

The Psychobiology of Trait Shame in Young Women: Extending the Social Self Preservation Theory

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Objective: The social self preservation theory (SSPT) proposes that social evaluative threat evokes the emotion of shame, which then shapes a coordinated psychobiological response. While this is supported in acute stress studies, there is no data on chronic experiences of shame. **Design:** We investigated the association of trait shame with activity of the hypothalamic-pituitary-adrenal (HPA) axis, of the sympathetic nervous system (SNS), and regulation of inflammation in $n = 56$ young women. **Main Outcome Measures:** Daily profiles of salivary cortisol and alpha-amylase were assessed as indices of HPA axis and SNS activity, respectively. Inflammatory regulation was assessed by lipopolysaccharide-stimulated production and glucocorticoid inhibition of interleukin-6 in vitro. **Results:** Trait shame was associated with SNS ($r = .49; p < .001$), but not HPA activity ($r = .14; ns$). Shame was associated with inflammatory activity ($r = .35; p = .006$) and glucocorticoid sensitivity ($r = -0.43; p = .001$). Relationships were not mediated by HPA and SNS activity. **Conclusions:** Results support SSPT predictions with respect to chronic shame experience and inflammation. Results further suggest the importance of SNS activation related to shame, and the possibility that HPA activation may be limited acute experiences of shame.

Keywords: social self preservation theory, shame, social evaluative threat, inflammatory activity, glucocorticoid sensitivity

Growing efforts have been made to delineate the psychological antecedents of neuroendocrine and immune responses to stressful experiences. Early theories postulated that biological responses to life stress were nonspecific and did not differ depending on the nature of the situation (e.g., Selye, 1956). More recently, some researchers have proposed that stressors elicit distinct cognitive, emotional, and biological responses, each of which has evolved to meet specific demands from the environment (Dickerson, Gruenewald, & Kemeny, 2004; Levenson, 1994; Weiner, 1992). For example, threats to the physical self are known to activate fearful emotions and the sympathetic nervous system, and thereby give rise to a fight-or-flight response that facilitates survival. Dickerson, Gruenewald, et al. (2004) extended this theory to social threats, arguing that these threats elicit the emotion of shame, which in turn shapes a coordinated psychobiological response, consisting of activation of the hypothalamus pituitary adrenal

(HPA) axis and peripheral pro-inflammatory activity. Their model is known as the “social self preservation theory” (SSPT).

Shame and Cortisol Response to Acute Stress

Dickerson, Gruenewald, et al (2004) have presented evidence for the view that social-evaluative threat is a key component in activating the HPA axis. In a meta-analysis of studies investigating acute stress responses in humans, Dickerson & Kemeny (2004) showed that laboratory stressors that included a social-evaluative component produced the greatest activation of the HPA axis (Dickerson & Kemeny, 2004). The highest cortisol responses occurred in stress paradigms in which social evaluation was high and controllability was low. Although none of the studies had included a specific measure of shame, cortisol responses were generally unrelated to other negative emotions, suggesting that it may in fact be necessary to evoke a specific type of emotion such as shame to elicit HPA reactivity. In accordance with these findings, we were able to show in a recent study that cortisol responses increased in real-life social-evaluative situations (competitive ballroom dancing) compared to a control (noncompetition) day, that training days did not evoke similar increases in cortisol, suggesting the importance of the evaluation component to the cortisol response. We further showed that HPA axis activation was lower during group dancing competitions, as compared to couple competitions, suggesting that the greater the social evaluative focus on any one individual, the greater the cortisol response. Unfortunately, however, we were not able to include a measure of shame in this study (Rohleder, Beulen, Chen, Wolf, & Kirschbaum, 2007).

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A limited number of studies on acute stress have included measures of shame. In a study by Gruenewald, Kemeny, Aziz, and Fahey (2004), a modified version of a public speaking and mental arithmetic stressor, the Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993), was performed in the presence and absence of an audience. Increases in shame ratings were significantly higher in the presence of an audience, and cortisol increased only in the presence of an audience. Cortisol increases were associated with increases in shame and decreases in self-esteem, but not with anxiety (Gruenewald et al., 2004). In another study of 4-year-old children, Lewis and Ramsay (2002) reported a weak and nonsignificant association of shame-associated behavior with adjusted cortisol increases during color-matching tasks. However, as these tasks did not activate the HPA axis, these results have to be interpreted with caution (Lewis & Ramsay, 2002). As far as we are aware, no further studies exist on linking shame and HPA responses to acute stress.

Shame and Pro-Inflammatory Response to Acute Stress

Dickerson, Gruenewald, et al.'s (2004) theory also implicates the immune system in emotional and biological responses to social threat. They argued that social threats activate the immune system in a way that gives rise to inflammation. The signaling molecules deployed by the immune system to coordinate this response, known as pro-inflammatory cytokines, are thought to accumulate in the central nervous system, where they serve to extend and prolong disengagement-related motivational, cognitive, and behavioral effects. Dickerson, Gruenewald, et al. (2004) further suggested that glucocorticoid resistance might be a critical mechanism underlying this circuit (Dickerson, Gruenewald, et al., 2004). The idea here is that social threats diminish the immune system's ability to respond to cortisol, which is a key hormonal signal the body uses to regulate the inflammatory response. In partial support of this idea, declines in sensitivity to glucocorticoids have been reported to occur in socially threatening acute and chronic stressors (Miller, Cohen, & Ritchey, 2002; Rohleder, Schommer, Hellhammer, Engel, & Kirschbaum, 2001). There is some evidence supporting a direct association of shame and pro-inflammatory activity. Dickerson, Kemeny, Aziz, Kim, & Fahey (2004c) experimentally induced self-blame by asking volunteers to write about traumatic experiences in their lives in which they felt bad about themselves or that they blamed themselves for. Participants in the control group were asked to write about everyday activities during the last 24 hr. The experimental condition successfully induced shame and guilt, as well as increases in oral concentrations of the soluble tumor necrosis factor- α receptor-II (sTNF- α RII) as an index of inflammatory activity. In addition, higher levels of shame were associated with higher levels of sTNF- α RII. Cortisol, however, did not increase in this task, probably due to the absence of direct social evaluation (Dickerson, Kemeny, Aziz, Kim, & Fahey, 2004). Several other studies have investigated inflammatory activity in response to acute laboratory stressors involving social evaluation, even though shame was not specifically measured. In most studies, stimulated inflammatory cytokine production in vitro increased after acute stress (as reported for IL-6 in a meta-analysis of stress effects on immune function; Segerstrom & Miller, 2004).

Daily Life Experiences With Shame and Psychobiological Responses

Many of the studies reviewed above have investigated responses to acute laboratory stressors. However, one would expect that any detrimental health effects of shame experience would only emerge after long periods of time of chronic dysregulation of stress systems or chronic increases in peripheral inflammatory activity (Hansson & Libby, 2006; Sjöholm & Nystrom, 2006). Thus an important and unanswered question remains of whether long-term experiences of shame in real life are associated with everyday increases in stress system activity and disinhibition of inflammatory activity.

Few studies have measured shame in association with basal HPA axis activity. Mason et al. (2001) reported that in a sample of combat veterans with posttraumatic stress disorder (PTSD), low urinary cortisol excretion was associated with a cluster of shame and guilt-related items in several psychiatric instruments. In addition, low levels of cortisol were also associated with high depression and disengagement (Mason et al., 2001). In a recent meta-analysis of chronic stressors and HPA activity, Miller et al. found that stressors that were rated as having a high likelihood of inducing shame and social threat were associated with higher afternoon/evening levels of cortisol (Miller, Chen, & Zhou, 2007). However, the studies in this meta-analysis were rated on shame and social threat dimensions by coders, and thus cannot answer the question of whether individual experiences of shame are associated with daily stress system and inflammatory activity.

Summary, Objective, and Hypotheses

We therefore set out in the present study to investigate the association of shame experienced during everyday life with the HPA axis and with the inflammatory system. Daily HPA axis activity was assessed by repeatedly measuring free cortisol in saliva relative to awakening. Activity of the inflammatory system was assessed by measuring LPS-stimulated production of interleukin-6 (IL-6). Although SSPT does not discuss the role of the SNS, there is good evidence from studies using social evaluative stress paradigms that the SNS is activated as well (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Furthermore, the SNS is also an important regulator of the inflammatory response because its products like norepinephrine are well-known to stimulate inflammatory activity (Bierhaus et al., 2003; DeRijk, Boelen, Tilders, & Berkenbosch, 1994). Secretion of the salivary enzyme alpha-amylase might be a surrogate marker for sympathetic nervous system activity, as its secretion from the salivary glands is controlled by direct sympathetic innervation (Garrett, 1999). Accordingly, several recent human studies have shown that salivary alpha-amylase increases in response to acute psychosocial stress (Bosch, de Geus, Veerman, Hoogstraten, & Nieuw Amerongen, 2003; Nater et al., 2005; Rohleder, Wolf, Maldonado, & Kirschbaum, 2006), and salivary alpha-amylase has been shown to be associated with sympathetic nervous system activation (Ehlert, Erni, Heibisch, & Nater, 2006) and blockade (van Stegeren, Rohleder, Everaerd, & Wolf, 2006). It furthermore has a distinct daily rhythm (Nater, Rohleder, Schlotz, Ehlert, & Kirschbaum, 2007; Rohleder, Nater, Wolf, Ehlert, & Kirschbaum, 2004). Thus, we measured salivary alpha-amylase as an index for SNS activity

at the same time points as cortisol. We hypothesized that shame would be associated with higher daily HPA axis and SNS activity, as well as increased pro-inflammatory cytokine production, mediated by lower glucocorticoid sensitivity of the inflammatory system.

To test whether these associations are specific to shame, we also tested whether similar patterns would be found with a related negative emotion, guilt. Finally, because shame is often a prominent symptom of affective disorders, and depression has been linked with dysregulated inflammation (Miller & Blackwell, 2006; Miller, Stetler, Carney, Freedland, & Banks, 2002; Raison & Miller, 2003), we also evaluated whether these effects were independent of depressive symptoms.

Method

Participants

Data for this article were collected as part of a larger research project on depression and atherosclerosis among young women at high risk for affective disorders. Adolescent women were recruited from the larger Vancouver, British Columbia community through advertisements in schools, newspapers, and magazines. Young women were eligible for the study if they were (a) between the ages of 15 and 19, (b) fluent in the English language, (c) free of acute and chronic medical conditions, (d) without a lifetime history of major psychiatric disorders, and (e) at high risk for developing an initial episode of major depression. High risk was defined as having a first-degree relative with a history of depression, or as scoring in the top quartile of the sample distribution on one of two indexes of cognitive vulnerability, the Dysfunctional Attitudes Scale (Weissmann & Beck, 1978) or the Adolescent Cognitive Style Questionnaire (Alloy et al., 1999).

The current article focuses on a subgroup of 56 young women in whom salivary cortisol and alpha-amylase assessments were available. They had a mean age of 17.4 years ($SD = 1.2$) and a mean body mass index (BMI) of 21.33 kg/m² ($SD = 2.89$). At the time of the examination 23% of the women were 15 or 16, 59% were 17 or 18, and 18% were 19 or 20. Fifty-eight percent of the women self-identified as East Asian descent, 33% as Caucasian, and the remaining 9% described themselves as East Indian, African, Aboriginal, or other. Participants came from homes in which mothers had an average of 14.4 years of education and fathers had an average of 15.3 years of education, and 55% of parents had at least a college diploma. Eighty-four percent of the participants came from a family in which their parents were currently married or common-law. Four of the participants were later excluded from analyses because of technical difficulties with assay procedures, or because their values on inflammatory concentrations indicated they were likely in the midst of an infectious episode. This project was approved by the Research Ethics Board of the University of British Columbia. Written consent was obtained from all participants 18 years or older; for those who were younger, a parent or guardian provided consent, and participants provided written assent.

Procedures

All participants reported to the laboratory between 0900h and 1200h for regular scheduled assessments as part of the larger

research project. During these laboratory visits, participants were interviewed regarding somatic health and life stress, filled out a battery of self-report scales, including those described below, and provided venous blood samples for assessment of biological parameters. After that, participants were handed out material to assess saliva samples on two consecutive weekdays (see below).

Measurement of Shame and Depression

Shame

We used a modified version of the State Shame and Guilt Scale (SSGS; Marschall, Sanftner, & Tangney, 1994). The SSGS is a 15-item scale that assesses the experience of shame (internal consistency, $\alpha = .76$) and guilt ($\alpha = .74$) with two separate subscales. Shame and guilt are considered distinct emotions, with shame being a global negative feeling about the self, and guilt being a negative feeling about a specific event rather than the self (Tangney, Miller, Flicker, & Barlow, 1996). Higher scores indicate higher shame and guilt. The scale was modified to assess long-term experience of shame by asking participants about how they felt during the past few months. In light of this change, we refer to this measure as the Trait Shame and Guilt Scale (TSGS). Typical items from the shame subscale are "I've wanted to sink into the floor and disappear," "I've felt like I am a bad person," and "I've felt humiliated, disgraced"; typical items from the guilt subscale are "I've felt tension about something I did," "I've felt remorse, regret," and "I've felt like apologizing, confessing." In our sample, the mean score on the shame subscale was 9.7 ($SD = 3$; Min = 5, Max = 16); the mean score on the guilt subscale was 11.9 ($SD = 3.2$; Min = 5; Max = 20). To evaluate whether the modified instrument successfully captured trait shame and guilt, we administered it again 6 months later to the larger cohort of 104 young women. The test-retest correlations were $r = .49$ ($p < .001$) for the shame subscale and $r = .49$ ($p < .001$) for the guilt subscale. This degree of stability is identical to what is seen with other personality characteristics assessed during teenage and college years (Roberts & DelVecchio, 2000).

Depression

Depressive mood was assessed using the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a 21-item self-report measure of depressive symptoms with excellent psychometrics (internal consistency, $\alpha = .86$). Higher scores indicated higher levels of depression.

Inflammatory Activity and Glucocorticoid Sensitivity

To assess systemic inflammatory activity and its sensitivity toward glucocorticoid suppression, 10 ml of venous blood were drawn from an antecubital vein into Vacutainer tubes with lithium heparin as an anticoagulant (Becton-Dickinson, Mississauga, Ontario, Canada). Within 30 min, blood was transferred to the laboratory and processed under sterile conditions. Blood was diluted 10:1 with saline (0.9% NaCl, Braun, Scarborough, Ontario, Canada) and five aliquots of 1.6 ml were transferred to a 6-well culture plate (Sarstedt, Montreal, Quebec, Canada). All five aliquots were co-incubated with 200 μ l of lipopolysaccharide (LPS; Sigma Chemicals; St. Louis, MO) at a final concentration of 50 ng/ml and

five different concentrations of hydrocortisone (final concentrations: 0, 2.76×10^{-5} , 2.76×10^{-6} , 2.76×10^{-7} , 2.76×10^{-8} M HC; Sigma Chemicals; St. Louis, MO). After 6 hr of incubation at 37°C and 5% CO₂, each aliquot was centrifuged at 14,000 rpm for 5 min. The resulting plasma supernatant was stored at -30°C until further analysis. IL-6 concentrations were quantified in duplicates using commercial ELISA kits (Quantikine human IL-6; R&D Systems; Minneapolis, MO) with a minimum detectable concentration of 0.7 pg/ml. Inter- and intraassay variability was below 10%.

The LPS-stimulated IL-6 production without hydrocortisone expressed as pg/ml is used as an index for inflammatory activity. As an index for glucocorticoid sensitivity, the inhibitory concentration 50% (IC₅₀) was calculated for each individual dose-response curve using GraphPad Prism version 4.00c for Macintosh (GraphPad Software, San Diego, CA). The IC₅₀ reflects the specific concentration of HC needed for 50% inhibition of cytokine production and is therefore inversely related to glucocorticoid sensitivity.

Daily Trajectories of Free Cortisol and Salivary Alpha-Amylase

To assess daily trajectories of free cortisol and salivary alpha-amylase, each participant was asked to provide six saliva samples on two consecutive days. To facilitate the collection process and enhance compliance, we lent participants a handheld computer (Palm Zire 21), which signaled them to collect saliva at waking, and at 1/2, 1, 4, 9, and 14 hr after waking. Specifically, when participants woke up, they took their first saliva sample and activated a customized software application on the Palm. This application "programmed" the computer so that it would sound alarms at the appropriate times for the rest of the day's samples. To collect the saliva samples, participants chewed lightly on a cotton dental roll for 1 min so that it became saturated in saliva (Salivette; Sarstedt, Nümbrecht, Germany). Participants were instructed to avoid taking saliva samples immediately following tooth brushing and food intake. The dental roll was then placed in a plastic container and stored in the refrigerator until the end of the ambulatory monitoring period. To ensure compliance with the saliva sample protocol, the computer flashed a three-digit code each time the alarm sounded. Participants recorded the code on the plastic container. When the Salivettes were returned to the lab, a research assistant matched the computer codes with those recorded by the participant, and samples without proper codes were excluded from analyses. On an a priori basis, we chose to define compliance as taking a sample within 20 min of target in either direction for the waking, 1/2 hour, and 1-hr samples, and within 60 min of target for the remainder of the samples. When this definition was applied, a total of 83% met our criteria for compliance. Only these values were used to compute morning cortisol response and daily cortisol output AUC scores. In the case of a missing sample at waking, 1/2 hour after waking, or 1 hr after waking, we did not compute a morning cortisol response score for that day. We computed daily output scores when we had at least four samples across the day.

Samples were returned through the mail and stored at -20°C until shipment to Clemens Kirschbaum's laboratory at the Dresden University of Technology (Dresden, Germany) for analyses of salivary cortisol and alpha-amylase. Cortisol concentrations were

determined using a commercial chemiluminescence immunoassay (CLIA; IBL-Hamburg, Germany). Salivary alpha-amylase was measured by a quantitative enzyme kinetic method as previously described (Rohleder et al., 2006). Inter- and intraassay coefficients of variation were below 10% for both analytes.

Statistical Analyses

All data were tested for normal distribution prior to analyses. Salivary cortisol, alpha-amylase, and LPS-stimulated IL-6 were log-transformed. The two daily profiles of salivary cortisol and alpha-amylase were aggregated to yield one mean cortisol and one mean amylase profile. Three indices were calculated for each analyte to represent distinct characteristics of daily variation: We calculated the area under curve (AUC) with respect to ground using all daily samples as an estimate of overall hormone system activity (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003); the slope of the daily hormone trajectory as an estimate of daily rhythm (excluding the 30-min post wake-up sample to avoid an impact of the wake-up response on the daily slope); and the slope of the morning response (between samples wake-up and 30-min post wake-up) as an estimate of the wake-up response. Analyses of variance (ANOVAs) for repeated measures were used to analyze diurnal variation of cortisol and alpha-amylase. To test specific hypotheses, we first calculated Pearson correlations to test bivariate associations of shame with biological variables (indices of cortisol and amylase activity, stimulated IL-6, and GC sensitivity of IL-6). Second, hierarchical linear regression equations were used to predict the biological outcome variables by shame, controlling for potential confounders (age, BMI, guilt, and depressive mood). All results are presented as means \pm standard error of the mean (SEM).

Results

Preliminary Analyses

As shown in Figure 1, daily activity of the HPA axis as measured by free cortisol in saliva was characterized by a marked wake-up response in the first hour after awakening and decreasing concentrations toward the evening. ANOVA revealed a significant effect of time, $F(3.46, 176.30) = 79.02, p < .001$. The daily trajectory of salivary alpha-amylase was characterized by a decrease during the first 30 min of the day, and relatively higher levels in the later half of the day, $F(3.77, 192.03) = 19.22, p < .001$. No correlations between daily cortisol and amylase concentrations were found.

Associations of Shame and Psychobiological Parameters Shame and Daily Free Cortisol

To test the hypothesis that shame would be associated with alterations in basal HPA axis activity, we first computed Pearson correlations between shame and each of the three indices for basal HPA axis activity. Shame was not associated with overall daily cortisol secretion (AUC; $r = .12; p = .41$), the daily slope ($r = .07; p = .64$), or the awakening response ($r = .11; p = .46$). These results were confirmed by hierarchical regressions controlling for

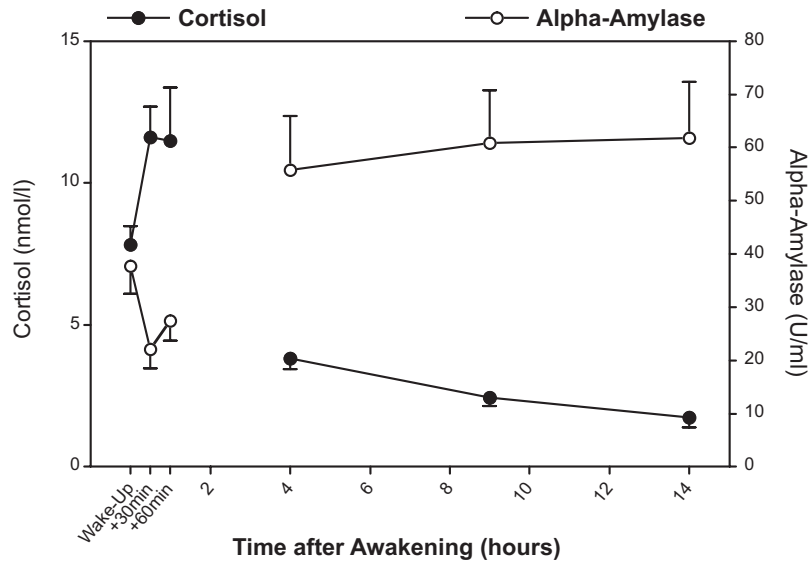


Figure 1. Daily trajectories of salivary free cortisol and salivary alpha-amylase (values shown are mean \pm SEM).

age and BMI (AUC: $\beta = 0.12$; $p = .42$; daily slope: $\beta = 0.07$; $p = .59$; awakening response: $\beta = 0.11$; $p = .46$).

ling for age and BMI (AUC: $\beta = 0.49$; $p < .001$; daily slope: $\beta = 0.14$; $p = .34$; awakening response: $\beta = 0.06$; $p = .67$).

Shame and Daily Salivary Alpha-Amylase

To test whether shame was associated with altered activity of the sympatho-adrenal-medullary system, as measured by concentrations of alpha-amylase in saliva, we calculated Pearson correlations between shame and each of the three activity indexes. As shown in Figure 2, greater shame was associated with greater AUC of daily amylase secretion ($r = .49$; $p < .001$). There was no association of amylase daily slope and wake-up response with shame measures ($r = .14$; $p = .33$; $r = .07$; $p = .66$, respectively). These results were confirmed by hierarchical regressions control-

Shame, Inflammatory Activity, and Glucocorticoid Sensitivity

Next we tested the hypothesis that shame would be associated with higher inflammatory activity. Pearson correlations revealed a positive association of shame with LPS-stimulated IL-6 production ($r = .35$; $p = .012$), indicating that the more shame participants felt, the greater their in vitro inflammatory response. Hierarchical regression controlling for age and BMI confirmed the positive association of shame with IL-6 production ($\beta = 0.36$; $p = .005$; see Figure 3A).

We next tested whether shame would be associated with altered glucocorticoid sensitivity by calculating Pearson correlations between shame and the IC_{50} of the cortisol induced suppression of IL-6 production. Contrary to expectations, shame was inversely associated with the IC_{50} ($r = -0.43$; $p = .001$), indicating that greater shame was associated with greater sensitivity to the anti-inflammatory properties of glucocorticoids. Because glucocorticoid sensitivity may be related to the extent of LPS-stimulated cytokine production in the absence of cortisol, we retested associations controlling for IL-6 production as well as age and BMI. Shame remained a significant predictor of glucocorticoid sensitivity ($\beta = -0.33$; $p = .02$; see Figure 3B) under these conditions.

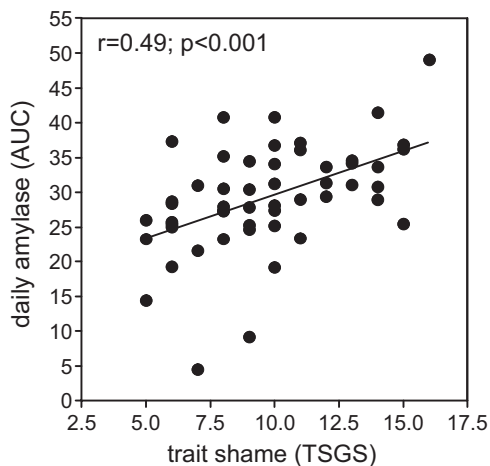


Figure 2. Association of trait shame with daily amylase output (area under curve, AUC).

Are the Effects Specific to Shame?

Testing the Role of Guilt

To test whether the associations with the biological variables were specific to shame, we added the guilt subscale to the regression models. Pearson correlations revealed that shame and guilt were positively associated, $r = .52$; $p < .001$.

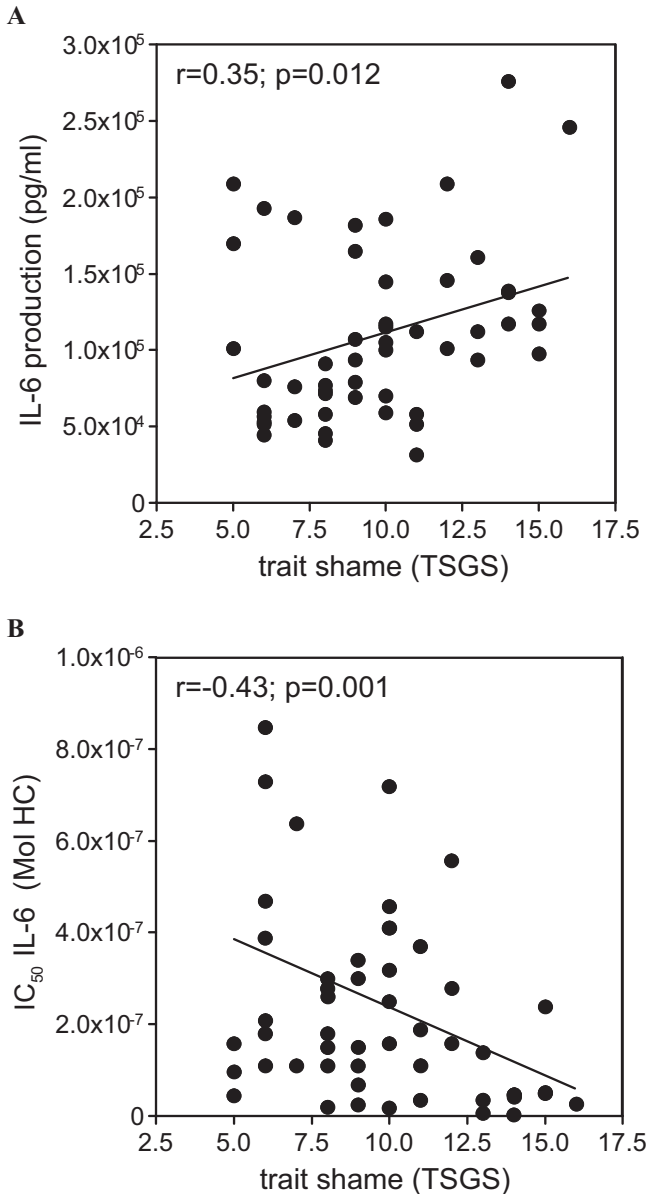


Figure 3. Associations of trait shame with (A) LPS-stimulated IL-6 production and (B) glucocorticoid sensitivity. Note that the IC₅₀ is inversely related to glucocorticoid sensitivity, that is, higher IC₅₀ indicates lower sensitivity and vice versa.

HPA axis. Guilt was not associated with cortisol AUC, daily slope, or awakening response, nor did it change associations of shame with any of the cortisol indices.

Sympathetic nervous system. Adding guilt as a predictor into the regression model revealed a significant association of higher guilt with lower amylase AUC ($\beta = -0.37$; $p = .012$). However, shame remained a significant predictor of amylase AUC even with guilt included in the regression model ($\beta = 0.86$; $p < .001$). Guilt was not associated with nor did it change the association of shame with the daily slope and the awakening response of salivary alpha-amylase.

IL-6 production and GC sensitivity. Guilt was not associated with in-vitro stimulated IL-6 production. When added to the regression model, the association of shame with IL-6 remained significant ($\beta = 0.39$; $p = .009$). Guilt was not associated with GC sensitivity of IL-6 production, but adding it as a predictor slightly decreased the association of shame with GC sensitivity ($\beta = -0.30$; $p = .06$).

Testing the Role of Depression

To test whether the associations with the biological variables were due to the potentially overlapping construct of depression, we added depression scores (BDI) to the regression models. Pearson correlations revealed that shame and depression were positively associated ($r = .66$; $p < .001$).

HPA axis. Depressive mood was a significant predictor of overall daily cortisol secretion (cortisol AUC; $\beta = 0.59$; $p = .003$), although no association of cortisol AUC and shame emerged. Depression was not associated with cortisol daily slope or the awakening response.

Sympathetic nervous system. Depressive mood alone ($\beta = 0.41$; $p = .004$), as well as shame alone (see above), were significantly associated with overall daily SNS activity (amylase AUC). Adding them into the model simultaneously abolished the association of depression with amylase AUC ($\beta = 0.13$; $p = .49$), although the association of shame with overall daily amylase secretion remained significant ($\beta = 0.41$; $p = .022$). Depressive mood was not associated with any of the remaining amylase indices.

IL-6 production and GC sensitivity. Depressive mood alone ($\beta = 0.38$; $p = .003$), as well as shame alone (see above) were significantly associated with IL-6 production. Adding them into the model simultaneously abolished both associations (BDI: $\beta = 0.25$; $p = .15$; shame: $\beta = 0.19$; $p = .26$). Depression was not significantly associated with GC sensitivity ($\beta = -0.01$; $p = .96$), but adding it to the model slightly decreased the association of shame with GC sensitivity ($\beta = -0.32$; $p = .07$).

Testing the Proposed Mediators

We next tested the hypothesis that the positive association of shame and inflammatory activity was mediated by altered basal HPA axis or SNS activity. According to Baron and Kenny (1986), three requirements have to be met to test for mediation: a significant association (a) of predictor and outcome variable, (b) of predictor and assumed mediator, and (c) of mediator and outcome variable. If these requirements are met, mediation exists if the previously significant direct relationship between the predictor and outcome variable is reduced in magnitude after the controlling for the mediator (Baron & Kenny, 1986).

Because cortisol levels were not associated with shame, the hypothesis that HPA axis alterations mediate the association of shame and inflammatory activity can be rejected without further tests. As reported earlier, shame was associated with IL-6 production and GC sensitivity, satisfying Criterion 1. In addition, shame was associated with total daily alpha-amylase secretion, thus fulfilling the second criterion. We next tested whether Criterion 3 (significant association of proposed mediator with outcome variable) was fulfilled. Pearson correlation did not reveal the

required association of amylase AUC and LPS-stimulated IL-6 ($r = .12$; $p = .39$), thus ruling out amylase as mediator between shame and inflammatory activity.

We then tested amylase as a mediator of the shame–GC sensitivity association. The association of amylase AUC and GC sensitivity was marginally significant ($r = -0.25$; $p = .07$). When added to the regression equation, the association of shame with GC sensitivity was reduced from $\beta = -0.43$ ($p = .001$) to $\beta = -0.28$ ($p = .07$). However, further mediational analyses using the Sobel test (Preacher & Hayes, 2004) revealed that amylase AUC was not a statistically significant mediator of the shame–GC sensitivity association ($z = -.36$; $p = .72$).

Discussion

In the present study we tested whether the predictions of SSPT regarding shame experience could be generalized to long-term conditions. We found that shame experienced over a period of several months was associated with greater SNS, but not greater HPA, activity. Second, greater experiences of shame were associated with increased in-vitro inflammatory activity and with increased glucocorticoid sensitivity of the inflammatory response. However, none of the stress systems appeared to be a mediator of the associations of trait shame with inflammation or GC sensitivity.

The results of our study show that the predictions of SSPT in terms of acute experiences of shame cannot uniformly be extended to the long-term or chronic experience of shame. Our findings of no association of trait shame with daily HPA axis activity are in contrast to findings using acute stressors characterized by social threat (Dickerson & Kemeny, 2004). We can think of several reasons for this discrepancy. One explanation might be that HPA activation only results from shame experiences that are elicited by social threats. Although most instances of shame are precipitated by social threats—that is, events that make a person feel devalued in relation to others—other experiences can bring about this emotion, too (Tangney, 1996). Because our measure did not differentiate between social and other antecedents of shame, we cannot determine whether this distinction accounts for the absence of HPA results. However, in future research it will be important to evaluate this issue.

Another explanation for the discrepancy between our findings and those on acute stress might be the differential effects of acute versus chronic psychological conditions on biological systems, especially the HPA axis. The ability of acute psychosocial stress to activate the HPA axis is well-documented (see, e.g., Dickerson & Kemeny, 2004). However, there is conflicting evidence about what happens to the HPA axis with longer exposures to stress. In some cases activity is increased (Baum, Gatchel, & Schaeffer, 1983), whereas in others it is decreased (Miller et al., 2002). To some degree this variability is explained by the duration of the stressor. Miller et al. (2007) showed in a meta-analysis that daily cortisol levels are inversely associated with the duration of chronic stress, that is, the longer the duration of the stressor, the lower the daily cortisol output. It might be assumed that the same temporal association exists between shame experiences and cortisol activity. Because we did not assess the duration of shame experiences in the present study, it might be speculated that some participants had been experiencing shame for shorter periods of time whereas

others may have been experiencing prolonged periods of shame, thus leading to no consistent association of shame with HPA axis activity. That said, the same meta-analysis found that chronic stressors rated as having a high likelihood of inducing shame and social threat were associated with higher afternoon and evening cortisol levels (Miller et al., 2007). It is not clear why a similar pattern of findings would not emerge in this context.¹

In contrast to cortisol patterns, we found a strong positive association of trait shame with daily sympathetic nervous system activity. Threats to the physical self are known to activate the SNS, and although SSPT focuses on HPA activation, there are no a priori reasons why social threats would not also activate the SNS. In fact, most situations that activate the HPA axis in the laboratory also strongly activate the sympathetic nervous system (Kudielka et al., 2004). Based on the current study, we would argue that SSPT needs to be modified to include predictions about SNS activation.

Our finding of a positive association of shame with LPS-stimulated inflammatory cytokine production is consistent with predictions of SSPT, and with the empirical data on acute stress responses. The impact of shame on salivary measures of inflammation was demonstrated in an acute setting using an experimental manipulation of shame (Dickerson et al., 2004). The present study represents the first test of longer term experiences of shame and inflammatory activity. This finding that trait shame is associated with activation of the inflammatory response system could have health implications. Disinhibition of the inflammatory system is proposed to be a mechanism in atherosclerosis, myocardial infarction, stroke (Hansson & Libby, 2006), insulin resistance/Type 2 diabetes (Sjoholm & Nystrom, 2006), and cognitive decline (Weaver et al., 2002). Hence, long-term experiences of shame might have the potential to negatively influence health and well-being.

In contrast to inflammatory activity, our findings were inconsistent with SSPT with respect to glucocorticoid sensitivity. Dickerson, Gruenewald, et al. (2004) had suggested that one possible mechanism permitting disinhibition of inflammatory activity during acute experiences of shame was via decreases in glucocorticoid sensitivity (Dickerson, Gruenewald et al., 2004). Our study on long-term experiences of shame showed the opposite. Higher levels of trait shame were associated with increased glucocorticoid sensitivity. Differences between acute and chronic effects on immune function are a well-documented phenomenon (Dhabhar & McEwen, 1997). For example, under conditions of acute social-evaluative stress, such as the TSST, women displayed a relative glucocorticoid resistance compared to men (Rohleder, Schommer, Hellhammer, Engel, & Kirschbaum, 2001). Although acutely, increased glucocorticoid resistance could facilitate inflammatory disinhibition, over time there may be a counterregulatory response that results in up-regulation of target tissue sensitivity after persistent experiences of shame. This type of counter-regulatory response has been found in populations experiencing chronic dis-

¹ We performed additional analyses looking specifically at single time point assessments of both salivary parameters. Results showed that salivary alpha-amylase levels measured 4 hr ($r = .50$; $p = .001$), 11 hr ($r = .44$; $p = .001$), and 14 hr ($r = .35$; $p = .01$) after wake-up were associated with shame. Amylase measured at earlier time points as well as cortisol was not associated with shame.

tress, such as PTSD patients (Rohleder, Joksimovic, Wolf, & Kirschbaum, 2004) and in depression (Miller, Freedland, & Carney, 2005), and could explain differences between acute and chronic effects of shame on glucocorticoid resistance.

We did not find evidence that either HPA or SNS activity mediates the relationship between trait shame and inflammatory activity. However, limitations of our measures may have precluded detecting mediational associations. Although salivary alpha-amylase measured in our study has been shown to be a good marker for SNS activity (Nater et al., 2005) and the only feasible method of ambulatory SNS assessment, it might not necessarily be a good predictor of SNS effects on immune cells, which are primarily exerted by circulating catecholamines. Furthermore, in parallel to the variation of glucocorticoid sensitivity of inflammatory cells, the sensitivity of inflammatory cells to catecholaminergic signaling is subject to variation as well (Kavelaars, Kuis, Knook, Sinnema, & Heijnen, 2000). Because we did not measure catecholamine sensitivity of inflammatory cytokine production, its role in mediating the observed effects remains open. Alternatively, behavioral factors could mediate the association of shame and inflammation. For example, persistent experiencing of shame could be associated with negative health behaviors, such as unfavorable dietary choices, smoking, or sedentary lifestyle, all of which are associated with peripheral inflammation (Bermudez, Rifai, Buring, Manson, & Ridker, 2002). However, none of our participants were smokers, and BMI was not correlated with shame or inflammation. Nevertheless, future research is needed to probe health behaviors in more depth as mediators between shame and inflammation.

Finally, we found evidence supporting the unique predictive value of shame over other similar negative emotions. Shame continued to predict SNS activity, inflammatory activity, and glucocorticoid resistance even after guilt was entered into the regression model. These findings are in line with the predictions of SSPT that the emotion of shame has unique physiological correlates that are distinguishable from related emotional states. (Dickerson, Gruenewald, et al., 2004). Shame and guilt are considered distinct emotions. Whereas shame is defined as a global negative feeling about the self, guilt is a negative feeling about a specific event or personal shortcoming and not about the self. Experiencing shame versus guilt also has different consequences. As shame is usually associated with low self-esteem, it is associated with withdrawal and passive coping, whereas guilt is more likely to be associated with adaptive behaviors (Tangney et al., 1996). These findings suggest that in addition to negative psychological effects, long-term shame also has potentially detrimental effects on biological systems that cannot be explained by other negative emotions such as guilt.

Shame and depression were also found to be unique in some respects. Depression was associated with activation of the HPA axis, whereas shame was not. Shame continued to predict SNS activity and glucocorticoid resistance even after depression was entered into the regression model. However, both shame and depression were associated with inflammatory cytokine production, and neither predicted above and beyond the effects of the other. This may be because shame is likely to co-occur with emotions like sadness, and with cognitions such as helplessness,

both of which are commonly found in depression, and hence it might be these common factors that drive the association of both shame and depression with inflammation.

The results of our study should be interpreted in the light of some limitations. First, the shame scale was originally designed to tap state shame, and more work needs to be done to insure that the modified version captures trait shame. Related, we cannot exclude the possibility that state shame experienced on a momentary basis might have influenced cortisol or amylase measurements. However, we were able to show in a previous study that daily amylase is relatively robust against momentary mood and stress ratings (Nater et al., 2007). Second, although we were able to show that our effects are specific for shame and not explained by guilt and depression, we did not measure anxiety. However, because depression and anxiety are usually strongly correlated, we think it is unlikely that anxiety drives the association of shame and alpha-amylase. Third, although there is emerging evidence to support using salivary amylase as an index for sympathetic nervous system activity in daily life, this measure requires further validation. Fourth, our immune measure utilized in-vitro stimulation of inflammatory activity, whereas basal plasma inflammatory markers have been identified as predictors of detrimental health outcomes. However, concentrations of plasma cytokines are relatively low in healthy young individuals, and thus it was necessary to use this in-vitro method in our sample. This measure assesses immune cells' potential for mounting an inflammatory response upon exposure to a stimulus, and could serve as a good predictor in young populations of later life systemic inflammation. Finally, the generalizability of our findings is limited because the sample consisted of young women at high risk for depression. Though this limits our ability to generalize to other populations, we do not see any reason that it would systematically bias our findings. For example, although age-related changes have been described for most of the biological parameters used in this study, that is, cortisol and inflammation, the literature does not suggest that there are significant differences between adolescent and premenopausal women (Ershler, 1993; Van Cauter, Leproult, & Kupfer, 1996). Finally, we should also note that there are gender differences in the shame proneness of adolescents (Lutwak, Panish, Ferrari, & Razzino, 2001), so future work will have to reveal whether these findings generalize to men.

In conclusion, we show here that some portions of SSPT generalize to long-term experience of shame, whereas others do not. Consistent with SSPT, greater experiences of trait shame were associated with greater in-vitro inflammatory activity. However, inconsistent with this theory, greater experiences of trait shame were not associated with HPA activity, but instead were associated with SNS activity, and neither HPA nor SNS activity mediated the relationship between shame and inflammatory activity. Overall, these results suggest the potential value of extending SSPT predictions to other biological systems such as the SNS. It also suggests that SSPT may need to be modified to make differential predictions for acute versus chronic experiences with shame. SSPT remains an important theory discriminating the biological responses of specific psychological states, and future work is needed to clarify the conditions and mechanisms by which social threats come to have implications for health.

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