

Cognitive Appraisals and Emotions Predict Cortisol and Immune Responses: A Meta-Analysis of Acute Laboratory Social Stressors and Emotion Inductions

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Models of stress and health suggest that emotions mediate the effects of stress on health; yet meta-analytic reviews have not confirmed these relationships. Categorizations of emotions along broad dimensions such as valence (e.g., positive and negative affect) may obscure important information about the effects of specific emotions on physiology. Within the context of the integrated specificity model, we present a novel theoretical framework that posits that specific emotional responses associated with specific types of environmental demands influence cortisol and immune outcomes in a manner that would have likely promoted the survival of our ancestors. We analyzed experiments from 66 journal articles that directly manipulated social stress or emotions and measured subsequent cortisol or immune responses. Judges rated experiments for the extent to which participants would experience theoretically relevant cognition and affect clustered around five categories: (a) cognitive appraisals, (b) basic emotions, (c) rumination and worry, (d) social threat, and (e) global mood states. As expected, global mood states were unassociated with the effect sizes, whereas exemplars from the other categories were generally associated with effect sizes in the expected manner. The present research suggests that coping strategies that alter appraisals and emotional responses may improve long-term health outcomes. This might be especially relevant for stressors that are acute or imminent, threaten one's social status, or require extended effort.

Keywords: stress, cortisol, immune system, emotion, appraisal

Models of stress and health suggest that appraisals and emotions mediate the effects of stress on health. Yet meta-analytic reviews have failed to confirm relationships between negative affect and cortisol or immune responses. One explanation is that categorizations of emotions along broad dimensions such as valence (e.g., positive and negative affect) may obscure important information about the effects of specific emotions on these systems. In line with this view, the integrated specificity model posits that distinct emotions are associated with distinct physiological responses (Dickerson, Gruenewald, & Kemeny, 2004; Kemeny, 2003; Weiner, 1992). Understanding the impact of specific appraisals and emotions on cortisol and immunity has important implications for health. Identifying the cognitive and

affective antecedents of hormonal and immune responses to stress is a crucial step toward informing evidence-based interventions designed to improve health. Cortisol is of particular interest because it is associated with dysregulation of the immune system. Repeated activation of the stress response and delayed recovery to stress are suspected of increasing risk for many negative health outcomes (Sapolsky, 1998, 2004). Over time, a weakened immune system leaves individuals susceptible to disease and chronic illness. Thus, the present research might eventually contribute to increasing health, quality of life, and longevity.

Early conceptualizations of physiological reactivity to stress focused almost exclusively on fight-or-flight responses (Canon, 1932). Clearly, physiological systems that would have supported these behaviors would have increased survival and hence reproductive success in nonhuman animals as well as *Homo sapiens*. Perhaps not surprisingly, humans share many physiological similarities with animals when exposed to stressful situations. However, two crucial distinctions between humans and most animals are that humans evolved in a highly complex social environment and our expanded frontal cortex supports unique cognitive abilities. As such, humans are capable of experiencing a number of unique appraisals and emotions that might activate specific hormonal, immune, and behavioral responses. It is likely that many of these responses promoted survival during complicated and stressful social situations, but in modern times, these responses tend to be unnecessary or maladaptive.

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Only limited research within the context of the integrated specificity model has investigated the effects of specific emotions on these outcomes. From this evolutionary perspective, the purpose of the present research was to explain how specific cognitive and emotional responses are related to acute physiological responses to social situations. In what follows, we describe our use of high-inference coding procedures to examine the influence of specific emotions and appraisals on cortisol and immune reactivity to emotional stress. This work represents the first meta-analytic support for the integrated specificity model.

Social Stressors and Emotion Inductions

Stressors are often divided into physical (e.g., cold or pain) and psychological categories. Psychological stressors refer to threats to psychological well-being (Kemeny, 2003; Lazarus & Folkman, 1984). Within the realm of psychological stressors, one potentially useful distinction is between mental and emotional stressors (Benschop & Schedlowski, 1999). Mental stressors predominantly consist of performing a cognitively demanding task such as the Stroop color-naming task, often under time pressure. Emotional stressors are characterized as having a predominant emotional component. Of particular interest is a type of emotional stressor known as social stressors. Social stressors are characterized by real or implied (but psychologically real) interaction with others and possessing a relatively stronger affective than cognitive component. Examples of social stressors include public speaking and marital conflict discussions. Another laboratory method of eliciting an emotional stress response from participants is to directly manipulate emotions independent of direct social contact. This relatively smaller body of work has employed a wide variety of methods including emotion-laden films (Mittwoch-Jaffe, Shalit, Srendi, & Yehuda, 1995), music (Hucklebridge et al., 2000), and reexperiencing emotional events (McCleery, Bhagwagar, Smith, Goodwin, & Cowen, 2000). The effect of such emotion inductions on the immune system has not yet been meta-analytically investigated. An additional methodological issue relevant for emotion induction studies is that the intended manipulation may simultaneously elicit a number of emotions. For example, recalling a happy experience might elicit happiness as well as pride. Moreover, this issue is likely exacerbated in stress experiments (i.e., when stress is the independent variable) because stress manipulations vary in social complexity and how they are implemented. Thus, through the use of high-inference coding procedures, the current meta-analysis individually examines the effects of a broad range of theoretically relevant emotions and cognitive appraisals on cortisol and immune outcomes.

Emotions and Appraisals

Initial conceptualizations of the stress response proposed that all stressors, regardless of their unique characteristics, should produce a uniform physiological response (Selye, 1975). This initial conceptualization is known as the generality model. Believing that specific emotions are likely related to specific physiological outcomes, more recent theorists have proposed the integrated specificity model, which postulates that distinct

emotions produce distinct physiological outcomes and that these effects are influenced by cognitive appraisals (Dickerson, Gruenewald, & Kemeny, 2004; Kemeny, 2003; Weiner, 1992). The model uses the term *integrated* because stressful experiences require a coordinated, integrated response on behalf of the entire organism. Such a response consists of physiological, psychological, and behavioral correlates (e.g., Weiner, 1992). When viewed from this perspective, psychological responses (i.e., emotions and cognitive appraisals) act to orient the organism to a threat and respond appropriately. However, when such an integrated system responds to everyday experiences that are not life threatening, unnecessary or maladaptive responses may occur.

In recent decades, a large body of evidence has emerged suggesting that experimentally induced stress influences cortisol and immune function (Dickerson & Kemeny, 2004; Segerstrom & Miller, 2004). Stress is presumed to produce these outcomes via cognitive appraisals and emotions. In his classic work on stress and coping, Lazarus (1966, 1999; see also Lazarus & Folkman, 1984) described the subjective experience of stress and its emotional sequelae as a result of the cognitive appraisal that one's available resources are not sufficient to deal with the present situational demands. Indeed, research has demonstrated that stressors perceived as uncontrollable, novel, challenging, or threatening contribute to the stress response (Dickerson & Kemeny, 2004; Lazarus & Folkman, 1984; Tomaka, Blascovich, Kelsey, & Leitten, 1993). Specific cognitive appraisals produce specific emotional responses (Frijda, 1986; Lazarus & Folkman, 1984). These emotional responses, in turn, are thought to mediate the effect of stressful events on cortisol and immune reactivity (e.g., G. E. Miller & Cohen, 2001; G. E. Miller, Dopp, Myers, Stevens, & Fahey, 1999).

Empirical evidence for the roles of emotions in affecting cortisol and immunity is limited. Researchers often manipulate stressful conditions and measure the emotions elicited with self-report measures (e.g., the Profile of Mood States; McNair, Lorr, & Droppleman, 1992). The researcher might examine correlations between composite variables of positive and negative affect and the physiological outcomes of interest. One particularly interesting finding from this work is that such global composite measures of affect are generally unassociated with cortisol and immune reactivity. For instance, Dickerson and Kemeny's (2004) meta-analytic work found that subjective self-reports of distress (i.e., negative affect) were uncorrelated with cortisol responses. Moreover, two meta-analyses concerning the effect of stress on the immune system reported that there was generally no relationship between self-report measures of perceived stress and immune outcomes in nonexperimental settings (Herbert & Cohen, 1993; Segerstrom & Miller, 2004).

One previously considered explanation for these results is that cortisol and the immune system may not be sensitive to emotional valence (e.g., Segerstrom & Miller, 2004, p. 619). In other words, classifying emotions into the broad classifications of positive and negative may not be a sensitive enough taxonomy for detecting such relationships. This suggests that specific emotions and cognitive appraisals that are otherwise obscured by broad dimensions (e.g., affective valence) may play an important role. Alternatively, other potential global dimensions of emotion such as level of emotional arousal (calm vs. excited)

and motivational action tendencies (approach vs. withdrawal) may prove to be significant predictors of cortisol and immunity. In the present meta-analysis, we therefore tested whether specific emotions, as well as these latter global dimensions of either arousal or action tendencies, influence cortisol and immune reactivity.

A Contextual Framework

In highly stressful situations, such as escaping from an enemy, the stress response ensures both energy allocation to potentially life-saving metabolic processes and energy diversion away from extraneous processes that do not aid in immediate survival. In the short term, the stress response is a finely tuned adaptation for survival. Within seconds of a stressful encounter, the brain signals the activation of the sympathetic nervous system (SNS) via the neurotransmitter epinephrine. The heart is stimulated to pump blood more rapidly to the muscles that need increased fuel. At the same time, nonessential processes such as digestion are inhibited. In addition to SNS activity, the hypothalamic-pituitary-adrenal (HPA) axis is activated. Cortisol is released into the bloodstream to promote the release of additional energy stores. Cortisol instigates the breakdown of fat and protein and triggers the liver to produce additional glucose. Although the energy burst supplied by the SNS lasts only minutes, cortisol can promote the release of additional energy for hours. Thus, at least during acute stress, cortisol reactivity to stress was likely selected for its survival value. However, if activated repeatedly, the HPA axis may exert detrimental effects on one's health, such as chronic immunosuppression or excessive levels of proinflammatory cytokines (Robles, Glaser, & Kiecolt-Glaser, 2005). Thus, chronic stress may produce long-term down-regulation of immune function, which could leave one susceptible to disease and depression (e.g., Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Robles et al., 2005).

During acute stress, immune function is altered in primarily adaptive ways. *Natural immunity* refers to elements of the immune system that are constantly present in the body and available to fight foreign substances. The natural immune system remains vigilant to the presence of nonself elements and is able to respond within minutes to a nonself element. Unlike the natural immune system, the cells of the *specific immune system* respond only to specific pathogens, termed antigens. The process of the specific immune response in which T lymphocytes are active is termed *cell-mediated immunity*. A full-blown specific immune response takes days to occur (vs. minutes for the natural immune system). In a wide-ranging meta-analysis of the literature, Segerstrom and Miller (2004) found that acute laboratory stressors were generally associated with enhanced natural immunity, whereas some measures of specific immunity were suppressed. Such a pattern of activity would have likely contributed to the survival of our ancestors. For example, when fighting or fleeing an enemy, a redistribution of the cells involved in natural immunity would decrease the risk of infection should harm occur, whereas suppression of specific immunity, which takes days to launch an assault, is energy efficient because those resources could be reallocated to more immediate needs (e.g., inflammation, providing energy to muscles).

Segerstrom's (2007) ecological model of immunity is consistent with such theorizing. Her model places immune activity in a broad context and suggests that immunity can be enhanced or suppressed depending on the immediate demands placed on the organism. Thus, when other activities are more important for the organism as a whole, immunity can be suppressed in order to allocate energy to more prepotent needs. By contrast, when infection is present, the immune system may take precedence over other systems by inducing sickness behavior. We expand upon this theorizing by specifying emotions and appraisals that attenuate or potentiate the effects of stressful social situations on cortisol and immunity.

Figure 1 presents our theoretical framework for understanding the role of emotions and appraisals in HPA and immune reactivity to emotional stressors. According to this perspective, increased cortisol output and immune system changes when under duress might have aided survival when confronted with certain types of stressors. We identify three nonorthogonal characteristics of emotional stressors that were likely to be associated with unique appraisal and emotion profiles (see Figure 1): stressors that are immediate or imminent, those that threaten one's social status, and those that require extended effort. According to this framework, in our evolutionary past, three unique characteristics of emotional stressors became associated with different patterns of emotions and appraisals. In turn, these emotions and appraisals then produced cortisol and immune responses in what would have been a primarily adaptive manner for our ancestors. Thus, depending on the emotion elicited, the physiological response can produce exacerbation of the cortisol response and/or either up-regulation or down-regulation of natural and specific immunity. The core idea is that appraisal, emotion, and physiology have become intricately linked through evolutionary forces. It is important to note that, in modern times, merely experiencing the associated appraisal or emotion may produce the selected physiological response even in the absence of the initial stressor characteristics (e.g., a stressor requiring extended effort).

Stressors Requiring Extended Effort

Our framework predicts that because one of the primary functions of cortisol is to promote the extended release of energy, appraisals and emotions associated with this need would be related to increased cortisol reactivity. At the same time, cortisol serves an immunosuppressive function. Thus, appraisals and emotions that are associated with increased cortisol output might also be associated with down-regulation of costly immune system activity such as cell-mediated immunity. Such a pattern would have been an efficient use of the body's limited energy.

Stressors That Threaten One's Social Status

Threats to social status signify real or potential social exclusion. Given the extremely high costs in our ancestral past associated with social rejection (e.g., banishment from the group, reduced access to resources) and social threat theory (Dickerson & Kemeny, 2004), we expected that appraisals and emotions associated with threats to one's social status would increase cortisol output. Such cortisol

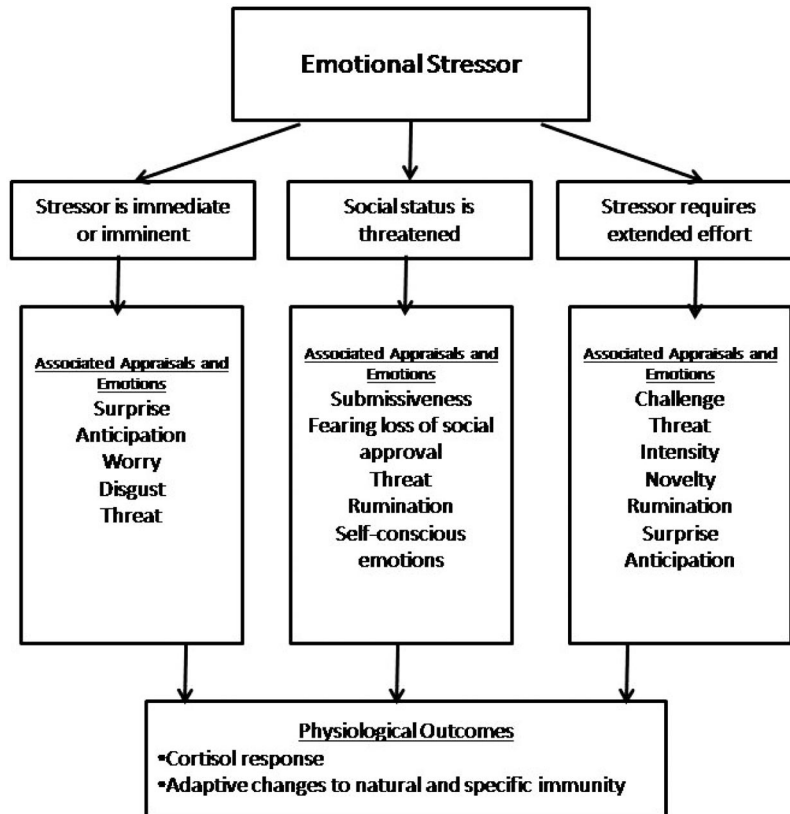


Figure 1. A theoretical framework for understanding adaptive changes in hypothalamic-pituitary-adrenal and immune reactivity to emotional stressors. According to this framework, different environmental demands (in this case, characteristics of the emotional stressor) produce different patterns of emotions and appraisals. In turn, these emotions and appraisals then influence the cortisol and immune response in a primarily adaptive manner. Thus, depending on the emotion elicited, the physiological response can include an exacerbation of the cortisol response and/or up-regulation or down-regulation of natural and specific immunity.

release would provide additional energy necessary to regain one's status in the group.

Stressors That Are Acute or Imminent

Consistent with an ecological view, imminent or acute stressors should initiate physiological reactivity. By contrast, specific appraisals and emotions that signaled an absence of immediate physical threat in the present environment might be associated with an attenuation of the stress response. For instance, whereas appraisals of threat maintain one's vigilance to a stressor, should a stressor reappear (e.g., a hungry predator), the natural immune system can respond within minutes and is therefore not needed when merely thinking about a stressor. However, rumination about a stressor might facilitate cortisol output as a means of energizing a resolution to the stressor.

Theoretical Categories of Appraisals and Emotions

To investigate the pathways whereby appraisals and emotions influence cortisol and immunity, we examined theoretically relevant appraisals and emotions clustered around five categories: (a) cognitive appraisals, (b) basic emotions, (c) rumination and worry,

(d) social threat, and (e) global mood states. We discuss each category and their expected influence on cortisol and immune outcomes in turn.

Cognitive Appraisals

Psychologists have long recognized that there is considerable variation in how people respond to equivalent stressors. It soon became apparent that individual differences in how people construe or appraise the stressor and its consequences were important contributors to the variability in psychological and physiological responses. Appraisal theory is now a core component of many models of stress and health (Dickerson & Kemeny, 2004; Lazarus & Folkman, 1984; Tomaka et al., 1993). These researchers have often focused on appraisals of uncontrollability, challenge, threat, and novelty—all of which have been shown to influence physiological responses to stress.

For instance, in their recent meta-analysis, Dickerson and Kemeny (2004) coded for three objective features of the experimental situations likely to influence cognitive and emotional responses. These were the presence or absence of social-evaluative threat, the controllability of the stressor, and whether the task was a moti-

vated performance task or a passive task. They found that motivated performance tasks with social-evaluative threat and uncontrollable manipulations were associated with the largest increases in cortisol reactivity, presumably by influencing cognitive appraisals and emotions.

In line with Dickerson and Kemeny's (2004) analysis, we expected that experimental procedures appraised as challenging, threatening, and intense would be associated with increased cortisol reactivity. Because novel stressors also generally require extended effort relative to familiar stressors, we also expected that this appraisal would be associated with increased cortisol. By contrast, we expected that these same appraisals would be associated with down-regulation of energy-intensive specific immune system activation.

Basic Emotions

The integrated specificity model has its historical roots in theories of basic emotions. These theories posit that there are a limited number of discrete emotions forged by evolution (e.g., Frijda, 1986; Izard, 1977; Panksepp, 1982; Plutchik & Conte, 1997; Tomkins, 1984). For instance, Plutchik's (1997) circumplex model specifies eight basic emotions (i.e., joy, sadness, surprise, anticipation, disgust, anger, fear, and acceptance) and notes that more complex emotions can be created from combinations of these eight basic emotions (e.g., joy and acceptance combine to form love). These models posit that the basic emotions have been selected for their survival value. Evidence in favor of this hypothesis is found in the universal recognition of facial expressions of basic emotions and the presence of basic emotions in many animal species (Darwin, 1872; Ekman & Friesen, 1971; Fridlund, Ekman, & Oster, 1987).

There is some evidence that individual basic emotions are also generally associated with unique patterns of physiological reactivity. Although there is some debate as to how successful the search for unique physiological indicators of the basic emotions has been (e.g., Ekman, 1992, 1994; Ortony & Turner, 1990), a number of investigations have reported unique patterns of physiological activity for several of the basic emotions (e.g., Ax, 1953; Ekman, Levenson, & Friesen, 1983; Schwartz, Weinberger, & Singer, 1981). Nonetheless, Ekman (1992) expressed doubts about whether all basic emotions would be associated with distinct physiological profiles and motor responses (p. 552).

We expected surprise, anticipation, and disgust to be associated with cortisol and immunity. It is likely that feelings of surprise in the presence of an unexpected, immediate threat would initiate a cortisol response to deal with the situation. Anticipation signals that stress is imminent but not yet occurring. Thus, anticipation may be associated with down-regulation of specific immunity that would allow energy conservation for one to deal with the threat (e.g., planning). Disgust was likely adaptive for avoiding threats to one's health (e.g., rotten food, sick people; see Park, Faulkner, & Schaller, 2003; Rozin, Haidt, & McCauley, 1993). However, once the emotion is elicited, the predominant behavioral tendency is to move away from the source of contamination. Thus, increased natural immunity would be superfluous. Oaten, Stevenson, and Case (2009) suggested that disgust functions to protect us from disease. Their theory predicts that disgust should elicit down-

regulation of immune activity. Indeed, one study found that lymphocyte proliferation in response to concanavalin A decreased following a film depicting combat surgery during the Vietnam War (Zakowski, McAllister, Deal, & Baum, 1992).

It is noteworthy that none of the other basic emotions tend to be overwhelmingly associated with specific types of emotional stressors. For instance, feelings of happiness and sadness are also not generally associated with immediate environmental threats and therefore do not require immediate responses on behalf of the organism.

Rumination and Worry

In line with the integrated specificity model, Brosschot, Gerin, and Thayer (2006) proposed that *perseverative cognition*, a common feature of rumination and worry, should be associated with increased endocrine (e.g., cortisol) and immune reactivity. Worry is generally characterized as trying to solve a problem in the future that could have potential negative consequences for the individual (Borkovec, Ray, & Stöber, 1998). Although there are many types of rumination, virtually all scientific and clinical definitions of rumination characterize the construct as being associated with uncontrollable, repetitive thoughts, usually about some distressing occurrence (Borders & Earleywine, 2006). Individual differences in such perseverative cognition have been linked to cortisol reactivity and immune alterations as well as increased endorsement of physical symptoms (for a review, see Brosschot et al., 2006; Thomsen, Mehlsen, Olesen, et al., 2004).

Repeatedly thinking about a stressor may lead to an extended stress response entailing dysregulation of the HPA axis and immune system. As with associative network theories of affect (e.g., Berkowitz, 1993), rumination might exacerbate the stress response by prolonging activation of the negative emotional response via continual reactivation of the associative network. In other words, each time a person thinks about or relives a stressful incident (or its accompanying affect), a new activation spreads through the network, which produces emotion-specific physiological responses. Rumination has been examined in the context of depression, anxiety, and anger, among others (Denson, Fabiansson, Creswell, & Pedersen, 2009; Denson, Pedersen, & Miller, 2006; Nolen-Hoeksema & Morrow, 1993; Rusting & Nolen-Hoeksema, 1998).

There is some empirical support for this importance of rumination in dysregulating cortisol and immune reactivity to stress. Brosschot et al. (2006) reviewed six published psychoneuroimmunology studies that contained a state or trait measure of worry or rumination. With the exception of one study with a failed manipulation, the studies reported that increased cortisol and some altered immune parameters were associated with worry or rumination. More recently, one study reported that participants who had experienced an interpersonal transgression in the past 7 days (e.g., romantic infidelity, insult, argument with a romantic partner) showed an increase in cortisol on days when they ruminated about the transgression more than usual over a 2-week period (McCullough, Orsulak, Brandon, & Akers, 2007). Moreover, the effect of rumination was mediated by fear of the transgressor. Another study found that experimentally induced brooding rumination following an insult

maintained high levels of cortisol for the duration of the experiment (Denson et al., 2009). Together, these results provide evidence that when social stress leads to rumination, cortisol and immune reactivity may be affected.

Thus, on the basis of recent theoretical and experimental work on rumination, we expected rumination to predict increased cortisol reactivity (Brosschot et al., 2006; McCullough et al., 2007). McCullough et al. (2007) speculated that rumination might enable individuals to “maintain a vigilant posture toward threats from the past that could become threats again” (p. 130). This view is consistent with experimental work demonstrating that cortisol is associated with increased attention to socially threatening stimuli (van Honk et al., 2000).

Social Threat

One instantiation of the integrated specificity model has focused on threats to the “social self” that occur in the presence of negative social evaluation, rejection, or potential loss of social status (Dickerson, Gruenewald, & Kemeny, 2004). Such threats are commonplace, given the ubiquitous nature of social hierarchies, the propensity toward which we have likely inherited from our evolutionary past. Indeed, Fiske’s (1991) model of social interaction asserts that authority ranking (i.e., hierarchical social structure) is one of the four universal means of social interaction, and even nonhuman primates evidence physiological stress responses depending on their place in the dominance hierarchy (Sapolsky, 2004). Thus, threats to the social self should be especially strong elicitors of emotional, physiological, and behavioral responses. Indeed, research on social rejection reveals that there is considerable neural overlap between physical and social pain (Eisenberger, Lieberman, & Williams, 2003). When rejected by others, individuals often respond with extreme, anger-driven behavior such as aggression (Leary, Twenge, & Quinlivan, 2006). However, when given alternative sources of social acceptance, individuals are not aggressive (Twenge et al., 2007), and social rejection often elicits behaviors designed to increase acceptance from others (Baumeister, 2005; Gonzaga, Keltner, Londahl, & Smith, 2001; Williams, Cheung, & Choi, 2000; Williams & Sommer, 1997).

Among humans, threats to the social self are sometimes accompanied by the presence of one or more of the self-conscious emotions. This group of emotions consists of shame, guilt, embarrassment, and pride (Tracy & Robins, 2004). Moreover, just as physiological responses to physical survival may be mediated by emotions such as fear, threats to social survival may be mediated by fear of losing social approval, self-conscious emotions, and feelings of submissiveness. When viewed from an evolutionary perspective, those individuals who reacted with guilt after having harmed a fellow ingroup member or shame after having violated social norms were likely to engage in behaviors that led to reacceptance into the group. Thus, emotions such as submissiveness, shame, and guilt, in conjunction with behaviors designed to regain group favor, may have enhanced the survival of our ancestors. Within the context of the integrated specificity model, the specific emotion elicited—regardless of its status as a basic or self-conscious emotion—is likely to influence the physiological responses as well as the motivational state of the individual. Indeed, shame

is associated with a motivation to withdraw socially and to exhibit submissive behavior (Tangney, 1990, 2002), and one study reported that feelings of shame were related to increased proinflammatory cytokine activity (Dickerson, Kemeny, Aziz, Kim, & Fahey, 2004).

In sum, we expected that threats to the social self would also be associated with cortisol increases. Submissiveness and sensitivity to threats to one’s status appear to be inherited from our primate ancestors (e.g., Sapolsky, 1998). Therefore, fearing the loss of social approval (e.g., Dickerson & Kemeny, 2004) and feelings of submissiveness were expected to be related to cortisol reactivity. Cortisol output might have served as a means of energizing behaviors designed to aid group reintegration. Indeed, increases in cortisol are associated with avoidant and submissive behavior in social contexts (Kagan, Reznick, & Snidman, 1988; Sapolsky, 1992; Shulkin, Gold, & McEwen, 1998). By contrast, because socially threatening situations are not generally associated with physical threats, it was expected that this cluster should be unassociated with immunity or associated with the down-regulation of some immune outcomes.

Global Mood States

We also examined the broad emotion dimensions of affective valence, psychological arousal, and subjective feelings of stress. Although a central assumption of stress and health models is that appraisals and emotions are hypothesized to mediate the effect of stressful situations on physiology, research has failed to confirm a relationship between broad composite classifications of emotions and cortisol and immune responses (Dickerson & Kemeny, 2004; Segerstrom & Miller, 2004). On the basis of this prior work, it was expected that these measures would generally be unassociated with physiological reactivity. Nonetheless, they were included to provide a comparison and to replicate prior meta-analytic findings demonstrating null associations among these global mood states, cortisol, and immune responses to stress. Table 1 summarizes our predictions for the five clusters.

Measurement of Cortisol and Immunity

To facilitate the interpretation of the results, we first provide some detail regarding the measurement of cortisol and immunity. Among studies included herein, cortisol was collected in saliva or blood samples. Once in the bloodstream, the majority of cortisol binds with carriers such as corticosteroid-binding globulin, albumin, and erythrocytes. The remaining cortisol is unbound. Measurement of this unbound cortisol is of interest to researchers because it is active biologically. Research demonstrates that salivary cortisol levels are highly correlated with blood cortisol levels (Kirschbaum & Hellhammer, 2000; Vining, McGinley, Maksvytis, & Ho, 1983). Furthermore, cortisol output is generally measured multiple times during an experiment. There is a delay between the initiation of stress and the appearance of cortisol in saliva or blood. Specifically, levels of cortisol in saliva and plasma taken at a given time point represent activity during the past 10–60 min. Furthermore, because there is diurnal variation in cortisol levels (i.e., a peak

Table 1
A Summary of Predictions Regarding the Effects of Appraisals and Emotions on Cortisol and Immune Reactivity to Social Stressors and Emotion Inductions

Variable cluster	Exacerbation of cortisol response?	Mechanism	Natural immunity	Mechanism	Specific immunity	Mechanism
Cognitive appraisals	Yes	Appraisals of challenge, novelty, and intensity signal immediate threats and the need for extended release of energy. Threat appraisals signal the need for vigilance and therefore the need for extended energy.	No relation	When exposed to a stressor, the natural immune response occurs very rapidly. This response is not costly in terms of energy expenditure. Thus, only appraisals and emotions that are associated with a lack of immediate environmental danger should be associated with down-regulation. None of the appraisals meet this criterion.	Attenuation of the stress response	Appraisals associated with immediate situational threats may be associated with an attenuation of the stress response. Such activity would conserve energy.
Basic emotions	Yes	Surprise and anticipation indicate immediate and imminent threats, respectively. Being able to mobilize energy reserves when surprised (e.g., predator or novel situation) or in anticipation of an imminent stressor likely aided in the survival of our ancestors.	Attenuation of the stress response	Happiness and sadness are likely to occur in the absence of immediate danger. Disgust motivates avoidance behavior, thus making natural immunity superfluous.	Attenuation of the stress response	Emotions associated with immediate situational threats may be associated with an attenuation of the stress response.
Rumination and worry	Yes	These variables signify the need to maintain a vigilant posture toward threats that might reappear (e.g., imminent threats) or unresolved problems, and therefore the need for extended energy.	Attenuation of the stress response	The presence of rumination and worry implies that a danger is not immediately occurring.	No relation	There does not appear to be any particular advantage of up-regulation or down-regulation of specific immunity associated with rumination and worry.
Threats to the social self and self-conscious emotions	Yes	Submissiveness and associated emotions (e.g., shame, fearing the loss of social approval) initiate "social survival" responses, which appear inherited from our primate ancestors.	No relation	These are generally not associated with physical danger.	No relation	There does not appear to be any particular advantage of up-regulation or down-regulation associated with social threats.
Global mood states	No effect	These broad states obscure unique information contained in specific appraisals and emotions.	No effect	These broad states obscure unique information contained in specific appraisals and emotions.	No effect	These broad states obscure unique information contained in specific appraisals and emotions.

after awakening), cortisol is best measured during the same time of day for all participants in a single study.

The two most common types of immune assays that were used in the studies and are included here are counts (or percentages) of immune cells and functional assays, also called cellular assays. Counts consist of counting the absolute numbers of immune cells or percentages of these cells (e.g., lymphocytes, natural killer [NK] cells, monocytes) in peripheral blood. Increases and decreases in numbers of immune cells are due to redistribution into the periphery from storage sites or from the periphery back into these storage sites, respectively. Functional assays, as the name implies, assess the functioning of immune cells. The effectiveness of NK cell function is assessed by an assay termed NK cell cytotoxicity (NKCC). This assay measures the ability of NK cells to lyse (i.e., kill) radio-labeled target cells. Higher values for all these functional measures reflect immune up-regulation. As with cortisol, immune responses also tend to be measured at multiple time points during an experiment.

The Current Meta-Analysis

For traditional meta-analytic reviews, the primary goal is to provide a quantitative summary of a research literature. Indeed, there have been two recent quantitative reviews on cortisol and immunity that summarize the effects of stress on these outcome measures across a wide range of stressors (Dickerson & Kemeny, 2004; Segerstrom & Miller, 2004). While acknowledging these works, we are particularly indebted to theoretical contributions that emphasize the unique contributions of specific emotions and cognitive appraisals (e.g., Brosschot et al., 2006; Dickerson, Gruenewald, & Kemeny, 2004; Dickerson & Kemeny, 2004; Kemeny, 2003; Lazarus, 1966; Lazarus & Folkman, 1984; Weiner, 1992). Thus, although we do report mean effect sizes for the sake of completeness, it is important to emphasize that our primary aim was to test theory through the use of high-inference coding procedures (e.g., Cooper & Hedges, 1994; N. Miller & Pollock, 1995). Consequently, we meta-analytically investigated the relationships between specific groups of emotions, cognitive appraisals, cortisol, and immunity. We used the high-inference coding procedures that have been advocated by N. Miller and Pollock (1995) for theory testing within the context of meta-analysis. These procedures, described by Cooper and Hedges (1994, p. 25) as “daring” and capable of producing rich inferences beyond the data that are reported in the source articles, rely on judges to assess the likely levels of key theoretical variables that were not intentionally manipulated within the articles composing the meta-analytic database. Specifically, by reading the method sections of the articles included in our meta-analysis, multiple coders made judgments concerning the extent to which each of a wide range of emotions and cognitive appraisals was likely to have been experienced by participants in the actual experimental settings. The convergent and construct validity of such high-inference judgments has now been well established (e.g., Bettencourt & Miller, 1996; Carlson, Charlin, & Miller, 1988; Carlson, Marcus-Newhall, & Miller, 1990; Carlson & Miller, 1987; Marcus-Newhall, Pedersen, Carlson, & Miller, 2000; N. Miller, Lee, & Carlson, 1991; Mullen, Salas, & Miller, 1991; Urban &

Miller, 1998). Other researchers have similarly provided evidence that supports the construct validity of judges' ratings of affect and cognition (e.g., Bowers & Chum, 1988; Eagly & Carli, 1981; Eagly & Crowley, 1986; Eagly & Steffen, 1986; Hull & Bond, 1986; Johnson & Eagly, 1989; Mullen et al., 1985; Steele & Southwick, 1985). Hence, the results based on these subjective coding procedures may inform future experimental work on the role of emotions and appraisals in the activation of cortisol and immune responses.

A secondary goal of the current meta-analysis was to facilitate generalization to daily events. Consequently, only types of stressors that people are likely to experience in their daily lives were included in the present investigation. These considerations meant that only social stressors and emotion inductions were chosen for analysis because they (a) provide a high degree of ecological validity,¹ (b) possess high levels of both experimental and mundane realism, and (c) elicit a broad range of emotional states. For example, crying during a sad movie (i.e., emotion induction), feeling nervous about giving a presentation at work or school (i.e., public speaking), and arguing with one's romantic partner (i.e., marital conflict) are all frequently experienced sources of stress in daily life, whereas performing either mental arithmetic in front of an audience or the Stroop task undoubtedly occurs most often in the laboratory but rarely in daily life. In sum, because our aim differed from the traditional meta-analytic goal of quantitatively summarizing a literature, we selected on an a priori basis a reasonable number of experimental manipulations ($k = 80$) that (a) provided a broad enough range of elicited emotions to detect meaningful relationships between emotions, appraisals, and physiological reactivity and (b) best allowed us to generalize to people's daily lives. The success of this strategy is reflected in our results.

It is important to note that this selection of studies in no way lessens the contribution made by research on mental stress. Indeed, cognitive tasks are very effective in inducing stress. Moreover, there may be little difference between the two in terms of physiological outcomes. Although Dickerson and Kemeny (2004) did find an additive effect of mental and social stress on cortisol reactivity, Segerstrom and Miller (2004) found that across a wide range of immune outcomes no differences existed between laboratory mental and social stressors: “Acute stressors elicited similar patterns of immune change . . . irrespective of whether they involved social (e.g., public speaking), cognitive (e.g., mental arithmetic), or experiential (e.g., parachute jumping) forms of stressful experience” (p. 612). Nonetheless, as our interests lay in theory testing and generalizability, we included only commonly encountered social stressors and emotion inductions. At the same time, however, although we recognize that we did reduce power by constraining our study selection criteria, we also note that we did have adequate power to detect meaningful differences (e.g., even

¹ Some have questioned the external validity of laboratory experimentation in psychology. However, meta-analytic work demonstrates that effect sizes in the laboratory and in the field are quite similar in domains as diverse as aggression, helping, leadership style, social loafing, self-efficacy, depression, and memory (Anderson, Lindsay, & Bushman, 1999).

small but reliable effect sizes such as $d = 0.16$; see the Results section).²

Method

Literature Search

MEDLINE and PsycINFO searches were conducted with the keywords *immun**, *stress*, *cortisol*, *affect**, *mood*, and *emot**. All abstracts were examined for inclusion according to the criteria presented below. Recent meta-analyses and review articles were reviewed for relevant references (e.g., Dickerson & Kemeny, 2004; Segerstrom & Miller, 2004). The individual-study articles were also examined for additional articles. Largely because, as described above, our goal differed from that of traditional meta-analyses, we did not attempt to obtain unpublished manuscripts to reduce file drawer effects.

Inclusion Criteria

An article was included in the current meta-analysis if it (a) was published in a peer-reviewed journal; (b) was a laboratory experiment; (c) was indexed in PsycINFO or MEDLINE by August 2005; (d) was conducted with healthy adult, human participants; (e) contained either a cortisol or immunological dependent variable; (f) contained sufficient statistical information for calculating effect sizes (or this information was obtainable from the authors);³ and (g) included an experimental manipulation of social stress and/or a direct emotion induction manipulation. Among studies that examined populations with mental or physical problems, only the data from the healthy control groups were included. Sixty-six articles met these inclusion criteria.

Variable Coding

We coded two types of variables: study characteristics and subjective variables. Study characteristics were coded from information directly available in the published articles or from e-mail correspondence with the authors. Levels of subjective variables were based on inferential judgments on the part of the judges. Such judgments allow one to “capture the psychological experience of subjects in the primary studies” (Cooper & Hedges, 1994, pp. 25–26). They allow the meta-analyst to make inferences beyond those reported explicitly in the individual study reports. As noted, these procedures provide a meta-analytic method of testing theory (N. Miller & Pollock, 1995; Mullen et al., 1991) and have been used effectively in a number of previous meta-analyses.

Over the course of a semester, nine judges (four women and five men, including Thomas F. Denson) read the method sections of the studies included in the meta-analysis. Two judges were graduate students in social psychology, and the remaining seven judges were undergraduate psychology majors. All judges, with the exception of Denson, were blind to the goals and hypotheses of the current meta-analysis. Denson provided the coding sheets, method section, and suggested time line for completion to each judge. Each judge rated all the studies on all the variables. To control for inflation of reliability due to shared variable or study order, each judge received a completely randomized order of variables and studies. Denson also met with each judge once or twice during the semester to discuss coding progress and answer questions. To

maintain independence of observations, we allowed no discussions or examples on how to judge specific studies.

The role of each judge was to evaluate one variable at a time for all the studies. For example, if working on the fear variable (i.e., “How afraid would participants feel in the experiment?”; 1 = *not at all*, 5 = *very much*), the judge would read the method section of the first study in his or her packet, imagine how the average participant would feel in this situation, and then make a rating on the ordinal scale. The judge would then read the method section of the second study, make the fear rating, and so on, until all the studies were coded for fear. Interrater reliability was assessed with

² To address potential differences in the appraisals and emotions elicited respectively by cognitive (e.g., Stroop task) and combined cognitive and social stressors (e.g., Stroop task plus public speaking), two coders rated all the subjective variables for a random sample of 20 cognitive and combined cognitive and social stressors from Dickerson and Kemeny’s (2004) meta-analysis. We then compared the mean differences on these subjective ratings between this first group of cognitive–combined stressors and another group of 20 randomly selected public speaking tasks included in the current work. Cognitive–combined stressors were judged as failing to elicit any sadness, happiness, or disgust. However, cognitive–combined stressors were rated as more challenging than public speaking alone ($M = 3.60$, $SD = 0.85$ vs. $M = 3.17$, $SD = 0.31$), $t(38) = 2.11$, $p = .04$. Consistent with the notion that cognitive–combined stressors are most often encountered in the laboratory, these stressors were rated as more novel than public speaking ($M = 4.33$, $SD = 0.47$ vs. $M = 3.35$, $SD = 0.31$), $t(38) = 7.77$, $p < .001$. Cognitive–combined tasks were rated as less likely to produce feelings of guilt than public speaking stressors ($M = 1.20$, $SD = 0.70$ vs. $M = 1.68$, $SD = 0.19$), $t(38) = -2.98$, $p = .01$, as well as less fear ($M = 1.68$, $SD = 0.67$ vs. $M = 2.25$, $SD = 0.34$), $t(38) = -3.72$, $p = .001$; less anticipation ($M = 2.00$, $SD = 1.00$ vs. $M = 2.99$, $SD = 0.44$), $t(38) = -4.05$, $p < .001$; less uncontrollable, repetitive thoughts ($M = 1.78$, $SD = 0.66$ vs. $M = 2.15$, $SD = 0.42$), $t(38) = -2.15$, $p = .04$; and less brooding ($M = 1.55$, $SD = 0.69$ vs. $M = 2.00$, $SD = 0.45$), $t(38) = -2.46$, $p = .02$, perhaps due to the cognitively demanding (and thereby distracting) nature of these tasks. There were no differences on any of the other subjective ratings. Thus, it does not appear that failing to include cognitive and combined stressors in the current meta-analysis is a serious threat to the validity of our findings. Furthermore, the ratings of greater challenge and novelty for the cognitive–combined tasks is consistent with our results demonstrating that increases in challenge and novelty were associated with changes in effect sizes for several of the outcomes of interest, despite being constrained to social stressors and emotion inductions. Another reason for excluding combined mental and social stressors was due to the redundancy inherent in including numerous similar manipulations. Given that public speaking is already the most common manipulation of social stress, including the studies with combined cognitive and social tasks would further exacerbate the preponderance of studies using this type of emotional induction. In other words, including an overwhelming number of similar studies would unfairly bias the results toward those emotions elicited by those stressors. Thus, even if the results for these different types of stressors were standardized before combining them, the over-weighting problem would still exist when examining emotions and appraisals.

³ Only five studies were omitted from analysis because of insufficient data for effect size calculations. These included one study in which only postmanipulation values were reported to examine between-groups differences (i.e., a group contrast). Because standardized mean change values may differ greatly from standardized group contrasts, Lipsey and Wilson (2001) suggested not including both effect sizes in the same meta-analysis. An additional two articles were eliminated from analysis because the data had been reported elsewhere.

Cronbach's alpha. For one of the variables, we eliminated data from one judge to increase interrater reliability. This is noted below.

As is apparent in the present meta-analysis, we made extensive use of variables created through human judgment. Specifically, we instructed judges to place themselves in the participants' role and imagine the consequences of experimental procedures. Despite the arguments and evidence presented above, researchers have sometimes had good reason to be skeptical about the validity of such role-playing procedures. As often as not, participants who served in role-playing replications of research were unable to predict the behavior of participants in the original study (A. G. Miller, 1972). In a meta-analytic assessment of this issue, we too were unable to find evidence that judges can reliably predict the behavior of study participants (N. Miller et al., 1991). Such failure with respect to judges' prediction of behavior within meta-analytic contexts, however, is not invariable (Aviles & Miller, 2006). By contrast, with this mixed outcome with respect to the prediction of behavior, and more important for our purposes here, judges can reliably predict the affective states and the cognitions induced in research participants by experimental manipulations (N. Miller & Pollock, 1995). Meta-analytically confirming the convergent validity of judges' ratings of study participants' affect, their ratings were positively and reliably correlated with the magnitudes of the manipulation check effect sizes that reflected the strength of each of two types of experimental inductions of affect (N. Miller et al., 1991). Similarly, such convergent validity also was reliably obtained for their judgments of each of two experimentally manipulated cognitions (N. Miller et al., 1991). In other work, we obtained positive convergent validity correlations between judges' ratings and manipulation check effect sizes of positive affect (Carlson et al., 1988), frustration and negative affect (Carlson & Miller, 1988), and arousal (Aviles & Miller, 2006). In addition to such evidence of convergent validity, the use of judges' ratings within meta-analytic contexts evidences substantial construct validity. In over 20 instances, judges' ratings of study participants' emotional or cognitive states, based on their reading of method sections, have evidenced theoretically predicted construct validity. These instances have included such diverse affective and cognitive states as happiness, anxiety, frustration, irritation, sadness, inhibitory response conflict, self-focus, responsibility, guilt, global negative affect, objective self-awareness, interpersonal similarity, anger, cognitive overload, personalization, and importance (Bettencourt & Miller, 1996; Carlson et al., 1988; Carlson & Miller, 1987, 1988; Ito, Miller, & Pollock, 1996; Miller & Carlson, 1990; Urban & Miller, 1998). Moreover, in some of this research there is evidence of discriminative construct validity for judges' ratings of closely related emotional states such as the negative mood states of sadness and guilt, self-focus and objective self-awareness, and anxiety and objective self-awareness. As previously noted, other researchers have similarly provided evidence that supports the construct validity of judges' ratings of affect and cognition (e.g., Bowers & Chum, 1988; Eagly & Carli, 1981; Eagly & Crowley, 1986; Eagly & Steffen, 1986; Hull & Bond, 1986; Johnson & Eagly, 1989; Mullen et al., 1985; Steele & Southwick, 1985). As a final point, it is worth noting that although some social psychological research demonstrates that predictive introspection (e.g., "How would I feel?") can be inaccurate, people are often much better at predicting others' reactions (e.g., "How would other

people feel?"), which is what our judges were asked to do. This holds true even for deeply intimate knowledge such as how long a romantic relationship will last (MacDonald & Ross, 1999). On the basis of both this evidence attesting to the convergent and construct validity of judges' ratings of the emotional experiences and cognitions of study participants, and the fact that many of the specific constructs for which we have cited evidence of valid judgments are ones identical to those used in the current meta-analysis, we had strong reason to anticipate that our judges would provide valid ratings of the relevant interpersonal, affective, and cognitive states of the participants in each of the studies that we sampled. The fact that many of those ratings did indeed give evidence of construct validity confirms this expectation.

Study Characteristics

Two judges recorded the following study characteristics, with their interrater reliabilities assessed via Pearson's correlation for continuous variables and kappa for categorical variables: (a) sample size ($r = .99$), (b) mean age ($r = 1.00$), (c) proportion of White participants ($r = .98$), and (d) proportion of male participants ($r = .96$). If gender was unstated, 50% male was assumed. If age was unstated but the participants were described as undergraduates, a mean age of 20 years was assumed. In two cases, age was replaced with the mean age of all the studies.

Two judges also coded the following methodological study characteristics: (a) control for time of day of the cortisol or immune assessment (yes/no dummy variable; $\kappa = .98$), (b) duration of the stressor or emotion manipulation in minutes ($r = .90$), and (c) type of independent variable manipulation (e.g., social stressor vs. emotion induction; $\kappa = .97$). Denson coded method of biomarker sample collection (i.e., saliva or blood) and type of dependent measures (e.g., cortisol, T lymphocytes).

Subjective Variables

Cognitive appraisals. In the tradition of cognitive appraisal theories of stress and emotion (e.g., Lazarus & Folkman, 1984; Tomaka et al., 1993), the judges were asked to rate six variables that participants could conceivably make in response to the stressor or mood induction. Specifically, they rated the extent to which the experimental manipulation was *challenging* (1 = not at all, 5 = a lot; $\alpha = .78$; values ranged from 1.00 to 4.50; $M = 2.73$, $SD = 0.84$), *threatening* (1 = not at all, 5 = a lot; $\alpha = .73$; range = 1.00–4.83; $M = 2.33$, $SD = 0.68$), *positively or negatively valenced* (1 = very negative, 5 = very positive; $\alpha = .89$; range = 1.17–4.50; $M = 2.35$, $SD = 0.69$), *intense* (1 = not at all, 5 = a lot; $\alpha = .73$; range = 1.00–5.00; $M = 3.05$, $SD = 0.76$), and *familiar versus novel* (1 = very familiar, 5 = very novel; $\alpha = .64$; range = 1.50–4.67; $M = 2.89$, $SD = 0.67$). They also rated the extent to which participants would have sufficient *psychological resources* to deal with the experimental manipulation (1 = not at all, 5 = a lot; $\alpha = .70$; range = 1.67–5.00; $M = 3.27$, $SD = 0.53$).

Basic emotions. Eight basic emotions were derived from Plutchik's evolutionary circumplex model of emotions (Plutchik & Conte, 1997). These were *afraid* (1 = not at all, 5 = very much; $\alpha = .76$; range = 1.00–4.83; $M = 1.97$, $SD = 0.64$), *happy* ($\alpha = .87$; range = 1.00–4.17; $M = 1.89$, $SD = 0.70$), *sad* ($\alpha = .89$; range = 1.00–4.67; $M = 1.93$, $SD = 0.84$),

disgusted ($\alpha = .80$; range = 1.00–4.00; $M = 1.65$, $SD = 0.49$), *surprised* ($\alpha = .70$; range = 1.00–4.83; $M = 2.46$, $SD = 0.76$), *angry* ($\alpha = .79$; range = 1.00–4.00; $M = 2.12$, $SD = 0.71$), *accepting* ($\alpha = .76$; range = 1.20–4.83; $M = 3.02$, $SD = 0.62$), and *anticipation* ($\alpha = .65$; range = 1.00–4.50; $M = 2.55$, $SD = 0.62$).

Rumination and worry. Four variables assessed the tendency for participants to experience different types of intrusive, perseverative cognition during the stressor or mood induction. They assessed the extent to which participants would *worry* (1 = *not at all*, 5 = *very often*; $\alpha = .81$; range = 1.00–4.83; $M = 2.45$, $SD = 0.77$), *focus on angry thoughts and feelings* ($\alpha = .81$; range = 1.00–4.17; $M = 2.04$, $SD = 0.79$), *engage in uncontrollable, repetitive thinking* ($\alpha = .69$; range = 1.00–4.33; $M = 2.29$, $SD = 0.62$), and *brood* ($\alpha = .72$; range = 1.00–4.67; $M = 2.29$, $SD = 0.84$) during the experiment.

Threats to the social self and the self-conscious emotions. Because social status has been linked to cortisol and immune outcomes in humans and nonhuman primates (Sapolsky, 2004), and social-evaluative threat predicted cortisol effect sizes (Dickerson & Kemeny, 2004), we included five variables to assess aspects of social standing. These were ratings of the extent participants would feel *submissive* (eight coders; 1 = *very dominant*, 5 = *very submissive*; $\alpha = .58$; range = 1.20–5.00; $M = 2.52$, $SD = 0.64$) and the extent to which participants would *fear losing social approval* (1 = *not at all*, 5 = *very much*; $\alpha = .80$; range = 1.00–4.17; $M = 2.17$, $SD = 0.76$). The remaining three items consisted of the self-conscious emotions (Tangney, 2002). These were *ashamed* (1 = *not at all*, 5 = *very much*; $\alpha = .79$; range = 1.00–4.67; $M = 2.09$, $SD = 0.67$), *guilty* ($\alpha = .85$; range = 1.00–4.83; $M = 1.80$, $SD = 0.66$), and *embarrassed* ($\alpha = .76$; range = 1.00–4.20; $M = 2.29$, $SD = 0.69$).⁴

Global mood states. Three additional descriptors assessed higher order mood judgments that are relevant to stressful experimental settings or mood inductions. These were ratings of feeling *stressed* (1 = *very bad*, 5 = *very good*; $\alpha = .85$; range = 1.00–4.83; $M = 3.07$, $SD = 0.84$) and *aroused* ($\alpha = .71$; range = 1.00–5.00; $M = 2.96$, $SD = 0.71$), as well as one additional item that assessed participants' *overall mood* ($\alpha = .90$; range = 1.00–4.50; $M = 2.21$, $SD = 0.77$).

Composite variables. Dimensional models of emotion frequently define emotions along continua such as valence (positive vs. negative), emotional arousal (calm vs. excited), and motivational action tendencies (approach vs. withdrawal). We therefore reverse-scored relevant variables (noted by *R* below) and created averaged composite variables for each of these dimensions. The *negative affective valence* composite consisted of shame, guilt, embarrassment, happiness (*R*), fear, worry, sadness, disgust, anger, stressed, focus on angry thoughts, uncontrollable repetitive thoughts, brooding, overall mood (*R*), and fear of losing social approval ($\alpha = .90$). Higher scores represent more negative affect. The *emotional arousal* composite consisted of shame, guilt, embarrassment, happiness, fear, worry, surprise, sadness (*R*), disgust, anticipation anger, submissive (*R*), aroused, stressed, focus on angry thoughts, uncontrollable repetitive thoughts, and fearing losing social approval ($\alpha = .79$). Higher scores represent greater excitement. The *approach-withdrawal* composite consisted of shame, guilt (*R*), embarrassment, happy (*R*), fear, sadness, disgust,

and anger (*R*) but did not form a reliable composite ($\alpha = -.18$). Higher values represent more withdrawal motivation.

Meta-Analytic Procedures

Effect Size Calculation

The effect size reported in this meta-analysis is the standardized mean change statistic, d (Becker, 1988; Lipsey & Wilson, 2001). The formula for this statistic is

$$d = \frac{\bar{X}_{T_2} - \bar{X}_{T_1}}{s_p}, \quad (1)$$

wherein the mean of Time 1 (baseline) is subtracted from the mean of Time 2 (postonset of the social stressor or mood induction). This difference score is then divided by the pooled standard deviation (s_p):

$$s_p = \sqrt{(S_{T_1}^2 + S_{T_2}^2)/2}. \quad (2)$$

The pooled standard deviation is simply the square root of the Time 1 and Time 2 variances divided by two. Thus, the statistic represents the difference in standard deviation units between the baseline assessment and the postmanipulation assessment. Convention suggests that d values of 0.2 are small, values of 0.5 are medium, and values of 0.8 are large (Cohen, 1988). Multiple effect sizes were usually calculated from a single study because it is common to take more than one postbaseline immune or cortisol sample. We recorded effect sizes up to 90 min following the onset of the experimental manipulation.

The large majority of effect sizes were calculated from means and standard deviations. If this information was not available in the articles, it was requested from the authors. The second choice was calculations based on F tests or dependent t tests. If these data were not available, p values were used as a last resort. If the phrase *no significant differences* was observed without statistical information, an effect size of zero was coded for that variable. In this manner, when means and standard deviations were unavailable, the effect sizes reported here represent conservative estimates. Once the raw effect sizes were calculated, they were corrected for small sample size bias with the following formula (Hedges & Olkin, 1985):

$$d_{\text{corrected}} = \left[1 - \frac{3}{4N-9} \right] d. \quad (3)$$

Only the corrected effect sizes are reported.

Inferential Data Analysis

We used a multilevel mixed-effects linear model as the inferential meta-analytic statistical method. Analyses were conducted with the HLM software package (Version 5.0; Raudenbush, Bryk, Cheong, & Congdon, 2000). This method is extensively detailed elsewhere (Kalaian & Raudenbush, 1996; Overton, 1998) and offers several advantages over traditional fixed-effects meta-analytic procedures. Dickerson and Kemeny (2004) used the same

⁴ Pride was also assessed but not analyzed because of relatively low reliability, and more important, restricted range ($\alpha = .51$; range = 2.00).

modeling method for their meta-analysis on stress and cortisol. Traditionally, in meta-analyses with multiple effect sizes, authors compute a mean effect size for each study. However, the multi-level mixed-effects model allows for correlations among multiple dependent measures from individual studies. Thus, none of the effect sizes were omitted from the analyses. The result is a maximization of the information available from each study and an increase in statistical power.

We first discuss the most basic form of this mixed effects model and then discuss models with moderator variables.⁵ For any given dependent variable type, the within-study effects constitute the Level 1 model and can be described as follows:

$$d_{ij} = \beta_{0j} + e_{ij}, \quad (4)$$

where d_{ij} is the calculated effect size i for study j , β_{0j} is the mean effect size of study j (i.e., the model intercept), and e_{ij} represents the error term for effect size i for study j . In this Level 1 equation, the errors are deviations from the mean effect size within each study. In other words, for each study, each effect size is modeled as consisting of two components: the mean effect size and the deviation from the mean effect size for that study. Thus, the Level 1 model is concerned with the variability of effect sizes within individual studies.

The Level 2 model represents the between-studies model:

$$\beta_{0j} = \gamma_{00} + U_{0j}, \quad (5)$$

where β_{0j} is the mean effect size of study j , γ_{00} is the mean effect size of all the studies, and U_{0j} is the error term—the deviation of study j 's mean from the grand mean. In other words, each study's mean effect size is modeled as a deviation from the mean effect size for all the studies. Thus, the Level 2 model is concerned with the variability of effect sizes between (or across) studies. The error term (U_{0j}) can be tested for significant variability. If the variance component is significant, this means that additional variability exists and may be explained by factors outside the model. One should then attempt to identify moderator variables that might explain this variability in between-studies effect sizes. Note that this is conceptually similar to the Q test for homogeneity of variances in fixed-effects meta-analytic work.

Once one has identified a significant residual at Level 2 (i.e., a significant test of U_{0j}), the model may include potential moderator variables. In the current meta-analysis, we specified the Level 1 model as follows:

$$d_{ij} = \beta_{0j} + \beta_{1j}(\text{Time of Sample}) + \beta_{2j}(\text{Time of Sample})^2 + \beta_{3j}(\text{Time of Sample})^3 + e_{ij}. \quad (6)$$

This Level 1 model controls for the time course of the cortisol or immune response for each study by introducing linear, quadratic, and cubic functions in sequential blocks. This model allows for multiple effect sizes within a single experiment representing the difference between the baseline assessment and assessments collected at various time points during the experiment. This is an advantage over traditional fixed-effects models in which only one effect size per study is permitted. Thus, the current mixed-effects model allows one to maximize the amount of information available for each study. It is noteworthy that some of the mean effect sizes are not significantly different from zero. This is to be expected

because of the rise and fall of the responses across the 90 min within experiments.

Of more interest is the test of the between-studies (Level 2) variance component and the tests of the coefficients for the time-of-assessment variables. At Level 2, we first introduced relevant methodological features to control for variables at the between-studies level that might conceivably affect the outcomes. We refer to this model as the control model:

$$\begin{aligned} \beta_{0j} = & \gamma_{00} + \gamma_{01}(\text{duration of manipulation}) \\ & + \gamma_{02}(\text{saliva vs. blood assessment method dummy}) \\ & + \gamma_{03}(\text{mean age of participants}) \\ & + \gamma_{04}(\text{percent male participants}) \\ & + \gamma_{05}(\text{experimental time of day control dummy}) + U_{0j}. \end{aligned} \quad (7)$$

If the variance component remained significant after controlling for these methodological variables, this suggested that additional variance remained that could be explained by additional moderators. Accordingly, we added the subjective variables individually to the model while those control variables that significantly or marginally ($p < .10$) predicted effect sizes remained in the equation.

The Level 2 between-studies model included potential moderators that might explain the significant residual variance observed in the basic model (see Equation 5). For example, a potential model attempting to explain the mean effect sizes for T cytotoxic cells across all the studies could be described as follows:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{duration of manipulation}) + \gamma_{02}(\text{surprise}) + U_{0j}, \quad (8)$$

where γ_{01} is the regression coefficient for the duration of the experimental manipulation and γ_{02} is the regression coefficient for the subjective variable "degree of surprise experienced by participants." A significant slope for γ_{02} would indicate that across all studies, the judged degree of surprise experienced by participants explained a significant amount of variability in T cytotoxic cell effect sizes. A positive value would mean that with increasing levels of judged surprise, T cytotoxic cell counts increase as well. A negative value would indicate that increasing levels of judged surprise produce decreases in T cytotoxic cell counts.

In the current article we reported significance tests for the residual Level 2 variance (U_{0j}) of the basic model. If the Level 2 variance component was significant, we examined the control variables for significance. If the Level 2 variance component remained significant at this stage, we conducted tests of the cognitive appraisal and emotion moderator variables. Unstandardized coefficients are reported along with their associated t statistics, standard errors, and two-tailed p values.

⁵ Although models of stress and health suggest a mediating role for emotions and appraisals, the meta-analytic methods for analyzing heterogeneity in effects sizes are called moderator analyses.

Results

Descriptive Statistics

Participant and Study Characteristics

Three thousand two hundred fifty-seven individuals participated in the studies ($M = 41$ per study, $SD = 32$). Participants were 54% male ($SD = 36\%$), and they ranged in age from 18.9 to 72.3 years ($M = 30.83$, $SD = 11.10$). The data for this meta-analysis consisted of 599 effect sizes culled from 80 experimental manipulations published in 66 peer-reviewed journal articles. On average each article contributed 9.08 effect sizes for analysis. We categorized 65% ($k = 52$) of the studies as social stress manipulations and the remaining 35% ($k = 28$) as emotion inductions. By far the largest number of social stress manipulations consisted of public speaking tasks ($k = 34$), but a variety of other manipulations were also represented. Examples include marital conflict discussions ($k = 6$), stressful interviews ($k = 5$), teaching a task to a bumbling confederate ($k = 2$), hostile role playing with a confederate ($k = 1$), and having participants think they severely injured someone ($k = 1$). The types of emotion inductions included films ($k = 7$) with the following content: humor ($k = 2$), sadness ($k = 1$), violence ($k = 2$), gruesome surgical procedures ($k = 1$), or horror or suspense ($k = 2$). Emotions were also induced by having participants reexperience, act out, imagine, or write about various mood states ($k = 5$). Other manipulations included positive and negatively valenced music ($k = 2$), imagining scenes from romantic relationships ($k = 1$), and imagining the death of a loved one ($k = 1$).

Among studies that assessed cortisol, 53% derived assessments from saliva, whereas 47% used plasma. Immune markers were always assessed through plasma samples. The average cortisol assessment was obtained 16.02 min ($SD = 10.05$) after the onset of the experimental manipulation, whereas the average immune assessment was taken 19.85 min ($SD = 20.67$) after manipulation onset. The average duration of the stressor or mood induction was 31.26 min ($SD = 37.41$; $Mdn = 15$; range = 3–135).

Validity of the Subjectively Rated Variables

Table 2 displays the correlations among the subjectively judged variables. These judgments were generally correlated with one another in the expected direction and magnitude. Table 3 displays the ratings for all the variables as a function of type of experimental manipulation. These ratings also demonstrated the expected pattern of results in that they discriminated between manipulation types on key variables. For example, studies that used interpersonal conflict manipulations were judged as eliciting the highest levels of anger, whereas those that manipulated positive mood were judged as eliciting the highest levels of happiness, and violent and horror films elicited the highest levels of disgust. Table 3 also provides evidence that different stress manipulations tend to be associated with different profiles of appraisals and emotions. Overall, the judgments demonstrated evidence of good convergent and discriminant validity.

Dependent Measures

Studies often assessed numerous dependent measures. The majority of the studies assessed cortisol ($k = 54$). Dependent mea-

asures specific to the innate immune system included total leukocytes, monocytes, and three types of polymorphonuclear cells (i.e., granulocytes, neutrophils, and eosinophils), all of which were analyzed together ($k = 14$). NK cells (surface markers CD16, CD56, and CD57; $k = 28$) were assessed and analyzed separately. A number of studies also assessed NKCC ($k = 17$). Specific immunity was assessed through lymphocyte subset counts or percentages of total lymphocytes ($k = 13$), T lymphocytes (surface marker CD3; $k = 22$), T helper lymphocytes (surface marker CD4; $k = 27$), T cytotoxic lymphocytes (surface marker CD8; $k = 26$), and B lymphocytes (surface markers CD19 and CD20; $k = 13$). Because Segerstrom and Miller (2004) found parallel results for cell percentages and cell counts for acute laboratory stressors, we analyzed effect sizes for these variables together. Table 4 displays each study, experimental manipulation, type of dependent measures, number of effect sizes, and the lower and upper bounds for the effect sizes for each dependent measure.

Inferential Data Analyses

Cortisol

The overall mean effect size averaged across the 90 min following the onset of the independent variable manipulation for the 54 manipulations in which cortisol was assessed (204 effect sizes) was $d = 0.11$, 95% CI $[-.03, .24]$. As expected, due to the time course of the cortisol response, this value did not differ from zero, $t(53) = 1.56$, $p = .13$. The maximum peak for the cortisol response occurred 46–60 min following the onset of the social stressor or emotion induction (see Figure 2). More important, because the test of the Level 2 variance component for cortisol was significant for the basic model, we proceeded to analyze the control model, $\tau_{00} = .21$, $\chi^2(53) = 432.85$, $p < .001$. Among the variables included in the control model, at the within-study level (Level 1), the cubic function for the time of assessment was significant, $\gamma = -.000004$, $t(200) = -1.99$, $p = .05$. At the between-studies level (Level 2), participants' mean age was negatively related to the cortisol effect sizes, suggesting that older participants evidenced decreases in cortisol reactivity, $\gamma = -.01$, $t(48) = -2.50$, $p = .02$. As is apparent from Figure 2, there was substantial variability in the effect sizes across the time course, suggesting a role for moderating variables. Indeed, after adding the control variables, the significance test of the variance component remained significant, $\tau_{00} = .19$, $\chi^2(48) = 376.10$, $p < .001$, suggesting that additional moderators might further explain the variability between studies. Thus, the coded variables were entered into the model while controlling for time course and age.

Among the cognitive appraisals, judges' ratings of the situation as challenging, threatening, and novel predicted effect sizes (see Table 5). Ratings of the overall intensity of the experience also predicted increased effect sizes. Among the basic emotions, judgments of surprise predicted effect sizes. As expected, variables related to threats to the social self were associated with increased cortisol reactivity as well. Specifically, manipulations judged to be likely to produce feelings of submissiveness and the fear of losing social approval were associated with increased effect sizes. Among the rumination items, experiments judged as likely to produce uncontrollable, repetitive thoughts were associated with increased cortisol reactivity. There was also a trend for brooding rumination

Table 2
Correlations Among the Subjectively Coded Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	
1. Resources																										
2. Challenging	-.48																									
3. Threatening	-.60	.68																								
4. Novelty	-.40	.50	.33																							
5. Valence	.62	.64	-.66	-.29																						
6. Intensity	-.39	.83	.72	.35	-.64																					
7. Happiness	.57	-.52	.63	-.21	.88	-.51																				
8. Sadness	-.24	.15	.29	-.18	-.51	.20	-.49																			
9. Surprise	-.41	.74	.58	.39	-.54	.73	-.49	.06																		
10. Anticipation	-.51	.56	.55	.56	-.46	.57	-.41	-.09	.65																	
11. Disgust	-.41	.21	.41	.05	-.43	.25	-.38	.41	.28	.24																
12. Anger	-.49	.50	.54	.13	-.54	.37	-.57	.40	.43	.31	.34															
13. Fear	-.49	.47	.71	.23	-.55	.59	-.55	.15	.44	.49	.43	.31														
14. Acceptance	.67	-.63	-.71	-.51	.76	.66	.68	-.29	-.57	-.62	-.38	-.55	-.54													
15. Worry	-.51	.75	.73	.34	-.73	.82	-.62	.37	.69	.54	.33	.51	.59	-.70												
16. Focus on angry thoughts	-.33	.56	.49	-.05	-.56	.50	-.51	.51	.44	.18	.25	.77	.17	-.45	.58											
17. Uncontrollable repetitive thoughts	-.20	.51	.39	.11	-.45	.60	-.33	.60	.50	.19	.30	.38	.26	-.41	.66	.56										
18. Brooding	-.20	.47	.39	.01	-.55	.54	-.47	.69	.47	.16	.29	.52	.18	-.42	.65	.76	.80									
19. Submissive	-.22	.64	.51	-.18	.44	.72	-.41	.06	.60	.38	.11	.05	.51	-.34	.65	.21	.47	.29								
20. Losing social approval	-.31	.73	.55	.41	-.50	.69	-.45	.08	.68	.40	.12	.48	.40	-.57	.71	.43	.51	.45	.62							
21. Shame	-.47	.70	.66	.30	-.61	.67	-.58	.31	.78	.50	.26	.61	.39	-.60	.76	.65	.58	.61	.52	.75						
22. Guilt	-.38	.54	.51	.03	-.49	.49	-.46	.48	.57	.22	.29	.70	.24	-.45	.61	.77	.57	.69	.27	.54	.81					
23. Embarrassment	-.57	.64	-.10	.44	-.52	.52	-.46	.09	.66	.52	.22	.55	.32	-.65	.61	.45	.33	.33	.30	.62	.81	.60				
24. Overall mood	.68	-.32	.59	-.32	.73	-.22	.71	-.44	-.22	-.41	-.39	-.57	-.38	-.67	-.39	-.37	-.12	-.29	.07	-.20	-.34	-.31	-.44			
25. Aroused	-.51	.48	-.48	.34	-.53	.61	-.40	.16	.42	.49	.34	.44	.50	-.68	.55	.32	.33	.29	.20	.52	.43	-.32	.55	-.47		
26. Stressed	-.62	.82	.60	.47	-.75	.77	-.62	.23	.68	.63	.31	.63	.54	-.82	.76	.60	.41	.45	.40	.66	.73	.56	.77	-.57	.71	

Note. Significant ($p < .05$) correlations are in bold.

Table 3
Means (and Standard Deviations) of the Subjective Variables as a Function of Type of Independent Variable Manipulation

Subjective variable	Public speaking	Interpersonal conflict ^a	Stressful interviews	Positive mood inductions	Negative mood inductions	Violent or gruesome film ^b
Cognitive appraisals						
Psychological resources	3.18 (0.28)	3.02 (0.31)	3.27 (0.30)	3.64 (0.77)	3.51 (0.65)	3.03 (0.43)
Challenge	3.18 (0.44)	3.46 (0.42)	2.72 (0.58)	1.67 (0.58)	2.14 (0.64)	1.80 (0.85)
Threat	2.60 (0.47)	2.81 (0.40)	2.27 (0.28)	1.38 (0.50)	2.12 (0.75)	2.12 (0.45)
Novelty	3.36 (0.44)	2.50 (0.32)	2.60 (0.32)	2.41 (0.51)	2.43 (0.55)	2.47 (0.64)
Valence	2.15 (0.24)	2.11 (0.15)	2.21 (0.30)	3.37 (0.83)	2.31 (1.03)	2.13 (0.43)
Intensity	3.46 (0.47)	3.19 (0.41)	3.17 (0.33)	2.13 (0.77)	2.65 (0.77)	2.45 (0.74)
Basic emotions						
Happy	1.77 (0.26)	1.50 (0.15)	1.81 (0.33)	3.00 (0.99)	1.74 (0.83)	1.43 (0.48)
Sadness	1.45 (0.23)	2.21 (0.43)	2.25 (0.37)	1.68 (0.72)	3.13 (1.24)	1.93 (0.44)
Surprise	2.78 (0.57)	2.98 (0.23)	2.37 (0.39)	1.64 (0.53)	1.79 (0.61)	2.37 (0.69)
Anticipation	2.92 (0.50)	2.56 (0.29)	2.11 (0.25)	2.07 (0.47)	1.93 (0.51)	2.65 (0.46)
Disgust	1.44 (0.18)	1.88 (0.20)	1.62 (0.14)	1.47 (0.48)	1.76 (0.50)	2.60 (1.09)
Anger	1.96 (0.32)	3.48 (0.45)	2.03 (0.37)	1.56 (0.45)	2.08 (0.81)	1.92 (0.22)
Fear	2.18 (0.38)	1.79 (0.15)	1.79 (0.28)	1.23 (0.34)	1.83 (0.82)	2.33 (0.54)
Acceptance	2.70 (0.40)	2.81 (0.57)	3.10 (0.25)	3.77 (0.65)	3.18 (0.71)	3.03 (0.48)
Rumination and worry						
Worry	2.75 (0.39)	2.71 (0.34)	3.06 (0.26)	1.47 (0.57)	2.20 (1.00)	1.67 (0.72)
Focus on angry thoughts	1.88 (0.17)	3.48 (0.55)	2.43 (0.42)	1.33 (0.41)	2.08 (1.14)	1.40 (0.38)
Uncontrollable repetitive thoughts	2.21 (0.42)	2.50 (0.22)	2.41 (0.27)	1.89 (0.56)	2.78 (0.83)	1.70 (0.67)
Brooding	2.13 (0.49)	3.00 (0.43)	2.75 (0.88)	1.62 (0.58)	2.67 (1.29)	1.85 (0.85)
Threats to the social self and self-conscious emotions						
Submissive	3.91 (0.30)	3.10 (0.37)	3.50 (0.22)	2.71 (0.95)	3.25 (0.47)	3.22 (0.33)
Fear losing social approval	2.55 (0.50)	2.42 (0.29)	2.65 (0.62)	1.26 (0.26)	1.70 (0.81)	1.35 (0.38)
Shame	2.22 (0.37)	2.73 (0.20)	2.53 (0.57)	1.28 (0.38)	1.80 (0.77)	1.65 (0.25)
Guilt	1.68 (0.17)	2.75 (0.46)	2.02 (0.34)	1.26 (0.36)	1.82 (0.82)	1.35 (0.38)
Embarrassment	2.49 (0.43)	2.94 (0.48)	2.64 (0.66)	1.70 (0.69)	1.92 (0.75)	1.55 (0.48)
Global mood states						
Overall mood	2.22 (0.25)	1.83 (0.15)	2.23 (0.19)	2.77 (1.48)	2.11 (1.04)	1.87 (0.92)
Aroused	3.13 (0.45)	3.06 (0.49)	3.10 (0.37)	2.53 (1.09)	2.67 (0.93)	2.72 (0.29)
Stressed	3.43 (0.52)	3.83 (0.38)	3.21 (0.37)	2.06 (0.83)	2.54 (0.76)	2.40 (0.56)

^a Interpersonal conflict includes manipulations of marital conflict discussions, teaching a task to a bumbling confederate, and role playing with a hostile confederate. ^b Although technically a form of negative mood induction, violent and horror films were included as a distinct category because of their ability to elicit unique emotions such as surprise.

(e.g., Trapnell & Campbell, 1999) to exacerbate cortisol reactivity. Finally, as expected, none of the three broad dimensions (valence of overall mood, arousal, and stress) were associated with cortisol reactivity.

Natural Immunity

NK cells. The overall mean effect size averaged across all time points following the onset of the independent variable manipulation for the 28 manipulations that assessed NK cells (81 effect sizes) was $d = 0.54$, 95% CI [.36, .72], $t(27) = 5.89$, $p < .001$. As was the case with cortisol, the test of the Level 2 variance component was significant for the basic model, $\tau_{00} = .13$, $\chi^2(27) = 69.17$, $p < .001$. Thus, we proceeded to analyze the control model. Among the control variables, the linear function for the time of assessment was significant, $\gamma = -.02$, $t(78) = -5.36$, $p < .001$. The maximum peak for the NK cell response occurred within 15 min of the onset of the social stressor or emotion induction (see Figure 3). It is interesting that manipulations of longer duration were associated with decreases in effect sizes, $\gamma = -.01$, $t(24) = -3.82$, $p = .001$,

probably due to a large initial response followed by a decrease in NK cells. Moreover, the test of the variance component revealed additional unexplained variance, $\tau_{00} = .21$, $\chi^2(23) = 168.39$, $p < .001$. Thus, the coded variables were entered into the model while controlling for time course and duration.

In contrast to cortisol reactivity, none of the cognitive appraisal judgments were related to NK cell responses. However, two negative basic emotions (sadness and disgust) were associated with decreases in NK cell counts. Thus, whereas the experimental manipulations produced a rapid, large increase in NK cell counts, variables likely to be associated with no immediate situational threat attenuated this response. As expected, the global mood states remained unassociated with NK cell effect sizes.

NKCC. NKCC data were based on 34 effect sizes from 17 manipulations. The mean effect size was $d = 0.30$, 95% CI [.15, .45], $t(16) = 3.65$, $p = .002$. The linear term for time of assessment indicated a significant negative linear time course, $\gamma = -.01$, $t(32) = -2.64$, $p = .01$ (see Figure 4). The variance component was not significant, $\tau_{00} = .02$, $\chi^2(16) = 22.82$, $p = .12$; thus, we did not proceed to analyze moderator variables.

(text continues on page 841)

Table 4

Study Characteristics and Standardized Mean Change Effect Sizes (Becker, 1988) Corrected for Sample Bias (Hedges & Olkin, 1985)

Study	Experimental manipulation	<i>N</i>	Dependent measures	Number of effect sizes	Effect size lower bound	Effect size upper bound
Abplanalp et al. (1977)	Stressful interview	21	Cortisol	1	1.10	1.10
al'Absi et al. (1997)	Public speaking	46	Cortisol	2	0.15	0.48
al'Absi et al. (2000)	Public speaking	46	Cortisol	4	1.03	2.15
Benschop et al. (1995)	Teach puzzle to confederate	40	NK cells	1	0.26	0.26
Berk et al. (1989)	Comedy film	5	Cortisol	9	-0.85	-0.30
Berry & Worthington (2001)	Imagine scenes from happy and unhappy romantic relationships	39	Cortisol	2	-0.50	-0.21
Bosch et al. (2003)	Public speaking	44	Lymphocytes	1	0.66	0.66
			CD3	1	0.17	0.17
			Monocytes	1	0.75	0.75
Bosch et al. (2005)	Public speaking	52	Leukocytes	2	0.10	0.49
			Lymphocytes	2	0.21	0.80
			NK cells	1	2.14	2.14
Brosschot et al. (1992)	Teach puzzle to confederate	50	Leukocytes	3	0.11	0.21
			Lymphocytes	3	-0.08	0.10
			Monocytes	3	-0.04	0.01
			CD3	3	-0.09	0.03
			CD4	3	-0.09	0.20
			CD8	3	-0.28	0.17
			NK (CD56) cells	3	-0.43	0.30
			NK (CD16) cells	3	-0.54	0.16
Brown et al. (1993)	Reading and experiencing positive or negative mood statements	26	Cortisol	22	0.13	1.28
Buchanan et al. (1999)	Public speaking	30	Cortisol	3	0.00	0.71
	Humorous video			3	-0.43	-0.79
Clark et al. (2001)	Positive and negative music	48	Cortisol	6	-0.24	0.14
Cohen et al. (2000)	Public speaking	115	CD3	1	0.16	0.16
			CD4	1	0.08	0.08
			CD8	1	0.71	0.71
			CD19	1	0.11	0.11
			NK (CD56) cells	1	1.23	1.23
			NKCC	1	1.31	1.31
			Cortisol	1	0.72	0.72
Demyttenaere et al. (1989)	Film about infertility featuring delivery of a baby	30	Cortisol	12	-0.54	0.17
Dickerson, Kemeny, et al. (2004)	Writing about past shame and guilt inducing situation	31	$\beta 2M^a$	3	-0.10	0.16
			Cortisol	3	-0.10	-0.29
Dopp et al. (2000)	Marital conflict	82	CD8	6	0.00	0.38
			CD8%	6	-0.03	0.22
			CD4	6	-0.23	0.08
			NK cells	6	-0.11	1.05
			NKCC	4	-0.04	0.47
Dutour et al. (1996)	Public speaking	19	Cortisol	15	-0.34	1.32
Fehm-Wolfsdorf et al. (1993)	Public speaking	24	Cortisol	4	-0.74	0.19
Fehm-Wolfsdorf et al. (1999)	Marital conflict	156	Cortisol	4	-0.49	0.14
Furlan et al. (2001)	Public speaking	35	Cortisol	24	0.00	1.16
Futterman et al. (1992)	Acting out situations associated with anxiety, happiness, and sadness	5	CD3%	3	-0.08	1.50
			CD4%	3	-0.29	0.59
			CD8%	3	-0.08	0.12
			NK% (CD57)	3	-0.82	0.42
			NK% (CD16)	3	-0.17	0.19
			NK% (CD56)	3	-0.20	0.25
			NKCC	3	-0.08	0.04

(table continues)

Table 4 (continued)

Study	Experimental manipulation	N	Dependent measures	Number of effect sizes	Effect size lower bound	Effect size upper bound
Futterman et al. (1994)	Reading and experiencing positive or negative mood statements	14	NKCC	2	-0.15	0.24
			NK% (CD16)	2	0.22	0.32
			NK% (CD56)	2	0.04	0.64
			NK% (CD57)	2	0.03	0.43
			CD3%	2	-0.16	-0.02
			CD4%	2	-0.09	-0.03
			CD8%	2	0.09	0.27
Gerra et al. (1996)	Violent film	20	Cortisol	3	-0.18	0.40
Gerritsen et al. (1996; Experiments 1 and 2)	Public speaking	79	Cortisol	2	0.00	0.15
			CD4%	2	-1.21	-1.08
			CD8%	2	-0.17	-0.15
			CD3%	2	-0.60	-0.25
			CD19%	2	-0.53	-0.11
			NK% (CD16)	2	0.43	1.48
			NKCC	2	1.06	1.09
Goebel et al. (2000)	Public speaking	45	Monocytes	2	0.26	0.43
			Granulocytes	2	-0.04	0.07
			Lymphocytes	2	0.11	0.28
			CD4	2	0.06	0.26
			CD8	2	0.12	0.32
			NK cells	2	0.05	0.23
			NK cells	8	0.18	0.97
Griffiths et al. (1997)	Emotional interview about positive or stressful life events	72	Cortisol	2	0.00	0.00
			CD3	2	0.00	0.00
			CD4	2	0.00	0.00
			CD8	2	0.00	0.00
			CD19	2	0.00	0.00
			Lymphocytes	2	0.00	0.00
			Leukocytes	2	0.43	0.44
Hengge et al. (2003)	Stressful interview	25	Neutrophils	2	0.36	0.45
			CD4	2	0.00	0.00
			CD8	2	0.05	0.11
			CD20	2	0.00	0.00
			NK cells	4	0.00	0.59
			Cortisol	8	-0.27	-0.04
			Cortisol	4	-0.66	0.43
Hubert & de Jong-Meyer (1989)	Suspense/horror film	27	Cortisol	4	-0.39	-0.14
			Cortisol	4	-0.39	-0.14
Hubert & de Jong-Meyer (1992)	Suspense/horror film	32	Cortisol	4	-0.66	0.43
Hubert et al. (1993)	Comedy film	52	Cortisol	4	-0.39	-0.14
Hucklebridge et al. (2000; Studies 1 and 2)	Imagining happy or guilty life experiences; positive or negative music	54	NKCC	6	-0.14	1.23
			Cortisol	6	0.00	0.00
			Cortisol	10	-0.90	1.13
			Cortisol	10	-0.46	0.67
			Leukocytes	2	0.01	0.16
			Lymphocytes	2	0.05	0.34
			NK (CD56) cells	2	0.00	1.82
Jacobs et al. (2001)	Public speaking	7	NKCC	2	0.00	0.25
			Cortisol	5	0.13	1.03
			Cortisol	5	0.11	0.67
			Cortisol	4	0.00	1.05
			Cortisol	4	-0.53	0.02
			Cortisol	4	0.00	0.00
			CD4	4	0.00	0.00
Jansen et al. (1998)	Public speaking	10	Neutrophils	4	0.00	0.31
			Lymphocytes	4	0.00	0.00
			Cortisol	2	0.22	0.37
Jansen et al. (2000)	Public speaking	21	Cortisol	5	0.11	0.67
Kemmer et al. (1986)	Public speaking	9	Cortisol	4	0.00	1.05
Kiecolt-Glaser et al. (1997)	Marital conflict	62	Cortisol	4	-0.53	0.02
Knapp et al. (1992)	Imagining positive and negative life experiences	22	NKCC	4	0.00	0.00
			CD4	4	0.00	0.00
			Neutrophils	4	0.00	0.31
			Lymphocytes	4	0.00	0.00
			Cortisol	2	0.22	0.37
Knight & Rickard (2001)	Public speaking	31	Cortisol	2	0.22	0.37
Larson et al. (2001)	Public speaking	56	Cortisol	1	-0.12	-0.12
			NKCC	1	0.81	0.81
			Cortisol	2	0.36	0.70
Lovallo et al. (2000)	Public speaking	10	Cortisol	2	0.36	0.70

(table continues)

Table 4 (continued)

Study	Experimental manipulation	<i>N</i>	Dependent measures	Number of effect sizes	Effect size lower bound	Effect size upper bound
Luecken (1998)	Public speaking and sad film	61	Cortisol	4	-0.32	-0.07
Lupien et al. (1997)	Public speaking	14	Cortisol	3	-0.28	-0.03
Malarkey et al. (1994)	Marital conflict	90	Cortisol	6	-0.40	-0.10
Marinari et al. (1976)	Public speaking	60	Cortisol	4	0.15	1.13
Marsland et al. (1995)	Public speaking	30	CD19	1	-0.13	-0.13
			NK cells	1	0.82	0.82
			CD3	1	3.11	3.11
			CD4	1	-0.08	-0.08
			CD8	1	0.22	0.22
Marsland et al. (2001)	Public speaking	84	CD19	1	0.03	0.03
			NK cells	1	1.11	1.11
			CD3	1	0.15	0.15
			CD4	1	0.03	0.03
			CD8	1	0.25	0.25
McCleery et al. (2000)	Imagining the death of a loved one	30	Cortisol	4	-1.15	-0.13
McDonald & Yagi (1960)	Thinking they accidentally severely harmed someone	17	Eosinophils	1	0.20	0.20
G. E. Miller et al. (1999)	Marital conflict	82	NKCC	1	0.02	0.02
			NK cells	1	-0.09	0.68
			CD4	1	-0.32	0.09
			CD8	1	-0.16	0.19
Mills, Berry, et al. (1995)	Public speaking	86	CD3	1	-0.05	-0.05
			CD4	1	-0.14	-0.14
			CD8	1	0.39	0.39
			NK cells	3	0.49	0.55
			CD19	1	-0.17	-0.17
			Lymphocytes	1	0.10	0.10
			Monocytes	1	0.22	0.22
Mills, Haeri, & Dimsdale (1995)	Public speaking	24	CD8	1	0.27	0.27
			NK (CD16) cells	1	0.67	0.67
			NK (CD56) cells	1	0.67	0.67
			NK (CD57) cells	1	0.65	0.65
			Leukocytes	1	0.13	0.13
			CD3	1	0.07	0.07
Mills et al. (1998)	Public speaking	110	NK cells	1	0.26	0.26
			CD19	1	0.00	0.00
			CD3	1	0.00	0.00
			CD4	1	0.16	0.16
			CD8	1	0.19	0.19
Mills et al. (2003)	Public speaking	23	CD4	1	0.15	0.15
			CD8	1	0.14	0.14
			Monocytes	1	0.41	0.41
			Neutrophils	1	0.08	0.08
			NK cells	1	0.54	0.54
			Lymphocytes	1	0.22	0.22
			Leukocytes	1	0.12	0.12
Moyna et al. (1999)	Public speaking	45	Monocytes	2	-1.07	0.00
			CD3	2	-0.20	0.12
			CD4	2	-0.09	0.09
			CD8	2	-0.15	0.45
			CD19	2	-0.14	0.00
			NK cells	2	0.00	1.69
			NKCC	2	0.25	0.98
			Cortisol	6	-0.24	0.20
Naliboff et al. (1995)	Role play with hostile confederate	20	CD20	1	-0.22	-0.22
			NK (CD57) cells	1	0.30	0.30
			NK (CD16) cells	1	1.16	1.16
			NK (CD56) cells	1	1.09	1.09
			CD8-CD57 ^b	1	0.30	0.30
			NKCC	1	0.58	0.58
Nejtek (2002)	Violent film	44	Cortisol	2	0.16	0.43

(table continues)

Table 4 (continued)

Study	Experimental manipulation	<i>N</i>	Dependent measures	Number of effect sizes	Effect size lower bound	Effect size upper bound
Nicolson et al. (1997)	Public speaking	55	Cortisol	6	-2.94	0.05
Pawlak et al. (1999)	Public speaking	30	CD4	2	0.28	0.36
			CD8	2	0.49	1.69
			NK cells	2	-0.12	2.73
			NKCC	2	-0.10	0.58
			Cortisol	3	-0.06	0.65
Rohrman et al. (1999)	Public speaking	60	Cortisol	3	-0.06	0.65
Sachs et al. (1993)	Stressful interview	36	Cortisol	2	-0.55	-0.21
Stones et al. (1999)	Stressful interview	15	Cortisol	1	-0.43	-0.43
VanderArk & Ely (1993)	Positive and negative music	60	Cortisol	2	-0.17	0.30
van der Pompe et al. (1996)	Public speaking	15	Cortisol	3	-0.50	-0.27
van der Pompe et al. (1997)	Public speaking	15	Lymphocytes	3	0.08	0.50
			CD3	3	0.11	1.95
			CD4	3	-0.49	0.24
			CD8	3	-0.15	0.47
			NK cells	3	-0.11	1.21
			NKCC	3	-0.91	0.86
			CD8	3	0.08	0.55
			NK cells	3	0.21	1.43
			NKCC	3	-0.15	0.87
			CD19	3	0.00	0.22
van der Pompe et al. (1998)	Public speaking	15	Lymphocytes	3	0.30	0.72
			CD4	3	0.16	0.29
			Cortisol	8	0.21	1.16
			Cortisol	1	0.49	0.49
van Eck et al. (1996)	Public speaking	87	Cortisol	8	0.21	1.16
Zakowski et al. (1992)	Gruesome surgical film	20	Cortisol	1	0.49	0.49

Note. NK = natural killer; NKCC = natural killer cell cytotoxicity.

^a β 2M is a general immune marker reflecting activity of monocytes and lymphocytes (analyzed with monocytes and polymorphonuclear cells). ^b CD8 cells with Leu 7 receptors.

Other measures of natural immunity: Leukocytes, monocytes, and polymorphonuclear cells. Total leukocytes, monocytes, and three types of polymorphonuclear cells were analyzed together, resulting in 41 effect sizes from 14 experimental manipulations. The mean effect size across all time points was $d = 0.16$, 95% CI [.08, .25], $t(13) = 3.76$, $p = .003$. There was a significant cubic time course, $\gamma = -.00001$, $t(37) = -4.50$, $p < .001$. Inspection of the variance component revealed no further unexplained variation, $\tau_{00} = .01$, $\chi^2(13) = 16.76$, $p = .21$. Accordingly, we conducted no further moderator analyses.

Specific Immunity

Total lymphocytes. The results presented here for total lymphocytes were based on 29 effect sizes from 13 experimental manipulations. The mean effect size was $d = 0.24$, 95% CI [.12, .36], $t(12) = 3.86$, $p = .003$. Among the control variables, the quadratic function for the time-of-assessment variable was significant, $\gamma = .0002$, $t(26) = 3.31$, $p = .003$. Lymphocyte reactivity occurred rapidly, peaking 1–15 min after the onset of the experimental manipulation and tapering off rapidly (see Figure 5). A significant test of the variance component revealed that control variables may explain additional variance, $\tau_{00} = .04$, $\chi^2(12) = 57.59$, $p < .001$. Among the control variables, an increasing percentage of male participants was associated with decreased total lymphocyte responses, $\gamma = -.004$, $t(8) = -2.32$, $p = .05$. After addition of the control variables, the variance component remained significant, $\tau_{00} = .02$, $\chi^2(8) = 23.09$, $p = .004$. We therefore added the coded variables controlling for the response

time course and gender. Among the subjective variables, two basic emotions—surprise, $\gamma = .21$, $t(10) = 2.21$, $p = .05$, and anticipation, $\gamma = .34$, $t(10) = 3.89$, $p = .003$ —were associated with increases in total lymphocytes. Thus, emotions associated with immediate or imminent threats (i.e., surprise and anticipation) were related to increased lymphocyte counts. As expected, the global mood state judgments were unrelated to effect sizes.

T lymphocytes. Data to evaluate T lymphocytes were derived from 34 effect sizes from 22 experimental manipulations. The mean effect size was $d = 0.29$, 95% CI [-0.03, .60], $t(21) = 1.80$, $p = .09$. The time course was not significant; however, the maximum T cell response occurred within the first 15 min after the experimental manipulation (see Figure 6). The variance component indicated significant residual variance, $\tau_{00} = .42$, $\chi^2(21) = 82.27$, $p < .001$. None of the control variables were related to the effect sizes. Exemplars from all the categories of variables except global mood states were related to effect sizes. However, the direction of the significant effects was generally the opposite to that observed for cortisol and total lymphocytes. For instance, appraisals of challenge and novelty were associated with an attenuation of the stress response, as was the basic emotion of anticipation. Among the rumination items, brooding also attenuated T lymphocyte reactivity. Finally, studies judged to produce high levels of embarrassment, an emotion associated with threats to the social self, also decreased T lymphocyte responses. Thus, variables associated with increased cortisol output and up-regulation of total lymphocytes were associated with the down-regulation of T lymphocyte activity, suggesting that the cell-mediated immunity may

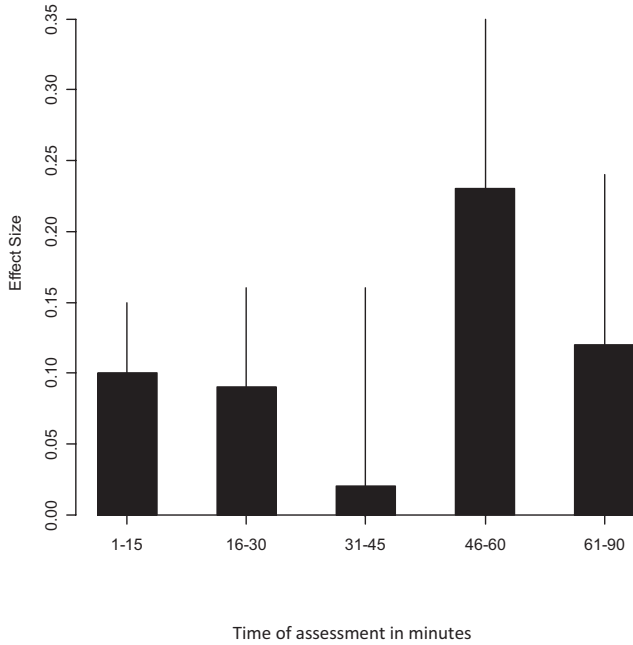


Figure 2. The time course of the cortisol response. Error bars represent standard error of the mean.

be temporarily suspended during times of acute or imminent stress, when the social self is threatened, and when stressors require extended effort. As expected, none of the global mood state judgments were related to the effect sizes. Table 6 displays these results.

T helper cells. Data on T helper cells were derived from 54 effect sizes across 27 experimental manipulations. The mean effect size across all time points was $d = -0.05$, 95% CI $[-.16, .05]$, $t(26) = -0.90$, $p = .38$. Time of assessment was not related to the effect sizes, suggesting equivalent effect sizes across the duration of the experiments (see Figure 7). Gender was marginally related to the effect sizes such that studies with larger proportions of male participants showed decreased T helper responses, $\gamma = -.004$, $t(22) = -2.01$, $p = .06$. None of the other control variables were related to T helper responses, and the variance component remained significant at this stage, $\tau_{00} = .06$, $\chi^2(22) = 62.41$, $p < .001$; thus, the moderators were analyzed while controlling for gender. Among the moderator variables, only one basic emotion was related to the effect sizes. Specifically, manipulations rated as likely to produce feelings of anticipation were associated with decreased T helper reactivity, $\gamma = -.19$, $t(24) = -2.71$, $p = .01$. Thus, although T helper cells did not seem overly responsive to the experimental manipulations, judgments of anticipation, which are associated with imminent environmental threat, were related to the down-regulation of this specific immune outcome. As expected, none of the global mood state judgments were related to the effect sizes.

T cytotoxic cells. T cytotoxic data were derived from 58 effect sizes from 26 experimental manipulations, all of which were motivated performance tasks and controlled for time of day. The mean effect size across all time points was $d = 0.19$, 95% CI $[.08, .29]$, $t(25) = 3.51$, $p = .002$. There was significant cubic time

course, $\gamma = .000009$, $t(53) = 2.41$, $p = .02$ (see Figure 8). None of the other control variables were significant. As expected, the variance component remained significant, $\tau_{00} = .06$, $\chi^2(21) = 150.32$, $p < .001$; thus, the moderator variables were analyzed while controlling for time course. None of the subjective variables were related to T cytotoxic reactivity.

B lymphocytes. The data for B lymphocytes were based on 19 effect sizes from 13 experimental manipulations. The mean effect size was $d = -0.07$, 95% CI $[-.16, .01]$, $t(12) = -1.64$, $p = .13$. In the control model, the cubic time-of-assessment function was significant, $\gamma = -.00002$, $t(14) = -8.31$, $p < .001$. The largest effect sizes were observed fairly late following the social stressor onset (see Figure 9). Gender also predicted B cell responses, suggesting that as the percentage of male participants increased, B lymphocyte responses decreased, $\gamma = -.004$, $t(9) = -3.97$, $p = .004$. The same pattern was observed for age, suggesting that studies with older participants produced smaller effect sizes, $\gamma = -.007$, $t(9) = -3.49$, $p = .008$. It is important to note that the variance component revealed that additional variables may explain the residual variance, $\tau_{00} = .02$, $\chi^2(8) = 157.45$, $p < .001$. However, none of the subjective variables were related to B lymphocyte reactivity.

Table 5
Subjectively Coded Predictors of Cortisol Responses to Laboratory Social Stressors and Emotion Inductions Controlling for Age

Subjective variable	γ	SE	t ratio
Cognitive appraisal ratings			
Psychological resources	.07	.13	0.51
Challenge	.24	.07	3.27**
Threat	.22	.10	2.08*
Novelty	.36	.10	3.63***
Valence	-.11	.09	-1.26
Intensity	.28	.07	3.93***
Basic emotions			
Happy	-.04	.09	-0.50
Sadness	-.01	.07	-0.14
Surprise	.19	.09	2.08*
Anticipation	.19	.11	1.77
Disgust	-.07	.14	-0.55
Anger	-.07	.11	-0.66
Fear	.13	.12	1.08
Acceptance	-.18	.10	1.74
Rumination and worry			
Worry	.14	.08	1.63
Focus on angry thoughts	-.00	.10	-0.02
Uncontrollable repetitive thoughts	.28	.10	2.74**
Brooding	.17	.08	2.12*
Social threat			
Submissive	.26	.10	2.66**
Fear losing social approval	.23	.09	2.65**
Shame	.18	.11	1.57
Guilt	-.03	.12	-0.23
Embarrassment	.14	.10	1.34
Global mood states			
Overall mood	.04	.08	0.54
Aroused	.16	.10	1.59
Stressed	.12	.08	1.61

* $p = .05$. ** $p = .01$. *** $p = .001$.

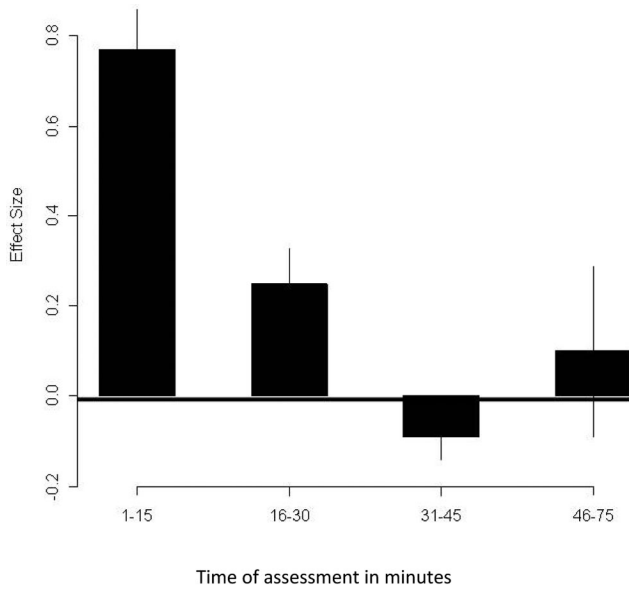


Figure 3. The time course of the natural killer cell response. Error bars represent standard error of the mean.

Additional Analyses of Composite Subjective Variables

As expected, the broad dimensions of negative affect, emotional arousal, and action tendencies (approach-withdrawal) were not associated with effect sizes for any of the measures. In summary, across all the various measures, none of the tests involving the broad dimensions and global mood states were significantly associated with cortisol or immune responses to social stressor and

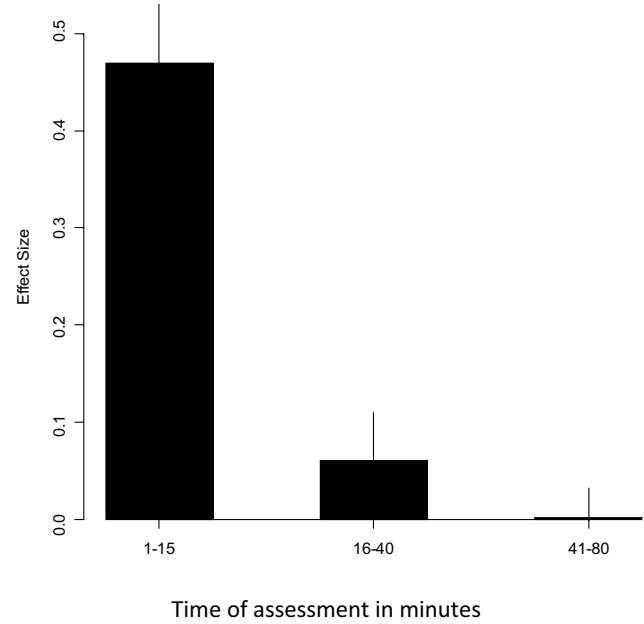


Figure 5. The time course of the total lymphocyte response. Error bars represent standard error of the mean.

emotion inductions. By contrast, numerous specific appraisals and emotions were associated with these physiological responses.

Discussion

This was the first meta-analysis to use high-inference coding procedures to systematically examine the effects of five clusters of

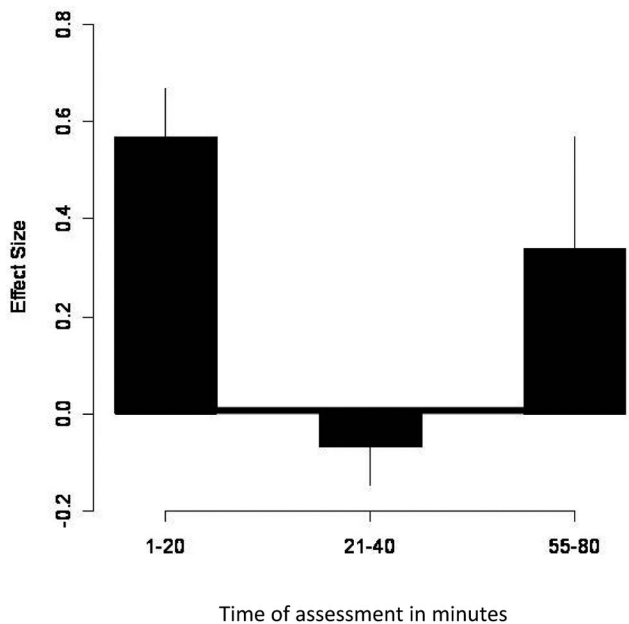


Figure 4. The time course of the natural killer cell cytotoxic response. Error bars represent standard error of the mean.

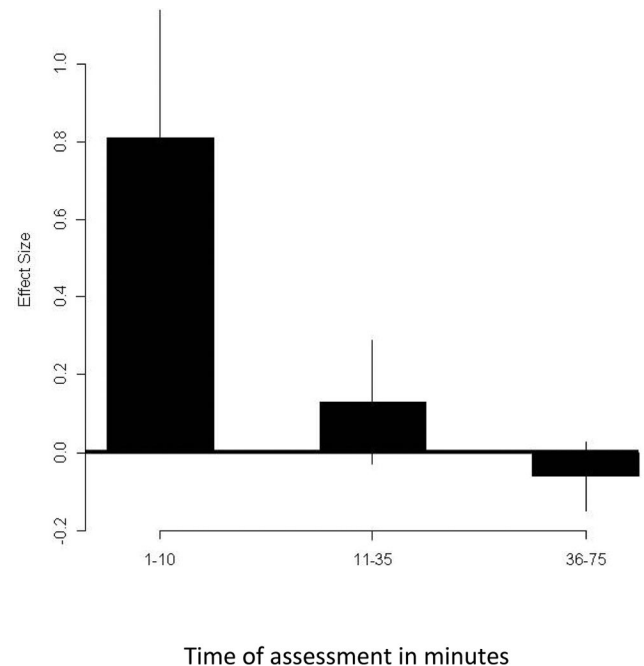


Figure 6. The time course of the T lymphocyte response. Error bars represent standard error of the mean.

Table 6
Subjectively Coded Predictors of T Lymphocyte Responses to Laboratory Social Stressors and Emotion Inductions

Subjective variable	γ	SE	t ratio
Cognitive appraisal ratings			
Psychological resources	.43	.37	1.14
Challenge	-.50	.23	-2.14*
Threat	-.21	.25	-0.84
Novelty	-.61	.19	-3.13**
Valence	-.38	.42	-0.89
Intensity	-.26	.33	-0.78
Basic emotions			
Happy	.20	.35	0.57
Sadness	.11	.32	0.35
Surprise	-.41	.20	-1.98
Anticipation	-.61	.20	-3.14**
Disgust	.29	.16	1.76
Anger	-.45	.34	-1.32
Fear	-.03	.28	-0.12
Acceptance	.18	.46	0.39
Rumination and worry			
Worry	.07	.28	0.26
Focus on angry thoughts	-.47	.28	-1.67
Uncontrollable repetitive thoughts	-.28	.42	-0.66
Brooding	-.57	.28	-2.06*
Social threat			
Submissive	-.08	.25	-0.34
Fear losing social approval	.04	.23	0.19
Shame	-.47	.30	-1.55
Guilt	-.24	.41	-0.58
Embarrassment	-.68	.24	-2.81**
Global mood states			
Overall mood	.36	.32	1.13
Aroused	.04	.30	0.13
Stressed	-.50	.31	-1.60

* $p = .05$. ** $p = .01$.

theoretically relevant cognitive and affective influences on cortisol and immune reactivity. The five groups consisted of (a) cognitive appraisals, (b) basic emotions, (c) rumination and worry, (d) social threat, and (e) global mood states. As expected, we obtained evidence that exemplars from each of the four specific categories were associated with cortisol and immune responses to social stressors and emotion inductions. By contrast, the global mood states and broad dimensional categorizations (e.g., approach-avoidance) were entirely unrelated to these same outcomes. These outcomes represent an empirical advance in that our results helped clarify the puzzling finding in the literature that self-reports of general negative affect and perceived stress are generally uncorrelated with cortisol and immune reactivity (Dickerson & Kemeny, 2004; Segerstrom & Miller, 2004), despite their prominence in models of stress and health.

Our theory and results suggest that emotions and appraisals do indeed impact HPA and immune reactivity to stress, yet these relationships are complex. Categorization of emotions along continua such as negative affective valence, arousal, and action tendencies does not appear informative in this context. Rather, we have found evidence in support of the integrated specificity model (Brosschot et al., 2006; Dickerson et al., 2004; Dickerson, Gruenewald, & Kemeny, 2004; Dickerson & Kemeny, 2004; Kemeny, 2003; Weiner, 1992) as well as cognitive appraisal models of stress

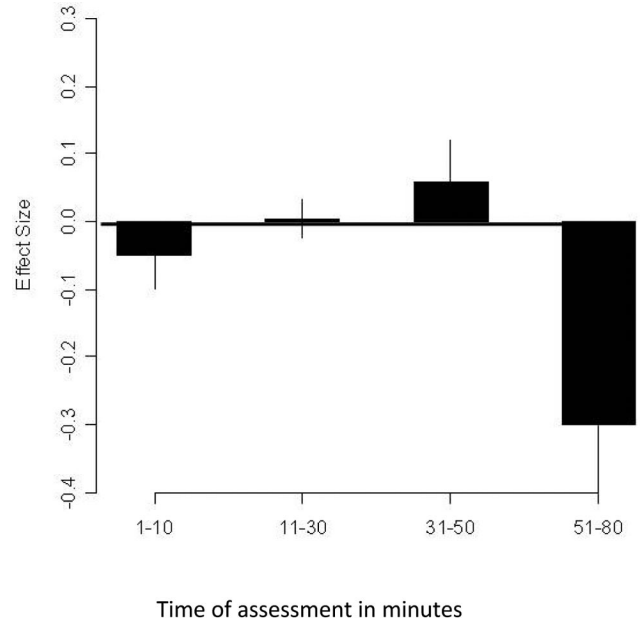


Figure 7. The time course of the T helper cell response. Error bars represent standard error of the mean.

and emotions (Lazarus & Folkman, 1984). Within this context, we have proposed a theoretical framework for understanding the puzzling null relationship between negative affect and cortisol and immunity. This framework specifies how certain physiological activity in response to specific environmental demands (e.g., characteristics of the stressor) likely promoted the survival of our ancestors. It appears that just as appraisals and emotions have long been known to initiate evolutionarily adaptive behaviors (e.g., Lorenz, 1966), so too do they appear to initiate adaptive physiological changes.

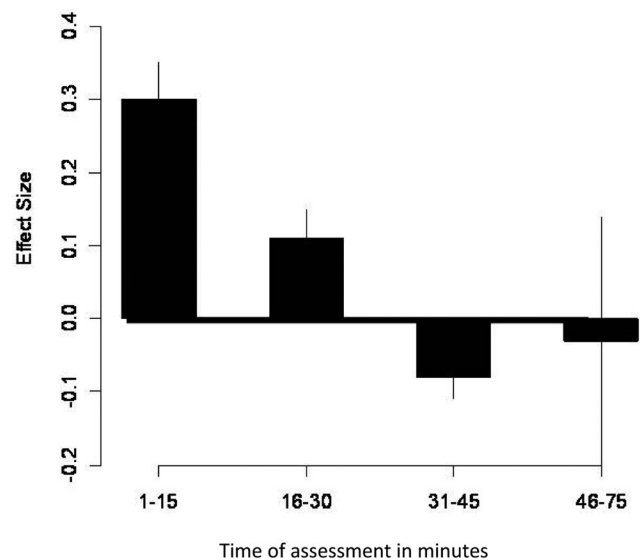


Figure 8. The time course of the T cytotoxic cell response. Error bars represent standard error of the mean.

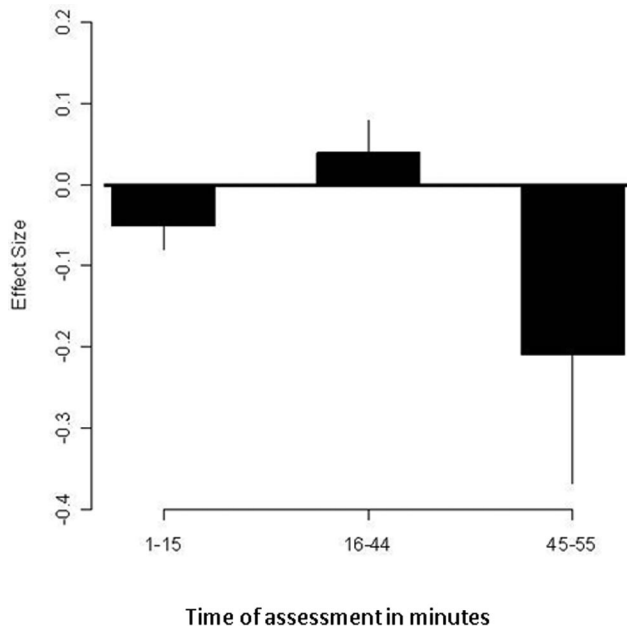


Figure 9. The time course of the B lymphocyte response. Error bars represent standard error of the mean.

In general, our results were consistent with our framework demonstrating that emotions and appraisals that are often associated with immediate or imminent stressors, threats to the social self, and stressors requiring extended effort elicit primarily adaptive changes in cortisol and immunity. For instance, rumination, which is likely associated with the need to maintain vigilance toward unresolved stressors, tended to exert an exacerbating influence on the HPA axis yet was consistent with the suppression of cell-mediated immunity (i.e., decreases in T lymphocytes). Consistent with the vigilance role of rumination, judgments of anticipation were also associated with down-regulation of cell-mediated immunity. Although rumination is relatively understudied in comparison with broad dimensions and other emotions (e.g., sadness), these effects are consistent with the notion that considering unresolved stressors would produce an adaptive release of energy when it is needed, while simultaneously suppressing energy expenditure on cell-mediated immunity. However, our findings also highlight the complexity and limited understanding of the role of specific immunity in response to acute stress. For instance, ratings of anticipation and surprise were associated with increases in total lymphocytes into the periphery. Because these two emotions may have been associated with events reflecting uncertain outcomes for our ancestors (e.g., anticipation of a long hunt, surprised by a snake), releasing increased numbers of lymphocytes into the periphery may have given our ancestors an increased chance of survival by acting as a second line of defense (albeit slow acting) should the natural immune system become overwhelmed. Future research will likely shed light on the complex role of the specific immune system during acute stress.

The current findings are remarkably consistent with prior theory and research that indicate the sensitivity of the HPA axis to social status and threats to the social self (e.g., Dickerson, Gruenewald, & Kemeny, 2004; Dickerson, Kemeny, et al., 2004; Sapolsky,

2004). Indeed, ratings of feelings of submissiveness and fearing the loss of social approval were related to exacerbated cortisol responses. Given the ubiquity of societal stratification, these findings may partially explain poor health outcomes among those lowest in status (e.g., the poor, older persons). Unexpectedly, the self-conscious emotions were generally unrelated to cortisol and immune outcomes. Recall that shame in particular is hypothesized to co-occur in the presence of threats to the social self (e.g., Dickerson, Gruenewald, & Kemeny, 2004). We believe that shame does indeed play a role in cortisol and immune reactivity, although the present meta-analysis failed to detect a relationship. Indeed, a promising line of research that has directly manipulated shame supports this notion (e.g., Dickerson, Kemeny, et al., 2004). The null effects of shame may have occurred because only a limited number of studies included in the current meta-analysis were likely to induce feelings of shame. For instance, public speaking in front of an audience might make participants feel submissive and worried about losing social approval but is unlikely to produce excessive levels of shame. The null results for the self-conscious emotions should be interpreted cautiously until further research directly examines their influences on human physiology. At a more general level, the lack of direct manipulations of some emotions may have impacted our findings by leading to increased rates of Type II error for emotions that are not often elicited by laboratory stress procedures.

The present work was also the first to meta-analytically demonstrate relationships between rumination and cortisol and immune reactivity. Specifically, rumination was associated with increases in cortisol and suppression of T lymphocytes. Our results provide a meta-analytic confirmation of Brosschot et al.'s (2006) hypothesis that rumination should dysregulate cortisol and immune reactivity. Furthermore, because rumination is a feature of numerous psychiatric disorders such as anxiety, depression, and posttraumatic stress disorder (Borders & Earleywine, 2006; Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Nolen-Hoeksema & Morrow, 1991), these findings may also help explain the HPA axis dysregulation observed in depression, posttraumatic stress disorder, and the additional adverse health outcomes associated with rumination (Gillespie & Nemeroff, 2005; Thomsen, Mehlsen, Christensen, & Zachariae, 2003; Thomsen, Mehlsen, Hokland, et al., 2004; Thomsen, Mehlsen, Olesen, et al., 2004).

A final novel contribution of the current meta-analysis was the examination of the time course of immune responses across a fairly large number of studies. Most of the immune outcomes demonstrated a rapid linear increase or decrease within minutes of the stressor or emotion induction onset. However, these results should be interpreted cautiously because of the small number of effect sizes for some of the outcome groups.

Support for the Integrated Specificity Model

Our results are consistent with the core concept behind the integrated specificity model. That is, within the context of a stressful event, organisms produce an integrated and coordinated response at multiple levels (i.e., cognitive, emotional, and physiological). Furthermore, appraisals and emotions influence the physiological stress response in a sophisticated manner. During acutely stressful occurrences, cortisol and immune reactivity to stress can be enhanced or attenuated depending on the emotions

and appraisals elicited in specific situations. These findings are also consistent with ecological models of stress and physiology (e.g., Segerstrom, 2007). Specifically, these systems respond in a manner that would have ensured efficient energy conservation of processes not immediately relevant to survival and promoted energy allocation to processes supporting the organism's immediate well-being. Such a pattern of physiological activity would have contributed to the survival of our ancestors. Unfortunately, our evolutionary past has not completely adapted to the demands of modern life. In modern times, the same flight-or-fight response activated in response to life-threatening situations that spared many of our ancestors can be activated in response to far more mundane events such as giving a brief presentation (Maier & Watkins, 1998; Sapolsky, 1998, 2004).

Despite the consistency of our findings with the integrated specificity model's core principles, a close inspection of our theoretical framework (see Figure 1) reveals that not all the relevant emotions and appraisal ratings predicted cortisol and immune outcomes in the expected manner. Thus, we cannot unequivocally claim support for the integrated specificity model. For example, not all the ratings associated with the lack of an immediate or imminent stressor such as acceptance or happiness were associated with down-regulation of immunity. Similarly, although most of the variables associated with imminent stress exacerbated cortisol output, not all showed this relationship. For example, ratings of anticipation were unrelated to cortisol output despite its relevance to stressors that are imminent. This contradicts one study that found increased cortisol output when anticipating a judo match (Salvador, Suay, González-Bono, & Serrano, 2003). It was also unclear why none of the appraisals or emotion ratings predicted increased NKCC. Perhaps some physiological responses are so ingrained in our physiology that they are impervious to mediating cognitive or affective influences. This was unexpected, as many appraisals and emotions would seem to indicate that the energy expended to increase NKCC would be better utilized by other systems. Future research will determine whether these and other null relationships are due to Type II error or gaps in our theorizing.

Another serious complication for the integrated specificity model concerns the dissociation between the effects of emotions and appraisals on total lymphocytes and T lymphocytes. Whereas judgments of emotions and appraisals associated with extended effort or immediate threat were associated with smaller T lymphocyte responses to stress, surprise and anticipation were associated with increased total lymphocyte responses. Both responses are arguably adaptive. Although simply redistributing lymphocytes into the periphery is not as costly as producing new lymphocytes, presumably an attenuated redistribution of lymphocytes (as was observed with T lymphocytes) would conserve energy for dealing with the immediate threat. On the other hand, it is equally plausible that increased lymphocytes in the periphery when surprised or in anticipation of a stressful encounter could facilitate a specific immune response in the event that the natural immune system becomes overwhelmed. Thus, it is unclear what is responsible for these opposing effects. Until further research clarifies the roles of various types of emotional stressors on lymphocyte redistribution, we cannot claim that the integrated specificity model fully explains these somewhat contradictory observations.

One serious consideration concerning the immunity results is that there is some ambiguity regarding the interpretation of

changes in immune cell counts from baseline. These changes do not indicate new cell production or elimination. Rather, these changes represent movement in the number of existing immune cells into the periphery from their respective storage sites such as the spleen and lymph nodes or movement out of the periphery and back into these storage sites. It is unclear to what extent transient stress-induced changes to immune cell numbers are broadly indicative of adaptive or maladaptive immune system functioning if at all. Thus, although researchers often regard the up-regulation of immunity as synonymous with increased cell counts, and vice versa for down-regulation, the bottom line is that the current state of the literature allows speculation only about the veracity of this assumption. It is also unknown how these small changes induced by acute stress might influence subsequent health. Ongoing and future research using functional assays of immune responsiveness to stress such as mitogenic proliferation, antibody production, and cytokine assays will likely help answer these important questions. Without clear answers to these questions, it is difficult to come to firm conclusions regarding the role of specific emotions and cognitive appraisals influencing immune system function.

Further exploration of the integrated specificity model should investigate the role of specific emotions and appraisals on cardiovascular reactivity and SNS hormone responses to acute stress. Indeed, inclusion of these measures would provide a more complete profile of acute responses to social-emotional stressors. The SNS hormones norepinephrine and epinephrine are quick acting in response to stress and have been implicated in altering immune responses such as NKCC and lymphocyte proliferation via surface receptor binding with immune cells (Madden & Livnat, 1991). The literature on SNS hormones and cardiovascular activity is larger than the stress and immunity literature, and its inclusion herein would have allowed for increased statistical power. It is important to note that there are also documented associations of SNS and cardiovascular outcomes with specific emotions such as anger (Suarez, Kuhn, Schanberg, Williams, & Zimmerman, 1998). Uncovering the effects of specific emotions and appraisals on SNS and cardiovascular reactivity would have been especially relevant for our meta-analysis of acute stress. Our failure to include these measures limits understanding of the complete, integrated acute response to social-emotional stressors. Thus, in answer to our original question regarding why research has failed to detect effects of general negative affect on cortisol and immunity, it is conceivable that SNS hormones are actually directly sensitive to general distress, whereas cortisol and immunity are not. We acknowledge the possibility that negative affective experience initiates SNS activity, which in turn mediates the effects of stress on cortisol and immunity.

One caveat concerning our theorizing is that we have at times oversimplified some of the complex phenomena that we seek to understand. For instance, our model implies a unidirectional relationship between cognitive appraisals, emotions, and subsequent physiological activity. Specifically, our model suggests that appraisals cause emotions, which then influence cortisol and immunity. We do, however, recognize that there is often a bidirectional relationship between physiological reactivity, especially SNS activity, and appraisals and emotional responses (Schachter & Singer, 1962).

We have also focused on only one function of cortisol (i.e., its ability to release stored energy). Cortisol serves additional func-

tions such as anti-inflammatory activity, the inhibition of lymphocyte proliferation, and catecholamine synthesis. We have focused primarily on the energy-releasing aspect of cortisol because of its relevance to acutely stressful situations. However, closer inspection of our data reveals that many of the same appraisals and emotions that exacerbated cortisol activity were associated with a reduction in the number of T lymphocytes in the periphery. Although we admit that the interpretation of cell counts is problematic, this dissociation between cortisol and cell-mediated immunity is consistent with the immunosuppressive role of cortisol.

A final caveat concerns the rational description of the evolutionary processes that we suspect of contributing to the effects of emotions and appraisals on cortisol and immune reactivity to stress. Although our theorizing seems to impart a rational motivation on behalf of evolutionary forces, in reality natural selection does not operate in such a manner. We do suggest that certain advantageous psychological and physiological responses have been forged via natural selection over a great period. However, far from implying the presence of a homunculus, we recognize that emotions and appraisals will not always influence cortisol and immunity in an entirely adaptive manner. Because of the nature of natural selection, puzzling side effects are also likely to occur. These side effects might explain some of our own curious findings. For instance, we found that ratings of brooding rumination were associated with decreased T lymphocyte counts. Considering the general negative effects of rumination on other physiological indicators (McCullough et al., 2007; Ray, Wilhelm, & Gross, 2008), this result is not surprising. However, we can think of no adaptive purpose for this result.

Implications

Our findings suggest numerous implications for future research on psychophysiological reactivity to stress. We strongly believe that stress researchers should broaden their array of stress manipulations to elicit a broad range of appraisals and emotions. The results and the conclusions of any meta-analysis depend on the nature of the individual studies included in the broader literature. Because there were numerous distinct experimental manipulations of stress, this allowed us to code for a wide variety of emotions. However, a sizable number of studies relied on public speaking to manipulate stress. Although there was substantial variability even within public speaking tasks in terms of implementation and the subsequent appraisals and emotions elicited, a broader range of social stressors, further theoretical refinement of the concept of stress, and controlled emotion induction studies intended to manipulate single emotions may help clarify and extend the results presented here. Furthermore, our results suggest that not all stress manipulations are created equal. Depending on the emotions and appraisals elicited by a certain manipulation, cortisol and immune responses may differ as a function of the types of laboratory stress manipulation.

Another implication concerns individual differences. To the extent that individuals vary in their affective styles or reactivity to life's stressors, they may be at risk for long-term, adverse health consequences. Individual differences in rumination may be particularly malignant. Rumination may be an adaptive cognitive-affective response to a stressful situation in the short term, because it focuses cognitive activity on resolving the issue at hand. Unfor-

tunately, over time chronic rumination may lead to HPA axis and immune dysregulation. These results are consistent with a growing body of research demonstrating adverse physical and mental health consequences of rumination (Thomsen, Mehlsen, Hokland, et al., 2004; Thomsen, Mehlsen, Olesen, et al., 2004) as well as recent theorizing on the subject (Brosschot et al., 2006). It is also interesting to note that although worry is similar to rumination in its focus on perseverative cognition, worry was not related to any of the outcomes. As was the case with shame, we believe that this may be due to the limited number of studies that were likely to induce substantial levels of worry.⁶ Alternatively, it may be that worry's future-oriented focus rather than rumination's past-oriented focus elicits distinct physiological profiles. Another possibility is that it is the emotion of anticipation and not simply worry that is responsible for eliciting a stress response. Future studies may yet elaborate on the role of worry in influencing cortisol and the immune system.

Our findings also have implications for human social life. Groups are a necessary element of primate life (e.g., Fiske, 1991). Maintaining good relations with one's primary group was necessary for the survival of our ancestors and is important for our psychological well-being today. It should come as no surprise then that a fight-or-flight reaction occurs when one's social status is threatened. Indeed, social rejection can be an emotionally intense experience, capable of eliciting aggressive behavior (Twenge, Baumeister, Tice, & Stucke, 2001). These results suggest that for groups that are low in socioeconomic status or are stigmatized, and consequently find themselves fearing social isolation or feeling submissive on a regular basis (e.g., those repeatedly needing to submit job applications), such repeated stress may be a contributor to their health deficits (Sapolsky, 2004).

Despite these implications, there is reason for hope. The theoretical framework that we have proposed suggests that physiological reactivity may be attenuated in at least two ways. First, if it is possible to reduce one's exposure to stressors that are immediate or imminent, threaten one's social status, or require extended effort, this would obviously lessen the frequency of stress responses. Second, if this is not possible, cognitive reappraisals (e.g., Gross, 1998; Ochsner & Gross, 2005) or direct training in reducing the intensity of emotional reactions associated with HPA and immune dysregulation may ameliorate such effects. For example,

⁶ As a method of addressing this issue, we conducted a post hoc test for the 22 subjective variables that were scored on unipolar scales (1 = *not at all*, 5 = *very much*; the remaining four variables were scored on bipolar scales and hence omitted). Specifically, we examined the percentage of studies that were coded above the scale midpoint of 3.00 and then correlated this percentage with the number of significant ($p < .05$) and marginally significant ($p < .10$) tests. If the correlation was significantly negative, this would indicate that a failure to detect significant relationships is associated with a lack of high levels of specific emotions and appraisals. However, this correlation was very close to zero ($r = -.01$, $p = .96$), thus suggesting that the failure of some dimensions of appraisals and emotions to elicit ratings of very high levels does not generally provide a threat to the validity of our findings. Nonetheless, we recommend that researchers interested in specific emotions and appraisals create laboratory situations designed to directly manipulate these subjective states. Such well-controlled experimentation could provide strong evidence for cause-and-effect relationships.

our data suggest that efforts designed to decrease feelings of submissiveness and the fear of losing social approval in response to status-relevant stressful situations may reduce or obviate excessive cortisol reactivity. Indeed, research suggests that self-esteem, optimistic thinking, and affirmation of one's values may reduce the harmful effects of stressors (Creswell et al., 2005), perhaps because they act to decrease feelings of submissiveness and lessened self-worth associated with threats to the social self. Interventions may also be implemented at the individual level or at the wider societal level. For instance, the promotion of egalitarian norms in broader society may eventually lead to improved health outcomes for low-status individuals. If our and other research findings are incorporated into a complementary, multipronged intervention, such approaches might provide substantial psychological and physical benefits.

Limitations

There were a number of limitations inherent in the current meta-analysis. As mentioned earlier, conclusions drawn from any meta-analysis are limited by the source studies. In the present work, there were simply not enough studies to conduct moderator analyses on all the immune system outcome variables. Specifically, the current work shed no light on the effects of cognitive appraisals and emotions on antibody production and the cytokines. This is unfortunate, as cytokines are the intercellular messengers within the immune system, and they have been implicated in mood alterations (Maier & Watkins, 1998; Robles et al., 2005). Furthermore, we did not assess additional outcomes such as cardiovascular reactivity or sympathetic adrenomedullary hormones. Thus, our meta-analysis is limited in the scope of the effects of emotions on the physiological stress response. Future meta-analyses might expand the integrated specificity approach to additional physiological outcomes. In addition, we did not make an attempt to obtain unpublished manuscripts to reduce file drawer effects.

The current meta-analysis is also limited in the choice of subjective variables that were coded. Although every effort was made to incorporate theoretically relevant cognitive appraisals and emotions, some potentially relevant items were not included. For instance, because most of the studies focused on negative affective states, we chose to include only two positive emotions (happiness and pride). Indeed, very few studies in the stress and emotion induction literature have examined distinct types of positive affect beyond *happy* or undifferentiated positive affect. Therefore, it did not make sense to rate other types of positive affect variables. (Pride was included to complete the set of self-conscious emotions.) Similarly, with the exception of sadness, there were no items assessing unactivated affect (e.g., calm). Future research could explore additional appraisals, positive emotions, and unactivated emotional states that might exert specific effects on cortisol and immune reactivity. We hope that future research will examine understudied appraisals and emotions.

Yet another concern relates to the limitations inherent in our subjective coding procedures. Specifically, we did not obtain materials (e.g., instructions, films, music) beyond the information reported in the method sections of the source articles. Although interrater reliability was generally adequate, these materials may have further increased the accuracy of the judges' ratings and thereby increased statistical power. In ad-

dition, it is important to keep in mind that these coding procedures assessed judges' inferences of appraisals and emotions that were *likely to occur* in the original studies and not the study participants' actual appraisals and emotions. This further highlights our suggestion that future research should directly manipulate specific appraisals and emotions and include manipulation checks to assess the actual levels of induced appraisals and emotions.

Conclusion

The current work broadly supported the integrated specificity model of stress, emotion, and health such that specific emotions and cognitive appraisals were associated with specific effects on cortisol and immunity, whereas broad emotion dimensions evidenced no relation to cortisol and immunity levels. It is important to note that the results here may help clarify why self-report measures of affective valence and general distress tend not to be informative about cortisol and immune outcomes. Future stress research should examine the effects of specific emotions on these outcomes with the ultimate aim of preventing adverse health outcomes. Indeed, the results reported here are intended to inform further experimental work on the effects of specific appraisals and emotions on cortisol and immunity.

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Call for Nominations:

International Perspectives in Psychology: Research, Practice, Consultation

The Publications and Communications (P&C) Board of the American Psychological Association and Division 52 (International Psychology) of the APA have opened nominations for the editorship of ***International Perspectives in Psychology: Research, Practice, Consultation***, for the years 2011–2016. The editor search committee is co-chaired by Lynn Collins, PhD, and Peter Ornstein, PhD.

International Perspectives in Psychology: Research, Practice, Consultation, to begin publishing in 2011, is committed to publishing conceptual models, investigative methodologies, and intervention strategies to help understand, study, and influence the world's major mental health problems. The journal will promote psychological science and practice that is contextually informed, culturally inclusive and serves the public interest. Recognizing that mental health problems are imbedded in economic, environmental, political, and social contexts, ***International Perspectives in Psychology*** is a multidisciplinary title that will incorporate empirical findings from education, medicine, public health, applied and basic psychology, sociology and other related disciplines.

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