SOCIAL NEUROSCIENCE, 2011, i*First*, 1–11

Psychology Press
Taylor & Francis Group

The empathic, physiological resonance of stress

Tony W. Buchanan[1], Sara L. Bagley[1], R. Brent Stansfield[2], and Stephanie D. Preston[3]

[1]Department of Psychology, Saint Louis University, St. Louis, MO, USA
[2]Department of Medical Education, University of Michigan, Ann Arbor, MI, USA
[3]Department of Psychology, University of Michigan, Ann Arbor, MI, USA

Physiological resonance between individuals is considered fundamental to the biological capacity for empathy. Observers of pain and distress commonly exhibit increases in reported distress, autonomic arousal, facial mimicry, and overlapping neural activity. An important, unstudied question is whether physiological stress can also resonate. Physiological stress is operationalized as activation of the hypothalamic pituitary adrenocortical (HPA) and sympatho-adrenomedullary (SAM) axes. People often report an aversive state resulting from the stress of another, but this could be conveyed through resonating arousal or distress, without activating the physiological stress response. Physiological stress is particularly important to examine since it commonly occurs chronically, with known negative effects on health. Salivary cortisol and salivary alpha-amylase (sAA) were measured in both speakers and observers during a modified Trier Social Stress Test (TSST) to assess activation of the HPA and SAM axes (respectively). Cortisol (but not sAA) responses resonated between speakers and observers. The cortisol response of observers increased with trait empathy and was not related to the speaker's subjective fear or distress. This study provides a novel method for examining physiological resonance, and indicates that we can indeed catch another's physiological stress, suggesting a specific health risk for those in the social network of stressed individuals.

Keywords: Empathy; Contagion; Stress; Cortisol; Salivary alpha-amylase.

The direct contagion of emotion has long interested philosophers and psychologists, suggesting perhaps that we are endowed with an evolved, biological preparedness to feel, understand, and be inspired by the states of others (Hume, 1739–1740/1990; Lipps, 1903; McDougall, 1908/1923; Nietzsche, 1895/1920; Smith, 1759). In evolutionary biology, this contagion or resonance of emotion across individuals is considered particularly adaptive for coordinating the behavior of groups and for fostering the motivated and tailored care of offspring (Bowlby, 1969; de Waal, 2008; Eibl-Eibesfeldt, 1971/1974). In human behavior, emotional contagion is described as a simple state in which one simply "catches" the emotions of another, producing a similar internal state in the observer that resulted directly from the observation (Hatfield, Cacioppo, & Rapson, 1993; Hoffman, 2000). Such contagion, also known as the "resonance" of emotion among individuals, is widespread, occurs early in development, and exists across species (Preston & de Waal, 2002a). However, it is also often derogated as a state that does not require advanced cognitive capabilities and can be associated with a self-oriented response to another's need, motivated more by the desire to reduce one's own distress than genuine concern for the other (reviewed in Eisenberg & Miller, 1987). The perception-action model (PAM) of empathy integrates both views by framing emotional

Correspondence should be addressed to: Tony W. Buchanan, Department of Psychology, Saint Louis University, 221 N. Grand Blvd., St. Louis, MO 63103, USA. E-mail: tbuchan7@slu.edu

We thank Michelle Hendricks, Dana Hamann, and Chris Schmank for help collecting data, and Clemens Kirschbaum for analyzing saliva samples. We are grateful for feedback on previous versions of this paper from Alicia Hofelich, James Abelson, Ethan Kross, and two anonymous reviewers. This research was funded by a grant to S.D.P. and T.W.B. from the Templeton Foundation.

www.psypress.com/socialneuroscience
DOI: 10.1080/17470919.2011.588723

contagion as one indication of an adaptive underlying neural architecture in which others' states are automatically mapped onto observers' representations for experiencing that state (Preston & de Waal, 2002b). According to this process view of empathy, this proximate mechanism is assumed critical for most forms of interpersonal understanding, but only produces visible or negative forms of contagion or sympathetic, prosocial responses under particular conditions (see Preston & Hofelich, in press).

Extensive research supports the PAM, with numerous demonstrations that others' distress or pain causes aversive arousal in observers, or activates their own neural regions for experiencing such states (e.g., Decety & Jackson, 2006; Eisenberg & Miller, 1987; Hatfield et al., 1993; Morrison, Lloyd, Di Pellegrino, & Roberts, 2004; Preston & de Waal, 2002b; Singer, 2006). For example, observers who believed themselves to be similar to confederates experiencing pain and pleasure showed concordant increases in autonomic activity, empathy, and helping (Krebs, 1975). Observers of a relationship conflict also showed linked autonomic increases to targets experiencing negative affect, and higher observer–target linkage was associated with greater empathic accuracy in observer ratings of the target's emotion (Levenson & Ruef, 1992). Observers have even shown linked changes in pupil size to sad targets that correlated with the degree of activation in their own brainstem region responsible for pupillary control (Harrison, Singer, Rotshtein, Dolan, & Critchley, 2006). At a specifically neural level, many studies have demonstrated common neural activation between experiencing and observing pain (see reviews in Decety & Jackson, 2006; Singer, 2006) and between imagining self and other experiences of fear and anger (Preston et al., 2007).

However, research has yet to examine the potential resonance of the physiological response to stress between a target and observer. We can surely become unnerved when observing a frightened public speaker or overworked partner. But these experiences may not reflect truly resonating stress, at the level of stress physiology. Elicitation of a true physiological stress response typically requires a combination of motivated performance, uncontrollability, and social evaluation (Dickerson & Kemeny, 2004) that is associated with reactivity in the hypothalamic-pituitary-adrenocortical (HPA) axis, measured through salivary cortisol. Physiological stress also includes a general arousal component that is associated with reactivity of the sympatho-adrenomedullary (SAM) axis and measured through autonomic nervous system indices such as salivary alpha-amylase (sAA) (Granger, Kivlighan, el-Sheikh, Gordis, & Stroud, 2007; Kirschbaum & Hellhammer, 1989; Rohleder & Nater, 2009).

To demonstrate true physiological resonance of stress between two people, a target and an observer, the observer's state must be similar in quality to the target and have resulted directly from the observation (after McDougall, 1908/1923). Thus, if the observer has an aversive state that does not match that of the target, such as feeling aroused, distressed, embarrassed, or irritated, the response is not considered true resonance. Resonance also does not occur if observers come to feel stressed indirectly—for example, from their perceived responsibility to help, through top-down simulation of how they would feel in the target's place, or from a conditioned association with some element of the stimulus (e.g., facial fear, images of bodily harm) (for a similar argument, see Krebs, 1975). To demonstrate true resonance, we need to demonstrate physiological stress of the observer that is *proportional to* that of the target, and not simply due to induced arousal, fear, or distress. Such proportional resonance would be very unlikely if the observer's state were unrelated or elicited indirectly.

Despite extensive evidence for perception-action processes, such as the emotional contagion of arousal and distress, or the common neural activation between experiencing and observing affective states like pain, physiological stress may not resonate for multiple reasons. For one, even in the literature reviewed above, the evidence for direct physiological matching is largely indirect, given that most studies only collect data from observers (not target–observer concordance) because they use distant, hypothetical, or feigning targets, such as pictures of needy children or confederates in pain (e.g., Batson et al., 1988; Eisenberg et al., 1991; Mehrabian & Epstein, 1972). Most studies also only measure autonomic activity, which indicates general arousal, but does not fully reflect physiological stress *per se*.

Stress is differentiable from arousal in a number of ways (Lundberg & Frankenhaeuser, 1980). Arousal can occur during both positive (e.g., happiness) and negative (e.g., anxiety) states (Bradley, Codispoti, Cuthbert, & Lang, 2001), while the physiological stress response occurs most commonly during the motivated performance of an uncontrollable task under conditions of social evaluation (Dickerson & Kemeny, 2004). Physiological stress, particularly when it is associated with an uncontrollable social evaluative challenge or threat, can be specifically linked to activation of the HPA. Physiological stress also includes a general arousal component reflected in activation of the SAM (Granger et al., 2007; Kirschbaum & Hellhammer, 1989; Rohleder & Nater, 2009), but

physiological stress as measured by the HPA is not one and the same as arousal or distress, since, for example, cortisol is not correlated with the degree of purported or self-reported distress and often does not show the same response profile as autonomic nervous system markers to the same event (e.g., with heart rate increasing but cortisol remaining unchanged) (e.g., Abelson, 1989; Buchanan, Bibas, & Adolphs, 2010). Therefore, the differences between stress and arousal are significant enough that we cannot assume that stress would truly resonate at a physiological level.

Stress is a particularly important state in which to examine contagion for multiple reasons. Stress is known to exist chronically in individuals, a problem that may be increasing according to recent large-scale surveys (American Psychological Association, 2010). In addition, the chronic state of stress is associated with severe health consequences, such as decreased immune functioning, increased morbidity, weight gain, trouble sleeping, and increased risk of cardiovascular disease (Cacioppo, 1994; Juster, McEwen, & Lupien, 2010). Individuals who are stressed are also surrounded by coworkers and family members who may not be stressed by their own life situation, but are clearly impacted adversely by social contact with the stressed other. If the stress of another is not only aversive but also directly contagious in the physiological sense, then individuals in a stressed target's social network are also at risk of the common mental and physical health consequences of stress. This is particularly important to examine in critical developmental relationships such as that between stressed parents and their children, because the health consequences of physiological stress are much more severe in early development when they can have long-lasting effects on the way that the nervous system develops to handle fundamental processes like metabolism, attachment, and resilience to adversity (Lupien, McEwen, Gunnar, & Heim, 2009).

To examine the potential resonance of physiological stress, salivary cortisol and sAA were measured in participant speakers and observing experimenters during a modified Trier Social Stress Test (TSST) (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST is a standardized laboratory task that reliably activates the HPA and SAM in participant speakers performing a speech and mental arithmetic task before observing experimenters. We hypothesized that during our empathic TSST protocol (the "eTSST," in which responses are measured from observers as well as speakers), speakers' neurohormonal responses to stress would resonate in the observing experimenters. We also hypothesized that the physiological stress response of observers would be greater in more trait-empathic observers, since trait empathy appears to reflect a higher motivation to attend to the affect of others (Hofelich & Preston, 2011) that should facilitate the perception–action process (Preston & de Waal, 2002b).

METHOD

Participants

The "observers" in this study were 20 student research assistants employed in the laboratory and trained on proper TSST experimenter behavior (after Kirschbaum et al., 1993). Of the 20 observers, approximately half were women (11 women, 9 men; age $M = 21.8$, range = 18–28), and each viewed both male and female speakers across multiple TSST experiments. Approximately two-thirds of the sessions included only one observer during the speech ($n = 112$) and one-third of the sessions included two observers ($n = 40$). Counting each speaker–observer pairing as a separate dyad, even if two observers watched the same speaker, produced a total of 152 speaker–observer combinations from 112 speakers (55 women and 57 men; age $M = 26.5$, range = 18–50). The "speakers" were recruited from the participant research pool of Saint Louis University and received course credit or were recruited from the local community and received payment for participation. All participants, speakers and observers, completed an informed consent document approved by the Institutional Review Board of Saint Louis University.

Procedure

The procedure for our modified empathic TSST included one experimenter, who initially interacted with the speaker, and one or two trained observers (research assistants from the laboratory), who observed the speaker during the TSST. The experimenter welcomed the speaker to laboratory room 1, obtained informed, written consent from the speaker, and initiated the testing procedure. To reduce potential social habituation effects, speakers and observers did not interact before the TSST.

Speakers

Speakers reported to laboratory room 1 between 1200 and 1600, to control for diurnal cycles of cortisol and sAA, and were administered through the phases of the experiment by an "experimenter," a separate research assistant who did not also act

as an observer for that participant. Speakers first spent 10 min completing demographic questionnaires, relaxed for 10 min, and provided pre-TSST measures of baseline affect—Positive Affect Negative Affect Schedule (PANAS); Watson, Clark, & Tellegen, 1988—and saliva samples (collected in Salivette collection tubes; Sarstedt, Rommelsdorf, Germany). After providing samples, speakers received instructions from the experimenter for our modified TSST: They were instructed to imagine being accused of shoplifting and having to defend themselves in front of the store manager. These instructions differ from the original TSST, in which speakers imagine a mock job interview; we selected these procedures after pilot testing determined that they were more effective in eliciting cortisol responses (after al'Absi et al., 1997). Speakers had 5 min to prepare the speech. At the end of the preparation period, speakers moved to laboratory room 2 where the observer(s) were waiting. Upon entering laboratory room 2, the experimenter left the room and the speaker was reminded of the instructions from one observer and was signaled to begin the speech. At the end of the 5-min speech, the observer gave instructions for the mental arithmetic task (serial subtraction of the numeral 13 from 1022). Finally, at the end of the 5-min mental arithmetic task, the observer indicated that the speaker's task was complete, and the experimenter returned and escorted the speaker back to laboratory room 1. Immediately after the task, speakers provided post-TSST PANAS measures; 10 min after the task, speakers provided post-TSST saliva samples.

Observers

Prior to their first testing session, observers completed the Interpersonal Reactivity Index (IRI) (Davis, 1983), a multidimensional trait empathy index with separate subscales for empathic concern, personal distress, perspective taking, and fantasy. Observers reported to laboratory room 2, 15 min after the speakers had arrived in order to avoid any pre-TSST interactions between speaker and observers. Observers provided pre-TSST saliva samples upon arrival at the laboratory, at the same time that the speakers provided their first saliva sample. The observers provided their second saliva sample 10 min after the speaker's completion of the TSST, again, concurrent with the post-TSST sample of the speaker.

Biochemical measurement

After the study, saliva samples were stored at −20 °C until assayed. Cortisol was measured with an immunoassay kit with chemiluminescence detection (CLIA; IBL Hamburg, Germany), and sAA was measured by the quantitative enzyme kinetic method (see Granger et al., 2007; Rohleder & Nater, 2009). Intra-assay and interassay coefficients of variation were less than 10%. The lower sensitivity for cortisol is 0.5 nmol/l; for amylase, 1.5 U/ml.

Data analysis

Because data were collected across multiple TSST experiments, using actual observing research assistants, the sample includes a heterogeneous number of observers per speaker, observations per individual observer, and combinations of males and females; this was accommodated during analysis by using general linear models (GLMs) and weighted regressions that controlled for the number of observations per observer and separately examined effects of observer and speaker sex. All analyses of resonant stress employed post-pre TSST difference scores (for cortisol, sAA, and PANAS). Cortisol and sAA were positively skewed and so were log-transformed to approximate normal distributions as recommended (Lovallo & Thomas, 2000). To test for physiological resonance in observers, separate GLMs were run to examine whether observer cortisol or sAA responses were similar to those of the speakers.

Speaker response was modeled with separate categorical fixed factors for cortisol and sAA (low, neutral, and high speaker response; see Table 1), parsing the separate effects of each on observer responses. Discrete categorization of speakers was used because cortisol and sAA responses were highly leptokurtic (Shapiro–Wilk W test rejected the normality of cortisol and sAA, respectively: kurtosis = 12.0, $W = .827$, $p < .0001$; kurtosis 17.4, $W = .763$, $p < .0001$). Low responders were equal to or less than 1 *SD* below the mean; neutral responders were between negative and positive 1 *SD*; high responders were equal to or above 1 *SD*. Five speakers had missing cortisol data, and nine speakers had missing sAA data and were excluded from these analyses. The number of speaker–observer pairs is higher than the number of speakers in most comparisons (Table 1) because some speakers were observed by more than one experimenter.

Observer identity was included as a random factor reduced with iterative REML to account for the heterogeneous number of observations per experimenter (observers viewed an average of 7.6 sessions, range = 1–34). To examine effects of speaker and observer sex, each comparison was recalculated with sex included as three fixed factors (speaker sex, observer sex, and

TABLE 1
Data on the speakers' response to the TSST for cortisol and sAA: speaker response characteristics. Data show log-transformed cortisol (originally in nmol/l) and sAA (originally in U/ml) response values. Speaker responses were categorized into low (equal to or less than 1 *SD* below the mean), neutral (between negative and positive 1 *SD*), and high (equal to or above 1 *SD*). The number of speaker–observer pairs is higher than the number of speakers when speakers were observed by more than one experimenter

	Speaker response		
Speaker descriptives	*Low*	*Neutral*	*High*
Cortisol			
Number of speakers	11	85	11
Speaker–observer pairs	16	115	15
Mean response	−0.3	0.04	0.7
Range response	−0.4 to −0.24	−0.23 to 0.39	0.4 to 2.07
sAA			
Number of speakers	7	89	7
Speaker–observer pairs	7	125	9
Mean response	−0.55	0.04	0.65
Range response	−0.95 to −0.33	−0.21 to 0.31	0.32 to 1.94

their interaction). To determine whether the speaker's self-reported affect was associated with the degree of physiological change in the observer, similar GLMs were used to examine the fixed effects of speaker changes in positive and negative affect on observer cortisol and sAA (again including observer identity as a random effects factor reduced with iterative REML). To examine effects of observer trait empathy on the observer's response, mean changes in observer cortisol and sAA across all observed speakers were correlated with their trait empathy per subscale, weighting each by their total number of observations. To examine potential effects of habituation in cortisol and sAA reactivity across repeated observations, intercepts and linear slope estimates were calculated for the sample and per individual observer, using GLM with random factors for observer identity and the number of observations nested within each observer. This model included 14 observers after removing six who had fewer than three observations, in order to maximize the reliability of slope estimates. All analyses were conducted with JMP 8.0.1 (SAS Institute, Cary, NC, USA) and R 2.9.1 (R Foundation for Statistical Computing), using the lme4 package (version .999375-31).

RESULTS

Speaker cortisol and sAA responses

Table 1 includes a breakdown of speaker cortisol and sAA response profiles. Speakers showed the expected cortisol increase to the TSST—pre-post repeated-measures ANOVA main effect: $F(1, 103) = 5.2$, $p < .05$—but increases in speaker sAA response were marginal, $F(1, 99) = 2.7$, $p = .11$.

Observer cortisol responses

Observer cortisol responses increased significantly from pre- to post-TSST, $t(147) = 2.77$, $p = .006$ (Figure 1), and increased relative to the amount of cortisol increase in the observed speaker (Figure 2). After accounting for 19% of the variance with observer identity, the response of the speaker predicted a significant amount of the remaining variance in the observer response, $F(2, 129.2) = 3.52$, $p = .03$, $d = 0.26$ (Figure 2). This resonance still existed after controlling for speaker and observer sex, $F(2, 126.4) = 3.72$, $p < .05$, and there were no main effects or interactions with sex—speaker sex: $F(1, 125) = 1.59$, *ns*; observer sex: $F(1, 19.6) = 0.22$, *ns*; interaction: $F(1, 124.6) = 0.52$, *ns*. Observer cortisol responses were not predicted by speaker sAA response, $F(2, 129.4) = 0.88$, *ns*, or speaker self-reported affect (after 15% of the variance explained by observer identity, no significant remaining amount was accounted for by speaker positive or negative affect, $F < 1.3$, *ns*). Even examining individual PANAS adjectives did not reveal any significant associations between observer cortisol response and speaker affect, $t < 1.29$ (for "determined"), *ns*. However, observer cortisol responses were significantly higher in observers with higher self-reported tendencies for empathic concern, $r = .46$, $p < .05$, and perspective taking, $r = .45$, $p < .05$, but *lower* fantasy, $r = -.64$, $p < .005$ (Figure 3). Personal distress was unrelated ($r = .12$, *ns*). Across observers, the

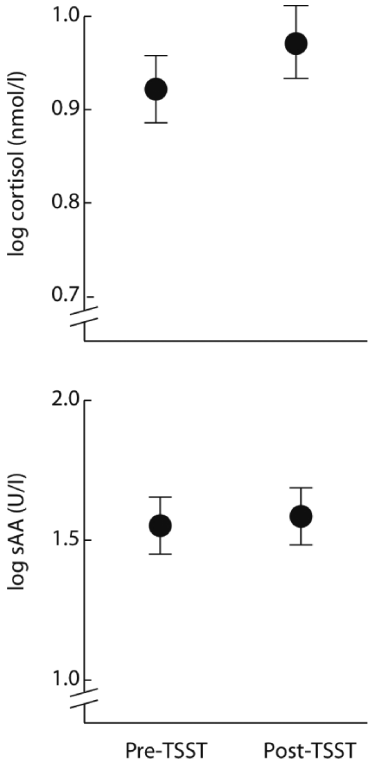

Figure 1. Speakers' mean, log-transformed cortisol (top) and sAA (bottom) responses before and after the stressful TSST speech and mental arithmetic task. Cortisol was originally measured in units of nanomoles per liter (nmol/l), and sAA was originally measured in enzyme units per milliliter (U/ml). Error bars represent 95% confidence intervals.

cortisol responses did not attenuate with experience, with an overall slope estimate that was near zero (estimate −.002 log nmol/l per observation, $SE = .003$, $t = -0.65$, *ns*). However, the distribution of individual observer estimates was right-skewed, with 13 of 14 observers (93%) having negative slopes.

Observer sAA responses

Observer sAA responses increased marginally from pre- to post-TSST, $t(144) = 1.87$, $p = .06$ (Figure 1), but were unrelated to the speaker sAA response (Figure 2). After accounting for 5% of the variance with observer identity, neither speaker sAA nor speaker cortisol response predicted a significant remaining amount of the variance, respectively, $F(2, 129.4) = 1.06$, *ns*; $F(2, 218.9) = 0.45$, *ns*. This lack of resonance was not affected by adding speaker sex and observer sex to the model—cortisol: $F(2, 126.2) = 0.26$, *ns*; sAA: $F(2, 126.5) = 1.40$, *ns*—and male and female observers responded similarly overall—main effect of sex: $F(1, 17.6) = 0.07$, *ns*; interaction with speaker sex:

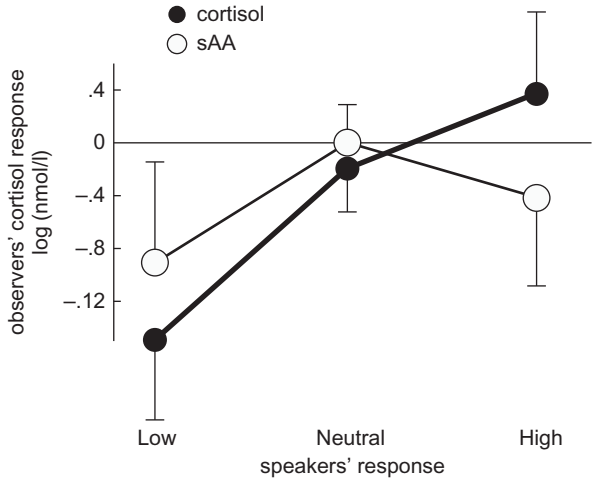

Figure 2. Observers' log-transformed cortisol and sAA responses classified by the level of response in the speaker they were observing (low, neutral, high) for both speaker cortisol (filled circles) and speaker sAA (unfilled circles) with *SE* bars ($n = 138$ observations). Both speaker responses (cortisol and sAA) were included in the GLMs to parse their separate effects on observers.

$F(1, 124.2) = 2.62$, *ns*. However, both male and female *observers* responded marginally stronger to female than male speakers, $F(1, 124.5) = 3.53$, $p = .06$; least squares mean response to females = 0.03, $SE = 0.05$; to males = −0.04, $SE = 0.05$, $d = 0.12$. In contrast to the lack of relationships between observer cortisol and speaker affect reported above, observer sAA responses *did* increase with speakers' self-reported increase in negative affect from the task. After accounting for 5% of the variance with observer identity, increased negative affect explained a significant remaining amount, $F(1, 141.9) = 3.97$, $p < .05$; parameter estimate .0078, $SE = 0.0039$). There were no such effects for positive affect, $F(1, 139.8) = .13$, *ns*. Similar to cortisol, increases in observer sAA were significantly higher for observers with higher tendencies for empathic concern, $r = .45$, $p < .05$, and marginally higher for perspective taking, $r = .42$, $p < .10$ (Figure 3). Fantasy ($r = -.28$, *ns*) and personal distress ($r = .16$, *ns*) were unrelated. Across observers, the sAA responses did not attenuate with experience, with an overall slope estimate that was near zero, estimate .001 log(U/l) per observation, $SE = 0.006$, $t = 0.12$, *ns*. Again, the distribution of individual observer estimates was right-skewed, but this time with only 7 of the 14 observers (50%) having negative slopes.

DISCUSSION

This is the first known demonstration of a resonating, physiological response in observers of stress.

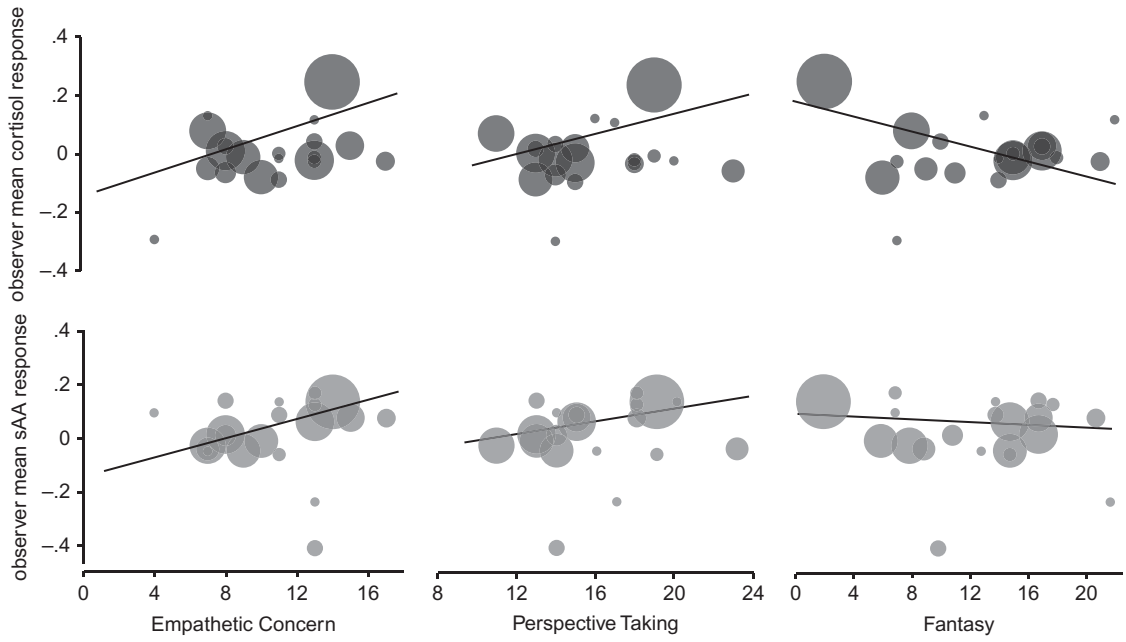

Figure 3. Weighted scatter plots showing associations between mean log-transformed observers' ($n = 20$) cortisol (top) and sAA (bottom) and trait measures of empathy from the Interpersonal Reactivity Index (IRI; see Davis, 1983), including subscales for empathic concern (left), perspective taking (middle), and fantasy (right). Bubble sizes indicate the weight (number of observations: range 1 to 34), and trend lines indicate weighted correlations. Statistics are provided in the text.

Previous research has demonstrated that we can "catch" the facial expressions of others (e.g., Dimberg, Thunberg, & Elmehed, 2000) and become aversively aroused from observing their distress, particularly when assayed through autonomic measurements such as skin conductance, heart rate, or pupil size (e.g., Harrison et al., 2006; Hatfield et al., 1993; Preston et al., 2007). Such empathic resonance has been central to many proximate models of empathy, which assume that intersubjective access to the feelings of others results from the spontaneous mapping of their perceived states onto our own representations for experience (e.g., Gallese, 2001; Preston & de Waal, 2002b). However, the physiological resonance of stress has not been demonstrated. In fact, multiple factors suggest that physiological stress would not resonate between individuals, including the difficulty of eliciting physiological stress in the laboratory, the specific conditions under which it is elicited, and the known dissociation between physiological stress and typical self-reported and autonomic measures of resonance (Dickerson & Kemeny, 2004).

Observers in our eTSST did indeed show increases in their own cortisol response, proportional to that of their paired speaker. This relationship occurred across observers, even before factoring in trait empathy, and was not affected by the sex of the observer or speaker. This suggests that it is a fairly robust phenomenon. Importantly, this effect demonstrates resonance, because it was measured simultaneously in interacting observer–target pairs, with the observer's response linked in both quality and quantity to the specific physiological state of the speaker. Because of this, the activation in observers is highly unlikely to reflect a state that does not match the target or was induced indirectly, or through top-down cognitive processes. Further supporting the empathic nature of the phenomenon, observers with higher self-reported empathic concern and perspective-taking had greater increases in cortisol and sAA to speakers. Interestingly, observers with a higher tendency to engage in fantasy actually had *reduced* cortisol responses, which may reflect the more inwardly directed nature of this particular subscale. Future work can help isolate the specific state elicited in empathic observers, perhaps focusing on measures of vagal tone via heart-rate variability, which has been associated previously with states of compassion and empathic concern (e.g., Eisenberg et al., 1996; Porges, 1999).

The empathic resonance of cortisol is particularly remarkable, given that the HPA response is not always elicited in laboratory stress studies. Indeed, the percentage of participants who show a cortisol response to the TSST, which is the most effective laboratory elicitor of cortisol responses (Dickerson & Kemeny, 2004), is only 70% (Kirschbaum et al., 1993). Further,

the cortisol response is difficult to predict from other measures of emotion that may be easier for observers to detect, such as the degree to which the speaker is distressed or aroused—-e.g., with increases in sweating or respiratory frequency (Bradley et al., 2001; Ritz, George, & Dahme, 2000). Instead, the cortisol response is more strongly associated with factors that are more internally driven, such as the degree to which the target feels threatened or socially evaluated, or perceives the situation as uncontrollable (Dickerson & Kemeny, 2004). Given these qualifications for producing a cortisol response in the laboratory, the finding of such a response from our observers, who were merely observing the performance of others, and not actively engaged in a task themselves, is noteworthy.

Our perception–action framework presumes that the observers centrally processed complex, multimodal indices of the speaker's affect that reflected their actual level of physiological stress (including vocal patterns, posture, facial expressions, the content of their speech), but was not linked to any one feature (e.g., a "stress expression"). According to this view, observers with higher trait empathy would have paid more attention to the affect of the speaker, which in turn enhanced any mimicry and reactivity to the stimulus (e.g., Avenanti, Miniopaluello, Bufalari, & Aglioti, 2009; Hofelich & Preston, 2011; Petrides & Furnham, 2003; Sonnby-Borgström, 2002).

One could take the alternative view that our results were caused by facial mimicry and feedback in our observers. Indeed, prior work has found that facial fear expressions in speakers, when analyzed frame-by-frame by trained coders, does predict their cortisol response (Lerner, Dahl, Hariri, & Taylor, 2007). Thus, a facial feedback model (e.g., Adelman & Zajonc, 1989; Basch, 1983; Freud, 1922/1990; Hatfield et al., 1993) could propose that our observers detected and mimicked the facial fear of the speakers, producing feedback to their own neural regions associated with facial fear, which caused both a resonating fear and proportional activation of the HPA. However, this cascade is unlikely, because none of our speakers' post-TSST ratings of affect, including fear, were associated with the cortisol response of the observers, and neither the recognition of fear in others nor the first-person experience of fear is associated with the release of cortisol in laboratory and controlled field experiments (Buchanan et al., 2010; Dickerson & Kemeny, 2004). Thus, even if our observers did notice and mimic the fear expressions of the speakers, this would not suffice to activate their HPA axis. Self-conscious emotions are also possible sources of speaker–observer concordance, as states like shame and submission are associated across species with easily observed nonverbal behaviors as well as with cortisol reactivity, particularly during threats to the social self (e.g., Carney, Cuddy, & Yap, 2010; Dickerson, Gruenewald, & Kemeny, 2004). Speakers' self-reports of shame (from an item analysis of the PANAS) did increase significantly from pre- to post-stress, but as with fear, this effect was unrelated to speakers' or observers' physiological responses.

Future work must determine which of the many possible cues in speakers were important for predicting their physiological stress response. Additionally, self-report data from the observers can help determine whether their subjective experience of the event reflects or explains the source of their physiological response. Of note, from a process-oriented view of empathy, a resonant state could still be considered to have occurred, even if speakers and observers consciously appraised their state differently. For example, speakers could report feeling motivated and activated by the speech challenge, and observers could directly catch this affect, but appraise and label it with cognitions of worrying about, or feeling uncomfortable for, the speaker. The resonant state is considered to be directly caught from the other, and enhanced in empathic observers who attend more closely to the affective cues of the other (Hofelich & Preston, 2011); however, once engaged, cognitive processes can cause observers to interpret this physiological state in different ways (e.g., Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Jakobs, Fischer, & Manstead, 1997; Russell, 2003), while still reflecting the basic perception–action mechanism.

Even though cortisol and sAA are often intercorrelated in response to stress (e.g., Nater et al., 2005), we did not find the same empathic resonance in sAA. This null effect must be examined further. The lack of resonance in sAA between observers and speakers may reflect the lower effect size of speaker sAA pre- to post-TSST responses or the time course of the post-TSST samples (the peak salivary response for sAA is faster than for cortisol and so our sample at 10 min post-stress may have missed the peak of the sAA response; Rohleder & Nater, 2009). However, these factors are probably not fully explanatory given that many relationships with observer sAA were detected (e.g., greater observer sAA responses for more empathic observers, in response to female speakers, and toward speakers who reported more negative affect). Given the nature of cortisol and autonomic response profiles, and the common divergence of the two systems during laboratory stress (e.g., Abelson, 1989; Buchanan et al., 2010), cortisol is more likely to reflect the speaker's response to an active, social, evaluative challenge (Dickerson & Kemeny,

2004), while sAA reflects the speaker's more transient, observable physical arousal and distress. This is supported by the fact that cortisol was most associated with the PANAS adjective "determined" (but still not significantly), while sAA was significantly associated with speakers' negative affect, and was more responsive to female speakers (who typically display more observable and pronounced responses; Fujita, Diener, & Sandvik, 1991).

The fact that we obtained these results in real laboratory experimenters during a commonly used task has multiple implications. For one, we have demonstrated a novel method for measuring empathic resonance, in an ecologically valid context, that can simultaneously address the interdependent behavior and physiology of both parties. This "empathy TSST" (eTSST) could be particularly useful in future experiments to examine the effects of personality, culture, and context on the empathic response, including not only the physiological, but also the psychological and prosocial elements of empathy. It would be difficult to examine true stress resonance in a neuroimaging environment, particularly since the slow response of salivary cortisol would preclude within-subjects comparisons across targets in a single session. However, once the specific behavioral cues of others' stress are isolated, one could use them to develop shorter, more efficient stimuli that can be administered within-subjects to examine the neural correlates of stress resonance.

Given the contagious effects of stress on observers, research needs to specifically examine the long-term consequences of and habituation to observing others' stress in a research environment. There was some evidence of habituation in observer cortisol responses, though not in sAA responses. The only observer to show an increase in cortisol reactivity over time was an observer with many more observations, indicating either a nonlinear effect over time, or trait effects that moderate this relationship. A more systematic test of habituation is required to examine this important question. Our ongoing work uses research participants specifically recruited as observers for a single session, which not only minimizes the potential for long-term effects but also allows us to test effects that were less controllable with our convenience sample of experimenter observers.

The contagion of stress likely occurs in a variety of common, everyday contexts including in partners and children of stressed family members and coworkers in stressful work environments (Hatfield et al., 1993). Indeed, recent studies reported that the vast majority of children say that they are negatively impacted by their parents' stress (American Psychological Association, 2010) and that low stress is one of the strongest predictors of successful parenting (Epstein, 2010). Such contagious transfer could have significant psychological, physiological, and health consequences (Cacioppo, 1994; Juster et al., 2010). Given that stress is widespread, and has clear negative effects on human health, its physiological resonance should be considered an important public health concern.

Original manuscript received 8 March 2011
Revised manuscript accepted 10 May 2011
First published online day/month/year

REFERENCES

Abelson, J. (1989). Cardiac and neuroendocrine responses to exposure therapy in height phobics: Desynchrony within the 'physiological response system'. *Behaviour Research and Therapy*, *27*(5), 561–567.

Adelman, P. K., & Zajonc, R. B. (1989). Facial efference and the experience of emotion. *Annual Review of Psychology*, *40*, 249–280.

al'Absi, M., Bongard, S., Buchanan, T. W., Pincomb, G. A., Licinio, J., & Lovallo, W. R. (1997). Cardiovascular and neuroendocrine adjustment to public speaking and mental arithmetic stressors. *Psychophysiology*, *34*(3), 266–275.

American Psychological Association (2010). *Stress in America findings*. Washington, DC: American Psychological Association.

Avenanti, A., Miniopaluello, I., Bufalari, I., & Aglioti, S. (2009). The pain of a model in the personality of an onlooker: Influence of state-reactivity and personality traits on embodied empathy for pain. *NeuroImage*, *44*(1), 275–283.

Basch, M. F. (1983). Empathic understanding: A review of the concept and some theoretical considerations. *Journal of the American Psychoanalytic Association*, *31*, 101–126.

Batson, C. D., Dyck, J. L., Brandt, J. R., Batson, J. G., Powell, A. L., McMaster, M. R., et al. (1988). Five studies testing the new egoistic alternatives to the empathy-altruism hypothesis. *Journal of Personality and Social Psychology*, *55*(1), 52–77.

Bowlby, J. (1969). *Attachment*. New York, NY: Basic Books.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion*, *1*, 276–298.

Buchanan, T. W., Bibas, D., & Adolphs, R. (2010). Salivary alpha-amylase levels as a biomarker of experienced fear. *Communicative & Integrative Biology*, *3*(6), 1–3.

Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology*, *31*, 113–128.

Cacioppo, J. T., Berntson, G. G., Larsen, J. T., Poehlmann, K. M., & Ito, T. A. (2000). The psychophysiology of emotion. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (2nd ed., pp. 173–191). New York, NY: Guilford Press.

Carney, D. R., Cuddy, A. J. C., & Yap, A. J. (2010). Power posing: Brief nonverbal displays affect neuroendocrine

levels and risk tolerance. *Psychological Science*, *21*(10), 1363–1368.

Davis, M. H. (1983). Measuring individual differences in empathy: Evidence for a multidimensional approach. *Journal of Personality and Social Psychology*, *44*, 113–126.

Decety, J., & Jackson, P. L. (2006). A social-neuroscience perspective on empathy. *Current Directions in Psychological Science*, *15*(2), 54–58. doi: 10.1111/j.0963–7214.2006.00406.x

de Waal, F. B. M. (2008). Putting the altruism back into altruism: The evolution of empathy. *Annual Review of Psychology*, *59*(1), 279–300.

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality*, *72*(6), 1191–1216. doi: 10.1111/j.1467–6494.2004.00295.x

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 355–391.

Dimberg, U., Thunberg, M., & Elmehed, K. (2000). Unconscious facial reactions to emotional facial expressions. *Psychological Science*, *11*, 86–89.

Eibl-Eibesfeldt, I. (1971/1974). *Love and hate* (G. Strachan, Trans. 2nd ed.). New York, NY: Schocken Books.

Eisenberg, N., Fabes, R. A., Murphy, B., Karbon, M., Smith, M., & Maszk, P. (1996). The relations of children's dispositional empathy-related responding to their emotionality, regulation, and social functioning. *Developmental Psychology*, *32*(2), 195–209.

Eisenberg, N., Fabes, R. A., Schaller, M., Miller, P., Carlo, G., Poulin, R., et al. (1991). Personality and socialization correlates of vicarious emotional responding. *Journal of Personality & Social Psychology*, *61*(3), 459–470.

Eisenberg, N., & Miller, P. A. (1987). The relation of empathy to prosocial and related behaviors. *Psychological Bulletin*, *101*, 91–119.

Epstein, R. (2010). What makes a good parent? *Scientific American Mind, November 4*, 46–51.

Freud, S. (1990). *Group psychology and the analysis of the ego*. (The Standard Edition). New York, NY: W. W. Norton & Company. (Original work published 1922)

Fujita, F., Diener, E., & Sandvik, E. (1991). Gender differences in negative affect and well-being: The case for emotional intensity. *Journal of Personality and Social Psychology*, *61*(3), 427–434.

Gallese, V. (2001). The 'shared manifold' hypothesis: From mirror neurons to empathy. *Journal of Consciousness Studies*, *8*(5–7), 33–50.

Granger, D. A., Kivlighan, K. T., el-Sheikh, M., Gordis, E. B., & Stroud, L. R. (2007). Salivary alpha-amylase in biobehavioral research: Recent developments and applications. *Annals of the New York Academy of Sciences*, *1098*, 122–144. doi: 10.1196/annals.1384.008

Harrison, N. A., Singer, T., Rotshtein, P., Dolan, R. J., & Critchley, H. D. (2006). Pupillary contagion: Central mechanisms engaged in sadness processing. *Social Cognitive and Affective Neuroscience*, *1*(1), 5–17. doi: 10.1093/scan/nsl006

Hatfield, E., Cacioppo, J. T., & Rapson, R. L. (1993). Emotional contagion. *Current Directions in Psychological Science*, *2*(3), 96–99.

Hofelich, A. J., & Preston, S. D. (2011). The meaning in empathy: Distinguishing true empathy from facial mimicry, attention to emotion, and trait empathy. *Cognition & Emotion*. Advance online publication. doi: 10.1080/02699931.2011.559192

Hoffman, M. L. (2000). *Empathy and moral development: Implications for caring and justice*. New York, NY: Cambridge University Press.

Hume, D. (1739–1740/1990). *A treatise of human nature* (7th impression of 2nd ed.). Oxford, UK: Clarendon Press.

Jakobs, E., Fischer, A. H., & Manstead, A. S. R. (1997). Emotional experience as a function of social context: The role of the other. *Journal of Nonverbal Behavior*, *21*(2), 103–130.

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, *35*(1), 2–16. doi: 10.1016/j.neubiorev.2009.10.002

Kirschbaum, C., & Hellhammer, D. H. (1989). Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology*, *22*, 150–169.

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*, 76–81.

Krebs, D. (1975). Empathy and altruism. *Journal of Personality and Social Psychology*, *32*(6), 1134–1146.

Lerner, J. S., Dahl, R. E., Hariri, A. R., & Taylor, S. E. (2007). Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. *Biological Psychiatry*, *61*(2), 253–260. doi: 10.1016/j.biopsych.2006.08.016

Levenson, R. W., & Ruef, A. M. (1992). Empathy: A physiological substrate. *Journal of Personality and Social Psychology*, *63*(2), 234–246. doi: 10.1037/0022–3514.63.2.234

Lipps, T. (1903). Einfühlung, innere Nachahmung und Organempfindung. *Archiv für die gesamte Psychologie*, *1*, 465–519.

Lovallo, W. R., & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology*. Cambridge, UK: Cambridge University Press.

Lundberg, U., & Frankenhaeuser, M. (1980). Pituitary-adrenal and sympathetic-adrenal correlates of distress and effort. *Journal of Psychosomatic Research*, *24*(3–4), 125–130.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445. doi: 10.1038/nrn2639

McDougall, W. (1908/1923). *An introduction to social psychology* (18th ed.). London, UK: Methuen & Co.

Mehrabian, A., & Epstein, N. (1972). A measure of emotional empathy. *Journal of Personality*, *40*, 525–540.

Morrison, I., Lloyd, D., Di Pellegrino, G., & Roberts, N. (2004). Vicarious responses to pain in anterior cingulate cortex: Is empathy a multisensory issue? *Cognitive, Affective & Behavioral Neuroscience*, *4*(2), 270–278.

Nater, U. M., Rohleder, N., Gaab, J., Berger, S., Jud, A., Kirschbaum, C., et al. (2005). Human salivary alpha-amylase reactivity in a psychosocial stress

paradigm. *International Journal of Psychophysiology*, *55*(3), 333–342. doi: 10.1016/j.ijpsycho.2004.09.009

Nietzsche, F. W. (1895/1920). *The Antichrist* (H. L. Mencken, Trans.). New York, NY: A. A. Knopf.

Petrides, K. V., & Furnham, A. (2003). Trait emotional intelligence: Behavioural validation in two studies of emotion recognition and reactivity to mood induction. *European Journal of Personality*, *17*(1), 39–57.

Porges, S. W. (1999). Emotion: An evolutionary byproduct of the neural regulation of the autonomic nervous system. In C. S. Carter, I. I. Lederhendler, & B. Kirkpatrick (Eds.), *The integrative neurobiology of affiliation* (pp. 65–81). Cambridge, MA: MIT Press.

Preston, S. D., Bechara, A., Damasio, H., Grabowski, T. J., Stansfield, R. B., Mehta, S., et al. (2007). The neural substrates of cognitive empathy. *Social Neuroscience*, *2*(3–4), 254–275. doi: 10.1080/17470910701376902

Preston, S. D., & de Waal, F. B. M. (2002a). The communication of emotions and the possibility of empathy in animals. In S. Post, L. G. Underwood, J. P. Schloss, & W. B. Hurlburt (Eds.), *Altrusim and altruistic love: Science, philosophy, and religion in dialogue* (pp. 284–308). Oxford, UK: Oxford University Press.

Preston, S. D., & de Waal, F. B. M. (2002b). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences*, *25*(1), 1–71.

Preston, S. D., & Hofelich, A. J. (in press). The many faces of empathy: Parsing empathic phenomena through a proximate, dynamic-systems view of representing the other in the self. *Emotion Review*.

Ritz, T., George, C., & Dahme, B. (2000). Respiratory resistance during emotional stimulation: Evidence for a nonspecific effect of experienced arousal? *Biological Psychology*, *52*, 143–160.

Rohleder, N., & Nater, U. M. (2009). Determinants of salivary alpha-amylase in humans and methodological considerations. *Psychoneuroendocrinology*, *34*(4), 469–485. doi: 10.1016/j.psyneuen.2008.12.004

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, *110*(1), 145–172.

Singer, T. (2006). The neuronal basis and ontogeny of empathy and mind reading: Review of literature and implications for future research. *Neuroscience and Biobehavioral Reviews*, *30*(6), 855–863.

Smith, A. (1759). *A theory of moral sentiments*. Oxford, UK: Clarendon Press.

Sonnby-Borgström, M. (2002). Automatic mimicry reactions as related to differences in emotional empathy. *Scandinavian Journal of Psychology*, *43*(5), 433–443.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–1070.