pathology (see Costa & McCrae's, 1985a, summary of work on heart disease).

Is It the State or the Trait That Is Associated With Health?

Earlier we referred to state NA as transient fluctuations in negative mood and trait NA as a stable individual difference in negative affective level. This is a standard distinction between state and trait, but somewhat more refined distinctions are helpful in this context. By trait NA, we mean a stable underlying disposition that characterizes affective response for months, years, or even a lifetime. It represents common variance in state NA that occurs across time but is also associated with a cluster of nonaffective trait characteristics including self-consciousness, inability to inhibit cravings, and vulnerability to stress (Costa & McCrae, 1985a). Trait NA is also referred to as neuroticism and is closely associated with concepts such as trait anxiety, poor morale, and low self-esteem. By state NA, we mean the affective response that occurs at any arbitrarily defined point in time. The notion of response suggests that state is best measured as the difference between the average response across time (trait) and the current response (e.g., Watson, 1988). However, most correlational studies of state NA and symptoms do not correct for trait and hence probably reflect both trait and state.

As discussed earlier, there are data implicating both trait and state NA in health. Are these relations independent of one another? Are they attributable to the same mechanism or to different mechanisms? First consider the possible independent role of state NA. Strong evidence for the independent effect of state NA on health complaints has been provided by the experimental literature. In two different laboratories, the manipulation of negative mood increased the number and severity of self-reported symptoms of illness (Croyle & Uretsky, 1987; Salovey & Birnbaum, 1989). In these studies, random assignment eliminated the usual natural correlation between trait and state NA and hence the possibility that trait NA might be spuriously driving the relation. How could state NA influence illness complaints? An effect of state NA on symptom reporting might occur because negative moods result in negative biases in the evaluation and categorization of stimuli (e.g., facilitating the labeling of physical sensations as representing negative consequences [symptoms] and defining symptom constellations as disease states; Cohen & Williamson, 1991; Pennebaker, 1982). Negative moods also facilitate access to memories of negative experiences such as illness (e.g., Bower, 1981; Clark & Isen, 1982; Salovey & Birnbaum, 1989). Evidence regarding associations between state NA and objective indicators of disease similarly suggests a relation independent of trait NA. Although trait NA has not been consistently found to predict objective indicators of disease (Watson & Pennebaker, 1989), recent evidence suggests that state NA is associated with both immune function (see review by Herbert & Cohen, 1993) and increased risk of developing biologically verified upper respiratory illnesses (Cohen, Tyrrell, & Smith, 1993). These relations are attributed to mood-related alterations in endocrine and, consequently, immune response.

As noted earlier, trait NA has been repeatedly associated with increased illness complaints. What is not clear is whether people high in trait NA report more complaints because they are experiencing a negative mood state at the point of symptom reporting or because of a more general influence of the NA personality. There are a number of other characteristics of people high in trait NA that can explain increased complaining. For example, Gray (1982) proposed that people high in trait NA (he used the term *trait anxious*) are hypersensitive, constantly scanning incoming stimuli as possible sources of impending trouble. This could result in greater attention to bodily sensations and aches and pains or even to the interpretation of normal stimuli as pathological (Watson, 1988). Relatedly, those high in trait NA are more introspective and ruminative (Watson & Clark, 1984). An association of this kind of internal orientation and reports of physical symptoms presumably occurs because introspective people are more likely to attend to their bodies and to interpret sensations as negative (Pennebaker, 1980, 1982; Pennebaker & Lightner, 1988). Although the mechanisms are not well established, characteristics of NA personality might also influence the pathogenesis of disease and, consequently, illness complaints. Examples of possible mechanisms include tendencies to engage in high-risk behaviors, to be differentially reactive to stressors, and to have resting levels of hormones or immune function that make one differentially susceptible to immune-mediated disease. Interestingly, there have been few studies attempting to determine whether relations between trait NA and somatic complaints are attributable to current mood or to other trait characteristics. An exception is the work of Watson (1988), who found more health complaints independently associated with greater state and trait NA.

Is NA Associated With Reporting of Real or Imagined Symptoms?

It is thought that trait NA is associated with a bias to report more symptoms (Watson & Pennebaker, 1989). As discussed earlier, this association is presumed to occur because people high in trait NA are more attentive to their bodies and are more likely to notice bodily sensations and to (mis)interpret these sensations as indicators of illness (Ward & Leventhal, 1993). However, this hypothesis is quite general in nature and does not distinguish between (a) mistaking normal sensations as symptoms and (b) increased sensitivity to true symptoms of underlying illness (Cohen & Williamson, 1991; Pennebaker, 1982; Watson & Pennebaker, 1989). Many of the existing studies relating

NA to health complaints have been conducted with healthy samples and have used "psychosomatic" symptom measures that assess the presence of relatively vague, nonspecific symptoms such as stomachaches, muscle aches, backaches, faintness, dizziness, and cold spells. It is plausible that NA results in reinterpretation of benign sensations into these kinds of nonspecific symptoms, especially when the symptoms are primed by a symptom inventory. However, of greater interest is whether similar "hypochondriacal" responses occur in the course of a specific disease. That is, do people high in NA who have a specific illness report symptoms (appropriate to the disease) that are not consistent with objective measures of disease progression and severity?

Do Associations Between NA and Illness Complaints Merely Reflect a Methodological Flaw?

Much of the existing literature on the relation between NA and somatic complaints is subject to a spurious explanation focusing on a bias that comes into play because of the method used to assess somatic complaints. Specifically, virtually all studies in this literature have asked for retrospective reports of illness (Larsen, 1992). As discussed earlier, negative emotional states are known to facilitate access to memories about negative experiences such as illness (Bower, 1981; Clark & Isen, 1982). Thus, elevated reports of illness may occur because people high in NA are primed to recall these states. Such an association presumably would not occur if health complaint data were collected concurrently. Consider, for example, a study assessing current symptoms and illnesses rather than those occurring over the last month. In fact, Larsen (1992) pursued this issue in a recent study. When he collected both concurrent and retrospective reports of health complaints, the relation of trait NA and illness reporting was primarily found for retrospective reports (recall bias) rather than concurrent reports (encoding of illness). In the study reported in this article, all somatic complaint data were collected concurrently (daily) and hence were not subject to recall bias.

We present a prospective study in which state and trait NA were measured in healthy people who we then infected with a dose of respiratory virus sufficient to cause illness. We assessed a cluster of health complaints and an associated biological marker of disease. This paradigm allowed us to address a number of conceptual and methodological questions raised by the existing literature, including the following: (a) Are state and trait NA associated with objective measures of disease expression or only with the experience and reporting of symptoms? (b) Are the commonly reported associations of trait NA and health attributable to the affective state associated with the trait or to other characteristics associated with trait NA? (c) Is NA associated with increased sensitivity to true symptoms of illness or with reports of symptoms with no pathophysiological basis? and (d) Are there associations between NA and health complaints when the biases associated with retrospective reports of health problems are minimized?

Method

Procedure

Volunteers were recruited from the community. Each was screened through a medical exam; only healthy (nonpregnant) adults who had tested negative for the human immunodeficiency virus and who were not on a medication regimen were eligible to participate. The mean age was 25.8 years (SD = 8.0, range = 18 to 53). Fifty-one percent were female. Volunteers who met selection criteria completed a series of questionnaires that assessed state and trait negative affectivity, had blood drawn for specific antibody assessments, and reported baseline upper respiratory symptoms. Subsequently, the participants were given nasal drops containing an infectious dose of one of two respiratory viruses. Fifty-three participants received rhinovirus 39, and 33 received the Kawasaki A influenza virus. Both of these viruses result in acute respiratory disease with upper respiratory symptoms. A saline (no virus) control group was not included in this study because earlier work indicated that those receiving saline do not report increases (over baseline) in disease-specific symptoms (e.g., Cohen, Tyrrell, & Smith, 1991). After viral exposure, participants were quarantined in a hotel and monitored daily for signs and symptoms of upper respiratory infection and objective indicators of pathophysiology. Measures collected daily during the trial included samples of nasal secretions for detection of infection, facial tissues for assessing mucus secretion weights, and daily self-reports of NA states and upper respiratory symptoms. Approximately 4 weeks after participants were exposed to the virus, a second blood sample was collected from each participant and analyzed for a second marker of infection: a fourfold increase over baseline in virus-specific serum antibody levels. This study addressed symptom reporting among those participants who developed biologically verifiable clinical upper respiratory illness.

Measures of State and Trait Affect

We chose to compare state and trait NA by having people rate the same adjectives under two different sets of instructions. This procedure allowed us to assess the same construct for both state and trait. The adjectives included subscales assessing anxiety (nervous, on edge, and tense), anger (hostile, angry, and resentful), depression (depressed, sad, and unhappy), and fatigue (fatigued, worn out, and tired). The subscales were derived from factor analyses of adjectives in the Profile of Mood States (POMS; Usala & Hertzog, 1989); each scale included three highly loading adjectives. The 12 adjectives were presented in random order to each participant twice just before exposure to the virus. In the trait version of the scale, participants were asked to rate how accurately each adjective described them as they are generally. Response alternatives ranged from *not at all accurate* (0) to *extremely accurate* (4). In the state version, they were asked to describe how often they had experienced each of the emotions in the previous 24 hr. Response alternatives ranged from *not at all* (0) to *a lot* (3). The trait version was administered in the morning of the day of viral exposure and the state version in the late afternoon immediately before the viral inoculation.

A factor analysis with varimax rotation of the four trait scales was conducted. Fatigue (.67), anxiety (.82), hostility (.84), and depression (.81) loaded on a single factor. As a result, we created a negative (anxiety, fatigue, depression, and hostility) trait affect scale by averaging the appropriate standardized scale scores. Thus, the mean of the combined scale was 0. This procedure equally weighed each of the factors contributing to overall NA in the calculation of the overall scale score. Factor analysis of the baseline state scale also resulted in fatigue (.62), anxiety (.75), hostility (.60), and depression (.78) loading on a single factor. We created the state NA scale using the same procedure used for the trait scale. Internal consistencies (alphas) were .75 for trait and .80 for state.

To assess the temporal stability of state NA and the importance of state NA at different stages of the disease, we also administered the state NA measure on each of the 7 days after exposure. These scales were similarly standardized within day. The distribution of trait NA scores was approximately normal, but state NA scores (both at baseline and during the trial) were subjected to square root transformations to provide approximately normal distributions. The transformed data were used in all of the analyses of state NA reported in this article.

The state NA measure was derived from standard scales of the POMS, which was designed for state measurement and for which adequate psychometric data are available (see review in Stone, in press). To provide evidence regarding the psychometrics of the trait measure, we collected data from three different samples. In the first, 45 participants completed the Eysenck neuroticism scale (Eysenck & Eysenck, 1964) as well as the state and trait NA scales described earlier. Trait NA was correlated .68 ($p < .001$) with neuroticism, and baseline state NA was correlated .29 ($p < .06$). In the second, 25 participants in the influenza trial reported here also completed the NEO Personality Inventory (NEO-PI; Costa & McCrae, 1985b) during the course of the trial. The neuroticism scale of the NEO-PI was correlated .54 ($p < .01$) with the trait NA measure but only .29 ($p < .16$) with the baseline state NA measure. The Eysenck scale's correlation with trait and state NA was reliably different, $t(42) = 3.32, p < .01$. However, the difference between the NEO-PI's correlation with state and trait did not reach statistical significance in this small sample, $t(22) = 1.34, p < .20$. Interestingly, correlations with neuroticism subscales of the NEO-PI suggested that the trait NA measure was tapping both affective (correlations of .53, .44, and .39 with anxiety [$p < .01$], hostility [$p < .05$], and depression [$p < .05$], respectively) and, to a lesser extent, nonaffective (correlations of .46, .36, and .29 with vulnerability [$p < .02$], impulsiveness [$p < .08$], and self-consciousness [$p = .16$], respectively) components of neuroticism. In the third sample, we administered the trait scale to 98 people twice to assess test–retest reliability. The trait NA scale was included in a large packet of scales administered twice between 2 and 10 days apart ($Mdn = 7$ days). The correlation between the two administrations was .86 ($p < .001$).

Infections, Clinical Illness, and Severity of Clinical Illness

Infection. Infectious diseases result from the growth and action of microorganisms or parasites in the body (see Cohen & Williamson, 1991). Infection is the multiplication of an invading microorganism. Clinical disease occurs when infection is followed by the development of symptomatology characteristic of the disease.

Biological verification of infection can be accomplished by establishing that an infectious agent is present or replicating in tissue, fluid, or both. We used two common procedures for detecting replication of a specific virus. In the viral isolation procedure, nasal secretions were cultured (put in a medium that stimulated virus replication). If the virus is present in nasal secretions, it will grow in the medium and can be detected. Alternatively, one can indirectly assess the presence of a replicating virus by looking at changes in serum antibody levels to that virus. Antibodies are protein molecules that attach themselves to invading microorganisms and mark them for destruction or prevent them from infecting cells. An invading microorganism (i.e., infection) triggers the immune system to produce antibody. Because each antibody recognizes only a single type of microorganism, the production of antibody to a specific infectious agent is evidence for the presence and activity of that agent.

Nasal washes were performed before viral exposure and daily after exposure to provide samples of nasal secretions for viral detection. Because of different temporal courses of viral shedding (replication) of the two viruses, washes were performed for 6 days after exposure for those receiving rhinovirus and for 9 days for those receiving influenza. Both sides of the nasal cavity were washed with a saline solution that was then collected for analysis. Standard methods were used to test nasal wash specimens for virus isolation; four cell culture tubes of human embryonic lung cells (WI 38) were used for rhinovirus (Gwaltney, Colonno, Hamparian, & Turner, 1989), and triplicate monolayers of Modin Darby canine kidney cells were used for influenza (Tobita, Sugiura, Enomoto, & Furuyama, 1975). Also, standard methods were used to test antibody titers to rhinovirus (homotypic neutralizing antibodies) and influenza (hemagglutination-inhibition antibodies) in preexposure and serum collected 28 days after viral exposure (Dowdle, Kendal, & Noble, 1979; Gwaltney et al., 1989). A participant was deemed infected if the virus was isolated in nasal secretions on any day after viral exposure or if there was a fourfold increase over baseline levels in the virus-specific serum antibody titer.

Symptom scores. On the day of (but before) the viral exposure (baseline) and on Days 1–7 after exposure, participants rated the presence and severity of eight upper respiratory symptoms during the previous 24 hr (Farr et al., 1990). These ratings were made between 4 p.m. and 6 p.m. The eight symptoms were congestion, rhinorrhea (runny nose), sneezing, cough, sore throat, malaise, headache, and chills. Ratings ranged from *none* (0) to *severe* (3) for each symptom.

Two different symptom measures were calculated from daily responses to these eight symptoms: the number of symptoms reported and the average severity of reported symptoms. We separated these two components of symptom reporting to distinguish between judgments of symptom severity and frequency (Ward & Leventhal, 1993). Daily number of symptom scores reflected the number of symptoms reported (rated as 1 or greater) each day. The possible range of scores on any day was 0 to 8. The total postexposure measure summed the number of symptoms reported across the 7 postexposure days. When we entered the number of times (days) each of the eight symptoms was reported in a principal-components factor analysis, all eight scores loaded on a single factor (loadings greater than .5). The internal consistency (alpha) for the scale was .75. Daily average severity scores reflected the average severity rating for symptoms reported by the participant on a single day (ratings of 1 to 3). The total postexposure measure took the average severity of symptoms reported across the 7 postexposure days. The average severity measure was intended to assess participant evaluation of the severity of experienced symptoms independent of the number of symptoms experienced. Unfortunately, we found little variation in average severity across participants ($M = 1.2$, $SD = 0.20$) and no association between either state or trait NA and severity. As a consequence, we report only data on number of symptoms. Participants were also asked each day whether they had a cold or flu.

Mucus weights. We also collected data on a clinical sign not subject to self-presentation bias: mucus weights. Mucus weights were determined by collecting tissues used by participants in sealed plastic bags for each day of the study. The bags were weighed and the weight of the tissues and bags subtracted. Preexposure mucus weight was assessed only among participants receiving the influenza virus and was based on the day before exposure. The postexposure measure was assessed for all participants and was based on the sum of mucus weights from Days 3 to 7 after exposure.

Criterion for Illness

Because we were concerned with symptom reporting among those with clinical illness, we used viral doses that normally result in most participants developing a clinical illness. However, evaluations of serum antibody levels in blood samples drawn before viral exposure indicated that some participants had antibodies to the virus with which they were to be inoculated, an indication that they had been naturally exposed to

the virus at some earlier time. Because these antibodies provide partial protection from infection, we expected that some of those with antibodies would not become ill. As a consequence, we used a criterion to exclude data from participants who did not develop an illness. Clinical upper respiratory illness was defined as the combination of verified infection and symptomatology. Those with verified infection were diagnosed as having a clinical illness if, after viral exposure, they either reported having a cold or flu or reported two or more upper respiratory symptoms not reported at baseline. (This was a modification of the Jackson criterion to accommodate symptoms from rhinovirus and influenza A viral infections [Farr et al., 1990].) Seventy-two of the possible 86 participants developed clinical illnesses. Logistic regressions indicated that neither baseline state NA nor baseline trait NA predicted the development of clinical illness. (Although a relation between state NA and illness was reported in an earlier study, the effect size was relatively small, and the sample was more than five times the size of the current sample [Cohen et al., 1993].) The mean age of these 72 participants was 26.1 years (SD = 8.1, range = 18 to 53); 2 were excluded from analysis because they did not complete the NA questionnaires. Thus, the analyses we report included 70 participants (45 receiving rhinovirus and 25 receiving influenza virus), 56% of whom were female. Analyses using a less stringent criterion for illness, infection alone (75 participants), are not reported here, but the results supported identical conclusions.

Control Variables

We collected data on a series of control variables that might provide alternative explanations for the relations between NA and illness. These variables included preexposure serostatus, age, gender, education, whether the participant received rhinovirus or influenza virus, and whether or not the participant received an experimental drug. Preexposure serostatus refers to whether a participant had antibodies to the virus before experimental exposure (i.e., was previously exposed to the virus). Serostatus was defined as positive in the case of the rhinovirus when a volunteer had a preexposure antibody titer greater than 2 and positive in the case of the influenza virus when the preexposure antibody titer was greater than 8. Age was scored continuously. Education levels were classified on an 8-point scale ranging from *didn't finish high school* (1) to *doctoral or other higher degree* (8), as reported by the participants. This research was piggybacked on studies of the development of symptoms for people exposed to respiratory viruses. As part of the original study, 16 of the 45 participants who received the rhinovirus were randomly administered nontherapeutic doses of an experimental drug. Whether or not they received a drug was also treated as a control variable.

We conducted preliminary analyses (analyses of variance and correlations) to determine whether any of the control variables were associated with both a predictor variable (state NA, trait NA, or both) and an outcome variable (mucus weights or symptoms). Control variables with both associations could provide spurious explanations for associations between the NA measures and outcomes. To be conservative, we set a significance level of $p < .10$ for selecting variables to be included as controls in later analyses. Administration of the drug was associated with trait NA ($r = .27$, $p < .03$), symptoms ($r = -.25$, $p < .04$), and mucus weights ($r = -.21$, $p < .08$). This finding was quite unexpected because drug was a randomly assigned condition. As a consequence, we included a variable (dummy coded as received drug or not) to control for possible spurious relations that could have been attributed to the drug in the regression, repeated measures, and path models.

Results

This study was designed as a strong prospective test of a number of hypotheses discussed earlier. Consequently, we focus on trait and state NA as measured before viral exposure (baseline) to address the questions we have raised. However, clarification of the role of baseline state NA is then provided by analyses of state NA as reported during the illness.

Table 1
Means and Standard Deviations for the Major Variables in the Study

Variable	*M*	*SD*
Trait NA (possible range: 0–12)	3.43	1.65
State NA (possible range: 0–9)		
Baseline (Day 0)	1.00	1.00
Day 1	0.88	0.79
Day 2	0.98	0.99
Day 3	0.90	0.79
Day 4	0.73	0.82
Day 5	0.67	0.92
Day 6	0.62	0.99
Day 7	0.72	1.35
Mucus weight (g) before viral exposure[a]	0.52	0.80
Total mucus weight (g) after viral exposure	12.53	16.18
Number of symptoms before viral exposure	0.63	0.97
Total number of symptoms after viral exposure	19.09	10.04

Note. NA = negative affect.
[a] Available for 25 participants.

Table 1 presents the means and standard deviations for the major variables in the study. Because the mean of standard scores is 0, we present descriptive statistics for NA measures based on the sums of the raw scores of the 12 adjectives.

Do State and Trait NA Predict Symptoms and Mucus Weights?

Baseline symptoms and mucus weights. As expected, there were virtually no upper respiratory symptoms reported before exposure to the virus. The mode and median baseline number of symptom scores were 0. Recall that we had preexposure mucus weights only for participants receiving the influenza virus. However, preexposure mucus weights are trivial and cannot explain postexposure differences (see Tyrrell, Cohen, & Schlarb, 1993). For the 25 participants for whom we had baseline mucus weights, the mode was 0 g, and the correlation between baseline and postexposure scores was .07 ($p > .73$).

Correlations between NA and outcomes. Table 2 reports the zero-order correlations between variables included in the major analyses. As expected, both state and trait NA, as assessed at baseline, were related to increased numbers of symptoms reported after viral exposure. Interestingly, although number of symptoms was moderately correlated with mucus weights, only state NA was associated with this biological marker of disease. The difference between the correlation of state NA and mucus weights (.26) and that of trait NA and mucus weights (−.02) was reliable, $t(67) = 2.31$, $p < .05$.

Regressions predicting total symptoms. In a second group of analyses, we fit a series of multiple regression models that

Table 2
Zero-Order Correlations Between Variables in Major Analyses

Variable	1	2	3	4	5	6
1. Baseline state NA	—	.46**	.10	.33**	.26*	.05
2. Trait NA		—	.07	.27*	−.02	.27*
3. Symptoms before viral exposure			—	.42**	.22	.07
4. Symptoms after viral exposure				—	.38**	−.25*
5. Mucus weight after viral exposure					—	−.21
6. Experimental drug						—

Note. Correlations with experimental drug (0 = did not receive drug, 1 = received drug) are point-biserial coefficients. NA = negative affect.
* $p < .05$. ** $p < .01$.

allowed us to adjust for the possible spurious effects of drug administration and to test whether the associations reported in Table 2 were equivalent across viruses. As a means of providing more conservative prospective analyses, these models also included a control for baseline symptoms. In each model, we forced baseline symptoms, drug administration (yes or no), and virus (cold or influenza) into the equation before NA score(s) and allowed the Virus × NA interaction(s) to enter last. The first two regressions individually examined the associations of state and trait NA with number of symptoms. The results of these conservative analyses were identical to those of the correlations reported earlier. Participants high in both state and trait NA reported more symptoms than their low NA counterparts: trait, $\beta = .34$, $F(1, 65) = 11.12$, $p < .01$ (10.8% of variance), and state, $\beta = .30$, $F(1, 65) = 8.59$, $p < .01$ (8.6% of variance). The Virus × NA interactions were not significant in either of the equations, indicating that the associations we found were consistent across the two virus groups.

Next we wanted to estimate the independent associations of state and trait NA and number of symptoms. Thus, we fit a single regression model in which baseline symptoms, drug administration, and state and trait NA were all entered simultaneously. We also allowed the interaction of state and trait NA to enter in a subsequent step. Although the overlap between these variables resulted in attenuation of their associations with symptom numbers, both maintained roughly equal and reasonable associations: 4.5% of the variance was attributable to trait, $F(1, 65) = 4.78$, $p < .04$, and 2.9% was attributable to state, $F(1, 65) = 3.09$, $p < .09$. There was no State × Trait NA interaction.

Regressions predicting mucus weights. Recall that state (but not trait) NA was correlated with mucus weights (see Table 2). To provide a more conservative analysis of this association, we fit two separate regression models in which either state or trait NA was entered in the prediction of postexposure mucus weights. In each model, we forced baseline symptoms, drug administration (yes or no), and virus (cold or influenza) into the equation before NA score(s) and entered the Virus × NA interaction(s) last. As with the zero-order correlations, state, $\beta = .26$, $F(1, 65) = 5.19$, $p < .03$ (6.6% of variance), but not trait NA was associated with mucus weights. There were no Virus × NA interactions.

Finally, in a model designed to assess the independent role of state NA in predicting mucus weights, we simultaneously entered baseline symptoms, drug administration, state NA, and trait NA in a single step and then entered the State × Trait NA interaction in a separate step. Again, only state NA predicted mucus weights, $\beta = .30$, $F(1, 65) = 5.40$, $p < .03$ (6.9% of variance). These data indicate that state NA is directly associated with the pathophysiological expression of illness and that it acts independently of trait NA.

Do Mucus Weights Mediate the Relations Between NA and Symptoms?

The data presented earlier indicate greater numbers of symptoms reported among people high in both state and trait NA but do not explain whether these complaints were based on underlying illness severity, represent biases in encoding or reporting symptoms, or both. To address the issue of mediation, we tested a path model containing both the direct and indirect (through mucus weights) paths between trait and state NA and number of symptoms using LISREL VII, a maximum likelihood estimation method (Jöreskog & Sörbom, 1988). (Sample size limitations did not allow us to test the measurement model [Bentler & Chou, 1988].) We first tested a saturated or fully recursive model that included all possible paths. This model is depicted in Figure 1. By definition, this model completely accounted for the associations among the variables, $\chi^2(0) = 0$. In the case of trait NA, there was a direct path to symptoms but no indirect path from trait NA to symptoms through mucus weights. In the case of state NA, there was an indirect path linking state NA to symptoms through mucus weights, but no direct path from state NA to symptoms. To test the hypothesis that the effects of state NA on symptoms were mediated by mucus weights, we tested the original model again, deleting the direct path from state NA to symptoms (Figure 2). The chi-square value associated with the trimmed model was not significant, $\chi^2(1) = 1.29$), indicating that a model that omits the direct path from state NA to symptoms explains the relations among the variables as well as the fully recursive model. The Tucker–Lewis (1973) fit index

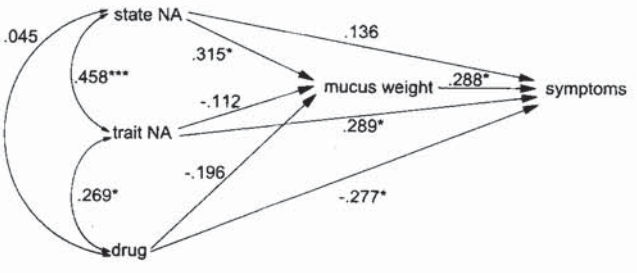

Figure 1. LISREL (path) fully recursive model testing direct and indirect (through mucus weights) paths between state and trait negative affect (NA) and total number of symptoms reported after viral exposure, including controls for drug administration. *$p < .05$; ***$p < .001$.

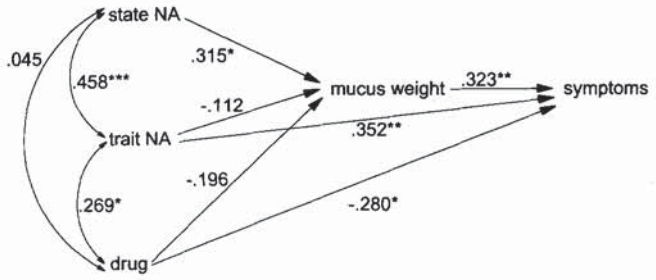

Figure 2. Trimmed LISREL (path) model testing direct and indirect (through mucus weights) paths between state and trait negative affect (NA) and total number of symptoms reported after viral exposure, deleting the direct path from state NA to symptoms. $*p < .05$; $**p < .01$; $***p < .001$.

for the second model was .950. Finally, we also tested the indirect effect of state NA on symptoms through mucus weights ($\beta = .102$, $p < .06$).

Trait NA and Symptom Course

Our analyses suggest that associations between trait NA and increased frequency of symptom reporting during viral illness are probably attributable to cognitive biases rather than to pathophysiological responses to infection. Our correlational data (Table 1) suggested that although a trait NA association with bias in symptom reporting occurred during illness, it did not occur before illness onset (baseline). To further illustrate this point and provide a formal statistical demonstration, we divided trait NA into low and high groups based on median score (−0.18) and calculated the number of symptoms before and after viral exposure for each group. The mean scores before exposure were 0.58 ($SD = 0.81$) and 0.68 ($SD = 1.12$), respectively, for low and high trait NA; the corresponding postexposure means were 16.25 ($SD = 9.71$) and 22.09 ($SD = 9.63$). A repeated measures analysis of variance with one between-subjects variable (low or high trait NA) and one within-subject variable (at baseline or after viral exposure) indicated main effects for trait NA, $F(1, 67) = 8.42$, $p < .005$, and time, $F(1, 68) = 278.00$, $p < .001$, as well as a Trait NA × Time interaction, $F(1, 68) = 6.67$, $p < .02$. (Analyses examining the individual postexposure days instead of the aggregate of the 7 days resulted in interactions with similar patterns of means on Days 1 [$p < .06$], 3 [$p < .06$], 4 [$p < .01$], and 5 [$p < .01$].) The Trait NA × Time interaction reflected the evidence that trait NA was associated with symptoms during but not before illness. This suggests that trait NA is associated with greater sensitivity to disease-related sensations but not with hypochondriacal response.

When in the Course of an Infectious Disease Does State NA Matter?

The analyses of the relation of state NA and symptoms presented up until now have included only state NA as measured at baseline, just before viral exposure. We were interested in both the course of state NA during the illness and whether state NA during the illness predicted subsequent symptoms. Table 1 presents the means and standard deviations for NA at baseline and on the 7 days after viral exposure. As is apparent from the table, there was a general decrease in state NA over the course of the trial. This probably reflects participants adjusting to being in the trial and to the various procedures involved.

Did the state NA measures on subsequent days (after exposure) predict symptoms? To maintain the prospective nature of the study, we calculated the lag correlations between state NA and subsequent symptoms for each of the consecutive days of the trial. Because current illness can influence current mood, we calculated partial correlations that controlled for symptoms on the day of the state measure. In essence, we were asking whether state NA on a certain day predicted changes in symptoms from that day to the subsequent remaining days in the trial. Because the number of days left in the trial (and the illness) decreased with each sequential day of the trial, a simple total symptom score would necessarily be based on different numbers of days for each lag. To assess whether this influenced the outcome, we calculated symptom totals based on the 1, 2, 3, and 4 days following each day of prediction. These data are presented in Table 3. As is apparent from the table, only Day 0 (baseline) state NA consistently predicted symptoms over the course of the trial. Although none of the postexposure NA measures predicted 1- through 3-day cumulative measures, Day 3 state NA did predict 4-day cumulative scores. This suggests the possibility that state NA on Day 3 did not influence symptoms until 2 or 3 days later.

Because mucus weight data were collected only on Days 3–7 of the trial, symmetric analyses were not possible. However, we were able to do lag zero-order correlations between state NA as measured on different days of the trial and mucus weights for the next 4 days (in some cases [e.g., predicting from baseline], total mucus weights were based on as little as 2 days of data). State NA at baseline was correlated with subsequent mucus weights (.23, $p = .05$). However, state measures on Days 1–3 were not (correlations ranged from −.06 to .10). Like the lag analysis predicting symptoms, these data suggest the preeminence of state NA at the time of viral exposure.

Discussion

Is NA Associated With Complaining or With Viral Illness Severity?

Individual analyses indicated that greater trait and state NA are independently associated with reporting more disease-specific symptoms. The relations occurred consistently across illnesses induced by both the rhinovirus and the influenza virus. That state and trait have independent relations with health complaints is consistent with Watson's (1988) earlier work predicting complaints that were not disease specific. Both our and Watson's data are consistent with the idea that state NA is only partly driven by trait (less than 25% overlapping variance in this study). Presumably, most state NA is determined by minor and major events that occur in day-to-day living, and this variance is sufficient to influence health complaints.

Table 3
Partial Correlations Between State Negative Affect and Number of Symptoms on Subsequent Days, Controlling for Number of Symptoms on Day of Prediction

Day of prediction	Correlation with symptom scores			
	Next day	Next 2 days	Next 3 days	Next 4 days
Day 0 (baseline)	.36**	.29*	.27*	.29*
Day 1	−.04	.06	.03	.03
Day 2	.08	.06	.11	.17
Day 3	.09	.18	.23	.27*
Day 4	−.04	−.03	−.01	
Day 5	.00	−.05		
Day 6	−.08			

* $p < .05$. ** $p < .01$.

Our path models tested both direct and indirect (through mucus weights) paths connecting both NA measures to number of reported symptoms. The association between state NA and greater symptom reporting was explicable in terms of actual underlying illness (indirect path). The association between trait NA and greater symptoms was explicable in terms of biases in encoding and reporting symptoms (direct path). These results were consistent with the independent contributions of state and trait to symptoms and the association of state but not trait NA to an objective marker of disease (mucus weights). In short, increased complaints among those high in trait NA seem to be relatively independent of objective illness, whereas increased complaints for people high in state NA are more closely tied to illness.

These results are consistent with the interpretation of many investigators that trait NA (trait anxiety or neuroticism) is associated with cognitive biases that influence symptom reporting (Costa & McCrae, 1985a; Watson & Pennebaker, 1989). The failure to find a strong bias in symptom reporting among those high in state NA is somewhat more puzzling, especially in light of experimental evidence that manipulation of negative mood increases the number and severity of self-reported symptoms (Croyle & Uretsky, 1987; Salovey & Birnbaum, 1989). However, it is possible that self-report biases found in experimental studies are attributable to the retrospective nature of their symptom measures or the vagueness or disease nonspecificity of the symptoms to which participants are asked to respond.

We also found that baseline state NA was associated with an increase in an objective indicator of respiratory illness: mucus production. Although there are few data addressing the role of state affect in illness severity, these data are consistent with other findings. Associations between negative emotional states and changes in immune function have been found in both laboratory experimental (Futterman, Kemeny, Shapiro, Polonsly, & Fahey, 1992; Knapp et al., 1992) and field correlational studies (e.g., see review by Herbert & Cohen, 1993). Moreover, state NA just before viral exposure has been associated with a greater risk for developing upper respiratory infections (Cohen et al., 1993). Even so, this is the first evidence for an association of negative mood with an objective sign of disease severity. This impact might be mediated through changes in acute behavioral responses such as smoking or drinking or through neuroendocrine response, with consequent alterations in the ability of the immune system to respond adequately to the viral exposure. Recall that baseline but not later state NA measures were associated with subsequent mucus production. These data are consistent with recent evidence on the speed of immune response to affective change (e.g., Herbert et al., 1994), as well as evidence that these immune responses may last 72 hr or more (Sieber, Rodin, Larson, Ortega, & Cummings, 1992). T-lymphocytes, natural killer cells, and B-cell production of antibody have all been established as responses to negative affective states (see review in Herbert & Cohen, 1993). Suppression of any of these functions at the time of viral exposure could result in more serious infection (greater viral replication) and, consequently, a broader range of symptoms. However, only future studies that assess appropriate immune response concurrent with state NA and then follow the course of the disease can provide definitive evidence for such mechanisms.

Is Trait NA Associated With Health Complaints Because of Associated State NA or Because of Other Trait NA Characteristics?

The data support the hypothesis that increased health complaints for people high in trait NA occur independently of state NA. This indicates that elevated negative affectivity at the point of assessment is not the primary mediator of relations between trait NA and health complaints. What is it about people high in trait NA that results in more complaining? There are no answers to this question in our data. As mentioned earlier, several explanations have been discussed in the literature. One is based on the argument that trait NA is related to a vigilant cognitive mode in which the individual scans the environment with uncertainty and apprehension (Gray, 1982; Tellegen, 1985). According to Gray, people high in NA have an overactive behavioral inhibition system (a brain structure postulated to identify all incoming stimuli as requiring careful checking). This hypervigilance is thought to result in a greater tendency to both notice and attend to bodily sensations. This could lead to mistaking normal sensations as symptoms (hypochondria), a mechanism not supported by our data, but could also lead to hypersensitiv-

ity to true symptoms of underlying illness. Alternatively, people high in trait NA have been found to be more introspective and ruminative (Watson & Clark, 1984). This internal orientation has been associated with reports of physical symptoms, presumably because these individuals are more likely to attend to their bodies and to interpret sensations as negative (Pennebaker, 1980, 1982; Pennebaker & Lightner, 1988).

Is NA Associated With Reporting of Real or Imagined Symptoms?

Our participants did not generally report upper respiratory symptoms of infectious disease when they were not actually ill (a mean of less than one symptom on the day of baseline). As a consequence, although trait NA was associated with illness complaints during illness, there was no association between trait NA and complaints before viral exposure. One possible conclusion from these data is that trait NA is associated with greater sensitivity to real symptoms but not with reporting of nonexistent symptoms. However, increased symptom reporting of those high in trait NA was also found to be independent of mucus weights, the objective indicator of illness severity. If people high in trait NA are hypersensitive, would they not show a stronger association with the objective disease indicator? One possibility is that people high in trait NA report symptoms in response to actual illness, but they are poor in discriminating specific symptoms and hence report a range of disease-relevant symptoms in response to sensations that people low in NA would identify as specific symptoms. In short, their problem might be symptom discrimination and overgeneralization rather than oversensitivity. At a cognitive level, this problem might be viewed as a manifestation of an illness representation (e.g., Leventhal, Nerenz, & Steele, in press) whose (over)activation results in biased and undifferentiated symptom interpretation.

How can we reconcile the lack of relation between trait NA and symptoms at baseline with existing literature showing that people high in trait NA report symptoms when they are not ill? The symptom measure in this study was specifically relevant to respiratory infections with a significant upper respiratory component. It is possible that inventories of nonspecific (psychosomatic) symptoms would be associated with trait NA independent of a pathological cause. That is, the process of interpreting ambiguous somatic sensations into vague, nonspecific symptoms may be more susceptible to the influence of trait NA. However, the associations found in this study are attributable to differences in complaints about what are, by all indications, true symptoms with an identifiable underlying pathology. In future work, asking people with specific diseases to report both disease-specific and disease-nonspecific (vague psychosomatic) symptoms could help clarify whether trait NA differentially influences these two types of symptom reports.

Do Associations Between Trait NA and Illness Complaints Merely Reflect a Methodological Flaw?

Larsen (1992) argued that the retrospective reports of health problems used in the vast majority of studies supporting relations between trait NA and health complaints might explain why trait NA is associated with increased complaining. In short, he attributed increases in retrospective complaints to a bias among people in a negative mood to remember negative events such as symptoms (Bower, 1981; Clark & Isen, 1982). Moreover, his own data comparing retrospective and concurrent reports of symptoms suggested that the relation between trait NA and health complaints was primarily attributable to this methodological flaw. In our study, symptom reports were collected concurrently and hence were not subject to recall bias. Even so, there were strong and consistent relations between trait NA and increased symptoms. Thus, the association between NA and increased symptom reporting in this case is attributable to differences in how physical sensations are perceived and interpreted (i.e., encoded). The daily report of symptoms not specific to a particular disease among healthy college students (as in the Larsen study) may be extremely sensitive to recall biases and involve less encoding bias. However, our own data suggest that trait NA is strongly associated with encoding biases in the case of disease-specific symptoms for sick people.

Conclusions

In sum, we found that trait and state NA are both associated with more disease-specific health complaints among people with viral respiratory illnesses. However, these associations occurred for different reasons. In the case of state NA, the association was primarily attributable to increased underlying illness. In the case of trait NA, the association was primarily attributable to overreporting of disease symptoms. Moreover, the relation between trait NA and symptom reporting appears to be driven by a component of the NA disposition other than trait-associated state affect.

It is important to emphasize that the generality of our results may be limited by several components of the study. First, we studied people who were currently ill and their reporting of disease-relevant symptoms. As discussed earlier, given the existing literature, it is likely that state NA influences on symptom reporting depend on these characteristics and that biases in reporting of more vague psychosomatic symptoms by healthy people may be directly altered by state NA. Second, we studied only two common respiratory-type illnesses. Our evidence indicating associations between more NA (both state and trait) and symptom reporting is consistent with data from other illnesses (Costa & McCrae, 1985a) and from symptom reporting outside of illness (Watson & Pennebaker, 1989). Our conclusion that trait NA influences are mediated through biases in perception and reporting is also consistent with the existing literature (Costa & McCrae, 1985a; Watson & Pennebaker, 1989). However, our data indicating that the influence of state NA occurs only through its impact on disease pathophysiology is not as well based in the current literature and requires replication. Moreover, our conclusion that trait NA does not alter the pathophysiology of disease must be tempered by our use of a less than optimal assessment of trait. Clearly, assessing trait NA with repeated state measures (e.g., Watson, 1988) or with repeated measurement with a more standard trait assessment tool (e.g., the NEO-PI or Eysenck's neuroticism scale) would provide stronger evidence for this conclusion.

Finally, we focused on undifferentiated subjective distress, ig-

noring the potential role of specific affective traits or states (e.g., depression and hostility) in disease. This is particularly important in regard to traits. Although, as discussed earlier, there is little evidence linking undifferentiated trait NA to verified disease states, there is a growing literature suggesting that some specific dispositions such as hostility (e.g., Siegman & Smith, 1994) and depression (e.g., Fraser-Smith, Lesperance, & Talagic, 1993) may be linked to verified disease, especially coronary heart disease. We were unable to ask enough questions of our participants to reliably assess differentiated trait affect and hence cannot address the potential roles of differentiated traits (or states) in this article. Clearly, research using psychometrically sound assessment of both differentiated and undifferentiated affective traits would add substantially to an understanding of what aspects of affective dispositions influence both illness and illness behavior.

References

Bentler, P. M., & Chou, C. P. (1988). Practical issues in structural modeling. In J. S. Long (Ed.), *Common problems/proper solutions* (pp. 161–192). Beverly Hills, CA: Sage.

Bower, G. H. (1981). Mood and memory. *American Psychologist, 36,* 129–148.

Clark, M. S., & Isen, A. M. (1982). Toward understanding the relationship between feeling states and social behavior. In A. Hastorf & A. M. Isen (Eds.), *Cognitive social psychology* (pp. 73–108). Amsterdam: Elsevier.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *New England Journal of Medicine, 325,* 606–612.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1993). Negative life events, perceived stress, negative affect, and susceptibility to the common cold. *Journal of Personality and Social Psychology, 64,* 131–140.

Cohen, S., & Williamson, G. (1991). Stress and infectious disease in humans. *Psychological Bulletin, 109,* 5–24.

Costa, P. T., & McCrae, R. R. (1985a). Hypochondriasis, neuroticism, and aging: When are somatic complaints unfounded? *American Psychologist, 40,* 19–28.

Costa, P. T., & McCrae, R. R. (1985b). *The NEO Personality Inventory Manual.* Odessa, FL: Psychological Assessment Resources.

Croyle, R. T., & Uretsky, M. B. (1987). Effects of mood on self-appraisal of health status. *Health Psychology, 6,* 239–253.

Dowdle, W. R., Kendal, A. P., & Noble, G. R. (1979). Influenza viruses. In E. H. Lennette & N. J. Schmidt (Eds.), *Diagnostic procedures for viral rickettsial and chlamydial infections* (5th ed., pp. 585–609). Washington, DC: American Public Health Association.

Eysenck, H. J., & Eysenck, S. B. G. (1964). *Manual of the Eysenck Personality Inventory.* London: University of London Press.

Farr, B. M., Gwaltney, J. M., Hendley, J. O., Hayden, G. G., Naclerio, R. M., McBride, T., Doyle, W. J., Sorrentino, J. V., & Proud, D. (1990). A randomized controlled trial of glucocorticoid prophylaxis against experimental rhinovirus infection. *Journal of Infectious Disease, 162,* 1173–1177.

Fraser-Smith, N., Lesperance, F., & Talagic, M. (1993). Depression following myocardial infarction: Impact on six month survival. *Journal of the American Medical Association, 270,* 1819–1825.

Friedman, H. S., & Booth-Kewley, S. B. (1987). The "disease-prone personality": A meta-analytic view of the construct. *American Psychologist, 42,* 539–555.

Futterman, A. D., Kemeny, M. E., Shapiro, D., Polonsly, W., & Fahey, J. L. (1992). Immunological variability associated with experimentally-induced positive and negative affective states. *Psychological Medicine, 22,* 231–238.

Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system.* New York: Oxford University Press.

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.

Herbert, T. B., & Cohen, S. (1993). Depression and immunity: A meta-analytic review. *Psychological Bulletin, 113,* 472–486.

Herbert, T. B., Cohen, S., Marsland, A. L., Bachen, E. A., Rabin, B. S., Muldoon, M. F., & Manuck, S. B. (1994). Cardiovascular reactivity and the course of immune response to an acute psychological stressor. *Psychosomatic Medicine, 56,* 337–344.

Jöreskog, K. G., & Sörbom, D. (1988). *LISREL VII: A guide to program and applications.* Chicago: SPSS.

Knapp, P. H., Levy, E. M., Giorgi, R. G., Black, P. H., Fox, B. H., & Heeren, T. C. (1992). Short-term immunological effects of induced emotion. *Psychosomatic Medicine, 54,* 133–148.

Larsen, R. J. (1992). Neuroticism and selective encoding and recall of symptoms: Evidence from a combined concurrent-retrospective study. *Journal of Personality and Social Psychology, 62,* 480–488.

Leventhal, H., Nerenz, D. R., & Steele, D. J. (in press). Illness representation and coping with health threats. In A. Baum & J. E. Singer (Eds.), *Handbook of psychology and health.* Hillsdale, NJ: Erlbaum.

Pennebaker, J. W. (1980). Perceptual and environmental determinants of coughing. *Basic and Applied Social Psychology, 1,* 83–91.

Pennebaker, J. W. (1982). *The psychology of physical symptoms.* New York: Springer-Verlag.

Pennebaker, J. W., & Lightner, J. M. (1988). Competition of internal and external information in an exercise setting. *Journal of Personality and Social Psychology, 39,* 165–174.

Salovey, P., & Birnbaum, D. (1989). Influence of mood on health-relevant cognitions. *Journal of Personality and Social Psychology, 57,* 539–551.

Sieber, W. J., Rodin, J., Larson, L., Ortega, S., & Cummings, N. (1992). Modulation of human natural killer cell activity by exposure to uncontrollable stress. *Brain, Behavior, and Immunity, 6,* 141–156.

Siegman, A. W., & Smith, T. W. (1994). *Anger, hostility and the heart.* Hillsdale, NJ: Erlbaum.

Stone, A. A. (in press). Measures of affective response. In S. Cohen, R. C. Kessler, & L. U. Gordon (Eds.), *Measuring stress: A guide for health and social scientists.* New York: Oxford University Press.

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In A. H. Tuma & J. D. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681–706). Hillsdale, NJ: Erlbaum.

Tobita, K., Sugiura, A., Enomoto, C., & Furuyama, M. (1975). Plaque assay and primary isolation of influenza A viruses in an established line of canine kidney cells (MDCK) in the presence of trypsin. *Medical Microbiology and Immunology (Berlin), 162,* 9–14.

Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika, 38,* 1–10.

Tyrrell, D. A. J., Cohen, S., & Schlarb, J. E. (1993). Signs and symptoms in common colds. *Epidemiology and Infection, 111,* 143–156.

Usala, P. D., & Hertzog, C. (1989). Measurement of affective states in adults: Evaluation of an adjective rating scale instrument. *Research on Aging, 11,* 403–426.

Ward, S., & Leventhal, H. (1993). *Explaining retrospective reports of side effects: Anxiety, initial side effect experience, and post-treatment*

side effects. Unpublished manuscript, University of Wisconsin, Madison.

Watson, D. (1988). Intraindividual and interindividual analyses of positive and negative affect: Their relation to health complaints, perceived stress, and daily activities. *Journal of Personality and Social Psychology, 54,* 1020–1030.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96,* 465–490.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96,* 234–254

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