International Journal of Cognitive Therapy, 5(3), 283–299, 2012

Biological and Cognitive Responses to an In Vivo Interpersonal Stressor: Longitudinal Associations with Adolescent Depression

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Social stress occurring during adolescence is considered a risk factor for the development of adolescent depressive symptoms. However, the specific mechanisms through which social stress may affect depressive symptoms are not well-established. The current prospective study considered hypothalamic-pituitary-adrenal (HPA) axis reactivity and social problem-solving deficits occurring in response to an acute interpersonally themed stressor as longitudinal predictors of depressive symptoms. Sixty-two adolescents (ages 12–16; 73% female) were recruited using oversampling techniques intended to include adolescents with a wide range

This research was supported by a grant from the American Foundation of Suicide Prevention awarded to the last author. Special thanks to Dan Bauer for his assistance with data analyses. <FN>Correspondence regarding this article may be addressed to Casey Calhoun or Mitch Prinstein at UNC Department of Psychology, Davie Hall, CB 3270, Chapel Hill, NC 27599-3270. E-mail: cdcalhou@email.unc.edu or mitch.prinstein@unc.edu.

of depressive symptoms. HPA axis reactivity was established by collecting cortisol samples before and after an in vivo interpersonally themed social stressor task. Similarly, adolescents completed a structured performance-based social problem-solving task before and after the stressor to provide a measure of stress-induced social problem-solving deficits. Depressive symptoms were assessed at baseline and at three follow-up time points (3, 6, and 9 months). Results indicated that a hyporeactive cortisol response to the interpersonal stressor task was associated with a later re-emergence of depressive symptoms. Also, the selection of more adaptive social problem-solving responses pre-stressor was related to greater decreases in depressive symptoms over time. Stress-induced deficits in problem-solving self-efficacy were associated prospectively with smaller decreases in depressive symptoms over time. Findings suggest that physiological and cognitive responses to social stress may be important to consider for interventions targeting depressive symptoms in adolescents.

The adolescent transition is a critical vulnerability period for increased symptoms of depression (Abela & Hankin, 2008; Costello, Egger, & Angold, 2005; Hankin & Abela, 2005), especially for females (Angold, Costello, & Worthman, 1998). Adolescence also is characterized by a marked increase in interpersonal stressors and emotional reactivity to interpersonal stress, particularly among females (Carter & Garber, 2011; Hankin, Stone, & Wright, 2010; Rudolph & Hammen, 1999). In support of the notion that these two developmental trends may be linked, substantial research has demonstrated that interpersonal stress is an important contributor to depression in adolescence (e.g., Prinstein & Aikins, 2004). Unfortunately, the processes by which interpersonal stress may affect the development of depressive symptoms remain unclear. The present study explored two potential mechanisms, one biological and one cognitive, through which interpersonal stress may affect the development of depressive symptoms over time. Building on previous research, it was hypothesized that both biological stress responses and social problem-solving deficits occurring in response to an interpersonally themed stressor would be associated with the development of depressive symptoms over time.

Biological Responses to Stress

Biological responses to stressors vary across individuals, and previous research has shown that certain response patterns may render individuals at greater risk for maladaptive emotional and behavioral responses. For instance, among adults and youth, research has suggested that dysregulated functioning of the hypothalamic-pituitary-adrenal (HPA) axis system, a primary stress response system, is associated concurrently with depressive symptoms (for a review, see Guerry & Hastings, 2011). The HPA axis stress response consists of multiple processes that are initiated by a perceived threat. Upon perceiving a threat, the amygdala triggers the hypothalamus to release corticotrophin-releasing hormone. This process in turn signals the pituitary gland to release adrenocorticotropic hormone, which increases production of glucocorticoids in the adrenal gland. Glucocorticoids assist in preparing an individual to cope with the perceived threat by increasing energy production in the body (Chrousos & Gold, 1992; Kaltas & Chrousos, 2007; McEwen, 2004). Cortisol, a glucocorticoid pro-

duced by the adrenal gland, is the best-established marker of HPA activity in humans. Both diurnal cortisol (i.e., daily patterns in cortisol levels) and cortisol reactivity (i.e., acute responses to a specific stressor) have been the focus of past research.

The study of the HPA axis system may be especially relevant during adolescence. Research has demonstrated that pubertal development is associated with greater cortisol reactivity to stressful or challenging tasks (Gunnar, Talge, & Herrara, 2009; Stroud et al., 2009; Stroud, Papandonatos, Williamson, & Dahl, 2011), which may facilitate the development of depressive symptoms (Cicchetti & Walker, 2001; Spear, 2000; Walker, 2002). Greater cortisol reactivity has been associated with depressive symptoms in adults (Burke, Fernald, Gertler, & Adler, 2005; Plotsky, Owens, & Nemeroff, 1998). However, relatively few studies have examined associations between HPA axis functioning and depressive symptoms among adolescents, and even fewer have examined these associations longitudinally (Guerry & Hastings, 2011).

Findings from cross-sectional studies suggest that depressed and dysphoric adolescents hypersecrete cortisol after an acute stressor, relative to their nondepressed peers (Hankin, Badanes, Abela, & Watamura, 2010; Rao, Hammen, Ortiz, Chen, & Poland, 2008). Prior longitudinal research, on the other hand, has yielded mixed results for the prediction of depressive symptoms from reactive cortisol. In healthy adolescents, cortisol hyperreactivity to acute stressors may be longitudinally related to the development of depressive symptoms (Susman, Dorn, Inoff-Germain, Nottelman, & Chrousos, 1997). However, this finding has been inconsistent across studies, as both hyper- and *hypo*reactivity have been found to predict increases in problems over time (Hastings et al., 2011). Given previous evidence of concurrent and longitudinal associations between depressive symptoms and dysregulated cortisol responses to acute social stressors, it is hypothesized that such effects will be observed in the current study.

Cognitive Functioning and Stress

Among adults, some evidence suggests that general social problem-solving skill deficits are associated with depressive symptoms both concurrently (Haugh, 2006; Marx, Williams, & Claridge, 1992) and prospectively (Nezu & Ronan, 1985). Additionally, problem-solving skills training approaches have been used to effectively treat depression in adults (Nezu, 1986; Nezu & Perri, 1989), and adolescents (Spence, Sheffield, & Donovan, 2003). Recent research suggests that the content of adolescents' responses to social scenarios as well as their perceptions of self-efficacy in carrying out effective responses may be particularly relevant to internalizing symptoms and self-injury (Nock & Mendes, 2008). Little work, however, has directly examined whether specific deficits in adolescents' social problem-solving skills may be associated longitudinally with adolescent depressive symptoms.

Further, it has been proposed that stressful conditions may compromise individuals' cognitive processing of social information, which in turn leads to dysregulated emotional and behavioral stress responses (Crick & Dodge, 1994; Lemerise & Arsenio, 2000). Adolescents may be especially vulnerable to the effects of stress on cognition given that the adolescent transition is associated with significant developments in cognitive processes such as judgment and decision-making (Blakemore, 2008; Steinberg, 2005). Moreover, adolescents may be more likely than adults to be influenced in their decision-making by social influences and emotional state (Spear, 2000; Steinberg, 2005). Given these insights, examinations of social problem-solving abilities in *stress-*

ful contexts would likely bear greater ecological relevance and relate more closely to psychological symptoms. This may be especially true for depressive symptoms given the aforementioned theoretical views suggesting that symptoms could be influenced by an inability to effectively regulate emotion while cognitively processing stressful situations. Hypotheses for the current study were two-fold. First, we hypothesized that general (i.e., pre-stressor) deficits in social problem-solving abilities would be longitudinally associated with higher levels of depressive symptoms among adolescents. We also hypothesized that diminished social problem-solving abilities in response to an acute stressor would be prospectively associated with higher levels of depressive symptoms.

Overall, the current study examined the effects of a social stressor task on HPA functioning and social problem-solving skills to determine if these effects are similarly, or uniquely, related to the development of depressive symptoms at the adolescent transition. For this study, adolescents' cortisol levels and social problem-solving abilities were assessed before and after an in vivo social stressor task. Participants completed measures of depressive symptoms at baseline, and 3-, 6-, and 9-month follow-up assessments.

METHOD

Participants

Participants included 62 youth (73% female) at the adolescent transition, between the ages of 12 and 16 years (M = 14.70; SD = 1.33). Approximately 76% of participants self-identified as White/Caucasian, 8% African American, 8% Latino American, 5% Asian American, and 3% Mixed or Other Ethnicity. Approximately 65% of adolescents lived in a two-parent household whereas 35% lived with their biological or adoptive mother only. Three percent of mothers reported that their highest level of education was a high school diploma or GED, 6% earned an associate's or trade degree, 29% attended some undergraduate college, 13% earned a bachelor's degree, 6% attended some graduate school, 26% earned a master's degree, and 16% had earned a doctoral degree. Participants reported current use of a variety of medications including stimulants (n = 7), anticonvulsants (n = 2), antipsychotics (n = 9), antidepressants (n = 15), hypnotics (n = 1), antibiotics (n = 5), antihistamines (n = 2), pain relievers (n = 3), corticosteroids (n = 1), and birth control (n = 3). A total of 50% reported no medication usage.

Oversampling procedures were used to yield a sample with a wide range of depressive symptoms. Specifically, adolescent participants were recruited from various clinical referral sources, including local inpatient units and outpatient clinics (n = 13; 21%), community mental health agencies (n = 2; 3%), local high schools (n = 16; 26%), and mass-email advertisements (n = 31; 50%). Potential participants initially were screened during recruitment for a number of exclusion criteria (i.e., psychosis, mental retardation, or pervasive developmental disorders) that would substantially alter the salience of our assessment of social stressors and social problem-solving tasks.

Of the 62 adolescents who completed baseline assessments, 55 (89%) participated in the 3-month follow-up assessment, 44 (71%) participated in the 6-month follow-up, and 41 (66%) completed the 9-month follow-up. Although many retention strategies were utilized (e.g., frequent phone, mail, and email contact with partici-

pants and their families, the provision of monetary incentives to encourage continuing participation, etc.), attrition over longitudinal intervals was due to reasons common to research of this type, such as family relocation, study drop-out, and hospital readmission. Attrition analyses revealed that data were missing at random (i.e., no statistically significant differences between participants with/without complete data), and analyses were able to utilize all available data on all 62 participants.

Procedure

Adolescents completed an initial baseline assessment in a laboratory setting. During this visit, participants completed questionnaire data, structured interviews, participated in an *in vivo* social stress-induction paradigm, and completed a performance-based social problem-solving task. Salivary cortisol samples were collected at regular intervals during this process (described in detail below). Subsequent to this baseline assessment, telephone follow-up interviews were conducted at 3, 6, and 9 months post-baseline to assess depressive symptoms. Adolescents received incrementally increasing monetary compensation for their participation at various stages of the present study (up to $80 for the completion of all lab and telephone-based data collection).

Adolescents were instructed to refrain from medication usage on the day of baseline testing until all baseline procedures were completed. Detailed information on prescription medication usage also was collected during the baseline assessment to ensure that certain medications would not be associated with target variables. Preliminary analyses were conducted to determine if specific medications were associated with primary variables (see Data Analytic Plan).

Social Stressor Task. Adolescents participated in a modified Trier Social Stressor task during the baseline laboratory assessment (e.g., Hastings, Zahn-Waxler, & Usher, 2007; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Participants who had been acclimated to an observational setting were oriented toward a camera connected to a closed-circuit "feedback screen" displaying their own live image. Adolescents were instructed to face this camera and feedback screen while preparing (for one minute) and subsequently delivering a three-minute speech. The explicit goal of the speech, as explained to participants, was to convince an audience of their peers (presumably watching the live video feed in a nearby room) that they should be selected to star in a fictional television show about teens' ability to form and maintain friendships. Immediately prior to the adolescents' delivery of the speech, an opposite gender undergraduate research assistant entered the room, ostensibly to evaluate participants' performance. Although this "observer" remained in the room at close proximity to the participant for the duration of the speech task, s/he was given instructions to fix his gaze on the feedback screen and withhold direct eye contact with the participant at all times. At approximate intervals of 20 seconds, the observer was instructed to make a small mark on a clipboard in order to give the appearance of continuous evaluation.

Cortisol Collection. Adolescents provided salivary cortisol samples using a passive drool procedure (see Klimes-Dougan et al., 2001) on four occasions throughout the baseline laboratory assessment described above: (1) immediately prior to the speech task (following a 20-minute relaxation period); (2) 20 minutes post-speech; (3) 30 minutes post-speech; and (4) 40 minutes post-speech. Cortisol reaches peak levels in human saliva approximately 20 minutes after the onset or peak of a stressor

(e.g., Adam, Sutton, Doane, & Mineka, 2008; Gunnar et al., 2009). Thus, samples yielded data regarding HPA reactivity before the speech stressor and at various intervals following the stressor. HPA reactivity was examined in this study, computed as a standardized difference score between the second and the first cortisol samples, with higher scores representing greater reactivity.

Salivary samples were frozen for storage at -25°C and then shipped on dry ice to Pennsylvania State University's Behavioral Endocrinology Laboratory for assay (Salimetrics, PA). Samples were assayed for cortisol using a 510-k cleared high-sensitive enzyme immunoassay designed to assess adrenal function. This test, which uses 25 μl of saliva (for singlet determinations), has a lower limit sensitivity of .007 μg/dl and a range of sensitivity from .007 to 1.2 μg/dl.

Social Problem-Solving Task. Immediately before and after the Trier Social Stressor task, adolescents completed the Social Problem-Solving Task (SPST; Nock, 2006), a performance-based measure of social problem-solving skills. The SPST measures a broad range of problem-solving skills on the basis of performance responding to eight social scenarios in four different domains. During the SPST, participants listened to a series of audio recordings involving potential problems with peers, a romantic partner, a parent, and a teacher or boss. After hearing each scenario, the participants performed various problem-solving tasks that examined different facets of their social problem-solving abilities. Participants were asked to generate as many responses as possible in a 15-second interval (response generation), and then were asked to choose which of the generated responses they would be most likely to enact (response selection). Finally, participants were asked to rate on a 5-point scale (0–4) their likely effectiveness at enacting their selected response to the problem (self-efficacy).

Participants' performance on each part of the SPST was audio recorded and scored by expert raters, blind to symptom ratings of participants, following a revised version of the manualized SPST coding system (Nock, 2006). Participants' responses to the SPST can be coded for attributional style, response generation, response content, and selected response content, and can be scored for perceived self-efficacy. The content of adolescents' selected response and their reported self-efficacy scores were utilized in analyses, consistent with prior work (Nock & Mendes, 2008; Oldershaw et al., 2009). As in previous research using this measure (Nock & Mendes, 2008), all tapes were coded to rate the content of adolescents' selected response (1= negative response; 2 = neutral response; 3 = positive or effective response). Responses were coded as negative if the content was aggressive, hostile, or self-disparaging (e.g., "I wouldn't say anything, because I'm ugly"); neutral if it was passive, avoidant, or unclear (e.g., "I would just forget that this happened . . . I wouldn't care"); and positive if the content was accommodating, agreeable, or appropriately assertive (e.g., "I would talk to my teacher about how I am feeling and ask for extra help in class"). Previous analysis of this rating system has revealed adequate inter-rater reliability for each construct examined in the current study (Nock & Mendes, 2008), and there was moderate agreement in the current study (ICC = .57). To assess adolescents' cognitive responses to stress, difference scores were computed to reflect change in the quality of adolescents' selected response and self-efficacy from before to after the stressor task, with higher scores representing a greater decrease in response content and self-efficacy skills subsequent to stress.

Primary Measures

Depressive Symptoms. Depressive symptoms were assessed at baseline and at each follow-up time point using the Mood and Feelings Questionnaire (MFQ; Costello & Angold, 1988). The MFQ was designed for use as a self-reported screening instrument for major depression among children and adolescents aged 8–18 years. The questionnaire, which consists of 33 items rated on a three-point scale (0 = Not True; 1 = Sometime True; 2 = Mostly True), includes content conforming to *DSM-IV* criteria for Major Depressive Disorder. Evidence from psychometric studies of the MFQ indicate that the questionnaire has strong internal consistency, acceptable test-retest reliability, and high convergent validity with semi-structured diagnostic measures of MDD such as the Kiddie Schedule for Affective Disorders and Schizophrenia—Child Version (Angold, 1989; Wood, Kroll, Moore, & Harrington, 1995). In the present sample, internal consistency was excellent ($\alpha = .97$). A mean score across all 33 items was computed at baseline and all subsequent time points.

Covariate Measures

Cortisol Timing. Cortisol levels observed immediately following a stressor represents the sum of the acute cortisol response together with the basal cortisol level for that particular time of day. Thus, adolescent participants reported their time of awakening, and laboratory personnel recorded the times of cortisol collections. For each individual, a "cortisol timing" variable was computed representing the duration of time elapsed between the time of awakening and the time at which the first cortisol sample was collected.

Pubertal Stage. Pubertal stage was assessed using adolescent' self-report on a pictorial questionnaire assessing body changes during the pubertal transition (Morris & Udry, 1980). This questionnaire presents two sets of five serial line drawings representing the development of two secondary sexual characteristics and corresponding to the five Tanner stages, from prepubertal (Stage = 1) to postpubertal (Stage = 5; Morris & Udry, 1980). Female and male participants were presented, respectively, with drawings depicting breast development/pubic hair growth and genital development/pubic hair growth. For each of the two sets, all participants were instructed to circle the picture that is "closest to your stage of growth." Adolescent self-ratings of pubertal stage on this questionnaire are highly correlated with physician assessment and are considered sufficient when a general estimation of pubertal stage is desired (Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990; Morris & Udry, 1980). For the purpose of the present study, and in accordance with other investigations (e.g., Negriff, Fung, & Trickett, 2008), pubertal stage was defined as the score on the pictures representing breast development or genital development.

Data Analytic Plan

Four sets of analyses were conducted to examine study hypotheses. First, descriptive statistics were conducted to examine the means and standard deviations on all study

variables over the 9-month longitudinal period. Correlational analyses also were computed between all study variables.

Second, independent samples *t*-tests were conducted to assess whether participants' medication usage was related to primary study variables and, accordingly, to determine if medication usage should be included as a covariate in subsequent analyses. Given that corticosteroids and birth control have demonstrated some influence on assessments of cortisol (Bouma, Riese, Ormel, Verhulst, & Oldehinkel, 2009; Hastings, Fortier, Utendale, Simard, & Robaey, 2009) and antidepressants likely bear direct influence on levels of depressive symptoms over time, these medications were selected as those of primary concern. Participants reporting and not reporting these medications were compared on primary study variables.

Third, analyses of longitudinal predictors of depressive symptoms were conducted using latent curve analysis in a structural equation modeling framework (Bollen & Curran, 2006). Due to a lack of normality in the distributions of depressive symptoms, scores at each time point were transformed using a logarithmic transformation prior to model identification. The four transformed scores for depressive symptoms (i.e., at baseline, 3, 6, and 9 months post-baseline) were used as indicators of a latent intercept (all path weights set to 1) and slope factor (path weights set to 1, 3, 6, 9). Analyses revealed that growth in depressive symptoms was best represented by a quadratic slope; thus a second slope factor also was estimated (path weights set to 1, 9, 36, 81). All latent curve analyses were conducted using AMOS 19.0 utilizing maximum likelihood methods to account for missing observations.

To maximize power, two latent curve analyses were conducted, to respectively examine cortisol reactivity and social problem-solving skills as prospective predictors of depressive symptoms. The prospective association between cortisol reactivity and depressive symptoms was examined by estimating paths between (1) pre-stressor cortisol levels; and (2) cortisol reactivity, each as predictors of the latent intercept, linear, and quadratic slope factors. Covariances among adolescents' pubertal stage, age, cortisol timing (i.e., time since morning waking) and pre-stressor cortisol measurement were estimated to yield an adjusted index of cortisol measurement. The prospective association between social problem-solving skills and depressive symptoms was examined by estimating paths between (1) pre-stressor measures of social problem-solving (i.e., content of selected responses and self-efficacy); and (2) difference scores reflecting pre-post stressor changes in these same dimensions, each as predictors of the latent intercept, linear slope, and quadratic slope factors.

Initial analyses also examined interactions between cortisol and social problem-solving skills as predictors of change in depressive symptoms over time; no significant effects were revealed. Given the absence of significant interactions, the latent curve models for cortisol and social problem-solving skills were conducted independently to allow for more parsimonious interpretations of findings.

RESULTS

Descriptive Statistics

Table 1 lists means, standard deviations, and intercorrelations for all primary variables. Results suggested that higher levels of pubertal development were associated with

TABLE 1. Descriptive Statistics and Correlations for Primary Study Variables

Variables	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	14.70	1.32	—												
2. Pubertal Stage	4.04	0.81	.65***	—											
3. Cortisol Timing	7.48	3.08	-.06	-.01	—										
4. Pre-Stressor Cortisol	-1.11	0.25	.06	.06	-.38**	—									
5. Pre-Stressor Self-Efficacy	2.74	0.52	-.08	-.29*	-.24	.24†	—								
6. Pre-Stressor Content	2.65	0.36	-.16	-.29*	-.15	-.09	.05	—							
7. Δ Cortisol	0.10	0.21	.16	.04	-.25†	.01	.22	.18	—						
8. Δ Self-Efficacy	0.05	0.55	-.13	-.20	-.16	.22	.35**	-.12	.08	—					
9. Δ Content	-0.02	0.38	-.08	-.13	.09	-.10	-.09	.65***	.01	-.06	—				
10. T1 Depressive Sx	0.47	0.47	-.16	.03	.13	.16	-.22	-.41***	-.21	.02	-.24	—			
11. T2 Depressive Sx	0.33	0.35	-.14	-.05	-.07	.12	.06	-.13	.00	.40**	-.05	.49***	—		
12. T3 Depressive Sx	0.30	0.37	-.27	-.12	.09	.12	.07	-.24	-.09	.41**	-.10	.61***	.73***	—	
13. T4 Depressive Sx	0.30	0.39	-.23	-.03	.02	.17	-.06	-.27	-.20	.29†	-.06	.78***	.68***	.81***	—

Notes: Cortisol Time = hours passed between awakening and pre-stressor cortisol sample; †$p < .06$, *$p < .05$, **$p < .01$, ***$p < .001$.

TABLE 2. Unstandardized and Standardized Regression Weights for Cortisol Predicting Latent Change in Depressive Symptoms Over Nine Months

	Intercept			Linear Slope			Quadratic Slope		
Predictors	b	S.E.	β	b	S.E.	β	b	S.E.	β
Medications	-.13	.03	-.75***	.02	.01	.63	-.00	.00	-.37*
Pre-stressor Cortisol	.08	.05	.26	-.02	.02	-.36	.00	.00	.17
Δ Cortisol	-.07	.06	-.17	.04	.02	.61	-.01	.00	-.36*

Note. *$p < .05$; ***$p < .001$.

higher levels of negative content in adolescents' selected responses to the social scenarios as well as lower self-efficacy in carrying out those responses. The timing of the pre-stressor cortisol sample was negatively related to the pre-stressor cortisol level such that samples taken later after wakening were lower in cortisol. Neither pre-stressor cortisol nor cortisol reactivity were associated with pre-stressor or change variables for self-efficacy and response content. Baseline depressive symptoms were negatively associated with the content of selected responses, indicating that individuals with higher levels of depressive symptoms were more likely to select responses with greater levels of negative content. Declines in self-efficacy from pre-to-post stressor were associated with depressive symptoms at 3- and 6-month follow-up.

To test for potential influences of medication usage on primary study variables, participants were divided into two groups. Individuals reporting the use of corticosteroids, birth control, and/or antidepressants were categorized into a "medication" group while all others were categorized into a "non-medication" group. Results from *t*-test analyses revealed that participants in the medication group did not differ on pre-stressor or change values for cortisol, self-efficacy, or content of selected response when compared to participants in the non-medication group. However, *t*-tests did reveal that the medication group showed significantly higher levels of depressive symptoms at baseline and all follow-up time points, than the non-medication group (all *t*s > 2.33). Given these significant group differences on levels of depressive symptoms over time, the medication classification was included as a predictor in subsequent analyses of longitudinal change in depressive symptoms.

Longitudinal Prediction of Depressive Symptoms

Before examining study hypotheses, an unconditional model using latent curve analysis was generated to examine growth in depressive symptoms over time. An initial model incorporated only an intercept and linear slope to characterize depressive symptom scores. This model was an adequate fit to the data, $\chi^2(5) = 10.68$, *ns*; $\chi^2/df = 2.14$; CFI = .95; RMSEA = .14; AIC = 28.68. However, inspection of the longitudinal data revealed a tendency for some adolescents' depressive symptoms to decrease over time, then increase at either 6 or 9 months post-baseline. Indeed, the measurement of depressive symptoms at four follow-up time points in this over-represented clinically referred sample was based on the expectation that adolescents may show a re-emergence of depressive symptoms following a period of decreasing symptoms. Thus, a second unconditional model incorporating a quadratic slope in depressive symptoms was computed. This model yielded a good fit to the data, $\chi^2(1) = 1.72$, *ns*;

TABLE 2a. Unstandardized and Standardized Regression Weights for Self-Efficacy and Response Content Predicting Latent Change in Depressive Symptoms Over Nine Months

	Intercept			Linear Slope			Quadratic Slope		
Predictors	b	S.E.	β	b	S.E.	β	b	S.E.	β
Medications	-.12	.03	-.61***	.02	.01	.35	-.00	.00	-.31*
Pre-Stressor Self-Efficacy	-.02	.03	-.09	.00	.01	.09	.00	.00	-.06
Pre-Stressor Content	-.11	.04	-.45*	.04	.02	.59*	-.00	.00	-.48*
Δ Self-Efficacy	-.01	.02	-.05	.03	.01	.60**	-.00	.00	-.44*
Δ Content	-.01	.04	-.06	.01	.02	.14	.00	.00	-.03

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

$\chi^2/df = 1.72$; CFI = .99; RMSEA = .11; AIC = 27.72. Thus, the model with both linear and quadratic slopes was utilized for hypothesis testing.

An initial conditional model examined prospective associations between cortisol reactivity and depressive symptoms, $\chi^2(16) = 12.18$, *ns*; $\chi^2/df = 0.76$; CFI = 1.00; RMSEA = .00; AIC = 110.18 (see Table 2 for model estimates). Results revealed that while controlling for associations among cortisol covariates (i.e., pubertal stage, age, cortisol timing), baseline medication usage, and pre-stressor cortisol, significant associations emerged for cortisol reactivity as a predictor of the quadratic slope of depressive symptoms, reflecting changes over time. Model implied trajectories of depressive symptoms were plotted at low (-1 *SD*), mean, and high (+1 *SD*) levels of cortisol reactivity (see Figure 1). Results revealed that low levels of cortisol reactivity (i.e., cortisol hyporeactivity) were associated with a re-emergence in depressive symptoms between 6 and 9 months post-baseline. Higher levels of cortisol reactivity, on the other hand, were associated with a slowly decreasing, more linear trend, predicting considerably lower levels of depressive symptoms at the 9-month follow-up.

A second model examined prospective associations between the two dimensions of social problem-solving skills (i.e., content of selected response and self-efficacy) and depressive symptoms, while also including medication use as a predictor, $\chi^2(5) = 4.82$, *ns*; $\chi^2/df = 0.80$; CFI = 1.00; RMSEA = .00; AIC = 100.82 (see Table 2a for model estimates). Results revealed two significant findings.

First, although no effect was revealed between stressor-related *changes* in the content of adolescents' selected response and depressive symptoms, results suggested that higher levels of negative content in problem-solving responses pre-stressor were associated with steeper increases in depressive symptoms over time (see Figure 2).

Results also were revealed for self-efficacy, as hypothesized. Pre-post stressor change in self-efficacy was significantly associated with both linear and quadratic slopes of depressive symptoms. As shown in Figure 3, larger pre-post stressor differences in self-efficacy (i.e., greater decreases in self-efficacy consequent to stress) were associated with steeper and more immediate increases in depressive symptoms over time.

DISCUSSION

Increases in interpersonal stressors are believed to be partially responsible for the increased risk for depressive symptoms observed during the adolescent transition period. However, specific mechanisms through which interpersonal stress may affect depres-

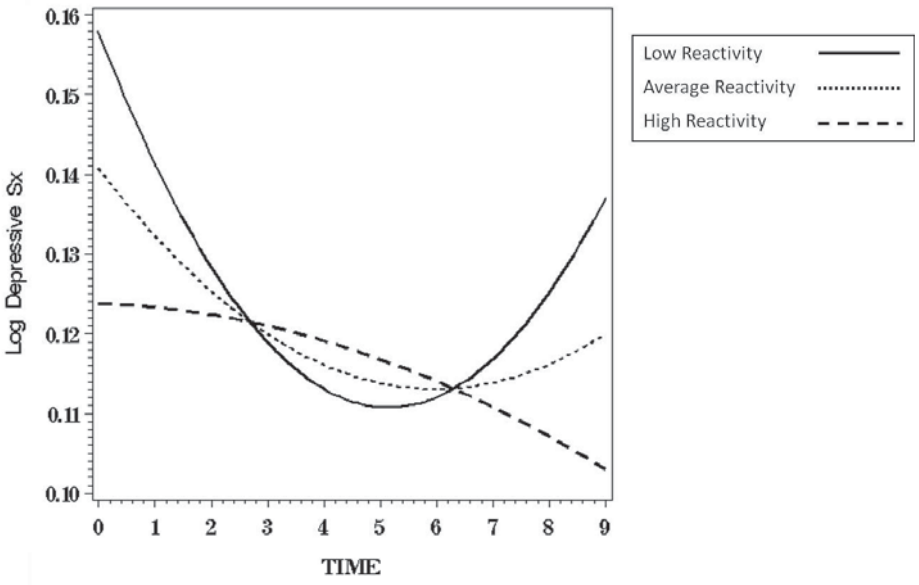

FIGURE 1. Quadratic functions for varying levels of cortisol reactivity (average = mean; high/low = mean +/- 1 SD) predicting depressive symptoms over nine months.

sive symptoms are not well-established. Some models have proposed that adolescents who are especially vulnerable to the biological and cognitive effects of stress may be more likely to develop depressive symptoms over time (Cicchetti & Walker, 2001; Crick & Dodge, 1994; Lemerise & Arsenio, 2000; Spear, 2000; Walker, 2002). Unfortunately, there are very few experimental or longitudinal tests of these models. The present study addressed this crucial gap by testing the prospective associations of cortisol reactivity and social problem-solving skill decrements, occurring in response to an acute interpersonal stress task, with the development of depressive symptoms over time.

Results revealed a significant quadratic association between biological responses to stress and later trajectories of depressive symptoms. Specifically, results indicated that a hyporeactive HPA response to an in vivo acute interpersonal stressor was associated with a later re-emergence of depressive symptoms. This finding was consistent with some prior work. For instance, Hastings and colleagues (2011) revealed that blunted HPA reactivity to an acute stressor predicted increasing internalizing problems over two years, but this was only for adolescent girls with relatively high blood pressure reactivity or low heart rate reactivity (e.g., dominance of sympathetic reactivity in autonomic regulation). In adults, an experience sampling study showed that depressed individuals had more blunted cortisol responses to negative daily events than healthy controls; this effect was especially pronounced for women (Peeters, Nicholson, & Berkhof, 2003). Similarly, blunted cortisol responses to experimentally administered stress tasks have been concurrently associated with depressive symptoms for women with very low incomes (Burke et al., 2005). Of particular relevance to the current study's findings, women remitted from recurrent major depression have shown more blunted cortisol responses when compared to healthy controls (Ahrens et al., 2008). Though the present study did not determine if adolescents later experienced Major

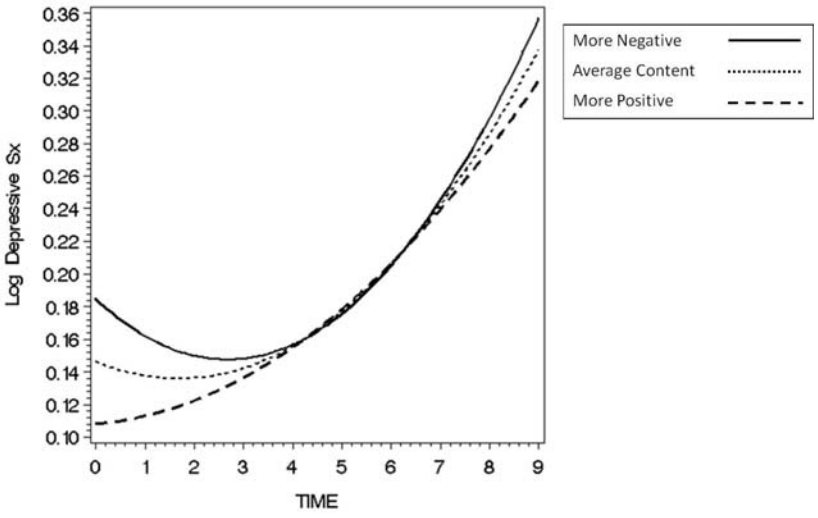

FIGURE 2. Quadratic functions for varying levels of response content (average = mean content; positive/negative = mean +/- 1 SD) predicting depressive symptoms over nine months.

Depressive "episodes," as determined by a structured diagnostic interview, the quadratic effects observed in the current study approximate the findings of Ahrens and colleagues (2008) and suggest that cortisol hyporeactivity to acute stress may be a unique biological disposition for some individuals at-risk for *recurring* instances of high depressive symptoms. For these individuals, blunted cortisol reactivity may foreshadow maladaptive biological responses to life stressors and subsequent presentations of elevated depressive symptoms.

It has been suggested that blunted HPA stress-responses may render individuals less prepared to manage stressful experiences (Yehuda, 1997). Adding to this theory, it could be that individuals with such a biological disposition are more likely to experience recurring depressive symptoms as they fail to adequately respond to stressful life events that may arise. It is also worth noting that low reactivity to social stress in adolescence is uncharacteristic of the normative maturational course of HPA reactivity (Gunnar et al., 2009), again suggesting that this could reflect HPA dysregulation that conveys risk for poor adjustment in adolescents. More longitudinal work is needed to determine if cortisol reactivity to acute social stress is indicative of stable or fluctuating trajectories for depressive symptoms during adolescence.

It is important to note that the quadratic nature of the findings could also be partially explained by the oversampling method used for study inclusion. Given that some adolescents participated shortly after discharge from an inpatient unit, the study's sample likely included adolescents with especially heightened levels of baseline depressive symptoms. While adolescents at the peak of their depressive symptoms are less likely to experience subsequent short-term increases in symptoms, they may be more likely to experience a recurrence of high levels of depressive symptoms over a more substantial period of time, such as 9 months.

Findings from the present study are among the first to demonstrate an association between adolescent social problem-solving and depressive symptoms. Results coincide with those found in adult samples (e.g., Nezu & Ronan, 1985) by revealing a pro-

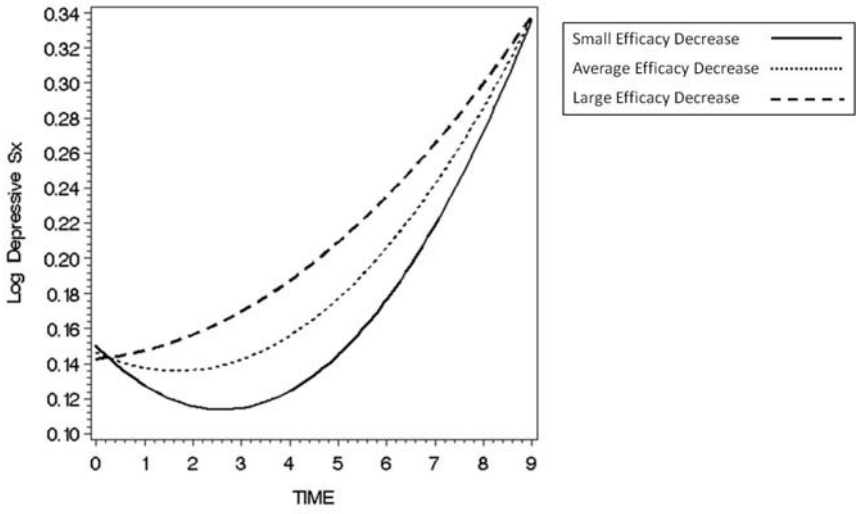

FIGURE 3. Quadratic functions for varying levels of self-efficacy change (average = mean change in self-efficacy; large/small = mean +/- 1 SD) predicting depressive symptoms over nine months.

spective association between social problem-solving deficits and depressive symptoms. More specifically, results suggest that the ability to select more adaptive responses to social problems may be protective against the development of depressive symptoms during adolescence. Results also suggest that the ability to maintain perceptions of high self-efficacy in the presence of stress may protect against increases in depressive symptoms over time. Identifying adolescents at risk for experiencing significant interpersonal stress and helping them develop appropriate problem-solving skills, as well as confidence to successfully enact these skills, may be important avenues for prevention efforts.

As they relate to depressive symptoms, it could be that stress-diminished perceptions of self-efficacy result from biological over- or underarousal, cognitive overload, or stress-induced reductions in self-esteem. Although this study did not reveal a significant relationship between cortisol reactivity and social problem-solving skills, it is possible that other biological stress response systems (e.g. autonomic) may be linked with cognitive manifestations of stress.

Future research on this topic would benefit by addressing the limitations of this study. First, the generality of study findings is limited by a predominately female and Caucasian sample. Also, parents' levels of education was fairly high. Replication with demographically diverse samples is needed. Second, participants in this sample did not report especially high levels of clinical symptoms. It is possible that observed processes may differ among adolescents with more severe levels of depressive symptoms. Further, the present study did not collect information regarding treatment (medicinal or psychotherapy) during follow-up assessments; post-baseline treatments could certainly prove influential on the developmental course of depressive symptoms. Regarding the study's methodology, the use of a single self-reported item to assess perceived self-efficacy may not have produced the most reliable measure of the construct. However, this method of assessing self-efficacy has previously demonstrated significant value, particularly in examinations of internalizing symptoms and self-injury (Nock &

Mendes, 2008). Lastly, though the use of an analog social stress task likely provides a more ecologically valid method of assessment compared to many other methods, such a task may yield different responses than the complex stressors adolescents experience in their actual lives.

In summary, the present study provides support for biological and cognitive models explaining how interpersonal stress may relate to the development of depressive symptoms. Specifically, findings suggest that HPA stress response and social problem-solving abilities may uniquely relate to depressive symptoms over time. Prevention efforts that focus on reducing, or correcting, maladaptive physiological and cognitive responses to social stress may be important for reducing the prevalence of depression in adolescence.

REFERENCES

Abela, J. R., & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents: A developmental psychopathology perspective. In J. Abela & B. Hankin (Eds.), *Handbook of depression in children and adolescents* (pp. 35-78). New York: Guilford.

Adam, E. K., Sutton, J. M., Doane, L. D., & Mineka, S. (2008). Incorporating hypothalamic-pituitary-adrenal axis measures into preventative interventions for adolescent depression: Are we there yet? *Development and Psychopathology*, *20*, 975-1001.

Ahrens, T., Dueschle, M., Krumm, B., van der Pompe, G., den Boer, J. A., & Lederbogen, F. (2008). Pituitary-adrenal and sympathetic nervous system responses to stress in women remitted from recurrent major depression. *Psychosomatic Medicine*, *70*(4), 461-467. doi: 10.1097/PSY.0b013e31816b1aaa

Angold, A. (1989). Structured assessments of psychopathology in children and adolescents. In C. Thompson (Ed.), *The instruments of psychiatric research* (pp. 271-394). Chichester, UK: Wiley.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status, and pubertal timing. *Psychological Medicine, 28*, 51-61. doi: 10.1017/S003329179700593X

Blakemore, S. J. (2008). The social brain in adolescence. *Nature Reviews Neuroscience*, 9(4), 267-277. doi: 10.1038/nrn2353

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation perspective*. Hoboken, NJ: Wiley.

Bouma, E. M., Riese, H., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2009). Adolescents' cortisol responses to awakening and social stress: Effects of gender, menstrual phase and oral contraceptives. The TRAILS study. *Psychoneuroendocrinology*, *34*, 884-893. doi: 10.1016/j.psyneuen.2009.01.003

Brent, D. A., Perper, J. A., Moritz, G., Allman, C., Friend, A., Roth, C., et al. (1993). Psychiatric risk factors for adolescent suicide: A case-control study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*(3), 521-529.

Burke, H. M., Fernald, L. C., Gertler, P. J., & Adler, N. E. (2005). Depressive symptoms are associated with blunted cortisol stress responses in very low-income women. *Psychosomatic Medicine*, *67*(2), 211-216. doi: 10.1097/01.psy.0000156939.89050.28

Carter, J. S., & Garber, J. (2011). Predictors of the first onset of a major depressive episode and changes in depressive symptoms across adolescence: Stress and negative cognitions. *Journal of Abnormal Psychology*, *120*(4), 779-796. doi: 10.1037/a0025441

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *Journal of the American Medical Association*, *267*(9), 1244-1252. doi: 10.1001/jama.1992.03480090092034

Cicchetti, D., & Walker, E. F. (Eds.). (2001). Stress and development: Biological and psychological consequences. *Development and Psychopathology*, *13*(3), 413-753.

Costello, E. J., & Angold, A. (1988). Scales to assess child and adolescent depression: Checklists, screens, and nets. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*(6), 726-737. doi: 10.1097/00004583-198811000-00011

Costello, E. J., Egger, H., & Angold, A. (2005). 10-year research update review: The epidemiology of child and adolescent psychiatric disorders: I. Methods and public health burden. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*(10), 972-986. doi: 10.1097/01.chi.0000172552.41596.6f

Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin*, *115*(1), 74-101. doi: 10.1037/0033-2909.115.1.74

DiClemente, R. J., Ponton, L. E., & Hartley, D. (1991). Prevalence and correlates of cutting behavior: Risk for HIV transmission. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*(5), 735-739. doi: 10.1097/00004583-199109000-00006

Dorn, L. D., Susman, E. J., Nottelmann, E. D., Inoff-Germain, G., & Chrousos, G. P. (1990). Perceptions of puberty: Adolescent, parent, and health care personnel. *Developmental Psychology*, *26*(2), 322-329. doi: 10.1037/0012-1649.26.2.322

Guerry, J. D., & Hastings, P. D. (2011). In search of HPA axis dysregulation in child and adolescent depression. *Clinical Child and Family Psychology Review*, *14*(2), 135-160. doi: 10.1007/s10567-011-0084-5

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, *34*(7), 953-967. doi: 10.1016/j.psyneuen.2009.02.010

Hankin, B. L., & Abela, J. R (2005). Depression from childhood through adolescence and adulthood: A developmental vulnerability and stress perspective. In B. Hankin & J. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 245-288). Thousand Oaks, CA: Sage.

Hankin, B. L., Badanes, L. S., Abela, J. R., & Watamura, S. E. (2010). Hypothalamic-pituitary-adrenal axis dysregulation in dysphoric children and adolescents: Cortisol reactivity to psychosocial stress from preschool through middle adolescence. *Biological Psychiatry*, *68*(5), 484-490. doi: 10.1016/j.biopsych.2010.04.004

Hankin, B. L., Stone, L., & Wright, P. A. (2010). Corumination, interpersonal stress generation, and internalizing symptoms: Accumulating effects and transactional influences in a multi-wave study of adolescents. *Development and Psychopathology*, *22*(1), 217-235. doi: 10.1017/S0954579409990368

Hastings, P. D., Fortier, I., Utendale, W. T., Simard, L. R., & Robaey, P. (2009). Adrenocortical functioning in boys with attention-deficit/hyperactivity disorder: Examining subtypes of ADHD and associated comorbidities. *Journal of Abnormal Child Psychology*, *37*, 565-578. doi: 10.1007/s10802-008-9292-y

Hastings, P. D., Shirtcliff, E. A., Klimes-Dougan, B., Allison, A. L., Derose, L., Kendziora, K. T., et al. (2011). Allostasis and the development of internalizing and externalizing problems: Changing relations with physiological systems across adolescence. *Development and Psychopathology*, *23*(4), 1149-1165. doi: 10.1017/S0954579411000538

Hastings, P. D., Zahn-Waxler, C., & Usher, B. A. (2007). Cardiovascular and affective responses to social stress in adolescents with internalizing and externalizing problems. *International Journal of Behavioral Development*, *31*(1), 77-87. doi: 10.1177/0165025407073575

Haugh, J. A. (2006). Specificity and social problem-solving: Relation to depressive and anxious symptomatology. *Journal of Social and Clinical Psychology*, *25*(4), 392-403. doi: 10.1521/jscp.2006.25.4.392

Hawker, D. S., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies. *Journal of Child Psychology and Psychiatry*, *41*(4), 441-455. doi: 10.1111/1469-7610.00629

Kaltas, G. A., & Chrousos, G. P. (2007). The neuroendocrinology of stress. In J. Cacioppo, L. Tassinary, & G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 303-318). New York: Cambridge University Press.

Klimes-Dougan, B., Hastings, P. D., Granger, D. A., Usher, B. A., & Zahn-Waxler, C. (2001). Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. *Development and Psychopathology*, *13*(3), 695-719. doi: 10.1017/S0954579401003157

Lemerise, E. A., & Arsenio, W. F. (2000). An integrated model of emotion processes and cognition in social information processing. *Child Development*, *71*(1), 107-118. doi: 10.1111/1467-8624.00124

Marx, E. M., Williams, J. M., & Claridge, G. C. (1992). Depression and social problem solving. *Journal of Abnormal Psychology*, *101*(1), 78-86. doi: 10.1037/0021-843X.101.1.78

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences*, *1032*(1), 1-7. doi: 10.1196/annals.1314.001

Morris, N. M., & Udry, J. R. (1980). Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence*, *9*(3), 271-280. doi: 10.1007/BF02088471

Negriff, S., Fung, M. T., & Trickett, P. K. (2008). Self-rated pubertal development, depressive symptoms, and delinquency: Measurement issues and moderation by gender and maltreatment. *Journal of Youth and Adolescence*, *37*(6), 736-746. doi: 10.1007/s10964-008-9274-y

Nezu, A. M. (1986). Efficacy of a social problem-solving therapy approach for unipo-

lar depression. *Journal of Consulting and Clinical Psychology*, *54*(2), 196-202. doi: 10.1037/0022-006X.54.2.196

Nezu, A. M., & Perri, M. G. (1989). Social problem-solving therapy for unipolar depression: An initial dismantling investigation. *Journal of Consulting and Clinical Psychology*, *57*(3), 408-413. doi: 10.1037/0022-006X.57.3.408

Nezu, A. M., & Ronan, G. F. (1985). Life stress, current problems, problem solving, and depressive symptoms: An integrative model. *Journal of Consulting and Clinical Psychology*, *53*(5), 693-697. doi: 10.1037/0022-006X.53.5.693

Nock, M. K. (2006). *Social Problem Solving Skills Test (SPSST): Administration and coding manual*. Unpublished manuscript, Harvard University, Cambridge, MA.

Nock, M. K., & Mendes, W. B. (2008). Physiological arousal, distress tolerance, and social problem-solving deficits among adolescent self-injurers. *Journal of Consulting and Clinical Psychology, 76,* 28-38. doi:10.1037/0022-006X.76.1.28

Oldershaw, A., Grima, E., Jollant, F., Richards, C., Simic, M., Taylor, L., & Schmidt, U. (2009). Decision making and problem solving in adolescents who deliberately self-harm. *Psychological Medicine*, *39*(1), 95-104. doi: 10.1017/S0033291708003693

Peeters, F., Nicholson, N. A., & Berkhof, J. (2003). Cortisol responses to daily events in major depressive disorder. *Psychosomatic Medicine*, *65*, 836-841. doi: 10.1097/01.PSY.0000088594.17747.2E

Plotsky, P. M., Owens, M. J., & Nemeroff, C. B. (1998). Psychoneuroendocrinology of depression: Hypothalamic-pituitary-adrenal axis. *Psychiatric Clinics of North America*, *21*(2), 293-307. doi: 10.1016/S0193-953X(05)70006-X

Prinstein, M. J., & Aikens, J. W. (2004). Cognitive moderators of the longitudinal association between peer rejection and adolescent depressive symptoms. *Journal of Abnormal Child Psychology*, *32*(2), 147-158. doi: 10.1023/B:JACP.0000019767.55592.63

Rao, U., Hammen, C., Ortiz, L. R., Chen, L., & Poland, R. E. (2008). Effects of early and recent adverse experiences on adrenal response to psychosocial stress in depressed adolescents. *Biological Psychiatry*, *64*(6), 521-526. doi: 10.1016/j.biopsych.2008.05.01

Rudolph, K. D., & Hammen, C. (1999). Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. *Child Development*, *70*(3), 660-677. doi: 10.1111/1467-8624.00048

Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews*, *24*(4), 417-463. doi: 10.1016/S0149-7634(00)00014-2

Spence, S. H., Sheffield, J., & Donovan, C. (2002). Problem-solving orientation and attributional style: Moderators of the impact of negative life events on the development of depressive symptoms in adolescence. *Journal of Clinical Child and Adolescent Psychology*, *31*(2), 219-229. doi: 10.1207/153744202753604494

Spence, S. H., Sheffield, J., & Donovan, C. (2003). Preventing adolescent depression: An evaluation of the Problem Solving for Life program. *Journal of Consulting and Clinical Psychology*, *71*(1), 3-13. doi: 10.1037/0022-006X.71.1.3

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, *9*(2), 69-74. doi: 10.1016/j.tics.2004.12.005

Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., & Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology*, *21*(1), 47-68. doi: 10.1017/S0954579409000042

Stroud, L. R., Papandonatos, G. D., Williamson, D. E., & Dahl, R. E. (2011). Sex differences in cortisol response to corticotrophin releasing hormone challenge over puberty: Pittsburgh pediatric neurobehavioral studies. *Psychoneuroendocrinology*, *36*, 1226-1238. doi: 10.1016/j.psyneuen.2011.02.017

Susman, E. J., Dorn, L. D., Inoff-Germain, G., Nottelman, E. D., & Chrousos, G. P. (1997). Cortisol reactivity, distress behavior, and behavioral and psychological problems in young adolescents: A longitudinal perspective. *Journal of Research on Adolescence*, *7*(1), 81-105. doi: 10.1207/s15327795jra0701_5

Walker, E. F. (2002). Adolescent neurodevelopment and psychopathology. *Current Directions in Psychological Science*, *11*(1), 24-28. doi: 10.1111/1467-8721.00161

Wood, A., Kroll, L., Moore, A., & Harrington, R. (1995). Properties of the Mood and Feelings Questionnaire (MFQ) in adolescent psychiatric outpatients: A research note. *Journal of Child Psychology and Psychiatry*, *36*(2), 327-334. doi: 10.1111/j.1469-7610.1995.tb01828.x

Yehuda, R. (1997). Sensitization of the hypothalamic-pituitary-adrenal axis in posttraumatic stress disorder. *Annals of the New York Academy of Sciences*, *821*, 57-75. doi: 10.1111/j.1749-6632.1997.tb48269.x