Depressive and Anxious Symptoms, Experimentally Manipulated Acute Social-Evaluative Threat, and Cortisol Reactivity

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Objective: Exposure to social-evaluative threat (SET) can elicit greater physiological responses, including cortisol, compared to non-SET stressors. An individual's level of depressive and anxious symptoms predicts variability in cortisol responses to stressors, and other research suggests that these individual differences may predict vulnerability to social evaluation. The current study integrates both lines of research, testing if there are different relationships between depressive and/or anxious symptoms and cortisol reactivity in the presence or absence of SET.

Methods: Healthy undergraduate students (N = 158, 65% female) were randomly assigned to deliver a speech in the presence (SET) or absence (non-SET) of two evaluators. Salivary cortisol was collected throughout, and self-reported depressive and anxious symptoms were assessed. We hypothesized that in the SET condition, higher levels of depressive and/or anxious symptoms would predict dysregulated cortisol responses compared to lower levels of symptoms and/or assignment to the non-SET group.

Results: In spite of inconclusive p values (which might be attributed to low statistical power), individuals with high depressive or high anxious symptoms appeared to have exaggerated cortisol responses in the SET condition, as indicated by more concave trajectories.

Conclusions: This study suggests that both depression and anxiety could be associated with increased cortisol reactivity to SET.

Key words: social-evaluative threat, cortisol reactivity, depressive symptoms, anxious symptoms, self-conscious cognitions and emotions

Abbreviations: **CES-D** = Center for Epidemiological Studies—Depression Scale, **non-SET** = nonsocial-evaluative threat, **SET** = social-evaluative threat, **STAI-T** = State Trait Anxiety Inventory—Trait, **TSST** = Trier Social Stress Test

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Given that dysregulated cortisol responses to acute stressors predict negative health outcomes (1), it is important to identify situational factors (e.g., social evaluation) and individual differences (e.g., internalizing symptoms) that may affect this psychobiological response. Social evaluation is one context that reliably leads to robust cortisol responses (2), and those with internalizing symptoms have shown increased sensitivity to social threat (3). Furthermore, internalizing symptoms predict dysregulated patterns of cortisol reactivity, although the results have been inconsistent across studies (4–7). Taken together, accounting for the social context of stressors may be key to elucidating the relationship between internalizing symptoms and cortisol reactivity. The current study manipulates social-evaluative threat (SET) and examines whether anxious and depressive symptoms are associated with different cortisol trajectories in different social contexts.

EMOTIONAL AND PHYSIOLOGICAL EFFECT OF SOCIAL-EVALUATIVE THREAT

Humans are motivated to preserve the social self by maintaining social connections and acceptance (2,8); therefore, stressors that threaten the social self or jeopardize this fundamental goal could elicit coordinated emotional and physiological responses. Threats to the social self could include situations that affect social standing (e.g., low social status or dropping social rank) or are characterized by social rejection, evaluation, or criticism (e.g., SET; where the self is or could be judged by others; 2).

There is evidence that SET is one condition that can elicit cortisol reactivity. Dickerson and Kemeny (2) conducted a meta-analysis demonstrating that acute stressors that incorporate SET (e.g., presence of an evaluative audience) lead to greater cortisol responses compared to non-SET stressors without this element. Other studies have experimentally manipulated SET in the lab by randomly assigning participants to deliver a speech and/or math task with or without the presence of an evaluative audience. These studies have demonstrated that SET contexts elicit a robust cortisol response compared to otherwise equivalent stressors without this evaluative component (2,9–11). This research has also found that self-conscious cognitions and emotions (e.g., shame, humiliation, embarrassment) may accompany cortisol responses to SET. Participants in SET conditions typically report greater increases in self-conscious cognitions and emotions compared to non-SET conditions (10,12), and those with greater increases in self-conscious cognitions and emotions have shown greater increases in cortisol. Therefore, increases in self-conscious cognitions and emotions and cortisol reactivity may be a coordinated response to SET.

Recently, Zoccola et al. (13) utilized a sophisticated data-driven approach to build on these findings. Specifically, latent group–based trajectory modeling was used, which identified five cortisol response profiles (including increased reactivity, blunted reactivity, and other profiles), indicating heterogeneity in individuals' cortisol reactivity to the Trier Social Stress Test (TSST; 14). Consistent with theory, socially evaluative stressors, greater perceived evaluation during the TSST, and

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increased self-reported experience of shame during the TSST predicted the most reactive cortisol response profiles. Incorporating both between-group (e.g., SET versus non-SET) and within-group (e.g., individual differences) factors may allow for a nuanced prediction of reactivity profiles.

Social or interpersonal threats may be particularly tied to internalizing symptoms. Social rank theory posits that depressive symptoms may be a response to low social rank and/or feelings of powerlessness (3,15,16); therefore, low social rank may be a precursor for depressive symptoms. Different forms of SET, including interpersonal loss, targeted social rejection, and active social demotion, can lead to depressive symptoms (17–20). Importantly, SET can provoke negative self-related appraisals that give rise to shame and humiliation (10). Slavich et al. (21) and others have demonstrated that this process can predict the occurrence of depressive symptoms (22,23). In sum, social threat, and accompanying emotions, may increase the likelihood of developing depressive symptoms.

Although this research has linked social threat to the onset of symptoms, other work has demonstrated that internalizing symptoms may increase the sensitivity to social threat, for example, leading to greater evaluative appraisals. Rapee and Heimberg (24) note that individuals who experience social anxiety scan the external environment for potential threats while also monitoring aspects of oneself that others may evaluate. Social anxiety has been conceptualized as evaluative or social comparative anxiety, characterized by fear of negative evaluation and social avoidance (25–27). Social anxiety symptoms (26) and depressive symptoms (3,28) have been shown to increase social comparative evaluations, in which the self is judged as inferior to a potential evaluating, rejecting, or attacking audience (29). Taken together, the experience of anxious symptoms and depressive symptoms may increase one's likelihood of experiencing negative social evaluations and continuously monitor for external threats or potentially threat-eliciting situations.

INTERNALIZING SYMPTOMS, SOCIAL EVALUATION, AND CORTISOL REACTIVITY

Studies examining the relationship between internalizing symptoms and cortisol reactivity to social-evaluative stressors have reported mixed results. For example, *blunted* cortisol reactivity has been observed following an acute social stressor for individuals experiencing depressive symptoms (4) (meta-analysis of seven studies with 196 participants), social anxiety symptoms (30,31), and anxious, depressive, or comorbid symptoms (5). On the other hand, other studies have reported *exaggerated* cortisol reactivity to acute social stress for individuals experiencing depressive symptoms (32) or social phobia (33). Additionally, others have found no relationship between cortisol reactivity and depressive symptoms (6) or comorbid social anxiety and depressive symptoms (31). These studies have utilized different stressors—some were socially evaluative, whereas others were not—but they also varied along other important dimensions (e.g., whether participants had a clinical diagnosis, or symptoms were assessed). Therefore, the presence and direction of relationships between anxious and/or depressive symptoms and cortisol reactivity are unclear; it is possible that considering the social context of the stressor could help delineate how and when associations may emerge.

Only two studies, thus far, has utilized a social-evaluative stressor manipulation when examining the impact of depressive symptoms on cortisol responses to test whether cortisol levels may differ based on the social context of the stressor. Morris et al. (34) randomly assigned remitted-depressed participants and never-depressed participants to complete a SET or non-SET speech and mental arithmetic stressor. Consistent with past research, cortisol responses were significantly higher in the SET condition compared to the non-SET condition. Furthermore, interaction effects (SET condition only) indicated that the remitted-depressed group had lower cortisol responses compared to the never-depressed group. These results suggest that SET (versus non-SET) stressors appear to lead to higher cortisol responses following an acute stressor for healthy individuals, whereas individuals who were previously depressed appear to have exhibited blunted cortisol responses in both conditions.

Wingenfeld (35) also examined between-group differences in cortisol reactivity, specifically investigating whether women who met the criteria for major depressive disorder or women who experienced abuse before the age of 18 would predict exaggerated cortisol reactivity following a randomly assigned acute stressor (TSST and Placebo-TSST). Cortisol levels increased for those who went through the SET stressor (TSST) but not for those in the non-SET condition (Placebo-TSST), and participants that met the criteria for current major depressive disorder (but no history of childhood abuse) exhibited lower cortisol in the TSST condition compared to the Placebo-TSST condition.

Taken together, these studies indicate that the social context may be important for understanding internalizing symptoms and cortisol reactivity, but more research is needed to clarify the nature and direction of these effects, particularly for those with depressive or anxious symptoms (rather than clinical diagnoses). Further, given that individuals with anxious symptoms and depressive symptoms tend to experience increased self-conscious cognitions and emotions (e.g., social threat and greater self-conscious cognitions and emotions may increase the likelihood of exacerbated internalizing symptoms; 19,28) and cortisol reactivity (10), self-conscious cognitions and emotions may function as a mediator between internalizing symptoms and cortisol reactivity.

THE PRESENT STUDY

The current study tested whether there are different relationships between anxious and/or depressive symptoms and cortisol reactivity in the presence or absence of SET. We assessed levels of depressive and anxious symptoms and randomly assigned participants to a social-evaluative or nonsocial-evaluative stressor condition. Based on previous research and due to the discrepancies in previous findings, we hypothesized that those with higher levels of depressive symptoms and assignment to the SET condition will show more dysregulated cortisol responses compared to those with lower levels or those in the non-SET condition (we did not hypothesize a direction for this relationship). Further, we hypothesized that exposure to SET and experiencing higher levels of anxious symptoms will predict exaggerated cortisol responses compared to those with lower levels of anxious symptoms or those in the non-SET condition. Finally, given that previous studies

have reported correlations between self-conscious cognitions and emotions and cortisol reactivity to SET (10,36), we examined whether the emotional responses to the stressor mediated the relationship between internalizing symptoms and cortisol reactivity.

METHODS

Participants

Healthy undergraduates were recruited for a study entitled, "Health Responses to Laboratory Tasks." All participants were at least 18 years. To avoid confounds with the cortisol data, participants were ineligible to participate if they were regular smokers, pregnant, or using hormonal contraceptives, or reported any chronic medical or diagnosed psychological disorders (37). Additionally, 10 participants were excluded on the basis of outlying or missing cortisol (see the Data Analysis section), leaving a final sample of 158 (79 participants in each condition). The 158 participants included in analyses (65% female, mean age = 19.9 ± 4.93 years) reported ethnically diverse backgrounds: 33% Southeast Asian, 30% East Asian, 14% European American or White, 12% Central American or Latino, 6% Middle Eastern, 3% African American or Black, and 2% Native American, Islander, or Eskimo.

Procedure

All procedures were approved by the University of California, Irvine's Institutional Review Board. Data were collected from March 2012 to May 2013. All sessions took place from 12:00 to 6:00 PM (to control for diurnal cortisol rhythms) and took approximately 2 hours to complete. Upon arriving at the laboratory, participants provided informed consent and then completed questionnaires and relaxed quietly for the first 40 minutes to habituate to the laboratory environment. Participants were then asked to deliver a speech about why they would be a good job candidate for their dream job (modified TSST; 14). Participants were given 10 minutes to prepare their speech and then they delivered the 5-minute speech. Participants were randomly assigned to one of two conditions.[1] In the social-evaluative condition, the speech was delivered in front of a video camera and in the presence of a two-member audience panel who listened to the speech with stoic facial expressions. In the nonsocial-evaluative threat (non-SET) condition, participants delivered their speech alone in front of a video camera (no evaluators present), and participants were told that the experimenter would not be able to hear what they were saying—only whether they were speaking or not. Therefore, the social nature of the evaluation was manipulated. Following the speech, participants completed additional questionnaires and rested with emotionally neutral magazines for 40 minutes to allow time to capture cortisol reactivity and recovery.

[1]The previously published study (11) reported the results of a 2 × 2 experimental design in which SET and cognitive load were manipulated. In the LOAD condition, participants delivered their speech while a soundtrack of 22 various tones played in the background. Participants were instructed to keep track of how many tones they heard while giving their speech because they would be asked to report their count upon speech completion. Participants in the no cognitive load (non-LOAD) condition did not hear tones or receive these instructions. Woody et al. (11) did not find a main effect for LOAD or a significant interaction between the SET and LOAD conditions indicating that cognitive load did not impact cortisol responses above and beyond social evaluative threat. Due to this finding, the current study collapsed across the LOAD conditions, leaving two experimental conditions, SET and non-SET.

Measures

The present investigation is part of a larger study in which additional measures were included. Only the measures used in the present study's analyses are reported below.

Demographics

Demographic information was collected via a questionnaire (e.g., age, gender, ethnicity, and educational background).

Health Behaviors

Participants self-reported recent engagement and frequency of various health behaviors that may influence cortisol responses (e.g., exercise patterns, prescription and nonprescription medication use, drug and alcohol use, eating behavior, smoking behavior).

Affect

Given that Dickerson (12) and Gruenewald et al. (10) found that emotional states correlated with changes in cortisol levels following social-evaluative stressors, we administered the 20-item Positive and Negative Affect Schedule (PANAS; 38) to assess state affect. Participants reported the degree to which they were currently feeling positive and negative mood adjectives along a five-point scale (1 = not at all to 5 = extremely). Furthermore, the 20-item PANAS scale was supplemented with the sadness and fear subscales from the longer 60-item version (38) to assess these additional emotions. Fear was assessed summing "afraid," "scared," and "frightened" items, whereas sadness was assessed summing "sad," "blue," and "downhearted" items. Self-conscious emotions were assessed with the "ashamed" item, plus three additional descriptors ("embarrassed," "humiliated," and "self-conscious"). Six items were also added to assess self-conscious cognitions or cognitive terms used to describe the shame experience ("foolish," "stupid," "defective," "awkward," "exposed," "defeated"; 11,38). Because the post-task self-conscious cognitions and emotions were highly correlated ($r(158) = 0.87$, $p < .001$), the scales were combined into one composite measure of self-conscious states (self-conscious cognitions and emotions; 10 items). This scale had excellent internal consistency in the current study, $\alpha = .92$.

Anxious Symptoms

The State Trait Anxiety Inventory-Trait (STAI-T; 39) is a 20-item self-report inventory that assesses how respondents typically feel in regard to trait anxious symptoms (e.g., "I worry too much over something that really doesn't matter"). The items were measured on a four-point Likert scale ranging from 1 (almost never) to 4 (almost always). This scale had excellent internal consistency in the current study, $\alpha = .92$.

Depressive Symptoms

The Center for Epidemiological Studies-Depression Scale (CES-D; 40) is a 20-item self-report scale that assesses

depressive symptoms in a general population (e.g., "I felt I could not shake off the blues even with the help from my family and friends"). The items were rated on a four-point Likert scale ranging from 0 (rarely or none of the time) to 4 (most or all of the time). This scale had adequate internal consistency in the current study, $\alpha = .76$.

Cortisol

Salivette sampling devices (Sarstedt, Inc., Newton, NC) were used to collect saliva at specific timepoints throughout the sessions (+0, +15, +25, +40, and +55 minutes following speech task instructions). Participants placed the cotton swab in their mouths for at least 2 minutes, allowing it to fully saturate before replacing it in the Salivette. Samples were stored in a freezer at −20°C until the end of the study, at which point the samples were shipped to a laboratory for cortisol measurement. Enzyme-linked immunosorbent assay (IBL-International, Hamburg, Germany) with lower limit of sensitivity of 0.005 μg/dl and, with average inter- and intra-assay coefficients of covariance of less than 10% was used to quantify cortisol concentrations. Due to nonnormality in the cortisol values, they were natural log transformed for analyses.

Analytic Plan

Participants' cortisol values were excluded if the sample taken immediately prior to speech task instructions (+0) was an outlier (+3 SD; $n = 4$) or if participants did not provide the majority of cortisol samples ($n = 6$) due to nonadherence to study protocols. Two other participants who reported oral contraceptive use after consenting were excluded from the current analyses. Thus, final analyses were conducted with a sample size of 158. The statistical significance and direction of the findings did not change based on inclusion or exclusion of these participants. Excluded participants were not significantly different from included participants in terms of demographics (i.e., age, gender), internalizing symptoms (i.e., anxious symptoms and depressive symptoms), or cortisol reactivity.

Cortisol trajectories were modeled using multilevel modeling with maximum likelihood estimation using lme4 (41), lmerTest (42), and bruceR (43) packages in RStudio (44). Multilevel modeling technique allows for the prediction of both within-subject and between-subject differences in physiological responses to the speech task, and to retain data from individuals who may be missing one or more measures of cortisol (45). To better capture reactivity and recovery from the speech task, the cortisol trajectory was examined from +15 to +55 minutes relative to speech task instructions (i.e., at time points + 15, +25, +40, and +55).

Prior to the main analyses that tested the effects of interest, we used statistical modeling to identify which time parameters to include as fixed and possibly random effects in the model. Because participants are expected, in the SET group, to have increased cortisol levels and to afterward recover, we considered both linear and quadratic time effects. To verify that a quadratic effect of time should be considered in later analyses, we first estimated a model with random intercept only, with fixed effects of wake time, sex, and linear and quadratic time effects. To reduce collinearity between random effects, we coded time effects using orthogonal polynomial contrasts (which was then applied throughout). In this first model, both linear and quadratic terms had significant effects (both $p < .001$). We thus decided to retain (linear and) quadratic time effects in all subsequent analyses.

Next, we examined whether a model with random intercept or random slopes (i.e., random intercept, slope linear and slope quadratic) should be included in the base model. The former accounts for within-person variability in baseline cortisol levels (controlling for all fixed effects), whereas the later also accounts for within-person variability in all three cortisol trajectory parameters (intercept, slope linear, slope quadratic). The random slopes model (AIC = −669.51, BIC = −613.85) significantly outperformed the random intercept model (AIC = −417.26, BIC = −384.79; $\chi^2(5) = 262.24$, $p < .001$). The multicollinearity of the effects, examined using the Variance Inflation Factor (VIF), calculated by the R package performance (46), ranged between 1.01 and 1.48, which was considered acceptable (despite a correlation of −0.78 between the linear and quadratic random effects).

We later used and present fixed effect using both random intercepts and random slopes models in parallel, which sometimes produced some discrepant results (in effect significance, but not in effect direction nor substantially in magnitude) and may both be of interest to the reader. However, it should be noted that a) ignoring random slopes in linear mixed models and including only random intercept can result in "catastrophically high type I error rates" (47), and b) the random slopes model is statistically justified here based on model fit comparisons. Therefore, the random intercepts models should be interpreted with more caution.

For results presented in text, time was centered at the first time included in analyses (+0 minute for cortisol). We then tested the interaction between SET and depressive symptoms on the linear (time) and quadratic (time by time) patterns of cortisol responses throughout the study. This resulted in the following interaction terms: SET × depressive symptoms × time by time (all lower-order terms were also maintained). Next, we tested the interaction between SET and anxious symptoms on the linear (time) and quadratic (time by time) patterns of cortisol responses throughout the study. This resulted in the following interaction term: SET × anxious symptoms × time × time (all lower-order terms were also maintained). Observed statistical power was calculated for the different effects of interest using the R package "simr" (48), in which we used 10,000 Monte Carlo simulations (it is reported along with the results). We conducted follow-up analyses, notably to study the effects of interest, controlling for the other trait (i.e., SET reactivity due to depression, controlling for anxiety, and vice versa). To illustrate effect magnitude, we computed plots of model-predicted trajectories by experimental group and by depression (and later anxiety) level (using a pick-a-point approach with the mean ± 1 standard deviation), resulting in four predicted trajectories.

Dickerson et al. (36) found significant effects of self-conscious cognitions and emotions on cortisol reactivity when examining the impact of SET on cortisol levels following an acute stressor. To build on this work, we conducted multilevel mediation analyses (bruceR package; 42) to explore if individual differences in emotional responsivity to SET could be driving differences in cortisol levels. We selected three emotions to

examine: sadness (based on theoretical connections to depressive symptoms; 49), fear (based on theoretical connections to anxious symptoms 50,51), and self-conscious cognitions and emotions (based on theoretical connections to SET-induced cortisol reactivity; 10,34). Given the underlying theory between sadness and depressive symptoms and fear and anxious symptoms, only sadness and self-conscious cognitions and emotions are tested as mediators for the depressive symptoms–cortisol model and only fear and self-conscious cognitions and emotions are tested as mediators for the anxious symptoms–cortisol model (i.e., no model was run, for example, testing if sadness mediates the relationship between anxious symptoms–cortisol associations).

The multilevel mediations were run with the following mediators: post-task self-conscious cognitions and emotions, post-task fear, and post-task sadness (controlling for pre-task levels of the emotions), and general covariates: sex, condition (SET, non-SET), wake time, cortisol time centered (time; linear), and cortisol time centered squared (time by time; quadratic). Multilevel mediation analyses' estimates, SE, and CI were estimated on 10,000 Monte Carlo Quasi-Bayesian samples. Data are available through Open Science Framework (https://osf.io/83tpc/?view_only=e9f9e41bc4a44622a37a1fa88f9ada7b; 52). Experimental protocol was designed in accordance with JARS-Quant Reporting Guidelines (53). The current study was not preregistered in an independent, institutional registry prior to data collection. All statistical tests had a significance level set at $p < .05$ and were conducted as two-tailed tests.

RESULTS

Previously Reported Preliminary Analyses

Woody et al. (11) previously reported that participants in the SET condition felt more evaluated compared to the non-SET condition, indicating that the manipulation was effective in inducing SET. Additionally, random assignment was successful as no differences emerged between conditions on morning wake time, body mass index, caffeine consumption, physical activity, sleep, session start time, use of prescription and nonprescription medications, or baseline cortisol. In line with past research (36), Woody and colleagues (11) also reported that the SET condition had greater cortisol responses and increases in self-conscious cognitions and emotions compared to the non-SET condition.

Depressive Symptoms

We tested whether higher levels of depressive symptoms and assignment to the SET condition showed more dysregulated (blunted or exaggerated reactivity) cortisol responses compared to the non-SET condition and found a marginally significant interaction between experimental condition (SET versus non-SET), depressive symptoms, and the quadratic form of time ($\beta = -0.084$, $t(593.47) = -1.825$, $p = .069$, observed power = 0.46) in the random intercept model, whereas it was nonsignificant ($\beta = -0.081$, $t(152.49) = -1.610$, $p = .109$, observed power = 0.37) in the random slopes model. Although these results are mixed, this indicates that condition and depressive symptoms may interact to predict the curvilinear change in cortisol throughout the experiment. We present predicted trajectories using the random slopes model in Figure 1. In spite of the lack of significance (which might be imputable to the low statistical power), it suggests that in this sample, cortisol trajectories for individuals with high depressive symptoms could be more affected by the SET condition than individuals with low levels of depressive symptoms.

Anxious Symptoms

Similar to what was found for depressive symptoms, there was a marginally significant three-way interaction between experimental condition (SET versus non-SET), anxious symptoms, and the quadratic form of time in the random intercept model ($\beta = -0.042$, $t(610.11) = -1.715$, $p = .087$, observed power = 0.37), which was nonsignificant in the random slopes

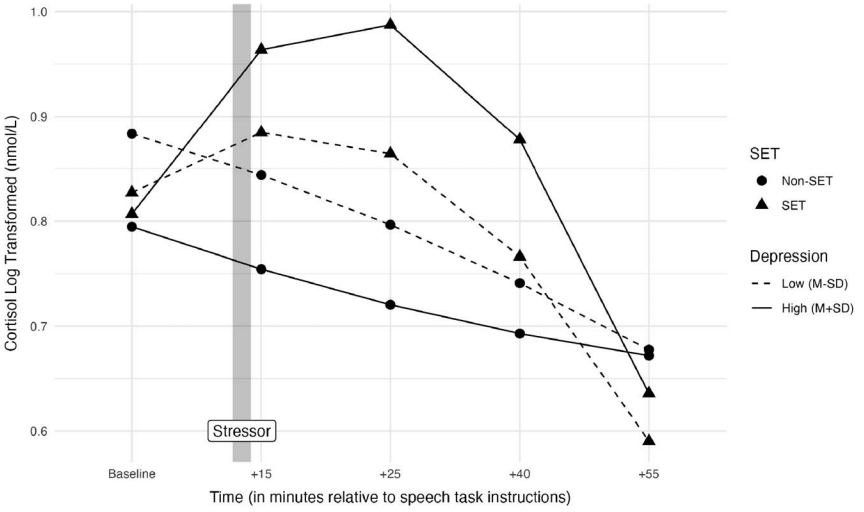

FIGURE 1. *N* = 158. Self-reported depressive symptoms (CES-D) are displayed in this graph as categorical (mean + 1 SD and mean – 1 SD) for visual purposes; the continuous depressive symptoms variable was used in analyses. CES-D = Center for Epidemiological Studies—Depression Scale; SD = standard deviation.

model ($\beta = -0.042$, $t(158.66) = -1.599$, $p = .112$, observed power = 0.34). Like previously, these are mixed results that suggest that the condition and anxious symptoms may interact to predict the curvilinear change in cortisol throughout the experiment. The effect is illustrated using model-predicted trajectories of the random slopes model in Figure 2.

Depressive and Anxious Symptoms

Given the substantial comorbidity between anxious and depressive symptoms, we ran the same model above with depressive and anxious symptoms included to examine whether there is a significant interaction between internalizing symptoms and cortisol reactivity. The SET × depressive symptoms × anxious symptoms × time × time interaction was not significant in the random intercept model ($\beta = -0.002$, $t(595.39) = 0.569$, $p = .569$) or the random slopes model ($\beta = -0.002$, $t(155.94) = 0.431$, $p = .667$). This indicates that the interaction of symptom by experimental effect (time × condition) did not depend on the other symptom levels (e.g., depressive symptoms does not depend on anxious symptoms levels, and anxious symptoms does not depend on depressive symptoms levels).

We also reran the models that investigated the effect of depression and anxiety on cortisol reactivity to SET, but this time each symptom controlling for the other. Controlling for anxiety, there was no significant interaction between depressive symptoms and experimental condition (SET and non-SET) on the quadratic form of time in the random slopes model ($\beta = -0.085$, $t(154.53) = -1.637$, $p = .104$), although the effect was marginally significant in the random intercept model ($\beta = -0.085$, $t(593.52) = -1.827$, $p = .068$). Controlling for depressive symptoms, there was no significant interaction between anxiety symptoms and experimental condition (SET and non-SET) on the quadratic form of time in the random slopes model ($\beta = -0.041$, $t(154.35) = -1.505$, $p = .134$) or random intercept model ($\beta = -0.041$, $t(594.07) = -1.625$, $p = .105$). Overall, these results are consistent with those obtained when studying depressive and anxious symptoms without controlling the other trait.

Self-Consciousness, Fear, and Sadness: Multilevel Mediations

Given that individuals with depressive and/or anxious symptoms undergoing a social-evaluative stressor appeared to show greater cortisol reactivity, we conducted ancillary mediation analyses to examine how depressive or anxious symptoms influence overall cortisol levels through changes in different emotional reactions to the stressor (i.e., sadness, fear, and self-conscious cognitions and emotions). These analyses controlled for wake time, sex, experimental condition, and linear and quadratic time parameters, as well as baseline levels of the emotions studied by including pre-stressor measures. Like before, both random intercept and random slopes (linear and quadratic) models were used each time. However, because the effect estimates, z values, and p values were the same when rounded at the third digit, we only reported them once. To note, for substantive reasons, self-consciousness and sadness were considered as mediators for the effect of depression, whereas self-consciousness and fear for anxiety.

Depressive Symptoms Multilevel Mediations

For self-conscious cognitions and emotions, the indirect path was significant in both the random intercept and random slopes model (estimate = 0.002, $z = 3.824$, $p < .001$, Monte Carlo CI = 0.001 to 0.003), with a nonsignificant direct effect in both models (estimate = −0.000, $z = -0.152$, $p = .880$, Monte Carlo CI = −0.004 to 0.003). This suggests a total mediation, such that higher self-reported depressive symptoms were associated with higher self-conscious cognitions and emotions, which in turn predicted higher cortisol overall. For sadness, the indirect and direct paths were both not significant (both $p > .10$), indicating that sadness does not mediate the relationship between depressive symptoms and cortisol reactivity. In sum, self-conscious cognitions and emotions totally mediated the relationship between depressive symptoms and cortisol levels, but sadness did not mediate this relationship.

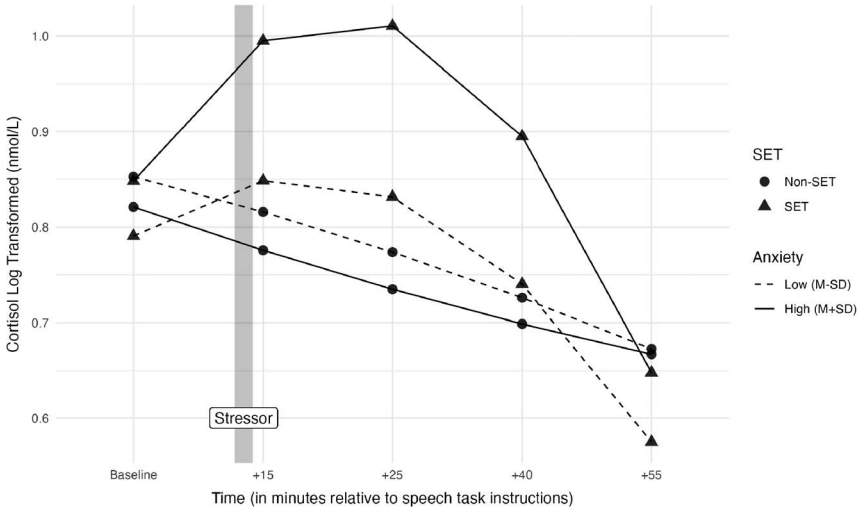

FIGURE 2. $N = 158$. Self-reported anxious symptoms (STAI-T) are displayed in this graph as categorical (mean + 1 SD and mean – 1 SD) for visual purposes; the continuous anxious symptoms variable was used in analyses. STAI-T = State Trait Anxiety Inventory—Trait; SD = standard deviation.

Anxious Symptoms Multilevel Mediation

For self-conscious cognitions and emotions, the indirect path (estimate = 0.001, $z = 2.995$, $p = .003$, Monte Carlo CI = 0.000–0.002) and the direct path (estimate = 0.004, $z = 3.558$, $p < .001$, Monte Carlo CI = 0.002–0.005) were both significant, in both random intercept and random slopes models. This suggests a partial mediation. Regarding fear, the indirect path (estimate = 0.002, $z = 5.211$, $p < .001$, Monte Carlo CI = 0.001–0.003) and the direct path (estimate = 0.002, $z = 2.337$, $p = .019$, Monte Carlo CI = 0.000–0.004) were both significant, in both random intercept and random slopes models, also indicating a partial mediation. In sum, self-conscious cognitions and emotions and fear partially mediated the relationship between anxious symptoms and cortisol levels.

DISCUSSION

The current study tested whether there was variability in cortisol trajectories for individuals experiencing depressive and/or anxious symptoms following an acute stressor and whether this would be dependent on its social-evaluative nature. We randomly assigned participants to either the SET (presence of an audience) or non-SET (no evaluative audience) condition and assessed cortisol reactivity as well as depressive and anxious symptoms. The observed power for the effects of interest was low, and the p values for these effects provided mixed findings, which notably depended on whether random slopes were included or not. Nevertheless, the results suggest that there could be interactions between experimental condition, depressive and anxious symptoms, and quadratic time effects. In other words, and as can be more clearly observed in the plots, higher levels of depressive (and anxious) symptoms seem to be associated with an exaggerated cortisol response in the SET condition. Furthermore, multilevel mediations demonstrated that higher levels of depressive symptoms were associated with increased self-conscious cognitions and emotions, which in turn predicted elevated cortisol overall. These findings indicate that considering the social-evaluative context of the stressor and self-conscious cognitions and emotions is critical for understanding the relationship between internalizing symptoms and cortisol reactivity.

Previous research has demonstrated that social-evaluative threat elicits greater increases in cortisol reactivity compared to non-SET conditions. The current study expands on this work by identifying how individual differences (i.e., depressive and anxious symptoms) that may increase vulnerability to emotional and physiological changes to SET. Specifically, heightened levels of depressive and anxious symptoms were marginally associated with increased cortisol reactivity following a SET stressor, but not for a non-SET stressor. This is aligned with other research showing interrelationships between internalizing symptoms and the stressor context. For example, depressive and anxious symptoms can increase sensitivity to social threat, including greater negative social-comparative evaluations (3,24). This sensitivity in terms of social evaluation may extend to physiological reactivity, given our findings that internalizing symptoms predict exaggerated cortisol responses to SET. There also could be reciprocal relationships between internalizing symptoms and sensitivity to social threat, as other work has shown that greater appraisals and perceptions of social threat can lead to depressive symptoms (20,21). Future research should further examine cognitive appraisals underlying this potential sensitivity to social threat, and explore the causal relationships between internalizing symptoms, sensitivity to social context, and physiological responses.

Previous studies that have examined the association between internalizing symptoms and cortisol reactivity have found different patterns of results, including exaggerated (32), blunted (4), and lack of an association with reactivity (6) following acute stressors. Our findings suggest that the social context of the stressor may contribute to understanding when and how associations between internalizing symptoms and cortisol reactivity emerge. Other avenues for future research could explore the impact of symptom severity or the presence or absence of psychological trauma (54) on cortisol reactivity providing deeper insights into the relationship between internalizing symptoms and cortisol reactivity. In addition, nonclinical versus clinical symptomatology has shown to predict differing levels of cortisol reactivity (4,5,31,34,55). It would be beneficial for future studies to specifically investigate how variations in nonclinical and clinical symptomatology contribute to distinct patterns of cortisol reactivity, thus providing a more nuanced understanding of the underlying mechanisms and implications for targeted interventions.

Given that emotions have strong theoretical connections to internalizing symptoms, we examined if individual differences in emotional reactivity may be leading to unique changes in cortisol reactivity for higher levels of depressive and anxious symptoms above and beyond the impact of SET. Drawing on literature that depressive symptoms are associated with the experience of self-conscious cognitions and emotions (3,36) and that increases in self-conscious cognitions and emotions are correlated with greater cortisol reactivity (10,36), we tested if pre versus post-stressor changes in self-conscious cognitions and emotions mediated the relationship between depressive symptoms and cortisol levels, controlling for (notably) the SET condition. As a comparison, we also tested if sadness could be a mediator, given the theoretical links between this emotion and depressive symptoms (49). We found a total mediation for self-conscious cognitions and emotions and depressive symptoms, indicating that depressive symptoms only predicted cortisol levels when there was an increase in self-conscious cognitions and emotions. Sadness did not mediate this relationship, demonstrating that another negative emotion often associated with depressive symptoms is not explaining the pattern of findings. This could be one reason why the previous literature examining the relationship between depressive symptoms and cortisol levels has found conflicting results—the emergence of exaggerated reactivity among those with higher levels of depressive symptoms could hinge on their experience of self-conscious cognitions and emotions.

One potential mechanism linking exaggerated cortisol reactivity for individuals presenting with increased depressive symptoms may be rumination, or repetitive negative thinking (56). Studies have demonstrated that social-evaluative conditions are more likely to elicit rumination compared to non-SET conditions when rumination is assessed 10 minutes (54) and up to 3–5 days (57) following the stressor, which is consistent with other research linking SET and rumination (58–60). Further, others have demonstrated (57) through mediation analyses that this increase in rumination under SET is uniquely

driven by increases in self-conscious cognitions and emotions (and not other negative emotions). Putting these findings that depressive symptoms are associated with higher levels of rumination (for a review, see 57) that rumination is associated with cortisol reactivity (54) together, a possible pathway is that internalizing symptoms lead to higher levels of self-conscious emotions, which in turn are associated with greater increases in cortisol and rumination. Future research should unpack these potential interrelationships between the social context, cognitive and affective processes, and physiological reactivity.

For anxious symptoms, we found that both fear and self-conscious cognitions and emotions partially mediated the relationship between symptoms and cortisol levels. The work of McLaughlin and Hatzenbuehler (61) on anxiety sensitivity, the extent to which one believes that anxious symptoms have negative consequences, may help explain these effects. A core component of anxiety sensitivity is fearing socially observable anxious symptoms (e.g., sweating, mind going blank, shortness of breath), which is thought to result from a fear that anxious symptoms will lead to social evaluation or rejection (62). Therefore, social-evaluative conditions may amplify anxiety sensitivity. Social-evaluative experiences may increase anxiety sensitivity such that one experiences increased thoughts about causes/consequences of anxious symptoms and social fears (61). It is plausible that an individual with increased anxiety and/or anxiety sensitivity may experience social evaluative stressors with higher levels of fear; because the physiological experience of fear in some contexts has shown to lead to cortisol reactivity (63), higher levels of fear resulting from anxiety sensitivity, in turn, could be associated with greater cortisol reactivity. As such, exposure-based interventions may be appropriate for individuals experiencing increased fear during social evaluative stressors with the goal to reduce or ameliorate the impact of anxious symptoms and fear on cortisol reactivity (64).

The potential connection between type of stress, stress response, and internalizing symptoms could be important in terms of health outcomes. Internalizing symptoms have been associated with negative health outcomes including asthma, hypertension, heart disease, and gastrointestinal problems (65). Further, this relationship may be particularly robust for social stressors; chronic interpersonal stress has been shown to predict increased risk of developing depressive symptoms and anxious symptoms, in comparison to non-interpersonal stressors, which have not shown to predict the onset or recurrence of symptoms (66). This dovetails with the results of the current study, which found marginal associations between depressive and anxious symptoms and cortisol reactivity, but only in response to a social evaluative stressor. Given evidence that exaggerated reactivity predicts negative health outcomes (1,32), this could be a mechanism explaining the association between internalizing symptoms and health. Additionally, this highlights that certain stressors may be more likely to lead to negative effects on health, ranging from increased attention to anxious thoughts and physical sensations (e.g., anxiety sensitivity) to exaggerated cortisol reactivity and physical health concerns (e.g., heart disease, asthma etc.).

Limitations

Our study recruited participants who reported a range of depressive and anxious symptoms, rather than recruiting those with diagnosed clinical disorders. Previous evidence demonstrates significant differences in cortisol reactivity for psychiatric patients and healthy controls (67–69); therefore, participants presenting with a history of psychiatric diagnoses may have different patterns of cortisol reactivity to SET versus non-SET contexts. As such, results from the current study can be reasonably generalized to a similar and healthy population with subclinical depressive and anxious symptoms but not to individuals diagnosed with clinical disorders. Future research should build on this work examining a range of internalizing symptoms to examining possible threshold effects of symptoms (subclinical and clinical levels of symptoms) on cortisol reactivity, specifically, whether the severity or chronicity of symptoms predicts different patterns of cortisol reactivity (e.g., exaggerated versus blunted reactivity).

Additionally, the study used a cross-sectional design, which does not allow for examination of causal relationships between internalizing symptoms and cortisol reactivity. We aimed to address some aspects of this limitation by conducting mediation analyses, which examines the impact of a variable on the relationship between other variables. Future research can address the limits of the current study's cross-sectional design by testing whether differences in cortisol reactivity could predict the development of depressive or anxious symptoms over time (e.g., testing reactivity at time 1 and following participants longitudinally), and/or whether differences in reactivity may co-occur with the presence of symptoms.

In this study, the effects of interest had low observed statistical power, which could have prevented us from obtaining clearer results in terms of statistical significance. Future research should recruit larger sample sizes to ensure appropriate power to examine additional interactions, including those probing potential gender differences, and test the replicability of higher-order interactions (e.g., the nonsignificant interaction with both depressive and anxious symptoms). Lastly, the current study only examined cortisol reactivity, but internalizing symptoms and SET have been found to affect other physiological systems, including inflammatory parameters and the sympathetic nervous system (20). As such, future research should examine the interaction between internalizing symptoms, social-evaluative threat, and reactivity in other biomarkers with health-relevant implications.

CONCLUSION

The current study suggests that individuals experiencing more depressive and/or anxious symptoms could exhibit exaggerated cortisol reactivity following a socially evaluative stressor but not for nonsocially evaluative stressors. This indicates that the social context of an acute stressor could affect the relationship between internalizing symptoms and cortisol trajectories. Ancillary analyses also suggested that self-conscious cognition and emotions mediate the relationship between cortisol levels and depressive, as well as anxious, symptoms. This provides potential pathways through which internalizing symptoms interact with social contexts of acute stressors (e.g., self-conscious cognitions and emotions) and lead to increased cortisol reactivity.

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