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An exploratory analysis of the joint contribution of HPA axis activation and motivation to early adolescent depressive symptoms

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Abstract

This study examines the interactive contribution of the hypothalamic-pituitary-adrenal (HPA) axis and approach-avoidance motivation systems to longitudinal changes in depressive symptoms across the adolescent transition. In the summer prior to, or fall of, 4th grade, 132 youth (68 girls; 64 boys; M age = 9.46 years) participated in a social challenge task and reported on their depressive symptoms. In the winter of 6th grade, youth completed a semi-structured interview of depression and a self-report measure of approach-avoidance motivations. Analyses revealed two profiles of risk for adolescent depressive symptoms, with some gender differences: (1) excessive disengagement, reflected in HPA underactivation along with low approach motivation or high avoidance motivation; and (2) excessive engagement, reflected in HPA overactivation along with high approach motivation. This research highlights the importance of a multi-system perspective on development, suggesting that the implications of HPA dysregulation for depressive symptoms are contingent on adolescents' tendencies toward approach versus avoidance.

KEYWORDS
adolescence, cortisol, depression, gender, motivation

1 | INTRODUCTION

The transition to adolescence is a period of heightened risk for depression (Hankin & Abramson, 2001; Rudolph & Flynn, 2014), stemming, in part, from stage-specific perturbations in the arousal and motivation systems undergirding emotion and behavior. In particular, adolescence is marked by enhanced sensitivity of approach and avoidance systems combined with compromised regulation of arousal (Casey et al., 2010; Crone & Dahl, 2012; Ernst, 2014). Despite the growth of complex theoretical models emphasizing the intersection of multiple systems in determining the development of depression across the adolescent transition, most empirical research focuses on these systems in isolation and relies on concurrent designs. This approach limits our understanding of how systems intersect to predict the unfolding of depression across this stage. To address this gap, the present study used a longitudinal design tracking youth from 4th to 6th grade to examine the interactive contribution of arousal (activation of the hypothalamic-pituitary-adrenal [HPA] axis) and motivations (self-reported approach vs. avoidance) to adolescent depressive symptoms. Given evidence for elevated depression risk (Hankin & Abramson, 2001; Rudolph & Flynn, 2014) as well as stronger links between these risk factors and depression (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Llewellyn & Rudolph, 2014; Natsuaki et al., 2009) in adolescent girls compared to boys, we examined gender differences in this joint risk for depression.

2 | HPA ACTIVATION AND DEPRESSION

During adolescence, youth face a range of psychological and social challenges (Rudolph, 2014). How they cope with these challenges depends on their ability to respond adaptively, by reestablishing equilibrium through appropriate and flexible regulation of biological stress response systems, a process called allostasis (Hastings et al., 2011; Marceau, Ruttle, Shirtcliff, Essex, & Susman, 2014; McEwen, 1998). The HPA axis serves as one of the body's key stress-response systems. Activation of this system triggers a cascade of events that begins with the secretion of corticotropin releasing hormone by the hypothalamus and culminates in the release of glucocorticoids (cortisol in humans) by the adrenal glands into the bloodstream. The HPA axis is a slow-acting system that responds to psychological stressors involving novelty, unpredictability, challenge, and social-evaluative threat (Chrousos, Loriaux, & Gold, 1988; Denson, Spanovich, & Miller, 2009; Dickerson & Kemeny, 2004; Lundberg & Frankenhaeuser, 1980). Healthy functioning of this system involves achieving an optimal balance between reactivity and regulation contingent on contextual demands. Although moderate, short-term elevations in cortisol are adaptive in novel or dynamic social environments, providing energy for facing ongoing demands (Weiner, 1992), excessive, repeated, or chronic activation can result in allostatic load, or cumulative "wear and tear," which is linked to a variety of health problems (McEwen & Gianaros, 2011).

Individual differences in sensitivity of the HPA system have been implicated in depression. In particular, some individuals are thought to display genetically or environmentally based abnormalities in the HPA system that make them vulnerable to depressive reactions to stress (e.g., Halligan, Herbert, Goodyer, & Murray, 2007). These atypical stress responses include over-activation (i.e., hyperarousal, indexed by very high levels of cortisol) or under-activation (i.e., hypoarousal, indexed by very low levels of cortisol). In either case, whether individuals mount too intensive a response to stress or an insufficient response to stress, they may be more prone to developing depression. HPA over-activation in response to stress may reflect dysregulated engagement with stressors, perhaps through cognitive perseveration (Denson et al., 2009; Gianferante et al., 2014; Huffziger et al., 2013; Rudolph, Troop-Gordon, & Granger, 2011), negative emotional arousal (Adam, 2006), and sensitivity to social cues, whereas HPA under-activation in response to stress may reflect dysregulated disengagement from stressors, perhaps through decreased energy (Hankin, Badanes, Abela, & Watamura, 2010), lack of motivation, helplessness, and blunting of emotion. Of note, both dysregulated engagement (e.g., rumination, intrusive thinking, emotional, and physiological arousal) and dysregulated disengagement (e.g., avoidance, inaction, emotional numbing) in the face of stress can interfere with effective coping and even exacerbate stress, leading to depression over time (Flynn & Rudolph, 2011). Indeed, research supports the role of both HPA over-activation and HPA under-activation in youth depression, particularly in the context of laboratory-induced or naturally occurring stress (for reviews, see Guerry & Hastings, 2011; Lopez-Duran, Kovacs, & George, 2009).[1]

With regard to over-activation, a number of studies link heightened cortisol reactivity to social stress with concurrent levels of depression (Gunnar et al., 2009, Trier Social Stress Test [TSST] in a mixed-age sample; Hankin et al., 2010, parent-child stressor and modified TSST in 9th graders; Rao, Hammen, Ortiz, Chen, & Poland, 2008, TSST in an adolescent sample) and internalizing symptoms (Klimes-Dougan, Hastings, Granger, Usher & Zahn-Waxler, 2001; Natsuaki et al., 2009, TSST in an adolescent sample). Although longitudinal studies are rare, there is some evidence supporting prospective effects of elevated cortisol reactivity to stress on subsequent depression and associated problems. In one study, Susman et al. (1997) found that heightened cortisol reactivity to a physical stressor (blood draw) predicted depressive symptoms 1 year later in early to mid-adolescents. In another study, Rudolph et al. (2011) found that heightened anticipatory cortisol while awaiting a laboratory peer stressor interacted with exposure to naturally occurring social stress to predict depressive symptoms 1 year later in late childhood. Hyper-activation of the HPA axis to a laboratory social stressor also predicts suicide risk across 3 months in adolescent girls (Giletta et al., 2015).

Although a smaller database, several studies also link attenuated cortisol reactivity to social stress with depression. Hankin et al. (2010) found evidence for hyporeactivity to social stressors in preschoolers with high levels of internalizing symptoms and in 3rd graders with high levels of depressive symptoms. In a sample that included the 3rd graders as well as 6th and 9th graders, Badanes, Watamura, and Hankin (2011) found that exposure to life stress more strongly predicted depressive symptoms across a 1-year period in youth showing attenuated than elevated cortisol (collapsed across five lab assessments that included a social stressor).[2] In a sample of early adolescent girls, Keenan et al. (2013) linked cortisol hyporeactivity to a physical stressor with higher levels of prior and concurrent depressive symptoms and with stability in depressive symptoms over time. Finally, Cicchetti, Rogosch, Gunnar, and Toth (2010) found that depressed children and early adolescents exposed to maltreatment show flat diurnal patterns of cortisol, and Harkness, Stewart, and Wynne-Edwards (2011) found that moderate to severely depressed adolescents exposed to maltreatment show blunted cortisol reactivity to a social stressor.

Several explanations have been proposed to shed light on this conflicting evidence regarding the contribution of HPA over-reactivity versus under-reactivity to depression, including differential patterns of activation associated with age and puberty (Hankin et al., 2010), chronic versus acute depressive symptoms (Booij, Bouma, de Jonge, Ormel, & Oldehinkel, 2013) and severe versus mild symptoms (Harkness et al., 2011), and different social contexts (Harkness et al., 2011; Rudolph et al., 2011). Conclusions from a review by Guerry and Hastings (2011) suggest several directions for resolving discrepant results and expanding our understanding of the role of the HPA axis in depression. First, they highlight the importance of studying cortisol activation in the context of acute psychological stress exposure. Second, they emphasize the use of longitudinal designs to examine the prediction of depressive symptoms over time. Third, they recommend examining how the HPA axis interacts with other risk factors to predict

depression, an area that has received minimal attention. Fourth, they underscore the need to examine gender differences, which has been done in only a handful of studies. The present study reflects an effort to address each of these concerns by: (i) assessing cortisol reactivity to an acute psychological stressor in the laboratory, specifically an in vivo peer stressor, which is likely to be particularly relevant to depression (Rudolph, Flynn, & Abaied, 2008); (ii) examining the contribution of cortisol reactivity to depressive symptoms approximately 2 ½ years later, across the adolescent transition; (iii) exploring the idea that the contribution of cortisol reactivity to depression may depend on youths' approach-avoidance motivations; and (iv) determining whether the interactive effects of HPA activation and motivations are moderated by gender.

3 | MOTIVATION AS A MODERATOR OF THE LINK BETWEEN HPA ACTIVATION AND DEPRESSION

Achieving optimal coordination of the approach and avoidance motivation systems represents another challenge of adolescence (Ernst, 2014). The approach or appetitive (behavioral activation) system modulates cognitive, emotional, and behavioral sensitivity to reward or incentive cues whereas the avoidance or defensive (behavioral inhibition) system modulates cognitive, emotional, and behavioral sensitivity to threat, novelty, punishment, or non-reward cues (Gray, 1991; Lang, 1995; Nigg, 2006). Whereas the behavioral activation system stimulates approach behavior, the behavioral inhibition system stimulates avoidance behavior (Ernst, 2014). Neuroscience models view approach-avoidance tendencies as involuntary reactions supported by distinct subcortical neural systems (Beauchaine, 2001; Beauchaine, Kline, Crowell, Derbidge, & Gatzke-Kopp, 2009; Nigg, 2000). Specifically, activation in the striatum drives reactive approach, whereas activation in the amygdala, hippocampus, and insula drives reactive avoidance (Ernst, 2014; Nigg, 2000, 2006). With appropriate regulation, these motivation systems can be effectively recruited for context-dependent, goal-directed behavior (e.g., engagement in prosocial approach behavior and positive emotions; appropriate avoidance of a dangerous situation; Ernst, 2014). When regulatory control is compromised (e.g., in the context of poor "top-down" executive function), as is often the case in adolescence (Ernst, 2014), one system may dominate over the other, leading to maladaptive cognition, emotion, and behavior (Carver, Johnson, & Joorman, 2008; Ernst, 2014; Nigg, 2000, 2006; Rothbart, Ellis, & Posner, 2004).

Because dysfunction of the HPA axis may reflect a breakdown in regulatory control (e.g., atypical processing in the prefrontal cortex (PFC); Kern et al., 2008), we hypothesized that adolescents' levels of approach versus avoidance motivations would moderate the effects of atypical HPA responses on depression, potentially creating two profiles of risk: (1) an *excessive disengagement profile*, involving youth who experience HPA underactivation in the context of an insensitive approach motivation system or a highly sensitive avoidance motivation system; and (2) an *excessive engagement profile*, involving youth who experience HPA overactivation in the context of a highly sensitive approach motivation system.

3.1 | Excessive disengagement profile

HPA underactivation signals diminished energy or resources for coping with stress (Hankin et al., 2010), perhaps leading to excessive disengagement from stressors in youth who are insensitive to reward cues (i.e., low approach motivation) and particularly attuned to aversive cues (i.e., high avoidance motivation). Individuals with a particularly weak approach orientation show low levels of positive emotions and low engagement in the environment (e.g., anhedonia, apathy, lack of energy, inaction) whereas individuals with a particularly strong avoidance orientation show high levels of negative emotions, vigilance, and withdrawal (Carver et al., 2008; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Nigg, 2006). Moreover, research at multiple levels—behavioral, psychophysiological, neural, and neurochemical—supports the role of an underactive approach system (e.g., Bress, Foti, Kotov, Klein, & Hajcak, 2013; Coplan, Wilson, Frohlick, & Zelenski, 2006; Davidson et al., 2002; Dunlop & Nemeroff, 2007; Forbes & Dahl, 2012; Henriques, Glowacki, & Davidson, 1994) and an overactive avoidance system (Coplan et al., 2006; Forbes, Phillips, Silk, Ryan, & Dahl, 2011; Rudolph, Miernicki, Troop-Gordon, Davis, & Telzer, 2016; Rudolph, Troop-Gordon, & Llewellyn, 2013; Yang et al., 2010) in depression (for a review, see Carver et al., 2008). Thus, immobilization stemming from HPA underactivation along with low approach or high avoidance motivations may block active coping efforts, instead leading to a sense of hopelessness, excessive disengagement from stressors, and consequent depression.

3.2 | Excessive engagement profile

HPA overactivation signals heightened arousal (Chrousos et al., 1988; Dickerson & Kemeny, 2004) and poorer regulation (Kern et al., 2008) in response to stress. For youth who are particularly high in approach motivation, this heightened arousal may lead to dysregulated engagement with stressors. Although high approach motivation can be beneficial, it also can increase risk (e.g., impulsivity) when it occurs in the context of poor regulatory control (Ernst, 2014; Heym, Ferguson, & Lawrence, 2008). Furthermore, it has been suggested that certain aspects of approach motivation may enhance cognitive perseveration, such as rumination (Heym et al., 2008). Thus, arousal stemming from HPA overactivation along with high approach motivation may impede efforts to effectively redirect attention away from stressors (e.g., cognitive reappraisal, distraction), instead leading to negative emotions, excessive engagement with stressors, and consequent depression.

4 | GENDER DIFFERENCES

The sharp increase in depression during adolescence is particularly pronounced among girls (Hankin & Abramson, 2001; Rudolph & Flynn,

2014). Moreover, relative to adolescent boys, adolescent girls show heightened exposure and reactivity to social stressors (Hankin, Mermelstein, & Roesch, 2007; Rudolph, Flynn, Abaied, Groot, Thompson, 2009; Rudolph & Hammen, 1999), which may amplify the adverse effects of a dysregulated HPA system. Indeed, two studies using a concurrent design reveal that cortisol reactivity to social stress is more strongly associated with depression (in a mixed age sample, Gunnar et al., 2009; cf. Hankin et al., 2010) and internalizing symptoms (in an adolescent sample, Klimes-Dougan et al., 2001; Natsuaki et al., 2009) in girls than in boys. Moreover, high avoidance orientation increases vulnerability to depression in girls but not boys (in late childhood, Llewellyn & Rudolph, 2014; Rudolph et al., 2013). Thus, we predicted that the joint contribution of HPA axis activation and motivation to depression would be stronger in adolescent girls than in boys.

5 | STUDY OVERVIEW

The present study used a longitudinal design to examine how HPA activation in the context of a naturalistic laboratory social stressor in 4th grade interacted with adolescent approach-avoidance motivations to predict depressive symptoms in 6th grade. This design enabled us to examine the emergence of depressive symptoms during the early adolescent transition. The study used an ecologically valid social stressor—a conflict-of-interest interaction with an unfamiliar peer—that involved a novel, unpredictable situation with the potential for social-evaluative threat, thereby maximizing the potential for individual differences in HPA axis activation (Dickerson & Kemeny, 2004). We assessed both children's level of pre-task cortisol while awaiting the impending stressor (i.e., after being informed that they would interact with an unfamiliar peer) as well as their task-related reactivity to the stressor. Although most research on biological stress responses focuses on task-related reactivity, increasing evidence underscores the importance of the anticipatory phase of the stress response, particularly in the context of naturalistic stressors (Afifi, Granger, Joseph, Denes, & Aldeis, 2009; Del Giudice, Ellis, & Shirtcliff, 2011; Klimes-Dougan et al., 2001; Laurent, Powers, & Granger, 2013; Stroud et al., 2009). In this case, children's level of pre-task cortisol was presumed to reflect activation while awaiting an impending interaction with an unfamiliar peer, which had the potential to trigger a sense of uncertainty, uncontrollability, and potential social threat. The motivation systems were assessed with a well-validated self-report measure of approach (behavioral activation) and avoidance (behavioral inhibition) motivations, which has established links to neural systems (Amodio, Master, Yee, & Taylor, 2008; Rudolph et al., 2016).

6 | METHOD

6.1 | Participants

Participants were 132 children (66 girls; 66 boys; M age = 9.46 years, SD = 0.33; 72.0% White, 13.6% African American; 8.3% Asian; .8% Native American; 5.3% multiracial) recruited from a larger longitudinal study. Families were economically diverse as reflected in annual income (50.4% under $60,000, 25.6% $60,000—89,999, and 24.0% over $90,000). For the larger study, parent consent forms were given to all eligible 2nd graders across several schools. Of the 725 eligible children, 576 (80%) received consent and participated. Children provided verbal assent at the time of data collection.

Families were invited to participate in a supplemental study involving an interaction with an unfamiliar peer. Of the 318 families contacted, 239 indicated an interest in participating; of those, 132 participated and 107 did not due to scheduling conflicts or exclusion based on medication usage that might influence cortisol (Granger, Hibel, Fortunato, & Kapelewski, 2009). Participants and nonparticipants did not significantly differ in gender, $\chi^2(1) = .04$, $p = 0.85$, age, $t(316) = -.29$, $p = 0.77$, or ethnicity (White vs. minority), $\chi^2(1) = 0.16$, $p = 0.69$. During the phone recruitment, parents completed a survey of children's use of over-the-counter and prescription medications; usage during the past 24 hr was confirmed at the assessment. Both members of the dyad were participants, allowing for a naturally occurring interaction. Participants from different school districts were paired to ensure lack of familiarity between partners; otherwise, children were randomly assigned to same-sex dyads.

6.2 | Procedure

All procedures for this study were approved by the university Institutional Review Board. During the summer following 3rd grade or the fall of 4th grade (referred to as 4th grade), children participated in a 3–4 hr session. Sessions occurred between 1:00 pm and 5:00 pm to avoid confounding effects of the steep decline in cortisol levels in the morning related to the diurnal pattern of HPA activity (Granger et al., 2012). Project staff described the study, explaining that the child would be playing a game with an unfamiliar peer, and parents and youth provided written consent/assent for participation. Youth were then separated from their parents and completed some questionnaires and neutral activities (e.g., art projects) while awaiting their turn to participate in the social challenge task. To ensure a lack of contact prior to their interaction, dyadic partners were kept in separate rooms during this time. Immediately prior to their participation, youth provided a saliva sample. There was a minimum of 20 min (M = 55 min, SD = 42) between the time when youth were informed about the impending interaction with the unfamiliar peer and the pre-task saliva sample.

In the first phase of the social challenge task, youth were told that whoever constructed a copy of a block model would win a prize. They were given a set of blocks that was sufficient to complete only one model, and were allowed to build for nine minutes. In the second phase, youth were informed that they would each receive a prize for their efforts, and were instructed to decide on the distribution of two prizes of noticeably unequal value. It was expected that this task would trigger individual differences in youths' anticipation of a social stressor (i.e., an impending interaction with an unfamiliar peer) and reactivity to a social stressor (i.e., potential conflict and perceptions of threat,

uncontrollability, and evaluation). Youth provided another saliva sample 20 min after the task to reflect peak activation levels related to the stressor. At the end of the session, participants were debriefed and the one who had received the less valuable prize was given the opportunity to exchange it for a higher valued prize.

In the winter of 6th grade, youth completed a semi-structured interview to assess depressive symptoms. Interviews were conducted at school or in the home by trained graduate students and postbaccalaureate staff with supervision by a clinical psychology faculty member. Of the youth who participated in the social challenge task, 123 completed the interview. Youth who did and did not complete the interview did not significantly differ in gender, $\chi^2(1) = 0.12$, $p = 0.73$, age, $t(126) = -1.13$, $p = 0.26$, ethnicity, $\chi^2(1) = 3.40$, $p = .07$, or lunch status $\chi^2(1) = .01$, $p = 0.92$.

6.3 | Measures

6.3.1 | Medication usage

Following recommendations by Granger et al. (2009), parents completed a checklist of medication usage. Youth were assigned a score of 1 if they had taken medications (e.g., steroidal or psychotropic) within the past 24 hr that may interfere with the cortisol assays and a score of 0 if they had not. A few children ($n = 7$) received scores of 1 despite the original exclusionary efforts; these children were included in the study, but all analyses adjusted for medication usage.

6.3.2 | Saliva sample collection and analysis

Saliva samples were collected and handled following Granger et al. (2007). To ensure against contamination from food consumption, youth were instructed not to eat for 1 hr prior to their assessment (Gibson et al., 1999). Youth provided two saliva samples: immediately prior to the task (pre-task) and 20 min after the completion of the task (reactivity). On average, the pre-task sample was collected at 2:19 pm (SD = 33.87 min) and the post-task sample was collected at 3:03 pm (SD = 34.23 min). Youth donated whole saliva by passive drool into a 2 ml cryogenic vial. Samples were frozen at −20°C until shipped overnight on dry ice for assay. They were then batched and stored at −80°C. On the day of assay, samples were brought to room temperature, centrifuged at 3,000 RPM for 15 min, and the clear top-phase of the sample was pipetted into appropriate test wells. All samples were assayed for salivary cortisol using a highly sensitive enzyme immunoassay US FDA (510k) cleared for use as an in vitro diagnostic measure of adrenal function (Salimetrics, State College, PA). The test used 25 µl of saliva, had a lower limit of sensitivity of .007 µg/dl, with a range of sensitivity from .007 to 3.0 µg/dl. Samples were assayed in duplicate; average intra- and inter-assay coefficients of variation were less than 5% and 10%. Averaged duplicate scores were used in all statistical analyses.

Because pre-task cortisol distributions were positively skewed, cortisol scores were log-transformed. Statistical outliers (±3 SD) were then recoded to the next highest value in the distribution. To ease interpretation, descriptive statistics (Table 1) are provided on the raw scores. Analyses of pre-task cortisol are based on the transformed scores. To assess cortisol reactivity, cortisol levels 20 min post-task were residualized on pre-task cortisol levels, with higher scores reflecting more reactivity.

6.3.3 | Approach-avoidance motivations

In 6th grade, youth completed the behavioral activation and behavioral inhibition subscales of the BIS/BAS. This measure was developed for adults (Carver & White, 1994) and modified for children (Muris, Meesters, de Kanter, & Timmerman, 2005). Both the adult (Coplan et al., 2006) and child (Bjørnebekk, 2007; Muris, Rassin, Franke, & Leemreis, 2007) versions show strong reliability and validity in youth. For this study, we primarily used the child version; for a few items, we adopted the adult version item or a slightly modified child version item to maintain the integrity of the original wording. The BAS (approach) subscale includes 13 items (e.g., "I feel excited and full of energy when I get something that I want."). The BIS (avoidance) subscale includes

TABLE 1 Descriptive statistics and bivariate correlations

	Girls		Boys				Bivariate correlations						
Variables	M	SD	M	SD	Min.	Max.	1	2	3	4	5	6	7
1. Pre-task cortisol	.10	.07	.10	.07	.03	.52	—	.54***	.15	−.17	.22	.08	.05
2. Post-task cortisol	.10	.08	.10	.08	.02	.62	.59***	—	.84***	.00	.16	.03	.11
3. Cortisol reactivity	−.02	1.11	.03	.87	−2.36	7.25	.01	.66***	—	−.06	.14	.05	.04
4. BAS	2.47	.49	2.49	.49	1.00	3.69	.12	.04	−.03	—	.24	.04	.16
5. BIS	2.29	.64	2.13	.60	1.00	4.00	.10	−.13	−.08	.12	—	.40**	.11
6. 4th grade depression	1.41	.40	1.42	.37	1.00	2.92	.06	−.06	−.05	−.09	.06	—	.13
7. 6th grade depression	3.48[a]	6.91	.95[a]	2.24	.00	31.00	−.04	−.09	−.02	−.15	.25*	.21	—

Means, standard deviations, minimums, and maximums are presented for pre-task and post-task raw scores. Bivariate correlations are based on transformed, winsorized scores. Correlations below the diagonal are for girls. Correlations above the diagonal are for boys.

[a]Means differ between gender at $p < .01$.

*$p < .05$. **$p < .01$. ***$p < .001$.

seven items (e.g., "I feel hurt when people scold me or tell me that I did something wrong."). Youth checked a box indicating how true each item was on a 4-point scale (1 = *Not true* to 5 = *Very True*). Scores were computed as the mean of the BAS (α = 0.83) and BIS (α = 0.80) items. Supporting the validity of this measure, BAS and BIS are associated in the expected ways with personality (Heym et al., 2008), achievement motivation (approach vs. avoidance; Bjørnebekk, 2007), and neurocognitive processes (Amodio et al., 2008).

6.3.4 | Depressive symptoms

During the lab social challenge task session (4th grade), youth completed the Short Mood and Feelings Questionnaire (SMFQ; Angold, Costello, Messer, & Pickles, 1995) to assess their recent depressive symptoms (13 items; e.g., "I felt unhappy or miserable."). Youth checked a box indicating how true each item was on a 4-point scale (1 = *Not at all* to 5 = *Very Much*). Scores were computed as the mean of the items (α = 0.80). This measure shows moderately high correlations with the Children's Depression Inventory and the Diagnostic Interview Schedule for Children (Angold et al., 1995), and differentiates depression from other psychiatric disorders (Thapar & McGuffin, 1998).

In 6th grade, youth completed an interview modified from the Mini-International Neuropsychiatric Interview for Children and Adolescents (MINI-KID), a structured clinical interview yielding diagnoses of psychiatric disorders according to the DSM-IV or ICD-10 (Sheehan et al., 1998). The interview has high reliability and high correspondence (Sheehan et al., 2010) with the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version-5 (K-SADS-E; Orvaschel, 1995). Interviewers assessed symptoms of major depressive disorder (MDD) and dysthymic disorder (DD) within the past year. The MINI-KID was modified to allow interviewers to ask detailed follow-up questions about the timing, duration, and context of symptoms. Interviewers rated each symptom on a scale modified from a yes/no criterion to a 2-point scale to enable ratings of subthreshold severity: 0 = Symptom absent, 1 = Symptom present at subthreshold levels (i.e., failed to meet required threshold for duration or severity under DSM-IV criteria), 2 = Symptom present at diagnostic levels. Finally, several categories of symptoms were divided into two separate prompts to increase the range of reported number and severity of symptoms. For example, cognitive symptoms of major depression consisted of separate prompts for low self-worth and excessive/inappropriate guilt, allowing participants to endorse none, one, or both symptoms at either threshold or subthreshold severity. The modified interview was administered individually to youth (and a few caregivers) to assess youths' levels of depression.[3]

Coding took place through consultation with a clinical psychology faculty member. Symptom ratings were summed within each depression diagnosis (MDD, Dysthymia). For participants with multiple MDD episodes, the totals across episodes also were summed. These totals were then summed across diagnostic categories (MDD + DD) to create a single continuous depression score reflecting symptoms over the past year. Higher ratings reflected more severe symptoms within a single diagnosis and/or presence of symptoms from separate episodes and/or separate depressive disorders (for a similar approach, see Hammen, Shih, Altman, & Brennan, 2003). Supporting the use of a continuous index, contemporary conceptualizations, derived in part from taxometric analyses, suggest depression is best represented on a dimensional continuum (Hankin, Fraley, Lahey, & Waldman, 2005). Providing evidence for concurrent validity, continuous scores for depression were significantly correlated (r = 0.73, p < .001) with self-reports on the SMFQ completed at the same time (Angold et al., 1995). Independent coding of a subset of interviews by an advanced graduate student in clinical psychology yielded strong inter-rater reliability (one-way random-effects ICC = 0.94). To characterize the level of depressive symptoms, we calculated how many children had experienced no symptoms, subthreshold symptoms, or threshold symptoms (i.e., diagnoses) of MDD and/or dysthymia over the past year. As would be expected in a community sample, most (70%) children experienced no depressive symptoms but a sizable minority experienced subthreshold (26%) or threshold (4%) levels of depressive symptoms.

7 | RESULTS

7.1 | Overview of analyses

For descriptive purposes, we examined mean level and changes in cortisol as well as bivariate correlations among all study variables. Using Mplus (Muthén & Muthén, 1998–2007), we conducted multiple regression analyses to test the interactive contributions of cortisol levels, BAS/BIS, and gender (0 = boys; 1 = girls) to 6th grade depressive symptoms (MINI-KID). Because post-task scores were nested within dyad, the cluster option was used when testing the independent and interactive contributions of cortisol and BAS/BIS to depressive symptoms for analyses involving stress reactivity. All analyses adjusted for 4th grade depressive symptoms (SMFQ), medication status (0 = no medication; 1 = medication; Granger et al., 2009), and time of the pre-task cortisol sample.[4] SMFQ scores, cortisol, and BAS/BIS scores were centered prior to inclusion in the regressions and prior to the creation of the interaction terms. Separate analyses were conducted for pre-task cortisol and cortisol reactivity. We therefore used a Bonferroni correction of p < .025 for each of the two sets of regression analyses (approach and avoidance). Significant interactions were decomposed by conducting simple slope analyses (Cohen, Cohen, West, & Aiken, 2003) for cortisol at ±1 SD from the mean of the moderator (BAS or BIS). Regions of significance tests were conducted to determine the level of BAS or BIS at which cortisol significantly predicted 6th grade depressive symptoms (Preacher, Curran, & Bauer, 2006). Two-way Cortisol × Gender and BAS/BIS × Gender interactions as well as the three-way Cortisol × BAS/BIS × Gender interaction were included in the initial analyses. For parsimony, these interactions were retained only if the three-way interaction was significant. Of the 132 participants, 4 were missing pre-task cortisol, 4 were missing post-task cortisol, 11 were missing BAS scores, 8 were missing BIS scores, 1 was missing 4th grade depressive symptom scores, and 9 were missing 6th grade depressive symptom scores. Because Mplus uses

full-information maximum likelihood (FIML; see Enders & Bandalos, 2001), data from all 132 participants were included in the analyses.

7.2 | Descriptive statistics and bivariate correlations

Table 1 presents the means, SDs, and intercorrelations for all study variables. *T*-tests revealed only one sex difference: As expected, girls reported higher levels of 6th grade depressive symptoms than did boys, $t(121) = -2.74$, $p = .007$. Cortisol levels were moderately correlated from pre- to post-task. Although paired-*t*-tests revealed no significant mean change from pre- to post-task for cortisol, $t(126) = 1.35$, $p = .09$, in previous papers (Rudolph, Troop-Gordon, & Granger, 2010), we have documented substantive individual differences in cortisol change (at least 10%) from pre- to post-task, including participants who evidenced increases (32.1%) or decreases (43.5%) in cortisol. Given this range of cortisol reactivity scores, we were able to test our hypotheses involving individual differences in HPA reactivity. Cortisol was not associated with BAS, BIS, 4th grade depressive symptoms, or 6th grade depressive symptoms in girls or boys. BAS and BIS were not significantly correlated; BIS was significantly positively associated with 4th grade depressive symptoms in boys and with 6th grade depressive symptoms in girls.

7.3 | BAS and BIS as moderators of pre-task cortisol

For pre-task cortisol, regression analyses yielded a significant main effect of gender on 6th grade depressive symptoms, reflecting higher levels in girls than boys. Because there were no significant two- or three-way interactions with gender, it was included only as a covariate. There was no significant main effect of cortisol or interactive effect with BAS. Analyses yielded a significant Cortisol × BIS interaction (see Table 2). Decomposition of this interaction revealed that blunted pre-task cortisol predicted more depressive symptoms in youth with high, $b = -3.25$, $t(122) = -2.43$, $p = .02$, but not low, $b = 1.89$, $t(122) = 1.27$, $p = 0.21$, avoidance. The region of significance test revealed that blunted cortisol significantly predicted depressive symptoms when avoidance was >0.35 SD above the mean. Of the children in the sample, 37 had avoidance scores in this range. As shown in Figure 1a, low pre-task cortisol was associated with elevated depressive symptoms at high levels of avoidance and with fewer depressive symptoms at low levels of avoidance. High levels of pre-task cortisol were associated with low levels of symptoms at both low and high levels of avoidance.

7.4 | BAS and BIS as moderators of cortisol reactivity

For cortisol reactivity, regression analyses yielded a significant main effect of gender on 6th grade depressive symptoms, reflecting higher levels in girls than in boys. The BIS × Gender interaction was significant (see Table 2). Decomposition of this interaction revealed that BIS predicted more depressive symptoms for girls, $b = 2.78$, $t(117) = 2.40$, $p = .02$, but not for boys, $b = -.28$, $t(117) = -.52$, $p = 0.60$. Analyses also yielded a significant three-way Cortisol × BAS × Gender interaction predicting 6th grade depressive symptoms (see Table 2). Decomposition of this interaction revealed that heightened cortisol reactivity predicted more depressive symptoms in girls with high approach, $b = 3.14$, $t(117) = 3.14$, $p < .001$, and fewer depressive symptoms in girls with low approach, $b = -2.63$, $t(117) = -2.04$, $p = .02$. The region of significance tests revealed that heightened cortisol predicted more depressive symptoms when approach was >.01 SD above the mean. Of

TABLE 2 Contributions of BAS/BIS, Cortisol, and their Interaction to the Prediction of 6th Grade Depressive Symptoms

	Pre-task Cortisol			Cortisol Reactivity		
Predictor	***β***	***b***	**SE**	***β***	***b***	**SE**
4th grade depressive symptoms	.15	1.98	1.14	.16	2.20	1.49
Medication (0 = no; 1 = yes)	.03	.68	2.05	−.01	−.32	1.34
Time of sampling	−.05	−.01	.01	.04	.01	.01
Gender (0 = boys; 1 = girls)	.20	2.08*	.89	.23	2.44**	.85
Cortisol	−.07	−.75	.92	.09	.50	.42
BAS	−.05	−.57	1.02	.06	.62	.55
BIS	.17	1.40	.78	−.03	−.28	.54
Cortisol × BAS	.00	−.00	1.91	.05	.71	.86
Cortisol × BIS	−.22	−4.02*	1.60	−.05	−.62	.50
BAS × Gender	—	—	—	−.16	−2.38	1.48
BIS × Gender	—	—	—	.27	3.06*	1.27
Cortisol × Gender	—	—	—	.01	.06	.50
Cortisol × BAS × Gender	—	—	—	.21	4.62*	1.81
Cortisol × BIS × Gender	—	—	—	.05	.82	1.86

*$p < .025$ (Bonferroni corrected). **$p < .01$.

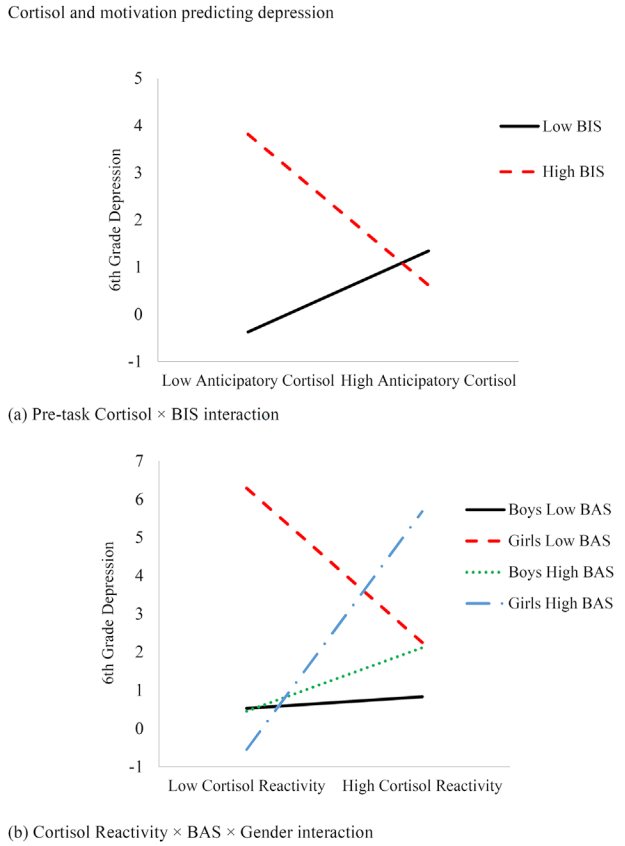

FIGURE 1 Interactions predicting depressive symptoms from (a) anticipatory cortisol and BIS; and (b) cortisol reactivity, BAS, and gender. Analyses adjust for 4th grade depressive symptoms

the girls in this sample, 28 had approach scores in this range. Heightened cortisol predicted fewer depressive symptoms when approach was <−.34 SD below the mean. Of the girls in this sample, 26 had approach scores in this range. As shown in Figure 1b, low cortisol reactivity was associated with elevated depressive symptoms at low levels of approach and with fewer depressive symptoms at high levels of approach. In contrast, high cortisol reactivity was associated with elevated depressive symptoms at high levels of approach and with fewer depressive symptoms at low levels of approach. Cortisol reactivity did not predict depressive symptoms in boys with either high, $b = 0.84$, $t(117) = 1.06$, $p = 0.29$, or low, $b = 0.15$, $t(117) = .58$, $p = 0.56$, approach.[5]

8 | DISCUSSION

Highlighting the importance of a multi-system perspective on development, the present study tested the novel hypothesis that the implications of HPA axis activation for adolescent depression are contingent on the approach-avoidance motivation systems. Specifically, we proposed two possible profiles of adolescent depression emerging from the intersection of dysregulated arousal and motivation: (1) excessive disengagement, reflected in HPA underactivation along with low approach motivation or high avoidance motivation; and (2) excessive engagement, reflected in HPA overactivation along with high approach motivation. Results supported this multi-system framework, revealing divergent profiles of adolescent depression stemming from the joint dysregulation of arousal and motivation. Moreover, this research partially supported the idea that adolescent girls are particularly sensitive to depressive symptoms as an outcome of dysregulation in these systems.

8.1 | Divergent profiles of depression

Consistent with an excessive disengagement profile, blunted HPA activation predicted depressive symptoms in girls with low approach motivation and both girls and boys with high avoidance motivation. Hypoactivation of the HPA axis in the context of a social stressor may characterize youth who lack the energy and resources for mounting a sufficient preparatory response to an upcoming stressor or who fail to engage in active efforts to cope with the stressor. When paired with a tendency to find social interactions unrewarding (insensitive approach system) or to find interpersonal stress highly aversive (highly sensitive avoidance system), youth may disengage from even minor social challenges. Excessive disengagement from stressors and associated problems—such as hopelessness and social isolation—may impede the ability of youths to deal effectively with stress, leading to depression (Agoston & Rudolph, 2011). Attenuated cortisol activation often is associated with prior exposure to chronic or severe stress, suggesting that it may be a key marker of allostatic load (Badanes et al., 2011; Gunnar & Vazquez, 2001; Miller, Chen, & Zhou, 2007). This attenuation may be adaptive in the short-term as it prevents persistent responses to repeated uncontrollable stressors, but may be maladaptive in the long-term as it discourages active engagement with, and resolution of, potentially controllable stressors, especially when paired with low motivation to approach rewards and high motivation to avoid punishment in the environment.

Consistent with an excessive engagement profile, heightened HPA reactivity to a social stressor predicted future depressive symptoms in girls with high approach motivation. Elevated arousal associated with hyperactivation of the HPA axis may cause girls with high approach motivation to mount too intensive a stress response and engage with stressors in a dysregulated, impulsive manner. Excessive engagement with stressors and associated problems—such as heightened emotional arousal—may inhibit efforts to redirect attention away from desired goals (e.g., cognitive reappraisal) and trigger cognitive perseveration (e.g., rumination) and extreme vigilance to social cues. Indeed, elevated HPA activation is associated with negative emotional arousal (Adam, 2006) and rumination (Gianferante et al., 2014; Huffziger et al., 2013; Rudolph et al., 2011). In turn, involuntary engagement with stressors predicts depression over time (e.g., Troop-Gordon, Rudolph, Sugimura, & Little, 2015; for a meta-analysis, see Rood, Roelofs, Bogels, Nolen-Hoeksema, & Schouten, 2009), particularly in girls (Jose & Brown, 2008). This joint profile of HPA overactivation combined with high approach motivation may therefore tip the balance from potentially advantageous correlates of engagement, such as effective problem solving and prosocial behavior,

to potentially disadvantageous correlates of engagement stemming from impulsive cognitive, emotional, and behavioral tendencies.

It is noteworthy that significant interactions were found between anticipatory cortisol and avoidance motivation but between cortisol reactivity and approach motivation. High avoidance may be particularly problematic during the anticipatory phase of a stressor because it prevents youth from preparing to cope, resulting in helplessness. In contrast, approach motivation may be particularly relevant to how youth respond once presented with a stressor. However, replication of this pattern of effects would be necessary before drawing any firm conclusions about whether different motivational systems are most active or relevant during different phases of stressors.

Admittedly, these hypothesized patterns of excessive disengagement or engagement are speculative and are difficult to study in the context of a single stressor. However, they are intriguing to consider as possible products of changing sensitivity to novel and potentially challenging events across development. Although a conflict-of-interest situation with an unfamiliar peer likely poses a challenge to many children, the extent to which this challenge activates the HPA axis may depend on individual differences in how children integrate past experience into their subjective perceptions of current stressors. The HPA axis is under the tonic inhibitory control of the hippocampus, which suppresses HPA activity until it detects a novel stimulus in the environment (Weiner, 1992). When novelty is detected, the hippocampus releases its tonic control over the HPA axis, which triggers its activation. Optimal levels of HPA activation may occur when youth apply knowledge and coping skills developed within prior stressful situations to adaptively adjust to a novel challenge (Weiner, 1986). Overactivation of the HPA axis may occur when youth fail to habituate to social stressors, resulting in hypervigilance and the excessive detection of novelty or the interpretation of a novel situation as highly threatening (Del Giudice et al., 2011). Underactivation of the HPA axis may occur when youth become over-habituated to social stressors or when they lack the resources to mount an effective response to challenge (Hankin et al., 2010), resulting in hypovigilance and immobilization in the face of stress, and disengagement from the environment. Approach-avoidance motivations may intensify the maladaptive effects of overactivation and underactivation by further calibrating the sensitivity, vigilance, or habituation of this signal detection system, thereby amplifying dysregulated engagement or disengagement responses to stress. Ultimately, understanding the joint contribution of arousal and motivation systems to depression will require tracking responses to multiple stressors over time and across different developmental stages.

8.2 | Future directions

This research provides an important step toward generating and testing multi-system frameworks of development by testing the novel hypothesis that the contribution of HPA activation to depression is contingent on the approach-avoidance motivation systems. Methodologically, the study is strengthened by the use of a naturalistic laboratory stressor to elicit HPA activation embedded within a longitudinal design examining the prediction of depressive symptoms across the transition to adolescence. However, future investigations examining the interactive contribution of arousal and motivation systems to depressive symptoms can build on this study in several ways.

First, we relied on a self-report measure of approach-avoidance motivations. Nevertheless, prior research supports associations between self-reported motivations (including the BIS/BAS measure used in this study) and distinct neural systems (Amodio et al., 2008; Rudolph et al., 2016) as well as associations between the sensitivity of neural systems guiding approach (Bress et al., 2013; Forbes et al., 2009; for a review, see Forbes & Dahl, 2012) and avoidance (Forbes et al., 2011; Rudolph et al., 2016; Yang et al., 2010) and depression. This study can therefore serve as a critical foundation for future research that directly tests the hypothesized interactions between arousal and motivation systems as reflected in various biological processes. Neuroscience theories view approach and avoidance as reflexive tendencies that are instantiated in specific neural systems. For example, Ernst's (2014) triadic model of development proposes three interrelated neural systems guiding adolescent motivated behavior: (1) approach, represented in the striatum; (2) avoidance, represented in the amygdala, hippocampus, and insula; and (3) regulatory, represented in the dorsolateral PFC, ventromedial PFC, and anterior cingulate cortex. Dysregulation is presumed to occur in the context of ineffective regulatory control over approach and avoidance tendencies. Future research using neuroimaging methods can therefore assess more directly whether the interaction between HPA activation and activation in neural systems involved in motivation predicts depressive symptoms in adolescence. It also will be important to further investigate the relative contributions of genetic, biological, and environmental factors to the development of dysfunction in the regulatory and motivation systems.

Second, it will be important for future research to determine the optimal level of HPA activation in response to particular stressors as well as the optimal balance between approach and avoidance motivations. Unfortunately, there is no consensus regarding a cut-off for adaptive or maladaptive functioning within these systems, leading us to adopt a statistical operationalization of high and low levels as reflected in 1 SD above and below the mean of the sample. Optimal functioning of these systems both individually and jointly is likely not absolute but rather may be determined by factors such as stage of development, exposure to prior stressful experiences, contextual demands of the stressor, and other individual and environmental differences.

Third, future research needs to identify the precise mechanisms through which HPA activation, motivations, and their interaction foster a greater risk for depressive symptoms. Here we suggest the possibility that arousal and motivations jointly contribute to children's cognitive, emotional, and behavioral responses to stress, perhaps accounting for divergent pathways to depression: one through excessive engagement with stressors and the other through excessive disengagement from stressors, both of which may

interfere with adaptive regulatory efforts. Future studies that directly test these and other possible mediating processes (e.g., attentional biases, goal-setting, interpersonal styles) will shed additional light on how these systems work together in depression risk. It also may be helpful to examine whether these two different pathways are reflected in different profiles of depression. For example, cortisol over-activation combined with high approach may predict an anxiety-depression syndrome whereas cortisol under-activation combined with low approach or high avoidance may predict an anhedonic depression profile (Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007).

Fourth, we interpreted pre-task cortisol levels as reflecting anticipatory stress because these assessments were taken at least 20 min after participants were informed that they would be interacting with an unfamiliar peer. Moreover, the pattern of results held after adjusting for baseline levels upon entrance into the lab as well as when using an anticipatory activation index that residualized pre-task scores on baseline scores. However, because this study did not include an assessment of daily cortisol levels or levels while in specific nonstressful contexts, we cannot be certain that pre-task cortisol levels reflect anticipation of the impending interaction. Additional research is needed to disentangle the differential effects of HPA dysregulation at baseline and during the various phases of stressor anticipation, exposure, and recovery to obtain a comprehensive picture of how the trajectory of cortisol intersects with approach and avoidance to contribute to depression risk.

Relatedly, it is important to note that there was variability in the elapsed time between children being informed about the impending interaction and their participation in the interaction, resulting in differences in the timing of the pre-task cortisol assessment. For children whose stress response system was reactive to the anticipated stressor, it is likely that their cortisol levels remained elevated during the waiting period. Moreover, our results replicated when adjusting for the elapsed time, suggesting they were robust to contextual variations. In this study, we chose to focus on a naturalistic and somewhat complex stressor involving an in vivo peer interaction. This type of stressor is rare in the field of stress physiology and thus studying the stress response system in this type of context is important. Unfortunately, naturalistic stressors do tend to involve less standardization due to practical constraints. Acknowledging the trade-offs of different research designs and triangulating results across different types of stressors, some of which involve naturalistic but less controlled contexts and others of which involve highly standardized paradigms, will be essential for developing a complete understanding of stress response systems.

9 | CONCLUSION

This research highlights the need for integrative models of adolescent depression to consider the interplay between developmental systems involved in arousal and motivation, and sheds light on prior conflicting findings regarding the implications of heightened versus blunted cortisol activation for adolescent depression. Specifically, heightened activation predicted future risk for depressive symptoms in girls with high approach motivation, whereas blunted activation predicted future risk for depressive symptoms in girls with low approach motivation and in youth with high avoidance motivation. This disjuncture suggests the potential need for prevention and intervention programs that target different pathways to depression among youth with HPA axis dysregulation.

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CONFLICT OF INTEREST

In the interest of full disclosure, DAG is founder and chief scientific and strategy advisor at Salimetrics LLC and Salivabio LLC (Carlsbad, CA) and the nature of these relationships is managed by the policies of the committees on conflict of interest at the Johns Hopkins University School of Medicine and the University of California at Irvine.

ENDNOTES

[1] Because the present study examined cortisol reactivity to stress, we focus here only on studies examining cortisol reactivity to a laboratory stressor and/or the interaction between cortisol activation and naturally occurring stress.

[2] Because age was not examined as a moderator, it is unclear whether these findings might differ in younger children versus adolescents.

[3] A few youth were unwilling to provide information or provided incomplete information. In these cases, caregivers were interviewed instead of, or in addition to, youth. Of the 123 interviews, 120 were administered to youth only, one was administered to caregivers only, and two were administered to both youth and caregivers. For interviews in which both types of data were available, consensual diagnoses were assigned using a best-estimate approach (Klein, Ouimette, Kelly, Ferro, & Riso, 1994).

[4] Because the time lapse between when children were informed about the task and their pre-task sample differed, we conducted an additional analysis for pre-task cortisol that adjusted for this time lapse. This analysis yielded the same results as those reported here.

[5] Because pre-task samples could partially reflect basal levels of cortisol or anticipatory activation in response to the lab visit more generally, we conducted two additional sets of analyses. In the first set, we adjusted for children's level of cortisol at a baseline sample taken immediately after the consent process. In the second set, we created a score reflecting the pre-task sample residualized on the baseline sample, and we adjusted for the time lapse between the two samples. Both sets of analyses yielded the same results as those reported here.

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