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Running Head: FACIAL EXPRESSIONS SIGNAL BIOLOGICAL RESPONSES TO STRESS

**Facial expressions of emotion reveal
neuroendocrine and cardiovascular stress responses¹**

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Abstract

Background

The classic conception of stress involves undifferentiated negative affect and corresponding biological reactivity. The present study hypothesized a new conception that disaggregates stress into emotion-specific, contrasting patterns of biological response.

Methods

Ninety-two healthy adults engaged in stress-challenge tasks, during which cardiovascular responses, hypothalamic-pituitary-adrenocortical (HPA) axis responses (i.e., cortisol), emotional expressions (i.e. facial muscle movements) and subjective emotional experience (self-reported) were assessed.

Results

Pronounced individual differences emerged in specific emotional responses to the stressors. Analyses of facial expressions revealed that the more fear individuals displayed in response to the stressors, the higher their cardiovascular and cortisol responses to stress. By contrast, the more anger and disgust (indignation) individuals displayed in response to the same stressors, the lower their cortisol levels and cardiovascular responses. Individual differences in optimistic appraisals appeared to mediate these correlated patterns.

Conclusions

Facial expressions of emotion signal biological responses to stress. Fear expressions signal elevated cortisol and cardiovascular reactivity; anger and disgust signal attenuated cortisol and cardiovascular reactivity, patterns that implicate individual differences in stress appraisals. Rather than conceptualizing stress as generalized negative affect, studies can be informed by this emotion-specific approach to stress responses.

Introduction

Stress is implicated not only in such nuisances as the common cold ([Cohen et al 1997](#); [Cohen et al 1991](#)), but also in the pathophysiology of major morbidity and mortality threats. For example, individual differences in stress reactivity have been implicated in the pathophysiology of depression, anxiety and heart disease – leading contributors to disability and death in the United States (e.g., [Charney and Manji 2004](#); [Coryell et al 1986](#); [Frasure-Smith et al 1995](#); [Kubzansky et al 1998](#)). Consequently, identifying and understanding behavioral and physiological indices of normal and pathological stress reactivity is important for improving physical and mental health.

Stress reactivity is typically assessed through active challenge paradigms during which participants are exposed to stressors such as difficult mental arithmetic or public speaking under harassing conditions (e.g., [Kirschbaum et al 1993](#)). An important but largely unexamined question about stress reactivity concerns individual differences in emotion-specific responses to stress challenges. When participants perform stressful tasks, such as serial subtraction tasks under harassing conditions, some participants may respond with fear of not measuring up to performance standards, whereas others may respond with anger or disgust, conveying their indignation at being badgered. These contrasting emotional responses may be associated with different physiological responses to the stressors in ways that have important clinical implications.

Although, fear, anger, and disgust are all negative emotions and might thus contribute to a generally negative stress response, fear differs from anger and disgust in ways that imply the possibility of diverging physiological and neuroendocrine stress responses. Specifically, whereas fear elicits the mood-congruent effects one might expect of a negative emotion --

leading people to perceive greater risk and a lack of individual control in stressful circumstances -- anger does not. Anger, by contrast, triggers optimistic perceptions of risk ([Lerner and Gonzalez 2005](#); [Lerner and Keltner 2001](#)). Differences in appraisals of certainty and control drive the divergent decision outcomes for fear and anger. Disgust, although comparatively less studied, resembles anger in terms of appraised certainty and control ([Smith and Ellsworth 1987](#)). Thus, we hypothesize that the disgust patterns might resemble those of anger, representing an indignation response. Taking past research together, therefore, one may predict that individuals who respond with anger and disgust to stressful circumstances will show lower biological responses to stress (e.g., lower blood pressure increases and cortisol levels) than will individuals who respond to stress with fear and that appraisals will mediate these patterns.

How such individual differences in emotional responses to stress can be assessed becomes an important issue. Emotion-specific behaviors -- i.e. facial displays of emotion -- provide an objective indicator of individual differences in the emotional experience of a stressor that can provide evidence as to whether such individual differences are predictive of differences in physiological response to stress.

Scientists as far back as Darwin (1872/1998) and Duchenne (1862/1990) recognized that specific facial expressions represent an output signal associated with specific emotional states ([Ekman et al 1983](#); [Ekman et al 1969](#); [Keltner et al 2003](#); [Levenson 1992](#)). As yet, however, almost no stress-reactivity studies have examined whether expressions of specific contrasting emotions may signal reliable individual differences in stress responses. One study on children has found promising evidence of systematic associations ([Quas et al 2000](#)). However, questions concerning the biological stress responses associated with contrasting emotion expressions in adult populations remain largely unexamined.

In the present study, we investigated whether individual differences in facial responses reflecting fear versus anger and disgust (indignation) were differentially associated with hypothalamic-pituitary-adrenocortical (HPA) axis responses (as assessed by cortisol) and cardiovascular responses to stress. For comparative purposes, trait and state measures of these emotions were also collected. As implied above, we predicted that facial displays reflecting fear in response to the stressors would be associated with increases in cortisol and cardiovascular stress responses, whereas facial displays reflecting anger and disgust would be associated with reduced cortisol and cardiovascular stress responses. We further predicted that the tendency to make optimistic appraisals would mediate these patterns.

Methods and Materials

Overview

At Time 1, participants completed self-report measures of trait affect. Within the following week (Time 2), participants engaged in stress-challenge tasks, during which cardiovascular responses and cortisol were assessed. Emotional behavior (i.e. facial muscle movements) and emotional experience (self-reported) were also assessed.

Participants

After obtaining approval from the Institutional Review Board from the University of California, Los Angeles, students and employees in a university community responded to an ad offering \$60 for participating. Because the tasks were intentionally stressful, the Human Subjects Protection Committee required screening out anyone who had been "diagnosed with a major mental health disorder or who was currently under treatment for a mental health disorder." (Individuals with a minor mental health disorder were not excluded.) Other exclusion criteria were use of medications affecting cardiovascular, monoamine, or endocrine function, and

current pregnancy or lactation. To avoid problems with demand awareness, people with training in psychology were also excluded.

Ninety-two participants (45 males and 47 females) comprised the final sample. Participants ranged from 18 to 29 years (mean age of 20.6 years). The sample was 43.5% European-American, 43.5% Asian American, 8% Latino, 3% African American, and 2% other. No participant dropped out during the study.

Questionnaire Session

Participants reported to a laboratory where they completed informed consent forms and a battery of demographic and psychosocial self-report scales. The self-report questionnaires included two measures of trait fear/anxiety, namely Spielberger's (1983) measure of trait anxiety and a Fear Survey Schedule ([Bernstein and Allen 1969](#); [Geer 1965](#); [Suls and Wan 1987](#)), and three measures of trait anger, the Cook-Medley Hostility Scale ([Cook and Medley 1954](#)), the Hostility Subscale of the Brief Symptom Inventory ([Derogatis and Savitz 2000](#)), and a 6-item Trait Anger Inventory ([Lerner and Keltner 2001](#)). The Life Orientation Test ([Scheier and Carver 1985](#)) was included to assess tendencies to make optimistic or pessimistic appraisals.

Stress-Challenge Tasks and Procedures

Within a week later, participants returned to the laboratory. Sessions were run in the late afternoon, to control for the circadian rhythm of cortisol. The challenge session began with collection of two saliva samples for assessment of cortisol levels. Samples were immediately placed on ice in a cooler and transferred within the next few minutes to a freezer. Participants then responded to a set of interview questions, material that is not part of the present analyses, after which the challenge began.

Participants were connected to a Critikon Dinamap Vital Signs Monitor Model 1846SX which automatically and continuously recorded heart rate and blood pressure every two minutes throughout the laboratory session. Before the challenge tasks began and immediately after connection to the monitor, participants were given 10 minutes of rest and acclimation time.

The stress-challenge tasks included: (a) counting backwards by seven's from 9,095; (b) mentally calculating arithmetic problems taken from the Wechsler Intelligence Test; and (c) counting backwards by thirteen's from 6,233. A two-minute rest period occurred between the second and third stress-challenge tasks. These kinds of stress-challenge tasks have been shown to induce stress in prior studies and constitute a part of the standardized Trier Social Stress Test (TSST) ([Dickerson and Kemeny 2004](#); [Kirschbaum et al 1993](#)). Participant responses were recorded and timed by the video camera.² To accentuate the socially stressful nature of the tasks, participants were informed of each error they made and urged to go faster by a harassing experimenter. Participants were also told that these tasks were diagnostic of general intelligence and that their responses would be compared to other participants' scores.

Immediately after the stress-challenge tasks, and again during the recovery period, participants completed the state emotion measures, rating the degree to which they had experienced 16 emotions ([Gross and Levenson 1995](#)) on a scale from 0 to 8 with labeled end points, including those assessing fear, anger, and disgust.

Immediately following completion of the stress-challenge tasks, a second saliva sample was taken. This time corresponded to approximately 25 minutes following the initiation of the stressors, a time period which would allow for stress-related increases in cortisol to be identified.

Following the stress tasks, a 30-minute recovery period began. During this time, participants listened to pleasant music and completed a demographics questionnaire and

questions assessing activities (e.g., exercise and eating) that might have influenced cardiovascular and cortisol responses. At the end of the recovery period, a third saliva sample was taken. Participants were then debriefed and dismissed.

Measures

Principal components factor analyses of the state emotion measures were conducted to create factors for each of the three emotional states of interest. (1) a *fear factor* (combining “anxiety,” “fear,” “afraid,” and “nervous”; $\alpha = .90$ at peak stress; $.86$ at recovery); (2) a *disgust factor* (“disgust” and “repulsion”; $\alpha = .84$ at peak stress; $.89$ at recovery); and (3) an *anger factor* (“anger,” “contempt,” “irritation,” and “frustration”; $\alpha = .88$ at peak stress; $.87$ at recovery).

Cardiovascular measures included heart beats per minute (BPM), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pulse (MAP), recorded automatically at 2-minute intervals throughout the laboratory session. One index for each of the cardiovascular dimensions (four indices total) was calculated by averaging readings taken during each of the stress tasks. The same procedure was followed for averaging across baseline and recovery.

Salivary Cortisol Assays

Saliva samples were shipped for overnight delivery on dry ice to the Behavioral Endocrinology Laboratory at the Pennsylvania State University. Salivary cortisol levels were determined from a 25- μ l sample, which was assayed in duplicate by radioimmunoassay using the HS-cortisol High Sensitivity Salivary Cortisol Enzyme Immunoassay Kit (Salimetrics LLC, State College, PA). All samples were tested with a single assay batch, eliminating error that might occur due to differences between batches. The HS-Cortisol Assay allows for robust results when the saliva samples have a pH within the range of 3.5 - 9.0. All samples were within this range.

Coding of Facial Expression

Participants' facial behavior was coded using the EMFACS version of the Facial Action Coding System ([Ekman and Friesen 1978](#)), an anatomically-based coding system. The reliability and validity of the EMFACS system have been demonstrated in prior investigations (e.g., [Keltner and Bonanno 1997a](#)). Fourteen segments from the session were sampled for each participant, including stressful tasks and relaxing time, constituting approximately 5.62 minutes of facial movement (2 hours of coding) per participant. Coders spent approximately 22 minutes coding each minute of actual muscle movement.

EMFACS criteria were used to code facial expressions of fear, anger, and disgust. Whereas anger and fear can be reliably represented in upper-face codes and/or lower-face codes, disgust is represented only in full-face codes. For each emotion, three dimensions were assessed: *frequency*, *intensity*, and *duration*. Coders scored the *intensity* of each muscle movement on a 5-point scale (1 = *minimal*, 3 = *moderate*, 5 = *extreme*). Expression *duration* was measured in milliseconds; *frequency* was a simple count of each emotion's occurrence.

Both coders were blind to all other study data including the time of day individual participants were run. Both coders passed a FACS reliability exam administered by Paul Ekman's laboratory, and both were blind to the hypotheses. One coded all the participants; another coded a randomly-selected 24% of participants. Following standard procedures (see [Keltner and Bonanno 1997a](#); [Keltner and Bonanno 1997b](#)), intercoder reliability was defined by a ratio in which the number of action units on which the two coders agreed was multiplied by 2 and then divided by the total number of action units scored by the two persons. This agreement ratio was calculated for each event observed by one or both coders. The mean agreement ratio was .82, demonstrating good reliability.

Results

Preliminary Analyses

Preliminary analyses compared expressions of emotions during the stress tasks with those during the three rest periods (baseline, in between tasks, recovery). As expected, participants displayed greater fear, anger, and disgust during the stress tasks than during the resting periods. Table 1 displays descriptive data for each emotion in terms of displayed frequency, displayed intensity, and displayed duration.³ Of all the emotions, anger expressions appeared with the greatest frequency, intensity, and duration. The stress tasks also produced significantly higher cardiovascular and higher cortisol levels than did the rest periods (see Table 2), confirming that the tasks had their expected effects on physiological functioning.

Sex of participant did not moderate the key relations between expression and physiology.⁴ Thus, we report the main results collapsing across males and females.

To test the main hypotheses most parsimoniously, thus reducing the likelihood of Type I errors, we aggregated measures across stress tasks and separately aggregated measures across rest periods. We also aggregated facial expression data by standardizing the measures of frequency, intensity, and duration, and averaging across them. We further aggregated by averaging the anger and disgust expression data into one composite called “indignation.” This aggregation was justified not only by similarity in the respective appraisals of control associated with anger and disgust but also by the fact that they were highly correlated with each other ($r = .64, p < .01$). If the predicted patterns appear in the aggregated form with the least possible number of significance tests, then it is justifiable to test associations with specific dimensions of specific emotions.

Emotions and Cortisol Responses

To test the main hypotheses, we calculated the relations between cortisol levels (at baseline, peak stress, and recovery) and: (a) Facial expression (i.e. EMFACS) measures of fear and indignation; (b) self-reported trait fear/anxiety and anger; (c) self-reported state fear, anger, and disgust. In support of the main hypotheses, facial expressions were reliably correlated with cortisol levels (see Figure 1, Panel A). As predicted, a generalized negative affect pattern did *not* appear. Rather, all of the correlations between fear displays and cortisol levels were *positive* whereas all of the correlations between indignation were *negative* (see Figure 1). At peak stress and at recovery, all of the correlations between facial display and cortisol were significant ($p < .05$). In all cases where a significant correlation between facial display and cortisol appeared, there was also a significant difference between the indignation correlations and the fear correlations (tested with Fisher r-to-z transformations; $p < .05$). Even at low levels of display when the emotions had not yet been activated (i.e., baseline), the opposing patterns still emerged. They were not significant at baseline, however, as one would expect. The results, thus, support the prediction that fear displays in the face are related to elevated cortisol levels in response to stress whereas indignation is related to lower cortisol levels in response to stress. If one disaggregates the indignation data into separate components of anger and disgust, all of the same patterns hold (see regression results of Table 3 column 1). Thus, indignation represents consistent patterns for anger and disgust.

Self-reported state and trait measures of fear, anger and disgust, for the most part, were not correlated with cortisol levels. One exception was the Trait Anger Inventory ([Lerner and Keltner 2001](#)); the more a person reported experiencing anger, the lower his or her cortisol level, but only during the recovery period ($r(90) = -.24, p < .05$).

Emotion and Cardiovascular Response

The evidence relating facial expressions to cardiovascular responses were, for the most part, also consistent with hypotheses (see Figure 1, Panel B). Whereas all of the correlations between fear expressions and cardiovascular responses to the stress tasks were *positive*, all of the significant correlations between indignation expressions and cardiovascular responses to the stress tasks were *negative*. More specifically, significant positive correlations were found with fear expression and systolic blood pressure, diastolic blood pressure, beats per minute, and mean arterial pulse ($ps < .05$). Significant negative correlations were found with indignation expression and systolic blood pressure as well as with beats per minute. There were also significant differences between the indignation correlations and the fear correlations (tested with Fisher r-to-z transformations; $p < .05$) on all four of the cardiovascular dimensions.

Finally, as one would expect, all significant correlations emerged during the stress tasks and not during rest periods. Thus, the overall pattern of results for the cardiovascular measures supported the hypotheses.⁵ Rather than a generalized negativity pattern as classic conceptions of stress might predict (Selye 1956), fear is associated with a different pattern than anger and disgust (indignation).

As with the cortisol data, none of the emotion self-report state measures were significantly correlated with the cardiovascular measures. Unlike the cortisol data, none of the self-report trait measures were correlated with the cardiovascular measures.

Do Particular Dimensions of Expression Matter for Predicting Biological Stress Responses?

To address whether particular dimensions of expression predict biological stress responses, we disaggregated the emotion expression data into specific emotions and specific dimensions of frequency, intensity, and duration.⁶ Results of three stepwise regression

equations, one for each emotion, reveal that each emotion did indeed have a particular dimension of expression that mattered most. For anger, the temporal *duration* of expression is the only dimension that mattered when it came to predicting physiological responses. The longer a person displayed anger on the face, the lower the cortisol responses at peak stress ($b = -.23$) and recovery ($b = -.23$). Similarly, the longer a person displayed anger on the face, the lower the systolic BP ($b = -.25$), the lower the diastolic BP ($b = -.44$), the lower the heart rate ($b = -.24$), and the lower the mean arterial pulse ($b = -.44$); all tests run at $p < .03$, 2-tailed.

By contrast, what mattered most with respect to fear was *frequency*. The more often a person displayed a fearful face, the higher the cortisol responses at peak stress ($b = .29$) and recovery ($b = .32$). Similarly, the more frequently a person displayed a fearful face, the higher the systolic BP ($b = .24$), the higher the diastolic BP ($b = .38$), and the higher the mean arterial pulse ($b = .33$). Frequency of fear expression did not predict heart rate at $p < .03$.

For disgust, what mattered most was *intensity*. The more intensely a person displayed disgust on the face, the lower the cortisol responses at peak stress ($b = -.25$) and recovery ($b = -.27$). Similarly, the more intensely a person displayed disgust on the face, the lower the systolic BP ($b = -.21$) and the lower the heart rate ($b = -.29$). Intensity of disgust did not predict diastolic BP or mean arterial pulse. All tests run at $p < .03$, 2-tailed.

Do Optimistic Appraisals Link Facial Expression to Biological Stress Responses?

As noted, previous research demonstrates that fear is associated with pessimistic stress appraisals whereas anger is associated with optimistic appraisals ([Lerner et al 2003](#); [Lerner and Keltner 2001](#)). Although disgust has not been studied in the context of optimism, it has been shown to have the same high appraisals of control, and appraisals of control have been shown to predict optimism ([Lerner and Gonzalez 2005](#); [Lerner and Keltner 2001](#)). Accordingly, we

examined whether chronic tendencies to make optimistic appraisals mediated the observed relations between facial expressions of emotion and biological responses to stress (i.e., cardiovascular and cortisol responses). The main hypothesis is that, in contrast to people who display fear during the task, people who display anger and disgust tend to make optimistic appraisals and these appraisals will mediate the association between facial expression and biological responses to stress.

A variable may be considered a mediator to the extent that it carries the influence of a given independent variable (IV) to a given dependent variable (DV). Following [Baron and Kenny \(1986\)](#), before mediation can be said to occur, at least the following four conditions must be met. (1) The IV significantly affects the DV in the absence of the mediator. (2) The IV significantly affects the mediator. (3) The mediator has a significant unique effect on the DV, and (4) the effect of the IV on the DV significantly shrinks upon the addition of the hypothesized mediator to the model.

Table 3 details the process used to test whether optimistic appraisals link facial expression to biological stress responses. Column 1 in Table 3 demonstrates that the IV (facial expressions of fear frequency, anger duration, or disgust intensity) is correlated with the DV (cortisol or cardiovascular responses to stress). This step establishes that there is an association that may be mediated. Column 2 in Table 3 demonstrates that the IV is correlated with the mediator (optimistic appraisals as measured by the LOT: [Scheier and Carver 1985](#)). Column 3 in Table 3 demonstrates that the mediator has a significant and unique association with the DV. It is important to note that it is not sufficient just to correlate the proposed mediator with the DV; the mediator and the DV may be correlated because they are both caused by the IV. Thus, the IV must be controlled in establishing the association of the mediator with the DV. Column 4 in

Table 3 demonstrates that the association of the IV with the DV shrinks to nonsignificance in the presence of the mediator.

Meeting these steps does not, however, establish that mediation has occurred. The amount of mediation is defined as the reduction of the association between the IV and the DV in the presence of the mediator (see column 4 in Table 3). To determine whether a mediator actually carries the influence of the IV to the DV a Sobel test can compare whether the reduction of the association between the IV and the DV without the mediator and with the mediator is significant (see column 5 in Table 3). If the Sobel test is significant, one may conclude that the mediator explains a significant percent of the variance between the IV and the DV (column 6 in Table 3). Following the logic outlined above ([Baron and Kenny 1986](#); [Sobel 1982](#)), the path models demonstrated that, for every significant relationship between facial expression and biological responses to stress (column 1), the tendency to make optimistic appraisals explained a significant percent of the variance (column 6).⁷

It is worth noting that a minority of participants expressed fear whereas almost all participants expressed indignation. Thus, the majority of participants have zeros for the fear correlations and regressions. The significance tests for r and b each assume that independent and dependent variables are normally distributed, which the present data therefore violate. The effect is to bias the fear-expression correlations to smaller magnitude, flattening the fit line, and thus creating a more stringent test of the hypothesis. Nonetheless, in order to make sure that the high frequency of zeros for fear did not in any unforeseen way favor our hypothesis, we conducted two sets of additional analyses. In the first set, we recoded the fear expression variable into a dichotomy (i.e., 0 = did not display the emotion versus 1 = did display the emotion) and re-ran all the regressions on this dichotomous variable. We found that all relations still held. For

Displayed Fear, the correlations were as follows: baseline cortisol ($r = .10$, *ns*); peak cortisol ($r = .25$); recovery cortisol ($r = .27$); systolic blood pressure ($r = .21$); diastolic blood pressure ($r = .37$); beats per minute ($r = .15$, *ns*); mean arterial pulse ($r = .28$); all significant $ps < .05$. To address the issue more comprehensively, we ran a set of correlations that included only those participants who showed any fear in the face. The correlations were as follows: baseline cortisol ($r = .10$, *ns*); peak cortisol ($r = .25$); recovery cortisol ($r = .30$); systolic blood pressure ($r = .23$); diastolic blood pressure ($r = .40$); beats per minute ($r = .19$, *ns*); mean arterial pulse ($r = .32$); all significant $ps < .05$. Thus, the sub-sample analysis is consistent with the other analyses in showing that fear expressions predict increasing cortisol and cardiovascular stress responses.

Discussion

The present study examined two prototypical responses to stressful challenges, namely fear versus anger and disgust (indignation), and related them to cardiovascular and hypothalamic-pituitary-adrenocortical (HPA) -axis responses to stress. Results support a hypothesis first ventured by Darwin (1872/1998): that emotion-relevant facial expressions reliably signal biological responses to stressors. This is the first study we are aware of that has tested this hypothesis in adults and related contrasting emotion expressions to both neuroendocrine (HPA-axis) and cardiovascular responses to stress.

Consistently, fear displays were positively associated with cardiovascular and cortisol stress responses, whereas anger and disgust displays were negatively associated with these same outcomes. Results of path-analytic regression models were consistent with the hypothesis that the tendency to make optimistic appraisals mediated the links from facial expression to physiology. This pattern points to the potential adaptiveness of indignant/confrontative responses to certain kinds of stressful events, as opposed to anxious/fearful responses. As

research addressing the role of perceived control in health shows (for reviews, see Seeman 1999; Taylor 2003), perceptions of individual control and certainty tend to be adaptive in situations where the contingencies allow some individual control and predictability.

Aside from the broad patterns, several smaller aspects of the results merit note. First, most of the significant relations emerged at peak stress and recovery, when the emotions were especially engaged, and not at baseline. These patterns suggest that the signaling function of facial expressions may be especially important during stressful times. A second aspect is the fact that self-reported emotional states for the most part did not show these relations (consistent with [Feldman et al 1999](#)), suggesting that facial expressions are distinctive in this signaling capacity.

A third aspect of note is the fact that all significant correlations between anger and fear and biological stress responses emerged with muscle movements in the lower face.⁸ Future studies can examine whether lower face muscles share more connections to biological stress response systems.

Clarifications, limitations, and unanswered questions

On the surface, the results could seem to conflict with research relating dispositional hostility to enhanced stress reactivity and to stress-related disorders, such as coronary heart disease. The present study found no association between Cook-Medley hostility (1954) and biological reactivity. It is notable, however, that a lack of association and even negative associations between hostility and cardiovascular response have been reported by several other researchers (e.g., [Carroll et al 1997](#); [Räikkönen et al 1999](#); [Sallis et al 1987](#); [Smith and Houston 1987](#)). The inconsistent findings for Cook-Medley hostility may be due to its poor internal validity ([Contrada and Jussim 1992](#)).

Measurement issues aside, however, the present results imply the need to expand investigations of anger and biological stress responses by examining anger not merely as a chronic dispositional quality, but also as a situation-specific behavioral response that may be justified and even adaptive under certain circumstances. Whereas behavioral medicine studies have typically examined self-reported *intensity* of a dispositional tendency to experience explosive and violent anger (for example, see Spielberger 1996)⁹, the present results examine the *duration* of situation-specific facial expressions. These differences highlight not only the heterogeneity inherent in anger but also the heterogeneity of anger-evoking situations and interactions thereof (Harmon-Jones et al 2003). It may be that certain kinds of anger are adaptive (and others are not). Specifically, a low intensity facial display of anger may be adaptive in the present study with a pesky experimenter. Feeling a sense of indignation when confronted with annoying badgering can confer a sense of control and can be seen as reasonable. It is probably not adaptive, however, to chronically approach the world with a hostile edge, as is typically assessed in the Cook-Medley Hostility scale (1954).

Another clarification pertains to the overall pattern of data. Although the data show that people who tend to display a preponderance of fear will show different biological profiles than people who display a preponderance of anger and disgust, it is worth noting that fear and anger/disgust expressions were not binary. As one would expect of negative emotions, they co-occurred at low levels among individuals.¹⁰

A limitation of this work is that it did not include a structured interview to assess mental health. Because of this methodology and because the population was relatively healthy, conclusions for clinical populations cannot yet be made with confidence.

Implications

At the broadest level, the results imply the importance of developing biobehavioral models that integrate emotion-specificity into both the appraisal of, and the responses to, stressful circumstances. Contrary to the classic idea of undifferentiated negative affect rising with biological stress responses ([Selye 1956](#)), it now appears that distinct appraisals and corresponding emotional displays signal distinct biological responses (see also [Kemeny 2003](#); [Lovallo and Thomas 2000](#)). Such differentiation may allow for more detailed understanding of the specific affective and stress-reactive dysfunctions implicated in specific pathological states. For example, laboratory studies of high risk samples might focus on emotion-specific responses to social stressors, rather than simply examining the magnitude of stress responses. This type of approach also lends itself to better integration with advances in cognitive and affective neuroscience and the activation of specific neural circuits involved with fear, anger, and disgust, as well as with other specific emotional states.

The results also imply that tendencies to appraise socially stressful situations optimistically or pessimistically may have significance for the pathophysiology of certain affective disorders and their relations to health outcomes. Further research is needed to study such links, as for example, by examining cortical modulation of amygdala responses ([Adams Jr. et al 2003](#); [Hariri et al 2003](#)). Although it has long been known that negative cognitive styles signal vulnerability to depression ([National Institute of Mental Health 2003](#)), the opportunity now exists to identify specific markers in the face for negative cognitive-affective patterns that are associated with heightened physiological responding to stress.

Facial expressions reveal biological responses in two major stress-response systems: the sympathetic nervous system and the HPA-axis. Facial expressions can be assessed from the first

moments to the last moments of life, across cultures, and across social contexts ([Keltner et al 2003](#)). These results therefore open up new opportunities for tracking developmental trajectories in stress responses, for assessing culture-specific appraisal patterns, and for assessing stress responses in naturalistic work and family settings.

Authors' Note

Lani Shiota and Ellen Yeung were expert coders; Dacher Keltner was an expert consultant. We gratefully acknowledge Nalini Ambady, Sheldon Cohen, Jason Dana, Baruch Fischhoff, Dacher Keltner, Lee Kirkpatrick, George Loewenstein, and Karen Matthews for insightful comments.

References

- Adams Jr. RB, Gordon HL, Baird AA, Ambady N, Kleck RE (2003): Effects of gaze on amygdala sensitivity to anger and fear faces. *Science* 300:1536.
- Allen MT, Stoney CM, Owens JF, Matthews KA (1993): Hemodynamic adjustments to laboratory stress: The influence of gender and personality. *Psychosomatic Medicine* 55:505-517.
- Baron RM, Kenny DA (1986): The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* 51:1173-1182.
- Bernstein DA, Allen GJ (1969): Fear survey schedule (II): Normative data and factor analysis based upon a large college sample. *Behavior Research and Therapy* 7:403-408.
- Carroll D, Smith GD, Sheffield D, Shipley MJ, Marmot MG (1997): The relationship between socioeconomic status, hostility, and blood pressure reactions to mental stress in men: Data from the Whitehall II Study. *Health Psychology* 16:131-136.
- Charney DS, Manji HK (2004): Life stress, genes, and depression: Multiple pathways lead to increased risk and new opportunities for intervention. *Science's STKE* 225:re 5.
- Cohen S, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM, Jr (1997): Social ties and susceptibility to the common cold. *Journal of the American Medical Association* 277:1940-1944.
- Cohen S, Tyrrell DA, Smith AP (1991): Psychological stress and susceptibility to the common cold. *New England Journal of Medicine* 325:606-612.
- Contrada RJ, Jussim L (1992): What does the Cook-Medley hostility scale measure? In search of an adequate measurement model. *Journal of Applied Social Psychology* 22:615-627.

Cook W, Medley D (1954): Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology* 38:414-418.

Coryell W, Noyes R, Jr., House JD (1986): Mortality among outpatients with anxiety disorders. *American Journal of Psychiatry* 143:508-510.

Darwin C (1872/1998): *The expression of the emotions in man and animals*, 3rd ed. New York: Oxford University Press.

Derogatis LR, Savitz KL (2000): The SCL-90-R and Brief Symptom Inventory (BSI) in primary care. In Maruish ME (ed), *Handbook of psychological assessment in primary care settings*. Mahwah, NJ, US: Lawrence Erlbaum Associates, pp 297-334.

Dickerson SS, Kemeny ME (2004): Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin* 130:355-391.

Duchenne de Boulogne CB (1862/1990): *The mechanism of human facial expression*. Cambridge: Cambridge University Press.

Ekman P, Friesen WV (1978): *Facial action coding system: A technique for the measurement of facial movement*. Palo Alto, CA: Consulting Psychologists Press.

Ekman P, Levenson RW, Friesen WV (1983): Autonomic nervous system activity distinguishes among emotions. *Science* 221:1208-1210.

Ekman P, Sorenson ER, Friesen WV (1969): Pan-cultural elements in facial displays of emotion. *Science* 164:86-88.

Feldman P, Cohen S, Lepore S, Matthews K, Kamarack T, Marsland A (1999): Negative emotions and acute physiological responses to stress. *Annals of Behavioral Medicine* 21:216-222.

Frasure-Smith N, Lesperance F, Talajic M (1995): The impact of negative emotions on prognosis

following myocardial infarction: Is it more than depression? *Health Psychology* 14:388-398.

Geer JH (1965): The development of a scale to measure fear. *Behavior Research and Therapy* 3:45-53.

Gross JJ, Levenson RW (1995): Emotion elicitation using films. *Cognition and Emotion* 9:87-108.

Hariri AR, Mattay VS, Tessitore A, Fera F, Weinberger DR (2003): Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry* 53:494-501.

Harmon-Jones E, Sigelman JD, Bohlig A, Harmon-Jones C (2003): Anger, coping, and frontal cortical activity: The effects of coping potential on anger-induced left frontal activity. *Cognition and Emotion* 17:1-24.

Keltner D, Bonanno GA (1997a): Facial expressions of emotion and the course of conjugal bereavement. *Journal of Abnormal Psychology* 106:126-137.

Keltner D, Bonanno GA (1997b): A study of laughter and dissociation: Distinct correlates of laughter and smiling during bereavement. *Journal of Personality & Social Psychology* 73:687-702.

Keltner D, Ekman P, Gonzaga GC, Beer J (2003): Facial expression of emotion. In Davidson RJ, Scherer KR, Goldsmith HH (eds), *Handbook of affective science*. New York: Oxford University Press, pp 415-432.

Kemeny ME (2003): The psychobiology of stress. *Current Directions in Psychological Science* 12:124-129.

Kirschbaum C, Pirke KM, Hellhammer DH (1993): The 'Trier Social Stress Test' -- a tool for investigating psychobiological stress responses in a laboratory setting.

Neuropsychobiology 28:76-81.

Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D (2001): How do risk factors work together?

Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry* 158:848-856.

Kubzansky LD, Kawachi I, Weiss ST, Sparrow D (1998): Anxiety and coronary heart disease: A

synthesis of epidemiological, psychological, and experimental evidence. *Annals of Behavioral Medicine Special Issue* 20:47-58.

Lerner JS, Gonzalez RM (2005): Forecasting one's future based on fleeting subjective

experiences. *Personality and Social Psychology Bulletin* 31:454-466.

Lerner JS, Gonzalez RM, Small DA, Fischhoff B (2003): Effects of fear and anger on perceived

risks of terrorism: A national field experiment. *Psychological Science* 14:144-150.

Lerner JS, Keltner D (2001): Fear, anger, and risk. *Journal of Personality and Social Psychology*

81:146-159.

Levenson R (1992): Autonomic nervous system differences among emotions. *Psychological*

Science 3:223-27.

Lovallo WR, Thomas TL (2000): Stress hormones in psychophysiological research: Emotional,

behavioral, and cognitive implications. In Cacioppo JT, Tassinary LG, Bernston GG

(eds), *Handbook of psychophysiology*, 2 ed. Cambridge: Cambridge University Press, pp 342-367.

National Institute of Mental Health (2003): Breaking ground, breaking through: The strategic

plan for mood disorders research (NIH publication 03-5121). Rockville, MD: US

Department of Health and Human Services, National Institutes of Health, National

Institute of Mental Health.

- Quas JA, Hong M, Alkon A, Boyce WT (2000): Dissociations between psychobiologic reactivity and emotional expression in children. *Developmental Psychology* 37:153-175.
- Räikkönen K, Matthews KA, Flory JD, Owens JF (1999): Effects of hostility on ambulatory blood pressure and mood during daily living in healthy adults. *Health Psychology* 18:44-53.
- Sallis JF, Johnson CF, Trevorrow TR, Kaplan RM, Hovell MF (1987): The relationship between cynical hostility and blood pressure reactivity. *Journal of Psychosomatic Research* 31:111-116.
- Scheier MF, Carver CS (1985): Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology* 4:219-247.
- Seeman T (1999): Personal Control, Retrieved March 30, 2005 from UCSF, MacArthur Research Network on SES & Health Web site:
<http://www.macses.ucsf.edu/Research/Psychosocial/notebook/control.html>
- Selye H (1956): *The stress of life*. New York: McGraw-Hill.
- Smith CA, Ellsworth PC (1987): Patterns of appraisal and emotion related to taking an exam. *Journal of Personality and Social Psychology* 52:475-488.
- Smith MA, Houston BK (1987): Hostility, anger expression, cardiovascular responsivity, and social support. *Biological Psychology* 24:39-48.
- Sobel ME (1982): Asymptotic intervals for indirect effects in structural equations models. In Leinhardt S (ed), *Sociological methodology 1982*. San Francisco, CA: Jossey-Bass.
- Spielberger CD (1983): *State-Trait Anxiety Inventory for Adults*. Palo Alto: Mind Garden.
- Spielberger CD (1996): *Manual for the State-Trait Anger Expression Inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources.

Suls J, Wan CK (1987): In search of the false-uniqueness phenomenon: Fear and estimates of social consensus. *Journal of Personality and Social Psychology* 52:211-217.

Taylor SE (2003): *Health psychology*, 5th ed. New York, NY, USA: McGraw-Hill.

Footnotes

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² To address another purpose of the study, participants also completed two projective tasks: (d) telling stories in response to Thematic Apperception Test (TAT) cards and (e) responding to a phrase association test.

³ It is worth noting that self reports of emotional experience at baseline were positively correlated with corresponding facial expressions of target emotions. Self reported *fear* correlated with lower face fear frequency .25 and with lower face fear intensity .22; self reported *anger* correlated with lower face anger frequency .33, with lower face anger intensity .24, and with lower face anger duration .40; self reported *disgust* correlated with disgust frequency .35, with disgust intensity .35, and with disgust duration .35; all $ps < .05$. The self reports of emotional experience for peak stress and for recovery showed small and largely non-significant relations with facial expression. It is not surprising that once social stress tasks commenced in this paradigm, self reports and facial expression lacked coherence. For example, the lack of significant association at peak stress most likely is explained by the fact that the self reports were retrospective and thus subject to error. We did not interrupt the stress tasks for people to concurrently report their feelings.

⁴ There were no sex differences in self reported state emotion and no sex differences in cortisol. There were sex differences in cardiovascular response, as others have found ([Allen et al 1993](#)). Specifically, males exhibited higher systolic blood pressure (SBP) and higher mean arterial pulse (MAP) than did females ($\text{male}_{\text{SBP}} = 138.07$, $\text{female}_{\text{SBP}} = 123.61$: $t(90) = 4.37$, $p <$

.001; $\text{male}_{\text{MAP}} = 101.06$, $\text{female}_{\text{MAP}} = 95.49$: $t(90) = 3.02$, $p < .01$). There were also sex differences in expression. During the stress tasks, males displayed less anger and disgust than did females ($\text{male}_{\text{anger}} = 15.72$, $\text{female}_{\text{anger}} = 23.46$: $t(90) = -2.23$, $p < .05$; $\text{male}_{\text{disgust}} = 2.18$, $\text{female}_{\text{disgust}} = 4.97$: $t(90) = -2.30$, $p < .05$).

⁵ One alternative possibility is that the changes in cardiovascular and cortisol response are driven by performance on the stress tasks, and that the facial displays of emotion are epiphenomenal. To explore this possibility, we scored each participant's performance (i.e., number of errors and the number of correctly counted numbers) and re-calculated the correlations described above, partialling out performance. All significant results held even after controlling for performance.

⁶ As a conservative measure, we adopted a more stringent p value (.03, 2-tailed) in order to account for the increased number of tests.

⁷ A limitation in the data merits note. Ideally for mediational analyses one would like a temporal sequence in which the hypothesized mediator is measured in between the independent and dependent variables ([Kraemer et al 2001](#)). In order to avoid interrupting the flow of the laboratory stress tasks, however, the present study required a different sequence. We therefore collected measures of dispositional optimism (the hypothesized mediator) prior to the laboratory session. Individuals who scored high on the measure are assumed to optimistically appraise the events throughout the lab tasks whereas people who scored low are not. The full statistical pattern one would want is obtained, but the study design implies the need to replicate the pattern with a different temporal sequence.

⁸ It is worth noting that EMFACS codes for each emotion contain at least one reliable -- i.e., non voluntary -- muscle movement. For anger, that movement is in the lower face whereas

for fear that movement is in the upper face. Because all significant correlations emerged with muscle movements in the lower face, voluntary regulation of expression is an unlikely cause of the overall data pattern observed here.

⁹ The dispositional tendency to suppress expression of intense angry feelings has also been correlated with cardiovascular reactivity. In such cases, the underlying anger is also described in explosive, violent terms (for review, see Spielberger 1996).

¹⁰ For example, 27% of participants who expressed anger more than once also expressed fear more than once. Future research with clinical samples may follow up on specific profiles showing strong co-occurrence as well as specific profiles showing the no co-occurrence (i.e., pure emotion). For example, individuals who display only anger/disgust versus individuals who display only fear would be interesting to study in terms of clinical implications.

Table 1

Characteristics of Emotional Expressions Displayed

Emotion	Participants Showing Expression	Average Frequency	Average Intensity	Average Duration (s)
Stress Tasks (Averaging across the counting and arithmetic tasks)				
Fear	31	1.87	2.02	1.58
Anger	53	2.63	3.19	2.41
Disgust	30	1.55	1.37	1.37
Resting Periods (Averaging across the three rest periods)				
Fear	8	.29	.38	.47
Anger	13	.41	.81	.36
Disgust	11	.16	.39	.07

Table 2

Physiological and Neuroendocrine Measures During Rest Periods and Stress Tasks

	Mean	Std. Deviation
A) Baseline Cortisol	.21	.11
B) Peak Stress Cortisol	.28	.23
C) Recovery Cortisol	.16	.08
D) Systolic Blood Pressure Across Rest Periods	113.99	11.37
E) Diastolic Blood Pressure Across Rest Periods	66.32	6.31
F) Beats Per Minute Across Rest Periods	68.76	9.04
G) Mean Arterial Pulse Across Rest Periods	84.04	7.71
H) Systolic Blood Pressure Across Stress Tasks	132.58	16.83
I) Diastolic Blood Pressure Across Stress Tasks	78.57	6.79
J) Beats Per Minute Across Stress Tasks	81.71	14.27
K) Mean Arterial Pulse Across Stress Tasks	96.28	9.15
Paired Comparisons	<i>t</i> (91)	<i>p</i>
A vs. B	2.83	.006
B vs. C	7.43	.001
D vs. H	18.60	.001
E vs. I	20.96	.001
F vs. J	13.90	.001
G vs. K	23.36	.001

Table 3

The Tendency to Make Optimistic Appraisals May Mediate Paths Between Physiology and Facial Expressions

Emotion Expression	Physiological Response	1 Expression to Physiology (Beta)	2 Expression to Optimism (Beta)	3 Optimism to Physiology (Beta)	4 Expression to Physiology with Optimism (Beta)	5 Change in Beta	6 Sobel Test Result
Relationships with Cortisol Responses							
Fear Frequency	Cortisol at Peak Stress	.29*	-.24*	-.22*	.15	.14	2.17*
Fear Frequency	Cortisol at Recovery	.32**	-.24*	-.21*	.13	.19	2.35*
Anger Duration	Cortisol at Peak Stress	-.23*	.26*	-.22*	-.07	.16	2.38*
Anger Duration	Cortisol at Recovery	-.23*	.26*	-.21*	-.12	.14	2.41*
Disgust Intensity	Cortisol at Recovery	-.25*	.23*	-.21*	-.10	.15	2.48*
Disgust Intensity	Cortisol at Peak Stress	-.27*	.23*	-.22*	-.08	.19	2.88*
Relationships with Cardiovascular Responses							
Fear Frequency	Systolic Blood Pressure	.24*	-.24*	-.22*	.12	.12	2.22*
Fear Frequency	Diastolic Blood Pressure	.38**	-.27*	-.24*	.19	.19	3.15*
Fear Frequency	Mean Arterial Pulse	.33**	-.26*	-.22*	.12	.21	2.89*

(Table 3 continues)

(Table 3 continued)

Anger Duration	Systolic Blood Pressure	-.25*	.26*	-.26*	-.04	.21	3.15*
Anger Duration	Diastolic Blood Pressure	-.44**	.26*	-.24*	-.15	.23	2.87*
Anger Duration	Beats Per Minute	-.24*	.26*	-.27*	-.03	.17	3.24*
Anger Duration	Mean Arterial Pulse	-.44**	.26*	-.22*	-.15	.16	2.38*
Disgust Intensity	Systolic Blood Pressure	-.21*	.21*	-.26*	-.05	.16	2.39*
Disgust Intensity	Beats Per Minute	-.28**	.21*	-.27*	-.09	.15	2.44*

Note. The tendency to make optimistic appraisals was assessed using the Life Orientation Test (Scheier and Carver 1985). In order to reduce the number of significance tests, the one facial display dimension that mattered most for each emotion was analyzed.

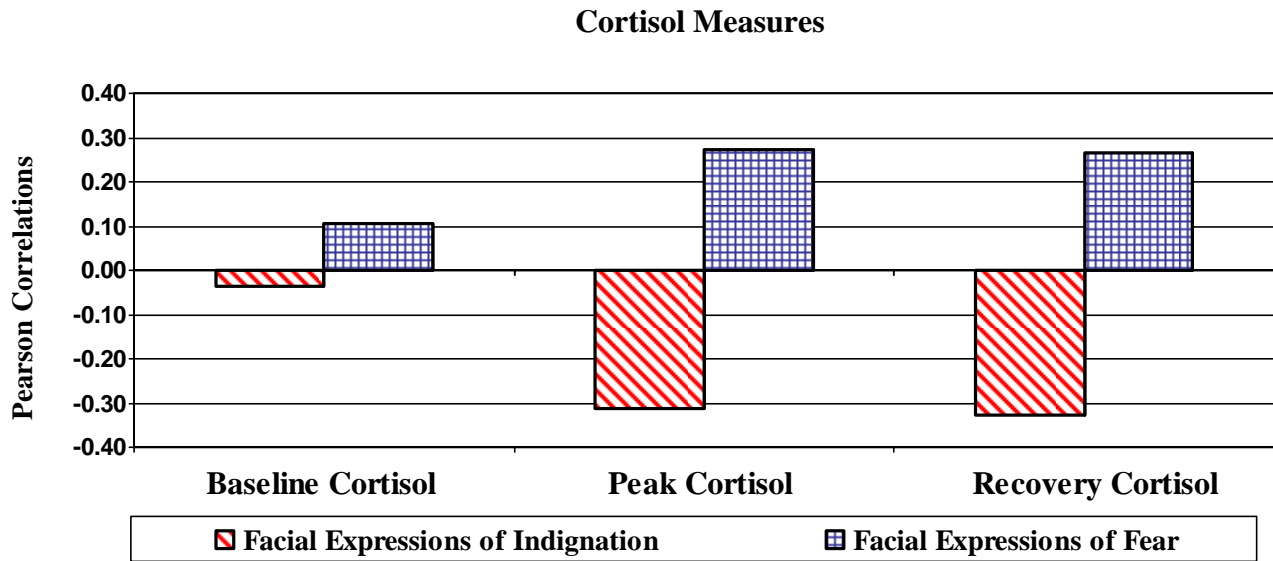
All *p* values are two-tailed. ** = $p \leq .01$. * = $p \leq .05$. ($N = 92$)

Figure Caption

Figure 1. Panel A: Pearson correlations between cortisol at baseline, peak stress, and recovery with emotion expressions of fear and indignation. Panel B: Pearson correlations between cardiovascular responses and emotion expressions of fear and indignation, all measured during stress tasks.

Note. Indignation expressions were computed by averaging anger expressions with disgust expressions.

Panel A



Panel B

