When the Social Self Is Threatened: Shame, Physiology, and Health

Sally S. Dickerson and Tara L. Gruenewald
University of California, Los Angeles
Margaret E. Kemeny
University of California, San Francisco

ABSTRACT Our program of research focuses on shame as a key emotional response to “social self” threats (i.e., social evaluation or rejection). We propose that shame may orchestrate specific patterns of psychobiological changes under these conditions. A series of studies demonstrates that acute threats to the social self increase proinflammatory cytokine activity and cortisol and that these changes occur in concert with shame. Chronic social self threats and persistent experience of shame-related cognitive and affective states predict disease-relevant immunological and health outcomes in HIV. Across our laboratory and longitudinal studies, general or composite affective states (e.g., distress) are unrelated to these physiological and health outcomes. These findings support a stressor- and emotional response-specificity model for psychobiological and health research.

Sally S. Dickerson, Department of Psychology and Social Behavior, University of California, Irvine; Tara L. Gruenewald, Department of Medicine\Division of Geriatrics, David Geffen School of Medicine, University of California, Los Angeles; Margaret E. Kemeny, Health Psychology Program and Department of Psychiatry, University of California, San Francisco. Preparation of this paper was supported in part by a Health Psychology Training Grant Pre-doctoral Fellowship from the National Institute of Mental Health, a National Science Foundation Graduate Fellowship, a Ruth L. Kirchstein National Research Service Award, and funding from the National Institute of Mental Health (MH42918). We thank Shelly Gable for her thoughtful comments on previous versions of this paper. Correspondence concerning this article should be addressed to: Sally S. Dickerson, Department of Psychology and Social Behavior, University of California, Irvine, 3340 Social Ecology II, Irvine, CA 92697-7085. E-mail: sdickers@uci.edu.

Blackwell Publishing 2004
A voluminous literature demonstrates that stressors can influence physiological and health outcomes. It has been commonly proposed that affective responses may mediate these associations; key questions, however, remain about the nature of the links between stressors, affect, physiology, and health.

The prevailing notion regarding these relationships can be described as a generality model (Kemeny, 2003). This model follows from the work of Hans Selye (1956), who argued that all stressors activate a generalized physiological “stress” response. Therefore, a variety of stressors (e.g., physical, psychological) would elicit a common set of physiological effects. More recently, affect has been incorporated into the generality model, in that stressful life events are thought to trigger the experience of distress, and distress then alters physiological processes that increase vulnerability to disease. Because generalized distress reactions are examined rather than distinct emotional states (e.g., fear, shame, sadness), this model is “nonspecific” in terms of the relationships posited between physiology, the eliciting conditions of the stressor, and the emotional responses engendered.

There is good reason, however, to suspect the existence of stressor and emotional response specificity with regard to the stressor-affect–health relationship. We have advocated the utility of an integrated specificity model (Kemeny, 2003), which is grounded in several basic premises. First, the nature of the stressor determines the specific psychobiological responses evoked; stressors are not interchangeable. Escaping a predator, coping with irrevocable loss, or maintaining one’s acceptance in a social group all require different sets of psychological, physiological, and behavioral changes to appropriately react under these diverse circumstances. Therefore, distinct patterns of psychobiological responses are necessary to adaptively cope with threats to different goals (e.g., Weiner, 1992).

Second, emotions are thought to play a key role in orchestrating these coordinated responses to different goal threats. Specific threats elicit prototypical emotional states; for example, one can experience fear when one’s safety or survival is threatened, sadness following the death of a loved one, and shame in response to social rejection. These emotions are associated with experiential, behavioral, and physiological components that provide a coordinated, adaptive response to these specific threats and/or opportunities (e.g., Ekman,
If different goal threats elicit specific patterns of emotional and physiological changes, it follows that discrete emotions may have distinct physiological correlates. Emerging data support this premise. For example, brain-imaging studies have demonstrated that specific emotions are associated with different patterns of central nervous system activity (e.g., Canli et al., 2001; Damasio et al., 2000; Lane, Reiman, Ahern, & Schwartz, 1997). The induction of different emotions can result in distinctive patterns of activation in peripheral neural systems such as the autonomic nervous system (e.g., Ekman, Levenson, & Friesen, 1983). These findings make sense when one considers that emotions “do different things” and thus require differentiated neurophysiological changes to support relevant actions and shifts in goals.

This integrated specificity approach is often illustrated with threats to the central goal of physical self-preservation (e.g., safety, survival), which can trigger the emotion of fear. Fear is thought to organize the behavioral and physiological changes necessary to address this survival threat. These changes could include a shift in the animal’s motivational state from finding food to finding an avenue of escape, increases in vigilance to threat-related cues, and focused attention on identifying and utilizing available resources. In concert with these changes, the sympathetic nervous system can become activated, increasing heart rate and respiratory rate in preparation for physical exertion. While this pattern of psychobiological changes may be optimal when physical self-preservation is threatened, threats to other central goals (e.g., social acceptance/inclusion) may elicit their own pattern of psychological, physiological, and behavioral responses that would be adaptive under those specific conditions.

Our program of research has focused on the emotional, physiological, and health consequence of threats to the central goal of preserving one’s “social self.” Our social self preservation model (Kemeny, Gruenewald, & Dickerson, 2004) proposes that threats to one’s social self (i.e., threats to one’s social esteem, status, and acceptance) are accompanied by a specific set of psychological and physiological responses. These include increases in shame (and other negative self-evaluative states), proinflammatory cytokine activity, and cortisol. We argue that these changes are not simply epiphenomena, but instead, that shame and accompanying physiology are
integral components of a coordinated psychobiological response to threats to social self preservation, just as fear and its physiological correlates are components of the response to threats to physical self preservation.

Although threat-specific emotional and physiological changes may be functional in an acute context, extreme or persistent experience of specific threats and corresponding patterns of psychobiological responses may have negative health consequences. Individual difference factors that increase one’s vulnerability to experience a specific emotional response, or that amplify threat-induced emotional reactions, may potentiate the adverse physiological effects of threat experiences. For example, chronic or repeated exposure to evaluative, rejecting conditions (i.e., social self threats), and accompanying increases in shame, proinflammatory cytokine activity, and cortisol, could lead to negative health outcomes. This may be particularly likely among individuals with dispositions that increase their vulnerability to experiencing shame-related cognitions and emotions. Therefore, the specific set of emotional and physiological responses that are persistently triggered, and the vulnerability factors that may increase such responses, may determine the pathways through which these health effects would occur. Examining specific emotional and physiological responses to specific eliciting conditions might elucidate coordinated patterns of psychobiological changes that could be health-relevant.

In this article, we will present evidence that shame may be a key affective component of a coordinated psychobiological response to threats to the social self. We will first review evidence that shame is elicited when the social self is threatened, and may be associated with motivational and behavioral changes that are adaptive in these contexts. Next, we will present studies from our laboratory that demonstrate that acute threats to the social self are associated with changes in specific physiological parameters, which may occur in concert with the emotion of shame. Finally, we will describe a series of studies in the chronic disease model of HIV. This research demonstrates that chronically experiencing social self threats or accompanying negative self-related cognitions and emotions predict negative long-term immunological and health effects. In describing our program of research, we hope to underscore how a specificity approach can reveal distinctive relationships among eliciting conditions, emotions, physiology, and health processes, leading to a more
comprehensive understanding of the pathways through which psychological states can influence health and disease.

**Threats to the Social Self and Shame**

Threats to the social self are situations that provide the potential for a loss of social esteem, social status, or social acceptance and are characterized by potential or explicit rejection. Prototypical threats to the social self are conditions in which an important aspect of the self-identity is, or could be, negatively judged by others (i.e., “social-evaluative threat”; Dickerson & Kemeny, 2004). Social-evaluative threat can occur in a variety of situations: in performance contexts that require displays of valued attributes, traits, or abilities in the presence of others (e.g., competence, intelligence), in rejection-laden contexts where one is (or could be) judged unworthy of acceptance or group membership, or in contexts where an uncontrollable characteristic or an unwanted identity is made salient (e.g., stigmatizing condition).

Shame may be a key affective response to social-evaluative threat, based in part on evidence that this emotion is preferentially elicited under these conditions.\(^1\) Shame is a self-conscious emotion, experienced when a core aspect of the self is judged as defective, inferior or inadequate (Gilbert, 1997; Tangney, 1995), and “is provoked by the realization that others consider one’s self deficient” (Schott, 1979, p. 1325). Negative self-evaluation is central to shame; this emotion is associated with negative characterological self-related cognitions, and its motivational and behavioral correlates are indicative of a devalued or damaged self (e.g., submission, withdrawal). Therefore, shame results when perceptions of negative *social* evaluation are transformed into negative *self*-evaluation.

The premise that shame is predicated on social evaluation is not new, but has had a prolific history within the scientific literature on emotion. Early influential theorists, such as Darwin (1871/1899), believed that this emotion “relates almost exclusively to the judgment of others” (p. 114). The symbolic interactionists of the early

---

\(^1\) Other emotions in the shame family, such as embarrassment, are also likely to be experienced in response to social self threat. Embarrassment, like shame, involves flaws in self-presentation (Schott, 1979), and these emotions are likely to be experienced together. However, following Gilbert (1997), we view shame as a prototypical emotion elicited under these specific forms of social threat.
20th century followed in this tradition, stating “there is no sense of ‘I,’ as in pride or shame, without its correlative sense of you, or he, or they” (Cooley, 1902/1983, p. 182). Others have also recognized that negative social evaluation is fundamental to shame, arguing that a characterological flaw or failure must be *publicly* revealed to elicit this emotion (e.g., Ausubel, 1955; Benedict, 1946; Buss, 1980; Gehm and Scherer, 1988). Even when the audience is not explicit, shame is still thought to originate from social sources, and is elicited when individuals evoke an image of a disapproving “imagined other” that negatively evaluates the self (Lewis, 1971). This rich theoretical tradition resonates with contemporary empirical evidence that negative social evaluation and appraisals of interpersonal rejection elicit the emotion of shame (e.g., Keltner & Buswell, 1996; Leary & Baumeister, 2000; Smith, Webster, Parrott, & Eyre, 2002; Tangney, 1995; Tangney, Miller, Flicker, & Barlow, 1996).

The motivational state, nonverbal displays, and behaviors associated with shame all support the premise that it is related to submission and withdrawal. When asked to recall shame-eliciting events, individuals report wanting to “hide,” “escape,” “disappear from view,” and “shrink into the floor,” during the experience, indicating the desire to flee the social situation and conceal the “defective” self from social scrutiny (Tangney et al., 1996; Wicker, Payne, & Morgan, 1983). Shamed individuals also show body postures that signal a submissive strategy. The characteristic nonverbal display associated with shame includes head down, slumped posture, and eye gaze avoidance (Gilbert, 2000; Keltner, 1995), which are similar to some of the displays that denote submission in primates. Thus, the motivational state and behavioral displays associated with shame indicate withdrawal and disengagement.

Theorists have argued that submissive displays and withdrawal behaviors are social signals that function as an appeasement strategy to reduce social conflict. Research in primates and other animals demonstrates that submissive displays serve as social communication that the animal is not going to contest resources or escalate conflict. This could signal to the attacker to de-escalate and not induce serious harm, which could facilitate control over aggression and maintain social cohesion (Gilbert, 2000; MacLean, 1990). Keltner and colleagues (Keltner, 1995; Keltner, Young & Buswell, 1997) have found that shame has similar appeasement functions in humans. They argue that shame occurs when social rules are violated that
could potentially disrupt social relations and lead to a heightened risk of conflict and aggression. Submissive displays indicative of shame evoke emotions and behavior from the interaction partner(s) that elicits cooperation and reductions in aggressive or punitive behavior (Keltner et al., 1997); therefore, submissive behavior could help maintain social cohesion and reduce overt hostility in the face of social threat.

Taken together, this theoretical and empirical evidence suggests that shame may be more than a non-functional affective consequence of social-evaluative conditions, but may be an integral component of a coordinated response to social self threats. This emotion is elicited under social-evaluative conditions and is sensitive to gradations in negative interpersonal appraisals. Shame may play a role in organizing motivational and behavioral changes that could be functional in the face of an antagonistic social encounter, namely promoting changes associated with withdrawal and disengagement and nonverbal behavioral displays denoting submission or appeasement. Finally, it is possible that shame could orchestrate physiological changes that may be adaptive in response to social self threats.

Physiological Responses to Social Self Threats

We propose that social self threats may provide one set of conditions that can lead to increased activity of the cortisol and proinflammatory systems, and further, that these changes may occur in concert with the shame family of emotions. In the following section, we provide an overview of these two systems, and review evidence that they are elicited in response to social-evaluative threat and may be co-activated with shame.

Cortisol

Cortisol, a hormone that is an end product of the activation of the hypothalamic-pituitary-adrenocortical (HPA) axis, plays an important regulatory role in normal physiological functioning. Hypothalamic neurons release corticotropin-releasing hormone (CRH), which in turn stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH). ACTH then triggers the release of cortisol from the adrenal cortex into the bloodstream, where it exerts effects on a number of physiological systems. For example, cortisol plays a primary role in metabolism; it releases energy stores
(primarily by elevating blood glucose levels), which can then provide metabolic “fuel” for the body. Cortisol can regulate other physiological systems; for example, cortisol can inhibit aspects of immune functioning and permits other systems, like the sympathetic nervous system, to function effectively.

In addition to its role in maintaining ongoing physiological processes, the HPA system can also be activated in response to certain stressful circumstances. This is thought to be adaptive, presumably because cortisol can mobilize energy resources and activate other physiological systems that are necessary to overcome a threat (Lovallo & Thomas, 2000; Sapolsky, Romero, & Munck, 2000). However, the HPA system does not appear to be activated indiscriminately in response to all negative stimuli; instead, stress-induced elevations in cortisol may be linked to the eliciting conditions or specific characteristics of the stressor (Blascovich & Mendes, 2000; Dickerson & Kemeny, 2004; Mason, 1968). There is growing evidence that one class of events that can be potent elicitors of HPA responses is threats to the social self.

In a meta-analytic review of 208 acute laboratory stressor studies, we tested whether threats to the social self would be associated with substantial changes in cortisol (Dickerson & Kemeny, 2004). We proposed that stressors with social-evaluative threat, in which others could negatively judge the self (e.g., evaluative audience present), would elicit greater cortisol responses than tasks without this component. We found strong support for this hypothesis; tasks in which others could negatively evaluate performance were associated with substantially greater cortisol responses (effect size $d = 0.67$) compared to tasks without this social-evaluative component ($d = 0.21$). These findings remained significant when controlling for other methodological factors that predicted cortisol changes (e.g., time of day, timing of cortisol assessment).

Additionally, cortisol responses were further heightened when social-evaluative threat was coupled with uncontrollability, in which participants could not succeed despite their best efforts. Social-evaluative, uncontrollable conditions induce a potent threat to the social self; not only can others evaluate performance, but failure is the likely outcome. These conditions of exposed failure elicited the largest cortisol responses observed in these laboratory studies ($d = 0.92$); this effect was nearly three times as large as tasks with one component alone (i.e., only uncontrollability or only social-evaluative
threat; \( d = 0.32 \) and \( 0.35 \), respectively). In addition, these social-evaluative, uncontrollable tasks were associated with a slower recovery of cortisol to baseline levels. Overall, the meta-analysis provides strong support for the premise that social-evaluative contexts that threaten the social self can elicit substantial, persistent changes in the cortisol system.

A subsample of studies included in the meta-analysis reported negative affective assessments pre- and poststressor. We calculated effect sizes on these affective measures to test whether generalized distress was associated with cortisol responses (Dickerson & Kemeny, 2004). Overall, the social-evaluative tasks were not more distressing than those without this evaluative component. Further, changes in general negative affect or distress were unrelated to the magnitude of cortisol responses. This provides support for the premise that more generalized emotional states may not be related to changes in the HPA system. As no studies in the meta-analysis assessed shame, we could not test whether this more specific emotion was associated with cortisol changes.

In a subsequent laboratory study, we experimentally tested the premise that tasks with social-evaluative threat would be more likely to activate the HPA and to induce the experience of shame compared to tasks without this evaluative component (Gruenewald, Kemeny, Aziz, & Fahey, in press). Participants were randomly assigned to give a speech and complete a computerized math task, with elements of uncontrollability, either alone or in front of an evaluative audience. There were no differences between the social evaluation and nonsocial evaluation conditions on post-task ratings of difficulty or anxiety. However, participants in the social-evaluative condition reported significantly more shame and other self-conscious emotions than those performing the same tasks alone. Additionally, it was only the social-evaluative threat condition that induced robust increases in cortisol from pre- to poststressor; those performing the identical tasks without an audience present showed no changes on average in cortisol from pre- to posttask. Consistent with our theoretical premise, increases in shame and cortisol occurred in concert under social-evaluative conditions; the experience of shame and other self-conscious emotions may be the “active ingredient” that led to cortisol responsivity within the social-evaluative condition.

We recently demonstrated a concordance between shame and cortisol in a study examining the avoidance achievement motive “fear
of failure” (Dickerson & Gable, 2004). Because this trait has been theoretically linked with the propensity to experience shame (e.g., Atkinson, 1957; Elliot & Thrash, 2004), we examined how fear of failure influenced shame and cortisol following an evaluative performance task. Consistent with hypotheses, we found that individuals high on this trait experienced more shame and had higher cortisol levels poststressor than those lower on this trait (Dickerson & Gable, 2004). However, measures of general negative emotion or other specific emotions (e.g., anxiety) could not differentiate those high and low on fear of failure. These findings suggest that individual difference factors that affect one’s vulnerability to experience shame also influence the cortisol system, and support the premise that shame and cortisol may be co-activated under certain evaluative conditions.

Other studies have found that the behavioral displays that accompany shame have been associated with cortisol changes. Children who displayed more shame and embarrassment behaviors during a series of laboratory tasks had greater cortisol changes during the session than those with other nonverbal behavioral styles (Lewis & Ramsay, 2002). Nonverbal shame behaviors observed in the children included head down, slumped posture, and eye gaze avoidance—behaviors quite similar to those that denote submission in nonhuman primates and other social animals (Keltner, 1995; Gilbert, 1997). It is particularly interesting that specific forms of social threat in animals (i.e., social defeat, subordination) are typically associated with concurrent increases in cortisol (e.g., Sapolsky, 1993; Shively, Laber-Laird, & Anton, 1997), and that the frequency of submissive behavioral displays correlate with cortisol activity (Haller, Kiem, & Makara, 1996; Shively et al., 1997). Threats to social status and submissive behavior may provide the animal analogue to social self threat and shame, and these processes may have been maintained and elaborated in humans (Gilbert, 1997; Price, Sloman, Gardner, Gilbert, Rohde, 1994).

Taken together, there is growing support that conditions that threaten the social self (i.e., social evaluation and rejection) can elicit cortisol responses. Several studies have shown a concordance between the conditions and vulnerability factors that specifically increase the shame family of emotions and the conditions and vulnerabilities that increase cortisol, while this relationship does not exist for more general negative emotional states or other specific
emotions. These findings suggest that shame and cortisol could be two parts of a coordinated response to social self threats.

Proinflammatory cytokine activity

Cytokines are mediators of the immune response. They are chemical communication molecules released from immune cells that alter the function of other cells. Cytokines play an important role in cell-to-cell communication among components of the immune system. Produced by lymphocytes and monocytes/macrophages, cytokines have a number of different functions including promoting cell growth, inhibiting viral replication, and promoting local responses in the tissue.

A subset of cytokines, called proinflammatory cytokines, initiate and maintain the inflammatory response. Inflammation is a coordinated bodily response to tissue injury or pathogen infection, which involves immune cells and their products entering the infected area, destroying the organism, and repairing injured tissue. Proinflammatory cytokines, such as tumor necrosis factor-α (TNFα), interleukin-6 (IL-6), and interleukin-1β (IL-1β), orchestrate the activities of different types of immune cells involved in inflammation. They also are responsible for initiating the processes that lead to signs of inflammation (e.g., redness, pain, edema).

There is a dynamic interaction between proinflammatory cytokines and cortisol; however, the exact nature of this relationship is complex. While cortisol can inhibit proinflammatory cytokine production, proinflammatory cytokines can activate the HPA axis. Additionally, both systems can be activated by other inputs, like the sympathetic nervous system, so it is possible to have elevations in both proinflammatory and HPA activity simultaneously. In fact, this is the case under certain situations; the systems can be co-activated in response to infection or in response to physical threats. One of the goals of our program of research is to test if threats to the social self provide another set of conditions that can activate these two systems in humans.

2. Recent studies have demonstrated that social stress in animals and humans can lead to glucocorticoid resistance (Rohleder, Schommer, Hellhammer, Engel, & Kirschbaum, 2001; Avitsur et al., 2001). Reduced downregulation of proinflammatory cytokine production in the presence of glucocorticoids following social stress could explain simultaneous elevations in these systems.
While biological stimuli (e.g., pathogens, injury) are most commonly associated with activation of the proinflammatory cytokine system, recent evidence from animal models and preliminary work in humans suggests that certain psychological states and psychological stressors can be linked to heightened inflammatory activity as well (e.g., Watkins, Nguyen, Lee, & Maier, 1999). Evidence for a relationship between psychological states and inflammatory processes stems primarily from studies of clinical depression. In some studies, individuals with major depression have shown increased levels of proinflammatory cytokines relative to non-depressed controls (e.g., IL-1 and 6; for review, see Maes, 1999). These differences have emerged even in carefully matched samples, which control for medical condition, medication, health behaviors, and other confounding factors (e.g., Miller, Stetler, Carney, Freedland, & Banks, 2002). There is also evidence that pharmacologic treatment for depression is associated with a reduction in proinflammatory cytokine levels in clinically depressed patients (Kenis & Maes, 2002).

In animals, research has documented a link between stressor exposure and activation of proinflammatory cytokines. Specifically, these cytokines appear to be produced in the context of social threats. The primary model of social threat utilized in this literature is social disruption stress, in which a very aggressive intruder is placed in the home cages of other animals, resulting in social defeat and subordination in the home-caged animals. Animals who are socially defeated show greater production of proinflammatory cytokines (Il-6, Il-1β and TNFα) compared to control animals that were not socially defeated (Stark et al., 2001; Stark et al., 2002; Avitsur, Stark, & Sheridan, 2001; Quan et al., 2001). This form of social stress can induce other immunological changes that are consistent with greater inflammation, such as increased numbers of circulating granulocytes and levels of nerve growth factor (Avitsur et al., 2001; Stefanski & Engler, 1998). Thus, specific forms of social threat (i.e., social subordination and defeat) cause increases in inflammatory processes in animals.

Less research has focused on the relationship between stressors and proinflammatory cytokine activity in humans. While some studies have investigated how stressful situations influence inflammatory responses, recent narrative reviews have highlighted the inconsistent effects in this area of research (Kronfol & Remick, 2000; Watkins et al., 1999). However, some of the contradictory findings can be
reconciled when the social-evaluative nature of the task is considered. A number of different types of laboratory stressors have been used in these studies; those that utilize social evaluative tasks (e.g., public speaking) have shown increases in proinflammatory cytokine activity (e.g., Ackerman, Martino, Heyman, Moyna, & Rabin, 1998; Altemus, Rao, Dhabhar, Din, & Granstein, 2001; Goebel, Mills, Irwin, & Zeigler, 2000). Those that used other tasks (e.g., watching a film, completing a Stroop task) have not consistently found increases in inflammatory parameters (e.g., Dugue, Leppanen, Teopp, Fyrquist, & Grasbeck, 1993; Peters et al., 1999; Zakowski, McAllister, Deal, & Baum, 1992). While tentative, this suggests that social-evaluative conditions may have a greater capacity to elicit proinflammatory cytokines in humans than the other types of laboratory stressors examined thus far.

We recently tested whether the negative self-related cognitions and emotions that can accompany these social-evaluative threats are associated with inflammatory processes (Dickerson, Kemeny, Aziz, Kim, & Fahey, 2004). We randomly assigned participants to conditions in which they wrote about an experience of self-blame or a neutral topic on three separate days over a week period. Participants in the self-blame condition wrote about a variety of stressful situations, including experiences of rejection and failing to live up to parental expectations. The self-blame induction elicited greater increases in shame and guilt than other emotions measured, and this increase was observed on each of the three experimental days. In addition, the receptor for TNF-α (sTNF-RII), a marker of proinflammatory cytokine activity, significantly increased from pre- to post-writing across the three days among the individuals in the self-blame condition, while there were no changes for those in the control condition. Furthermore, among the participants in the self-blame condition, those who reported the greatest increases in shame also showed the greatest increases in proinflammatory cytokine activity, but guilt, anger, anxiety, sadness, and general negative emotion were unrelated to this parameter. Together, these findings suggest that the experience of self-blame and self-related emotions can elicit changes in proinflammatory cytokine activity and that there could be a specific relationship between shame and inflammatory markers.

In the context of social self threat, this association between specific psychological states and proinflammatory cytokine activity may
function to promote disengagement and withdrawal. Recent evidence has demonstrated a bidirectional relationship between behavior and inflammatory processes. Psychological states and other stimuli can lead to alterations in proinflammatory cytokines, and proinflammatory cytokines, in turn, can exert central effects that influence cognition, affect, motivation, and behavior. For example, these bidirectional relationships can be seen in the context of infectious illness. In response to a pathogen, animals release proinflammatory cytokines, which can have local immune effects. Additionally, these cytokines can act on the brain to orchestrate processes directly related to fighting infection (e.g., fever) and to induce a constellation of behavioral changes called “sickness behavior.” This behavioral pattern includes reductions in exploratory, sexual, and social behavior, immobility and locomotor retardation, and decreases in food and water intake—changes that are associated with disengagement and withdrawal from the environment (Dantzer et al., 2001; Hart, 1988). These behavioral changes are known to result from the activity of proinflammatory cytokines because peripheral injections of these proteins can induce sickness behaviors in healthy, uninfected animals and humans (e.g., Dantzer et al., 2001; Yirmiya et al., 1999). Therefore, the release of proinflammatory cytokines can lead to motivational and behavioral changes reflecting disengagement or withdrawal.

This type of behavioral disengagement may be adaptive under certain conditions or in response to certain stimuli. Cytokine-induced sickness behavior is believed to be an adaptive response to infection because it reduces energy expenditure (by reducing activity) so energy resources can be diverted to mounting an effective response to the pathogen (Maier & Watkins, 1998). We have proposed that uncontrollable, social-evaluative threats may provide another set of conditions under which activation of the proinflammatory cytokine network and subsequent behavioral disengagement may also be adaptive (Kemeny et al., 2004). In contrast to controllable situations of threat to the social self, where employing active behavioral responses could successfully circumvent adverse social outcomes, uncontrollable threats to the social self provide a context where active social responses may be ineffective and, in some cases, even exacerbate conflict. Therefore, disengagement, submission, and withdrawal may be an adaptive response to uncontrollable, social-evaluative threat.
Research suggests that these uncontrollable, socially threatening conditions may result in the release of proinflammatory cytokines and elicit phenomenological and behavioral responses indicative of withdrawal or disengagement. The central effects of proinflammatory cytokines may facilitate these behavioral changes. Specifically, these cytokines may support the withdrawal and disengagement behaviors that are observed in animals under social threat (e.g., the subordinate animal) and in humans experiencing shame in response to social self threats.

Summary: Psychological and physiological responses to social-evaluative threat

The evidence presented demonstrates that social evaluative contexts—those in which an aspect of the self could be judged negatively by others—may be accompanied by specific psychobiological changes. A growing body of research in the self-related processes and emotion literature indicates that shame may be experienced particularly in conditions characterized by negative social evaluation and rejection. The cortisol and proinflammatory cytokine systems also appear to be responsive to social-evaluative threat. While tentative, there is support for the notion that the activation of these systems under the very specific condition of threat to the social self may hinge on the experience of shame and related emotions. Furthermore, it is possible that this pattern of psychobiological changes represent an integrated response, which is adaptive in socially threatening, uncontrollable conditions.

Social Self Threats, Self-Evaluations, and Health

The pattern of psychobiological responses detailed above may be adaptive in short-term situations of uncontrollable social threat; however, repeated or prolonged exposure to social self threats could place individuals at risk for adverse psychological and physical health outcomes. While little research has examined specific health effects of chronic threat to the social self, the cognitive, affective, and physiological sequelae of such threats may be important pathways to adverse health outcomes. Chronic or persistent shame experiences may have implications for mental health; shame is thought to be a central component of a number of psychological conditions, including depression (Lewis, 1971; Scheff, 2001), social anxiety (Gilbert &
Trower, 1990; Schwarzer, 1986), and suicidal ideation (Mokros, 1995). In addition, the tendency to easily experience shame, as well as more frequent shame experiences, has been shown to predict depressive symptoms cross-sectionally (Tangney, Wagner, & Gramzow, 1992) and prospectively (Andrews, Qian, & Valentine, 2002).

Chronic or repeated activation of the HPA and proinflammatory cytokine network may increase susceptibility to disease or moderate disease course (e.g., McEwen, 1998; Kronfol & Remick, 2000). Elevated basal cortisol levels may increase susceptibility to disease incidence and progression through suppression of some components of immunity (Munck, Guyre, & Holbrook, 1984). Increased activation of the cortisol and inflammatory systems has been implicated in a number of disease conditions, including the metabolic syndrome (Brunner et al., 2002; Ford, 2003) and major depression (Connor & Leonard, 1998; Gold, Licinio, Wong, & Chrousos, 1995; Maes, 1999; Maes et al., 1995). Accumulating evidence also associates heightened inflammatory processes with the incidence and progression of cardiovascular disease (Black & Garbutt, 2002; Danesh, Collins, Appleby, & Peto, 1998; Danesh et al., 2000; Pasic, Levy, & Sullivan, 2003), HIV infection (Kedzierska, Crowe, Turville, & Cunningham, 2003), and other chronic inflammatory diseases (e.g., rheumatoid arthritis; Feldmann, Brennan, & Maini, 1996). Elevated levels of the proinflammatory cytokine IL-6 in elderly individuals have been shown to predict increased mortality over extended follow-up periods (Harris et al., 1999). Thus, there are multiple pathways through which these physiological systems, which we propose are activated under conditions of threat to the social self and the experience of shame, may influence health outcomes.

In a series of studies, we have examined how individual differences in sensitivity to social self threats, and the self-related cognitions and emotions that can accompany these threats, predict immunological and health outcomes in a specific disease model—that of HIV infection. This is an ideal model in which to examine physical health correlates of repeated or prolonged threats to the social self. The adults we have studied have a life-threatening, stigmatizing disease, and, in most of our samples, are gay or bisexual, a stigmatizing sexual orientation. Therefore, they are subject to chronic threat to the social self and must navigate their way through a world that is often unaccepting, and sometimes explicitly hostile, regarding a core, uncontrollable aspect of their identity. Additionally, HIV/AIDS is
characterized by a lengthy but variable progression and known immunologic and virologic markers prognostic of accelerated disease course and mortality (e.g., CD4 T-cell decline, increased viral load; Fahey et al., 1990; see also Cole & Kemeny, 2001, for overview); this also makes this an excellent model in which to examine stress-affect–physiology relationships.

In one line of research, we have found that individuals who are particularly sensitive to social self threats, especially social rejection, are more likely to evidence poorer HIV-specific immunological and health outcomes. In a 9-year longitudinal study, Cole and colleagues found that individuals who were particularly sensitive to rejection based on their homosexual identity showed faster CD4 T-cell declines (an important marker of HIV progression) and faster times to AIDS diagnosis and death, compared to those lower on this trait (Cole, Kemeny, & Taylor, 1997). The results were quite striking: highly rejection-sensitive individuals died on average 2 years before those less sensitive to negative evaluation and rejection. These effects were observed in a carefully selected group of HIV-positive individuals who were healthy at baseline and when controlling for a variety of confounding factors (baseline immune status, demographics, health behaviors, medication usage, etc.).

In a separate sample of HIV-positive gay men, rejection sensitivity predicted elevated HIV viral load and poorer virologic and immunological outcomes over time (Cole, Kemeny, Zach, Fahey, & Naliboff, in press). The sample included men who were not on the highly effective highly active antiretroviral therapy (HAART) medication regimen at the baseline point of the study. All were assessed for immunological and virological status at baseline and one year later, during which time they initiated HAART therapy. Those high on rejection sensitivity showed a weaker response to the medication; the beneficial reductions in viral load and increases in CD4 T-cells were significantly less pronounced in the rejection-sensitive men. These findings suggest that a heightened sensitivity to negative social evaluation is associated with physiological states that may have profound consequences for disease progression and mortality.

We have also found parallel associations between perceptions of rejection and immunological declines in a multi-ethnic sample of HIV-positive women (Lewis, Kemeny, Myers, & Wyatt, 2004). In this study, specific components of depression (as measured by the CES-D) were predicted to be differentially associated with
immunological outcomes. Consistent with theory, the interpersonal rejection factor predicted CD4 T-cell declines over a 2-year period, whereas other components of depression, such as vegetative symptoms or depressed affect, did not (controlling for relevant confounding factors). In other words, strong endorsement of items such as “I felt people disliked me” or “people were unfriendly,” was associated with poorer immunological outcomes, whereas strong endorsement of items such as “I felt sad” or “my sleep was restless” was unrelated to CD4 T-cell decline. Taken together, these investigations indicate that perceptions of social rejection—or a heightened sensitivity to social rejection—are associated with immunological states indicative of poorer disease prognosis, as well as HIV health outcomes and mortality.

Several other studies from our research group have demonstrated that the negative self-related cognitions that can be elicited in response to social self threats are associated with immune declines. HIV-positive gay or bisexual men who characteristically blamed themselves for negative events (i.e., made negative characterological attributions) had faster declines in CD4 T-cells than those without this self-blaming attributational style over a 1 1/2-year follow-up period (Segerstrom et al., 1996). In a separate sample of HIV-positive, gay men, the self-reproach aspect of depression predicted CD4 T-cell declines (Kemeny & Dean, 1995), whereas other components that lacked a self-blame component (e.g., depressed mood, vegetative symptoms) were not associated with immunological change. These studies suggest that these specific cognitions, centered on self-blame, are associated with negative immunological outcomes in the context of HIV.

In all of our studies, the findings remain significant when controlling for a host of biobehavioral variables (e.g., demographics, health behaviors) as well as more general personality variables (e.g., neuroticism). Furthermore, general affective states, such as negative affectivity and depressed mood, do not explain the relationship between sensitivity to social self threats, negative self-related cognitions, and HIV-related outcomes. This is parallel to the results obtained in our experimental studies, in which distress or general negative emotion were unrelated to physiological parameters. However, as reviewed above, rejection sensitivity, perceptions of rejection, negative characterological attributions, and self-reproach can all elicit shame (e.g., Leary & Baumeister, 2000; Weiner, 1985); thus,
it is possible shame may underlie associations between these constructs and HIV outcomes.

To more specifically examine the relationship between shame and immunological outcomes in HIV, we tested whether chronically experiencing different specific negative emotions was associated with immune declines among HIV-positive gay and bisexual men. We found that persistent experience of shame and guilt surrounding HIV infection predicted CD4 declines over a 7-year follow-up, while other HIV-specific and general emotions, such as anxiety, anger, sadness, and negative affectivity, did not (Weitzman, Kemeny, & Fahey, 2004). Ancillary analyses indicated that this relationship hinged on persistent feelings of shame. These findings support the premise that shame, experienced in response to possession of a stigmatizing condition, is an important predictor of disease-relevant immunological change. It is plausible that shame may be an important affective component of previous findings linking individual differences factors and negative self-related cognitions to immunological parameters as well.

Taken together, these findings suggest that how individuals perceive and respond to social self threats are associated with disease progression and mortality in HIV-infected individuals. The immunological and virological changes assessed in our investigations are natural consequences of the course of HIV infection. However, our research suggests that individual difference factors and cognitive responses relevant to social-evaluative threat, as well as shame-related affective responses, may moderate the course of HIV disease progression. Thus, this work supports the potential importance of social self threats, and their cognitive and affective consequences, on physical health.

Conclusion

Our research is guided by our theoretical model that posits that conditions characterized by social evaluation or rejection, or those that threaten the social self, elicit a coordinated psychobiological response. We have focused on shame as a key affective component of this response, which may orchestrate specific patterns of physiological and behavioral changes under these conditions. Support for this model stems from our laboratory studies which demonstrate that acute social self threats increase proinflammatory cytokine activity
and cortisol, and these changes occur in concert with shame. In other work, we have shown that chronic threats to the social self and persistent feelings of shame-related cognitions and emotions predict disease-relevant immunological and health outcomes in the chronic disease model of HIV. These two areas of research link together because extensive research has documented that the proinflammatory cytokines IL-1β, TNF-α and IL-6 stimulate HIV replication and predict disease progression (Kedzierska et al., 2003).

Across our laboratory and longitudinal studies, we have found associations between shame and related self-evaluative states and physiological and health outcomes; however, more general affective states, such as distress, have been unrelated to these parameters. This is consistent with an integrated specificity approach (Kemeny, 2003), in which specific eliciting conditions are associated with distinct patterns of emotional and physiological changes. The shame family of emotions is vastly understudied in psychobiological research, and only a handful of studies have examined its physiological or health correlates. However, our findings suggest that shame may be important for elucidating pathways through which certain stressful conditions influence physiological responses and health outcomes.

REFERENCES


