Self-Discrepancy and Natural Killer Cell Activity: Immunological Consequences of Negative Self-Evaluation

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The study tested whether self-discrepancy theory could account for changes in natural killer (NK) cell activity after exposure to self-referential stimuli. Anxious, dysphoric, and control Ss were pretested and 1 month later covertly exposed to their own self-guides as well as those of another S. Blood samples were drawn for analysis of NK cytotoxicity and cortisol. The dysphoric Ss manifested the greatest actual-ideal discrepancy, whereas the anxious Ss manifested the greatest actual-ought discrepancy. Content analysis of written responses showed that activating discrepancies induced specific negative states; priming discrepancies also increased cortisol for the anxious Ss. NK activity was lower after self-referential priming for both distressed groups, particularly the anxious Ss. The control Ss showed a trend toward increased NK activity after self-referential priming. The study represents the first experimental demonstration that negative self-evaluation can alter immune responses.

Psychoneuroimmunological research has shown that psychological processes can induce acute and prolonged changes in immune responses (Ader, Felten, & Cohen, 1991; Jemmott & Locke, 1984). Momentous life events such as divorce or loss of a spouse are associated with decreases in immune indices (Irwin, Daniels, Smith, Bloom, & Weiner, 1990; Kiecolt-Glaser et al., 1987; Stein, Keller, & Schleifer, 1985). Intermittent situational stressors (such as college examinations also can cause decrements in immune function (Kiecolt-Glaser & Glaser, 1988). Recent studies indicate that some immune responses can change significantly even during brief stress (Kiecolt-Glaser, Cacioppo, Malarkey & Glaser, 1992). Thus, the focus of psychoneuroimmunological research is shifting toward elucidating the conditions and mechanisms that account for these findings.

A critical mediating factor in determining biological responses to stressful life events is the meaning of the stressor for the individual (Hochn-Saric & McLeod, 1988; Rose, 1984). Recent discussions of psychological factors in stress responsiveness (Cohen & Edwards, 1989; Lazarus & Folkman, 1984; Watson & Pennebaker, 1989) emphasize the importance of individual differences in stimulus appraisal. People who are prone to interpret events as having negative consequences for them will be more vulnerable to emotional, neuroendocrine, and immunological sequelae than others not prone to negative appraisal. Personality variables such as optimism—pessimism (Kamen-Siegel, Rodin, Seligman, & Dwyer, 1991), hardness (Funk, 1992), and coping styles (Lazarus & Folkman, 1984) all have been shown to predict stress reactivity.

To the extent that tendencies to interpret situations in certain ways are associated with specific physiological responses to stress (Rose, 1984), it should be possible to predict differences in stress reactivity from the construals people characteristically generate. The appraisal process involves evaluation of the significance of a situation for the individual, predominantly concerning dimensions such as loss or threat (Rose, 1984). In social—cognitive terms, appraisals involve comparisons between selected features of the situation and relevant personal goals or standards (Carver & Scheier, 1990; Higgins, Strauman, & Klein, 1986). Because self-standards are among the most highly accessible and frequently used standards, the appraisal process invariably entails self-evaluation (Higgins, 1990). Hence, we postulate that self-evaluation is implicated in responses to life events. Insofar as self-evaluation constitutes an important aspect of appraisal, we predict that patterns of positive or negative self-evaluation will be associated with distinct stress-response profiles. The more severe and chronic the tendency for negative self-evaluation, the greater the adverse response to stress.

Furthermore, we hypothesize that the link between self-evaluation and stress reactivity is mediated by affective responses to the outcome of the evaluation process (Cohen & Edwards, 1989). We propose the following model of self-evaluation and stress reactivity: (a) Individuals characterized by chronic negative self-evaluation will manifest chronic negative affect and will be vulnerable to acute exacerbations of their characteristic emotional state in situations triggering self-evaluative cognition (Carver & Scheier, 1990; Strauman & Higgins, 1993); (b) acute
and chronic negative emotional states involve physiological manifestations, including sympathetic and neuroendocrine activation (Rose, 1984); and (c) both sympathetic and neuroendocrine activation, in turn, are associated with acute as well as prolonged changes in various immune responses (O'Leary, 1990). Thus, if negative self-evaluation leads to negative affect, and negative affect is associated with sympathetic and neuroendocrine activation, then negative self-evaluation may lead to alterations in immune responses. Although there is abundant evidence supporting causal links between negative self-evaluation and negative affect as well as between negative affect and change in immune function, to our knowledge, a model such as this has not yet been tested experimentally.

Self-Discrepancy: A Theory of Self-Evaluation and Affect

Our study used self-discrepancy theory (Higgins, 1987) to examine the impact of self-evaluation-induced anxious and dysphoric states on immune function. We chose to conduct the study with groups of anxious and dysphoric subjects because they are likely to possess substantial chronic self-discrepancy (Strauman & Higgins, 1988) and to manifest heightened emotional responses to negative self-evaluation, thus increasing our likelihood of detecting an effect of anxiety or dysphoria on immune function (discussed later in this article). Self-discrepancy theory is a model of the relation between self-beliefs and affect. The theory distinguishes among domains of self-beliefs, including the actual self (a person's representation of the attributes that she or he or a significant other believe she or he actually possesses), the ideal self (a person's representation of the attributes that she or he or another would ideally like her or him to possess), and the ought self (a person's representation of the attributes that she or he or another believes she or he should or ought to possess). The actual self constitutes what is typically meant by the term self-concept; the ideal and ought self-states are self-evaluative standards or self-guides.

Discrepancies between the actual self and a self-guide evoke negative psychological situations involving particular motivational and emotional states. In an actual-ideal (AI) discrepancy, the person is vulnerable to dysphoria (sadness, disappointment, and dissatisfaction), resulting from the appraisal that one's hopes and wishes are unfulfilled (the loss of positive outcomes). In an actual-ought (AO) discrepancy, the person is vulnerable to anxiety (fear, worry, and agitation), resulting from the appraisal that one has failed to meet one's obligations and is liable for punishment (the [anticipated] presence of negative outcomes). Both correlational (Higgins, Klein, & Strauman, 1985; Strauman & Higgins, 1988) and experimental (Higgins, Bond, Klein & Strauman, 1986; Strauman, 1990, 1992; Strauman & Higgins, 1987) studies in analog and clinical samples support the contention that AI and AO discrepancies are causally linked with dysphoric and anxious states, respectively. These anxious and dysphoric states, in turn, lead to changes in cognition, physiology, and behavior.

Self-discrepancies represent proximal causal factors for the negative affective states implicated as a pathway between appraisal and alterations in immune responses. In addition, self-discrepancies, although conceived of nonothetically, can be idiosyncratically assessed (Higgins, Bond, et al., 1986), and individually tailored stressor stimuli can be covertly presented by use of cognitive priming techniques, permitting controlled experimentation. This study was intended to test a specific sequence leading from exposure to a personally relevant stressor to acute change in immune function in vulnerable individuals: Contextual activation of self-discrepancy → negative psychological situation (i.e., negative appraisal) → negative affect → alteration in immune response. We used a priming technique adapted from Higgins, Bond, et al. (1986) that was previously shown to activate targeted self-discrepancies and induce specific negative emotional states.

Affect and the Immune System

Considerable research has explored the impact of emotional states on various immune indices. Knapp et al. (1992) observed alterations in lymphocyte responses after the induction of negative mood by trained actors. Ironson et al. (1990) noted affect-dependent changes in several indices of immune status among individuals awaiting and receiving notification of human immunodeficiency virus antibody status. Of direct relevance in this context is a recent review summarizing the immunological effects of acute psychological stressors, including mental challenge, the viewing of unpleasant films, and conflict situations (Kiecolt-Glaser et al., 1992).

In addition to the immune effects of acute stressors, there have been a number of studies indicating an association between immunological abnormalities and various types of psychopathology. A recent meta-analysis of the association between depression and immune alterations (Herbert & Cohen, 1993) supported the conclusion that both clinical depression and depressed mood are reliably associated with decreased natural killer (NK) cell activity and other alterations in immune function. A similar literature has emerged describing an association between anxiety and immunity. Although few studies of immunity in anxiety disorders described in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1987) have appeared (e.g., Brown, Charney, Woods, Heniger, & Tallman, 1988), available data indicate that anxiety-producing situations influence immune function in both animals and humans (Hoehn-Saric & McLeod, 1988). Overall, despite some objections (e.g., Stein, Miller, & Trestman, 1991), when methodological issues are properly addressed, there is strong support for an effect of depression and anxiety on important aspects of immune function.

NK Cells as an Index of Immune Function

No single immune response provides a comprehensive index of the functional state of the entire system (Dorian et al., 1982; Kiecolt-Glaser & Glaser, 1988), and the choice of immune measures has varied across studies. We examined NK activity because it can be readily and reliably assessed and is sensitive to acute psychological stress (Darko, Wilson, Gillin, & Golshan, 1991). NK cells represent a small percentage of lymphocytes in circulation (10% to 15%) with the capability of lysing a broad range of tumor and virally infected cells (Whiteside & Herbertman, 1989). NK cells provide the body with a first line of de-
fense and may also act as a continuous tumor surveillance mechanism. Research also suggests that they play a role in the regulation of B cell functions (a subset of lymphocytes responsible for antibody production; Lotsova & Ades, 1989). Thus, the NK cytotoxicity assay provides a biologically relevant in vitro assessment sensitive to stress-related physiological changes. Many studies have examined the impact of negative emotional states on NK activity in both hospitalized (Irwin et al., 1990; Maes, Bosmans, Suy, Minner, & Raus, 1989; Miller, Asnis, Lachner, Halbreich, & Norin, 1991; Schleifer, Keller, Bond, Cohen, & Stein, 1989) and nonhospitalized (Ironson et al., 1990; Kiecolt-Glaser et al., 1987) populations.

Immune changes often are due to alterations in soluble substances in the bloodstream emanating from the sympathetic nervous system and the endocrine system. In particular, hormonal secretions from the adrenal gland have been implicated in psychosomatic phenomena, with cortisol being the most commonly studied neuroendocrine moderator of stress effects on immune competence (Rabin, Cohen, Ganguli, Lysle, & Cunnick, 1989). Given the association between elevated cortisol levels and both depression and anxiety (Hoehn-Saric & McLeod, 1988; Rose, 1984), we also examined plasma cortisol to evaluate whether our stress manipulation was associated with increased endocrine activity. Such an increase would indicate that a negative emotional state had been induced and suggest one possible pathway between affect and alterations in immune function (Munck, Guyre, & Holbrook, 1984).1

**Design and Hypotheses**

In a repeated measures procedure, we covertly exposed anxious, dysphoric, and nondistressed subjects to their own self-guides and (in a separate session) to those of another subject. Subjects were selected on the basis of chronic mood status rather than self-discrepancies, with the prediction that the anxious and dysphoric groups would manifest greater AO and AI discrepancies, respectively. Three measures of the impact of self-reference versus control priming were obtained: (a) a content analysis of subjects’ written protocols in response to each set of primes, to verify the induction of predicted negative affective states; (b) plasma cortisol, an index of endocrine activity (presumably in response to negative affect), and (c) NK cytotoxicity. A complete blood count and leukocyte differential also were obtained to determine whether any observed differences in NK cell activity were due to differences in the number and types of white blood cells rather than to acute affective response to priming.

Three hypotheses were tested. First, the two distressed groups should possess substantial self-discrepancies, the dysphoric subjects having the greatest AI discrepancy and the anxious subjects having the greatest AO discrepancy. The controls should manifest lower levels of both AI and AO discrepancy. Second, covert priming with subjects’ own self-guides should induce discriminable negative affective states in the distressed groups: increased anxiety for the anxious group and increased depressive affect for the dysphoric group. Third, the distressed groups should manifest decreased NK cytotoxicity in response to the covert self-referential priming.

**Method**

**Subjects**

Potential subjects were recruited by means of advertisements displayed throughout the University of Wisconsin—Madison campus during summer session. Thirty-eight Caucasian adults participated in the study, which was described as an investigation of personality and health. Thirty-two were summer session students and received either cash payments or course credit for their participation, and the other 6 were Madison residents and received cash payment. The 6 nonstudents were proportionally distributed across the groups. Although the gender and age distributions differed among the three groups, neither gender nor age was significantly associated with any dependent measure.

**Instruments and Procedure: Subject Selection Phase**

To identify dysphoric, anxious, and nondistressed individuals, screening sessions were conducted 6 weeks before the study under the guise of a comparison of self-report personality tests. A total of 145 individuals (110 students and 35 nonstudents) responded to the questionnaires and completed the screening, which was conducted in groups of 3–6 subjects. Each subject received a packet containing the following (in counterbalanced order): Selves Questionnaire (Higgins, Bond, Klein, & Strauman, 1986), Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), State-Trait Anxiety Inventory—Trait Scale (STAI; Spielberger, Gorsuch, & Lushene, 1970), Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978), and Survey of Immunological and General Health (SIGH; Kang et al., 1991).

The BDI and STAI have been used extensively in the personality and clinical literatures, and detailed descriptions of the use and scoring of these measures may be found elsewhere (Beck et al., 1961; Spielberger et al., 1970). We have used the BDI and STAI in previous research (Strauman, 1992) to identify chronically dysphoric and anxious subjects, and the measures show excellent test-retest reliability when used for this purpose.

The LES, a 57-item self-report inventory, assesses an individual’s perception of the life stresses experienced during the preceding 12 months. In addition to listing specific events, the LES provides an index of the positive or negative character of the event and the impact of each event. A total score was derived by subtracting the absolute value of the total impact of negative events from the total impact of positive events. The LES was used to determine whether any differences in NK cytotoxicity could be accounted for by the amount of life stress subjects had recently experienced.

With the lack of immunologically relevant health surveys, the SIGH was used to assess subjects’ recent health status. The questionnaire asks respondents to indicate the frequency of selected illness conditions during the preceding 2-month and 12-month periods. Illness categories include respiratory (cold or flu), viral (e.g., herpes or mononucleosis), and fungal infections (e.g., vaginal yeast or athlete’s foot), as well as dermatological (e.g., eczema) and allergic (e.g., airborne and food) reactions. Subjects also report family history of autoimmune disease.

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1 With regard to physiological mediators of immune alterations in depression, recently there has been a shift from the hypercortisolism hypothesis to a greater emphasis on sympathetic activation (Rabin, Cohen, Ganguli, Lysle, & Cunnick, 1989). Because of the considerable increase in difficulty associated with directly monitoring the effects of sympathetic activation on NK activity (e.g., Brown, Charney, Woods, Heniger, & Tallman, 1988), and to facilitate comparison of our study with existing research, we chose to focus on cortisol.
number of days absent from school or work due to illness, and medical or recreational drug and alcohol use.

The Selfes Questionnaire asks subjects to list traits or attributes for three self-states (actual, ideal, and ought selves) that comprise different domains of the self. For the sake of brevity, only the own standpoint was included in the questionnaire (i.e., subjects were not asked to describe their actual—other, ideal—other, or ought—other self-states; Higginso Bond, et al., 1986). Each page of the questionnaire concerned a particular self-state for example, “Please list the attributes of the type of person you think you actually are” (the actual—own self-state). Because subjects spontaneously listed the attributes associated with their self-states (instead of a checklist-type procedure), the likelihood that the attributes obtained would be chronically accessible and personally significant was increased (Higgins, King, & Mavin, 1982). Subjects also rated the extremity of each attribute within each domain of the self, using a scale ranging from slightly (1) to extremely (4).

A two-stage process (Higgins, Bond, et al., 1986) was used to calculate the discrepancy between the actual—own self-state and the ideal—own and ought—own self-guides. First, each attribute in subjects’ ideal—own and ought—own self-states was classified according to its relation to the attributes in the subject’s actual—own self-state: as matching (synonymous with an actual—own attribute), mismatching (antonymous with an actual—own attribute), or nonmatching (either non synonymous nor antonymous with any actual—own attribute). Matches and mismatches were operationalized using the thesaurus contained in the WordPerfect (Version 5.1) word processing program. The second step was to quantify magnitude of discrepancy. For each self-guide, self-discrepancies were operationalized as the number of mismatches minus the number of matches. An absence of matches were weighted as 2; mismatches of degree (i.e., where an actual—self and self-guide attribute) were synonymous but differed in extent rating by more than 1 point) were weighted as 1. Nonmatches were not included in the calculations. Subjects listed an average of 66.1 attributes for each self-state (each page permitted a maximum of 10 attributes); 67% of these attributes were classified as nonmatches. The overall interrater reliability (intraclass correlation) of the self-discrepancy measure was .84. The correlation between AI and AO discrepancy was r(36) = .50, p < .01.

Subjects for the second phase were recruited by phone by a different experimenter on the basis of their BDI and STAI scores. Subjects whose BDI scores were at the top 25% of the sample and whose STAI scores were not in the top quartile were recruited for the dysphoric group. Conversely, subjects whose STAI scores were at the top 25% of the sample whose BDI scores were not in the top quartile were recruited for the anxious group. Finally, subjects whose BDI and STAI scores were both in the bottom 25% of the sample were recruited for the nondistressed control group. Of those individuals contacted, 71% (10 of 14) of the dysphoric individuals, 53% (8 of 15) of the anxious individuals, and 91% (20 of 22) of the potential controls agreed to participate.

**Instruments and Procedure: Experimental Phase**

**Experimental setting and task.** The experimental phase was presented as an ostensibly unrelated study described as an investigation of how personality is related to health. Each subject participated individually in two sessions approximately 1 week apart (mean interval between experimental sessions = 7.2 days, SD = 1.1). The methods described below were adapted from Higgins, Bond, et al. (1986) and were intended to ensure that subjects would not recognize that they were being exposed to their own self-guides under the self-referential priming condition. Subjects were told that during each session they would be given a booklet containing open-ended questions about personality traits and that after completing the booklet a blood sample would be drawn. The content of the questions represents the experimental manipulation for the study.

Each booklet contained the following four standard questions, one question per page, with the set of questions listed four times (each set containing a different, positively valenced trait attribute): (a) “Would you say it was important for you to be X? If so, why? If not, why not?” (b) “Would your parents say that they want you to be X? If so, why? If not, why not?” (c) “What might be some advantages for you of being X?” and (d) “Have there been any changes over the course of your life in whether or not you were X? If so, please describe them briefly.” Unknown to each subject, the booklet they received during each session (counterbalanced within group for order) contained either self-guide attributes that the subject had listed approximately 6 weeks earlier (self-referential priming) or self-guide attributes of another subject that had not been listed by the target subject (yoked control priming). The quasimatched procedure helped to rule out the alternate hypothesis that any effects were due to the specific attributes used rather than their status as self-guides.

The self-referential booklet for each distressed subject contained some of their own self-guide attributes from the self-guide domain of interest (i.e., ideal or ought attributes), including any attributes that were mismatches (discrepant) with an actual-self attribute. The dysphoric subjects’ booklets contained self-guides unique to the ideal domain; overall, 78% of the self-referential attributes for this group were mismatches. The remaining 22% were chosen from among unique ideal self-guide attributes that were neither matches nor mismatches with the actual self. The anxious subjects’ booklet contained self-guides unique to the ought domain; overall, 68% of the self-referential attributes for the anxious group were mismatches. The remaining 32% were chosen from among other unique ought self-guide attributes that were neither matches nor mismatches with the actual self. The self-referential booklets for the controls contained either all ideal or all ought self-guide attributes, including any that were matches (congruent) with an actual-self attribute. Overall, 57% of the self-guide attributes for the control group were matches; the remaining 43% were chosen from among ideal (or ought) attributes that were neither matches nor nonmatches with the actual self.

Attributes for the yoked control booklets were chosen as follows: Each dysphoric subject was assigned four ideal self-guide attributes taken from the mismatching self-referential attributes of other dysphoric subjects. Each anxious subject was assigned four ought self-guide attributes taken from the mismatching self-referential attributes of other anxious subjects. Ten of the control subjects were assigned four ideal self-guide attributes taken from the mismatching self-referential attributes of dysphoric subjects, and the other 8 control subjects were assigned four ought self-guide attributes taken from the mismatching self-referential attributes of anxious subjects.

Each subject was escorted to a private clinic room where informed consent was obtained and instructions for completing the booklet were provided. Subjects were asked to be as frank and open as possible in answering and were told that after approximately 4 hr the experimenter would return and escort them to a nearby room where the blood sample would be drawn. Subjects also were instructed that there were no “correct” answers and that they should concentrate on responding to each question in detail rather than on completing the entire booklet. Subjects were then left alone to work on the booklet. At the completion of the second priming session, subjects underwent a brief interview to determine whether they had discovered the actual hypotheses. The interview was constructed to begin with general questions (e.g., “Do you have any ideas about what we’re looking at in this study?”) and move on to more specific ones (e.g., “Do you know where

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2 The method used to select anxious and dysphoric subjects reflects the high intercorrelation between the BDI and STAI in our sample, $r = .56$. 

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the words in the booklet came from?). No subject reported having discovered, or had suspicions about, the study hypotheses.

We analyzed the incidental negative emotional content of the written responses by searching for dysphoric and anxious content. We scored for anxious content by using the anxiety content rating scale of Gottschalk and Gleser (1969); two undergraduate raters blind to subject group and study hypotheses rated the presence or absence of anxious content in each verbalized memory. Interrater reliability (intraclass correlation) for the presence or absence of anxious content was .89; in cases of disagreement, a third blind rater’s judgment was used. Dysphoric content was scored using the depressive content rating scale of Gottschalk and Hoagard-Martin (1986). Interrater reliability for the presence or absence of dysphoric content was .90. Because the two coding systems were developed for joint use, there was no overlap of anxious and dysphoric content. Each successive phrase was assigned to one of three categories (no emotional content, anxious content, or dysphoric content).

**Blood sampling and immunological and endocrine measures.** Immediately after each 30-min writing period, the experimenter escorted the subject to a room designated for blood sampling. All sampling took place between 8:30 a.m. and 10:30 a.m. to control for diurnal variation. Each subject donated approximately 15 mL of venous blood drawn by a trained phlebotomist. The groups did not differ significantly on time interval between experimental sessions.

A commercial laboratory conducted the complete blood counts (CBCs) including differentials, using a Cell Dyn. CBC results provided an analysis of the white cells into the percentages of lymphocytes, neutrophils, monocytes, eosinophils, and basophils. The CBC results provide an index of acute changes in cell distribution in peripheral blood that may reflect psychological stress (Coe & Scheffler, 1989).

Plasma cortisol levels were examined as a measure of neuroendocrine response to primum-induced negative affect. Frozen aliquots of plasma were thawed and analyzed using a Clinical Assays Gamma Coat competitive protein binding assay.

To assess NK cytotoxicity, a standard assay using chromium-labeled K562 target cells, an erythroleukemia cell line, was used (Whitson, Bryant, Day, & Herberman, 1990). Although the concentration of target cells remained constant, the concentration of effector cells was varied to produce triplicate wells of four effector-to-target ratios (100:1, 50:1, 25:1, and 12.5:1) for each sample. So that we could determine the maximum values possible, control wells consisted of target cells and a 4% solution of centimide detergent; so that we could determine the minimum possible values in the absence of the subject’s lymphocytes, some wells contained the target cells suspended in 10% RPMI supplemented with fetal calf serum. After 5 hr of incubation, 200 μL of supernatant were harvested and the amount of chromium released from the target cells into the supernatant was quantified on a Beckman gamma counter. Higher gamma counts (in counts per minute) indicated a higher level of NK cytotoxicity. Lysis of target cells at each of the four ratios was calculated by the following formula:

\[
\%\ lysis = \frac{\text{Experimental cpm} - \text{spontaneous release cpm}}{\text{maximum cpm} - \text{spontaneous release cpm}} \times 100.
\]

**Experimental cpm** refers to each individual’s count; **spontaneous cpm** and **maximum cpm** refer to the control wells.

Data from individual effector-to-target ratios often are combined to compute an overall value, *lytic units* (LUs), indicating NK potency (Whiteside et al., 1990). A regression line fit to a subject’s percentage lysis across the four ratios estimated the effector-to-target ratio needed to produce a predetermined percentage of lysis (20%) per standard number of target cells. These values were then converted to an index that increases with potency (Whiteside et al., 1990). We report LUs per 10^7 effector cells. Because the LU measure produced a highly skewed distribution (skewness was 6.42; kurtosis was 12.6), analyses were performed on log-transformed LU data. NK data for three of the control subjects were partially lost because of technical difficulties, so the statistical analysis was conducted using 35 subjects (10 dysphoric, 8 anxious, and 17 control).

### Results

**Characteristics of the Subject Groups**

Table 1 describes the subjects by age, gender, emotional status, recent life events, and self-discrepancies. Neither gender nor age was significantly associated with any dependent measure, despite the predominance of women in the two distressed groups (p < .01 by chi-square test) and a marginally significant difference in age among the groups, F(2, 35) = 2.9, p < .08.3

Analysis of variance (ANOVA) indicated a significant group (dysphoric, anxious, or control) difference on the BDI, F(2, 35) = 17.43, p < .001. A Newman-Keuls test indicated that the dysphoric subjects (M = 17.1) scored significantly higher on depressive symptoms than the anxious subjects (M = 9.3), who in turn scored significantly higher than the control subjects (M = 2.2). For STAI, a significant group effect also was obtained, F(2, 35) = 27.19, p < .001. The difference between the anxious (M = 57.3) and dysphoric (M = 46.8) groups was marginal (p < .09), although both groups were significantly higher on anxious symptomatology than the controls (M = 31.9).

For self-discrepancy, a repeated measures ANOVA with group as a between-subjects factor and discrepancy type (ideal or ought) as a within-subject factor revealed a discrepancy type effect, F(1, 35) = 7.29, p < .02, as well as a Group X Discrepancy Type interaction, F(2, 35) = 7.11, p < .01. Univariate ANOVAs found a significant group effect for both AI discrepancy, F(2, 35) = 13.61, p < .001, and AO discrepancy, F(2, 35) = 22.92, p < .001. The dysphoric subjects had the highest level of AI discrepancy (M = 2.9), although it was not significantly higher than the anxious group (M = 1.8); both groups reported significantly greater AI discrepancy than the controls (M = −0.4). A contrast F test indicated that the dysphoric group manifested significantly greater AI discrepancy than the average of the other two groups, F(1, 35) = 10.86, p < .005. The anxious group possessed significantly greater AO discrepancy (M = 1.6) than both the dysphoric (M = 0.7) and nondistressed (M = −1.9) subjects.

To further test the hypothesis of discriminant AI-BDI and AO-STAI associations, we obtained partial correlations between each discrepancy type and each symptom measure, with the other discrepancy type and symptom measure statistically controlled. In this manner, AI and BDI were significantly correlated, pr(36) = .34, p < .05, after controlling for AO and STAI. Likewise, AO and STAI were significantly correlated, pr(36) = .45, p < .01, after controlling for AO and BDI.

For the SIGH, no group differences were found for incidence, severity, or family history of immune-related illness.

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3 In addition, neither gender nor age was involved in any statistically significant interaction effects for any dependent measure.
There was a group difference in the reported number of over-the-counter medications consumed, $F(2, 35) = 3.08, p < .05$. The mean number of over-the-counter medications used did not differ between the anxious ($M = 1.25$) and dysphoric ($M = 1.3$) groups and both used more than the controls ($M = 0.3$). Two dysphoric subjects were taking tricyclic antidepressants during the study; their NK and cortisol data were excluded from the final statistical analysis.

For the LES, the distressed groups had higher negative life event scores than the controls. A univariate ANOVA indicated a significant group effect for LES total score, $F(2, 35) = 7.76, p < .002$. The dysphoric and anxious groups had approximately equal scores ($M_s = -11.3$ and $-11.1$, respectively), both of which were significantly more negative than the control subjects’ scores ($M = 3.4$).

**Negative Affect Content of Written Responses**

Table 2 summarizes the incidental negative affect content of subjects’ written responses. A repeated measures ANOVA was conducted, with group as a between-subjects factor and affect type (dysphoric vs. anxious) and priming condition (self-referential vs. yoked) as within-subject factors. A significant Group × Priming × Affect Type interaction was found, $F(2, 32) = 7.00, p < .005$. Group differences were detected for dysphoric and anxious content during self-referential priming but not during yoked control priming, so each content type was analyzed separately.

For anxious content, repeated measures ANOVA found a significant Group × Priming effect, $F(2, 32) = 6.27, p < .01$. The interaction resulted from a significant group effect under self-referential priming, $F(2, 32) = 12.26, p < .001$, with no group effect in the yoked condition, $F(2, 32) = 1.24$. The anxious group generated significantly more anxious content after self-referential priming ($M = 3.3$ instances) than both the dysphorics ($M = 2.0$ instances) and controls ($M = 0.5$ instances). For dysphoric content, a repeated measures ANOVA revealed a significant Group × Priming effect, $F(2, 32) = 9.09, p < .001$. The interaction was caused by a significant group effect under self-referential priming, $F(2, 32) = 17.84, p < .001$, with no group effect in the yoked condition, $F(2, 32) = 1.15$. The dysphoric group generated significantly more dysphoric content after self-referential priming ($M = 3.6$ instances) than both the anxious group ($M = 1.5$ instances) and controls ($M = 0.8$ instances)

We then examined correlations between type of discrepancy and negative content during each session. AO was more highly correlated with anxious content during self-referential priming, $r(36) = .55, p < .01$, than during yoked priming, $r(36) = .17$. AO remained significantly associated with anxious content during self-referential priming after anxious content during yoked priming was controlled, $pr(35) = .43, p < .05$. AO was more highly correlated with dysphoric content under self-referential conditions, $r(36) = .44, p < .05$, than in the control condition, $r(36) = .24, n.s$. With dysphoric content following yoked priming controlled, AO remained correlated with dysphoric content in response to self-referential priming, $pr(35) = .38, p < .06$.

### Plasma Cortisol

Table 3 lists mean plasma cortisol levels by subject group and priming condition. Mean levels were within the normal range

**Note.** Group means in rows 4–13 with different subscripts differ at $p < .05$ by Newman-Keuls test. BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory Trait Scale. AO = actualown-ideadown self-discrepancy. AI = actualown-oughtown self-discrepancy.

* Distribution of gender by group differs at $p < .01$ by Cochran-Mantel-Haenszel test.

**Table 2**

<table>
<thead>
<tr>
<th>Content and condition</th>
<th>Dysphoric</th>
<th>Anxious</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anxious</strong> Self</td>
<td>2.0, 0.5</td>
<td>3.3, 1.5</td>
<td>2.0, 0.7</td>
</tr>
<tr>
<td><strong>Yoked</strong> M</td>
<td>1.6, 1.6</td>
<td>1.7, 1.6</td>
<td>0.7, 1.5</td>
</tr>
<tr>
<td><strong>Dysphoric</strong> Self</td>
<td>3.6, 0.8</td>
<td>1.5, 0.8</td>
<td>1.8, 0.8</td>
</tr>
<tr>
<td><strong>Yoked</strong> M</td>
<td>1.7, 0.7</td>
<td>1.9, 0.9</td>
<td>1.8, 0.9</td>
</tr>
</tbody>
</table>

**Note.** Numbers refer to the mean incidence of dysphoric or anxious statements per protocol. Means in each row not sharing subscripts differ at $p < .05$. Self = self-referential priming condition; yoked = yoked control priming condition.
Table 3
Mean Plasma Cortisol (µg/dL) by Subject Group and Priming Condition

<table>
<thead>
<tr>
<th>Condition</th>
<th>Dysphoric</th>
<th>Anxious</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self M</td>
<td>12.7ₐ</td>
<td>17.2ₙ</td>
<td>13.3ₙ</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td>2.1</td>
<td>1.4</td>
</tr>
<tr>
<td>Yoked M</td>
<td>12.9</td>
<td>13.4</td>
<td>15.7</td>
</tr>
<tr>
<td></td>
<td>2.4</td>
<td>2.0</td>
<td>1.5</td>
</tr>
</tbody>
</table>

Note: Significant differences within a row are indicated by different subscripts. Self = self-referential priming condition; yoked = yoked control priming condition.

(approximately 5–20 µg/dL). A repeated measures ANOVA with group as a between-subjects factor and priming condition as a within-subject factor found a Group × Priming interaction, F(2, 31) = 4.55, p < .03; univariate repeated measures ANOVAs within each group were then performed. For the anxious group, an ANOVA indicated a trend for an effect of priming, F(1, 6) = 3.66, p < .08; cortisol tended to be higher following self-referential priming (M = 17.2 µg/dL) than following yoked control priming (M = 13.4 µg/dL). For the dysphoric group, no reliable difference was observed between the self-referential (M = 12.7 µg/dL) and yoked control (M = 13.5 µg/dL) conditions, F(1, 9) = 1.42, p > .25. For the controls, a significant priming effect opposite to the trend for the anxious group was found, F(1, 19) = 4.84, p < .05, with cortisol after self-referential priming (M = 13.3 µg/dL) lower than that after control priming (M = 15.7 µg/dL).

To test for the relation between negative affect and cortisol, we obtained partial correlations between negative affect content and cortisol under each priming condition for the anxious and control groups (no effect was found for the dysphoric group). For yoked control priming, no significant correlations between affect content and cortisol were obtained (all ps > .25). For the anxious group, the only trend was an association between incidental anxious content and cortisol under self-referential priming, r(7) = .61, p < .10.

NK Cytotoxicity

Table 4 lists mean NK cytotoxicity at each effector-target ratio by group and priming condition; lytic units (LU) are shown in Figure 1. A repeated measures ANOVA for LU with group as a between-subjects factor and priming condition as a within-subject factor found a Group × Priming interaction, F(2, 30) = 3.53, p < .05. The interaction was due to a significant group effect for self-referential priming, F(2, 30) = 5.60, p < .01; no group effect was found for yoked control priming, F(2, 30) = 0.72. The anxious group (M = 23.9 LU) manifested significantly lower cytotoxicity than the controls (M = 69.3 LU), with the dysphorics (M = 32.1 LU) not significantly different from either.

Repeated measures ANOVAs for priming effects were then performed within each group. The anxious group showed a significant within-subject effect of priming, F(1, 7) = 6.55, p < .05, indicating that cytotoxicity was significantly lower under self-referential than under control priming. For the dysphoric group, no within-subject effect of priming condition was found, F(1, 7) = 0.30. For the controls, a trend was observed in the opposite direction: a marginal within-subject effect of priming was found, F(1, 16) = 3.33, p < .09, with relatively greater cytotoxicity following self-referential priming than following yoked control priming.

We tested the mood-mediation hypothesis by examining correlations between incidental negative affect content and LU. Within the self-referential condition, combined anxious–dysphoric content was significantly correlated with NK cell activity, r(33) = −.37, p < .05; the greater the incidental negative content, the lower the NK cytotoxicity. Conversely, negative content was not significantly correlated with NK cell activity in the control condition, r(33) = .17, p > .25.

Table 4
Mean Percentage Natural Killer Cell Cytotoxicity by Subject Group and Priming Condition for Each Effector-Target Ratio

<table>
<thead>
<tr>
<th>Effector-target ratio and condition</th>
<th>Dysphoric</th>
<th>Anxious</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>100:1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self M</td>
<td>29.5</td>
<td>30.1</td>
<td>52.9ₙ</td>
</tr>
<tr>
<td>SE</td>
<td>5.9</td>
<td>10.7</td>
<td>6.1</td>
</tr>
<tr>
<td>Yoked M</td>
<td>40.1</td>
<td>39.4</td>
<td>43.0</td>
</tr>
<tr>
<td>SE</td>
<td>5.0</td>
<td>7.9</td>
<td>6.3</td>
</tr>
<tr>
<td>50:1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self M</td>
<td>17.2</td>
<td>11.4</td>
<td>24.1</td>
</tr>
<tr>
<td>SE</td>
<td>5.2</td>
<td>5.1</td>
<td>4.6</td>
</tr>
<tr>
<td>Yoked M</td>
<td>24.5</td>
<td>23.9</td>
<td>22.3</td>
</tr>
<tr>
<td>SE</td>
<td>4.4</td>
<td>5.7</td>
<td>4.2</td>
</tr>
<tr>
<td>25:1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self M</td>
<td>7.3</td>
<td>4.7</td>
<td>12.0</td>
</tr>
<tr>
<td>SE</td>
<td>2.3</td>
<td>2.0</td>
<td>2.9</td>
</tr>
<tr>
<td>Yoked M</td>
<td>13.6</td>
<td>12.9</td>
<td>13.7</td>
</tr>
<tr>
<td>SE</td>
<td>3.1</td>
<td>3.4</td>
<td>3.5</td>
</tr>
<tr>
<td>12.5:1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self M</td>
<td>3.5</td>
<td>3.7</td>
<td>5.8</td>
</tr>
<tr>
<td>SE</td>
<td>1.9</td>
<td>2.0</td>
<td>2.1</td>
</tr>
<tr>
<td>Yoked M</td>
<td>9.1</td>
<td>10.6</td>
<td>10.0</td>
</tr>
<tr>
<td>SE</td>
<td>3.0</td>
<td>2.5</td>
<td>3.2</td>
</tr>
</tbody>
</table>

Note: Significant differences within any row of the table are indicated by different subscripts. Self = self-referential priming condition; yoked = yoked control priming condition.

No main effect or interaction involving order of priming was found for the NK measure.

We conducted a similar analysis using the NK cytotoxicity data for each effector-target ratio (with ratio as an additional within-subject factor). The results closely paralleled the analysis of the lytic units data, with the impact of self-referential priming strongest at the 100:1 ratio.
group was found, $F(2, 30) = 7.07, p < .01$. The anxious (adjusted $M = 20.3$ LUs) and dysphoric (adjusted $M = 27.4$ LUs) groups manifested significantly lower cytotoxicity than the controls (adjusted $M = 77.9$ LUs). For the yoked condition, the covariate was significantly associated with LU, $F(1, 29) = 4.17, p < .05$, but no main effect of group was found, $F(2, 30) = 0.38, ns$. No differences were found among the adjusted means after yoked control priming (anxious, 64.5 LUs; dysphoric, 39.9 LUs; control, 62.4 LUs).

**Discussion**

Contemporary theories of stress postulate that appraisal is a critical process linking life events and physiological responses. The present study demonstrated that negative self-evaluation, a predominant form of appraisal, can lead to immunological changes by means of the induction of negative affect. On the basis of previous findings that priming self-discrepancies induces negative emotional states in vulnerable individuals, we hypothesized that presenting stimuli relevant to individuals’ self-state conflicts would induce anxious or dysphoric affective states. In turn, we observed that such states, as indexed by analysis of written content and assessment of plasma cortisol, were associated with alterations of NK cytotoxicity. We believe this study is the first experimental demonstration of the immunological consequences of negative self-evaluation.

Our three-part model of the relation between self-discrepancy and altered NK cytotoxicity was supported. Chronic dysphoric states were discriminantly associated with AI discrepancy, whereas chronic anxiety was discriminantly associated with AO discrepancy (Hypothesis 1). Covert, idiographic self-referential priming induced the predicted negative affective states (Hypothesis 2). Most important, the priming-induced negative states were associated with alterations in NK cytotoxicity (Hypothesis 3). Despite the subtlety of the priming, the exclusive use of positively valenced trait attributes in the writing exercise, and the fact that subjects’ self-beliefs had been assessed approximately 6 weeks before the experimental phase, reliable effects of self-referential priming on NK cell activity were observed. On debriefing, no subject indicated awareness of the study hypotheses or the nature of the attributes used in the self-referential priming.

The inhibitory effect of self-discrepant priming on NK cytotoxicity was greatest for the anxious group, which showed significantly increased incidental anxious content and a marginal increase in cortisol. In fact, all 8 anxious subjects showed a lower NK cell response after self-referential priming, whereas 6 of the 8 dysphoric subjects included in data analyses showed this pattern of change in NK cytotoxicity. The dysphoric group was less physiologically responsive to self-discrepant priming but did show the predicted effect of self-referential priming on incidental dysphoric content. In contrast, the nondistressed subjects, whose self-referential priming included attributes related to positive psychological states (i.e., self-congruencies;

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8 Analyses of covariance determined that the observed Group × Priming effects on NK cytotoxicity remained after controlling for individual variability in the number and type of WBCs.
Table 5

<table>
<thead>
<tr>
<th>Measure</th>
<th>Dysphoric</th>
<th>Anxious</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBCs (1,000/μL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>5.2</td>
<td>5.7</td>
<td>5.1</td>
</tr>
<tr>
<td>SE</td>
<td>0.4</td>
<td>0.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Yoked</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>4.7</td>
<td>6.1</td>
<td>5.6</td>
</tr>
<tr>
<td>SE</td>
<td>0.3</td>
<td>0.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Lymphocyte %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>28.8</td>
<td>33.6</td>
<td>34.5</td>
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<tr>
<td>SE</td>
<td>2.0</td>
<td>2.9</td>
<td>1.4</td>
</tr>
<tr>
<td>Yoked</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>M</td>
<td>30.1</td>
<td>31.4</td>
<td>31.0</td>
</tr>
<tr>
<td>SE</td>
<td>1.5</td>
<td>2.6</td>
<td>2.3</td>
</tr>
<tr>
<td>Neutrophil %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>63.6</td>
<td>57.3</td>
<td>56.6</td>
</tr>
<tr>
<td>SE</td>
<td>2.1</td>
<td>2.6</td>
<td>1.7</td>
</tr>
<tr>
<td>Yoked</td>
<td></td>
<td></td>
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<tr>
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<td>61.9</td>
<td>58.4</td>
<td>60.3</td>
</tr>
<tr>
<td>SE</td>
<td>1.8</td>
<td>2.8</td>
<td>2.4</td>
</tr>
</tbody>
</table>

Note. Self = self-referential priming condition; yoked = yoked control priming condition.

Higgins (1990), showed a trend toward increased NK cytotoxicity after self-referential priming. This trend suggests the possibility that positive, self-congruent appraisals (positive self-evaluation) may have stress-buffering, immune-enhancing effects.

It should be emphasized that the differential response to the two priming conditions did not represent a nonspecific effect of low self-esteem or dispositional negative affectivity. Rather, the pattern of results implicates a predictable unintended cognitive response to the priming stimuli. Here, self-discrepancy theory offered a precise and testable operationalization of the self-evaluation process that could account for the Group × Priming condition interactions.

The data also are consistent with correlational studies relating self-discrepancies to health problems. Higgins, Tykocinsky, and Vookles (1990) observed that one form of chronic AO discrepancy predicted subjects’ subsequent reports of a variety of physical symptoms, including poor appetite, diarrhea, migraine headaches, menstrual problems, and acid stomach—in-digestion. Higgins et al., (1985) reported similar findings in a survey of college students. Although this study did not find a significant group difference in reported health history, the data do suggest increased sympathetic activation following AO-discrepant priming in vulnerable individuals. Such a mechanism for the relation between AO discrepancy and health problems is also consistent with skin conductance data obtained in other priming studies (Strauman, 1989; Strauman & Higgins, 1987), whereby covert presentation of ought-discrepant self-guides caused increases in skin conductance number and amplitude.

Our data add to the growing body of evidence for significant alterations in immune responses after brief stressors (Kiecolt-Glaser et al., 1992). Many of the previous findings can be explained by (or at least are associated with) changes in lymphocyte numbers due to stress-induced variation in cell trafficking. However, we did not observe a lymphocytopenia in any of the three groups. Moreover, other studies have shown that NK cell numbers tend to increase in peripheral blood circulation after acute stress (e.g., Darko et al., 1991). Thus, the decrease in lysis after self-discrepent priming most likely reflected a hormone-induced change in the functioning of the NK cell. The prevailing evidence from the psychoimmunology literature points to release of catecholamines by the adrenal medulla (Rabin et al., 1989) as the dominant hormonal influence on NK cytotoxicity. We observed increased plasma cortisol following self-referential priming within the anxious group, which also manifested a greater decrease in NK activity than the dysphorias. Either catecholamines or cortisol (or both) are plausible explanations for our findings.

The study design was noteworthy in several respects. First, it involved an individually tailored acute stressor that was distinct in nature and time course from the stressors used in previous research. The term acute has often been used to characterize stressors or psychological states that may have lasted anywhere from hours to days; for example, Pennebaker, Kiecolt-Glaser, and Glaser (1988) have subjects write about traumatic personal experiences during each of 4 consecutive days. In contrast, in the present study, subjects simply wrote about positively valenced attributes such as "outgoing" or "successful" for 30 min. Blood sampling was performed immediately after the writing task, and significant immune alterations were found to occur even within such a brief time frame. Our study also was unique in terms of the idiographic and subtle nature of the stressors and the covert manner in which they were presented. Finally, the use of self-discrepancy theory in formulating our model of self-evaluation and stress responses provided a comprehensive psychological framework that is compatible with physiological perspectives on stress.

The results argue for a conceptualization of stress in which the individual’s unintended or habitual construal of social stimuli (which are typically multifaceted, complex, and somewhat ambiguous) can determine her or his emotional reaction and the physiological sequelae of that reaction. Self-discrepancy theory postulates a motivational basis for appraisal-mediated stress vulnerability in the individual’s history of self-other social reinforcement contingencies (Higgins, 1989). Self-discrepancies have been shown to function as cognitive structures, which when activated by features of the environment can induce the psychological situations they symbolize. Our findings support this assertion and extend it into the realm of biological responses to stress. In essence, what people think about themselves (both explicitly and implicitly) may matter ultimately for both emotional and physical health. The data exemplify the importance of considering psychological processes as equipotent with biological processes in the domains of emotion and stress. What is emotion inducing (and ultimately stressful) depends on the goals or standards each individual possesses and on the previous consequences for meeting or not meeting them. The acquisition of self-discrepancies reflects a combination of temperament-based emotionality and the individual’s cumulative experience of self-other contingencies. In this respect, self-
evaluation represents a locus for the interaction of psychological and biological processes subserving adaptation.

Demonstrating that brief manipulations of psychological states can alter indices of immune function also highlights the importance of comparing stress and control conditions. This design feature permitted us to observe that the distressed and nondistressed subjects differed primarily in response to self-referential priming, suggesting that the findings were due to chronic differences in baseline affective or immune status. However, the present study did not include a true baseline condition, and we cannot presume cross-groups or within-group equivalence in baseline NK cytotoxicity. One would predict from the literature lower baseline levels of NK cell activity for clinically anxious and depressed individuals (Herbert & Cohen, 1993). Future studies should include all three conditions—baseline, control, and provoked—to more clearly delineate the effects of appraisal on immune responses.

A few other caveats are in order. The limited sample size may have precluded detecting more subtle group differences or priming effects; this will be addressed in replication studies. Similarly, future studies should include more extensive assessment of mood change to further evaluate the mood-mediation hypothesis linking self-discrepancies, negative affect, and alterations in immune responses. (Still, it should be noted that priming self-discrepancies has reliably induced specific negative affective states in a number of previous studies that involved a variety of mood-relevant measures.)

Finally, we offer a word of caution concerning speculations on the possible clinical significance of these findings. At present, little is known of the health impact of deviations in NK cell activity within the normal range. Prospective studies designed to clarify the possible impact of chronically low NK cell activity have only recently been undertaken (e.g., Levy et al., 1991; Levy et al., 1989). Whereas there is some indication that persistently low NK cell activity may be associated with a greater risk for infectious diseases in young adults, our subjects did not show sustained decreases in NK cytotoxicity. A larger sample would be required to determine the consequences of differences in either basal or psychologically altered NK cell activity. Nevertheless, it is clear that experimental designs targeting the cognitive processes underlying appraisal (along with the collection of cross-validating physiological measures) will help increase our understanding of the relation between psychological processes and the immune system.

References


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