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Running head: BIOLOGICAL STRESS RESPONSE AND COGNITIVE
VULNERABILITY TO DEPRESSION IN ADOLESCENCE

Biological Stress Response and Cognitive Vulnerability to Depression in

Adolescence

A Thesis

Presented in Partial Fulfillment of the

Requirements for the Degree of

Master of Arts

By

Bridget M. Brush

July 2018

Department of Psychology

College of Science and Health

DePaul University

Chicago, IL

Thesis Committee

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To my family, who always inspire me to never give up.

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Biography

The author is from St. Augustine, Florida. She received her Bachelor of Science degree in Psychology from Florida State University. She began her doctoral work in 2015 in the Clinical/Child Psychology Program at DePaul University, Chicago, Illinois.

Table of Contents

Thesis Committee	i
Acknowledgements	iii
Biography	iv
List of Tables	vi
List of Figures	9
Abstract	1
Introduction	3
Negative Cognitive Style and Depression	5
Biological Stress Response and Depression	8
Negative Cognitive Style and Biological Stress Response.....	12
Rationale	14
Statement of Research Questions & Hypotheses	15
Method	15
Research Participants	15
Procedures.....	16
Measures	18
Results	21
Descriptive Statistics.....	21
Preliminary Analyses	22
Hierarchical Multiple Regression	22
Discussion	28
Cognitive Vulnerability and Cortisol Moderation	29
Sex Differences	30
Strengths and Limitations	32
Summary	34
References	35
Appendix A. Additional Mediation Analyses	48
Appendix B. Measures	48

List of Tables

TABLE 1. BIVARIATE CORRELATIONS BETWEEN MAIN STUDY VARIABLES	22
TABLE 2. NEGATIVE COGNITIVE STYLE AT T1 PREDICTING DEPRESSIVE SYMPTOMS AT T2	24
TABLE 3. CORTISOL STRESS RESPONSE AT T1 PREDICTING DEPRESSIVE SYMPTOMS AT T2	24
TABLE 4. INTERACTION BETWEEN NEGATIVE COGNITIVE STYLE AND CORTISOL STRESS RESPONSE AT BASELINE PREDICTING DEPRESSIVE SYMPTOMS AT FOLLOW-UP	25

List of Figures

FIGURE 1. INTERACTION BETWEEN NEGATIVE COGNITIVE STYLE AND CORTISOL
RESPONSE PREDICTING PROSPECTIVE DEPRESSIVE SYMPTOMS27

Abstract

Depression is the leading cause of worldwide disability. Rates of Major Depressive Disorder (MDD) increase exponentially over the adolescent transition, suggesting adolescence represents a key period of risk for the onset of depression. Previous research has associated both biological stress response and cognitive vulnerability with symptoms of depression; however, there is little research examining the joint effects of these two risk factors and symptoms of depression, especially during adolescence. The present study examined the association between symptoms of depression and two established risk factors for depression: cognitive vulnerability, as measured by negative cognitive style, and biological stress response, as measured by cortisol reactivity, in a diverse sample of 187 adolescents (52% female, mean age = 14.4 years-old). Participants completed interviews and questionnaires to assess depressive symptoms and cognitive styles, as well as a laboratory social stress task to elicit a biological stress response. Results showed that neither negative cognitive style at T1 (time 1) nor cortisol response to stress at T1 were statistically significant independent predictors of adolescents' depressive symptoms at T2 (time 2), when controlling for T1 depression, gender, and life stress. However, as hypothesized, a significant interaction effect between cortisol response to stress and negative cognitive style emerged. At lower levels of physiological reactivity to stress, a more negative cognitive style predicted more T2 depressive symptoms, but this relation was not found at higher levels of physiological reactivity. Additionally, sex, but not previous

stress exposure further moderated this relationship such that the effect was present for girls, but not for boys. Findings from the current study provide evidence that cognitive and biological factors interact to influence the onset of depression in adolescence. Results also shed light on potential mechanisms that contribute to observed sex differences in rates of depression over adolescence.

Introduction

Depression is the leading cause of worldwide disability and rates of major depressive disorder (MDD) increase exponentially over the adolescent transition, from 2% in childhood to approximately 20% of youth meeting criteria for MDD by the end of adolescence (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Thapar, Collishaw, Pine, & Thapar, 2012). Adolescence also marks the emergence of sex differences in rates of depression. In childhood, girls and boys show similar rates of depression, but by the end of adolescence, girls become twice as likely as boys to experience depression (Hankin & Abramson, 2001). Moreover, adolescent MDD is associated with impairments in social and academic functioning, comorbidity with other psychiatric disorders, increased risk of suicide (Brent, Baugher, Bridge, Chen, & Chiappetta, 1999), and shows substantial continuity with adult MDD (Weissman et al., 1999). The adolescent transition is associated with an increasing number of social, psychological, and academic stressors in addition to the biological changes associated with puberty (Eiland & Romeo, 2013). Previous research has also characterized adolescence as a period of increased stress sensitivity (Dahl, 2004; Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Romeo, 2010; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010), leaving adolescents particularly vulnerable to the deleterious effects of stress. Given the continuity between adolescent and adult functioning, as well as increased stress exposure, it is important to

identify causal pathways in order to pre-empt the disorder, and thus improve adjustment, productivity, and adult trajectories for at-risk adolescents.

Previous research has demonstrated a clear relationship between stress and onset of depression, such that greater stress exposure is associated with an increased likelihood of developing depression. Researchers have also uncovered a number of cognitive and biological factors implicated in adolescent depression. For example, some researchers have focused on individual differences in the processing of emotional information in depressed youth (e.g., (Taylor & Ingram, 1999), while others have attempted to delineate individual differences in neuroendocrine responses to stressors (Goodyer, Herbert, Tamplin, & Altham, 2000b). These studies have significantly advanced our understanding of depression in childhood and adolescence and helped refine our hypotheses regarding potential risk factors for the disorder.

However, a major limitation of this research is that these literatures have been developing independently of one another. This schism has left a need to integrate the cognitive aspects of depression with investigations of biological factors in order to gain a more comprehensive understanding of mechanisms of risk. For example, (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008; Hankin, 2012) have suggested a multidimensional approach across disciplines may be more informative when considering risk for depression. Drawing on previous research indicating that both biological and cognitive factors influence depression in adolescents, the current study will use an integrative framework to examine the joint effects of cognitive and

physiological predictors of depression in adolescence in terms moderating mechanisms of impact.

Negative Cognitive Style and Depression

Cognitive vulnerability has been proposed as one factor that may increase risk for depression, especially when faced with stressful life events. According to cognitive vulnerability theories of depression (Abramson, Metalsky, & Alloy, 1989), individuals have different styles of explaining and interpreting negative life events and some of these styles can be maladaptive. Maladaptive cognitions impact not only how individuals with see the world around them, but also how they view themselves. One prominent cognitive vulnerability theory- the Hopelessness Theory of Depression (HT) proposed by Abramson et al. (1989), argues that cognitive vulnerability is best characterized as a negative cognitive style by which individuals tend to make negative inferences about the cause of an event, consequences following the event, and self-worth implications related to the event. According to the HT, people who attribute negative life events to stable, global, and internal causes are more likely to develop depression than individuals who do not exhibit such negative inferential styles (see Abramson et al. 2002). In line with diathesis-stress models of depression, these depressogenic cognitive styles are activated by stressors, which initiates a pattern of negative information processing that leads to feelings of hopelessness and depression (Segal & Shaw, 1986).

Results from a number of studies have supported this hypothesis, providing evidence that negative cognitive styles may indeed contribute vulnerability to depression. Negative cognitive style has been associated with depression in cross-sectional studies (Ingram et al., 1998) as well as prospective cognitive vulnerability-stress research (Hankin, Abramson, Miller, & Haefffel, 2004), suggesting the cognitive vulnerability-stress interaction precedes and predicts depressive symptoms (Hankin & Abela, 2010). For example, Alloy and colleagues (2006) found that negative cognitive style increased vulnerability for first onsets of depression in undergraduates with no prior history of clinical depression. The same group also found that negative cognitive style predicted recurrences of depression, such that the high cognitive risk group was more likely to exhibit a recurrence of major and minor depression than the low cognitive risk group. Interestingly, the risk conferred by negative cognitive style specifically predicted depression and not any other disorder. This finding is consistent with other studies (e.g., Carter & Garber, 2011) showing a negative cognition by stress interaction predicts significant changes in depressive symptoms, suggesting negative cognitive style as a unique risk factor for depression and not psychopathology in general.

Cognitive vulnerability factors (e.g. negative cognitive style) may also interact with negative life events to predict depression over the adolescent transition. Youth who encounter many negative life events are hypothesized to adopt a pattern of attributions for these negative events that, over time,

become stable and global. Once this attributional pattern becomes more trait-like, these youth are more likely to respond with hopelessness to future stressors, increasing their likelihood of developing depression. This notion is supported by the work of Morley & Moran (2011) showing that depression in adulthood is associated with the development of these vulnerability factors in childhood. In addition to being highly prevalent, depression is also a highly recurrent disorder, with over 80% of depressed individuals experiencing more than one depressive episode (Belsher & Costello, 1988). Some researchers suggest the high rate of recurrence likely reflects the presence of stable vulnerability factors which may increase an individual's risk for depression (Gotlib & Neubauer, 2000). Therefore, adolescence may represent the ideal period to target interventions aimed at changing maladaptive cognitive styles, before they become stable traits.

There is emerging evidence suggesting that cognitive vulnerabilities may play an important role in explaining sex differences in depression, as sex differences in cognitive vulnerability factors appear to emerge in conjunction with sex differences in depression. In childhood, girls and boys show similar rates of depression; however, by the end of adolescence, girls become twice as likely as boys to experience depression (Hankin & Abramson, 2001).

Similarly, sex differences in cognitive vulnerability factors, such as negative inferential styles about causes and consequences of events, have not been found in studies using young children (Abela & McGirr, 2007), whereas they have been consistently demonstrated in adolescent populations, with girls

reporting higher levels of negative cognitive style (Abela & McGirr, 2007; Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008; Mezulis, Funasaki, & Hyde, 2011). For example, Hankin and Abramson (2002) used a cross-sectional design to study of 219 public high school students and found that negative cognitive style explained gender differences in depressive symptoms such that general cognitive style, attributional style, and negative inferences mediated girls' greater levels of depressive symptoms compared to boys. Together, these studies provide compelling evidence that negative cognitive style may contribute to gender differences in depression that emerge over the adolescent transition.

Biological Stress Response and Depression

Previous research has also demonstrated a clear link between stress and depression (Calabrese, Molteni, Racagni, & Riva, 2009; Hammen, 2005; Mazure, 1998), such that individuals experiencing a greater number of life stressors are more likely to develop depression. Additionally, major stressors are known to precede the first onset of major depression (for review see Stroud, Davila, & Moyer, 2008), such that individuals experiencing their first episode of depression are more likely to have experienced a recent stressful event. However, not everyone who encounters a major stressor develops depression; therefore, a large body of research has focused on examining the various mechanisms involved in regulating stress response and factors that may cause some individuals to become particularly vulnerable to the effects of stress.

The hypothalamic-pituitary-adrenocortical (HPA) axis and its byproduct (cortisol) are markers of stress regulation and coping. When an organism interprets a situation as being stressful the autonomic nervous system (ANS) and the (HPA) axis both regulate physiological response to stress. These stress response systems activate the “fight-or-flight” response and promote adaptation to stress (McEwen, 2007). During stress reactivity, the activation of the HPA axis causes neurons in the hypothalamus to release a hormone called corticotropin-releasing hormone (CRH). The secretion of CRH initiates the release of another hormone called adrenocorticotropin (ACTH) from the pituitary gland, which subsequently triggers the synthesis and release of two main stress hormones: glucocorticoids and catecholamines. Glucocorticoids (called corticosterone in animals, and cortisol in humans) and catecholamines (epinephrine and norepinephrine) are considered the primary stress response “mediators” (Romeo, 2013). Therefore, cortisol levels produced in response to a stressor may reflect an individual’s ability to cope with that stressor (Gotlib, Joormann, Minor, & Hallmayer, 2008). Further, receptors for glucocorticoids and catecholamines are present in cells throughout the body and brain and have therefore been implicated in a variety of physical and mental health outcomes. Thus, interest in the roles of biological stress response regulation and dysregulation in normative and psychopathological development has burgeoned in the last two decades. Specifically, research has begun to focus on neuroendocrine bases of stress regulation, and their relationships to psychological adjustment. In adult

studies, several lines of research have provided evidence of stress hormones as depressogenic variables (Van Praag, 2002; Plotsky et. al., 1998) suggesting that dysregulation of stress hormones such as cortisol may play a role in the pathophysiology of depression.

A healthy stress response is characterized by a quick rise in cortisol levels followed by a sharp decline upon the end of the stressor. However, continual activation of stress response systems has been shown to cause downregulation of the HPA-axis, leading to a blunted stress response. Therefore, much like cognitive vulnerability factors, it can be conceptualized as a product of experiences with our environment and the individuals in our environment, in addition to our genetic makeup. There is increasing evidence for recalibration of HPA-axis activity in response to chronic stress exposure. For example, chronic stress has been associated with elevated baseline (resting) cortisol levels (Juster, McEwen, & Lupien, 2010), blunted cortisol response to acute stressors (McEwen 2000), and delayed cortisol recovery to pre-stress levels after removal of the stressor (Wingenfeld et al. 2009). Therefore, the HPA-axis may be heavily influenced by negative life events over childhood and adolescence, thus altering HPA profiles into adulthood. Such alterations in stress responsivity may help explain why childhood adversity is a risk factor for the development of depression in adulthood (McLaughlin et al., 2010).

So far, studies have demonstrated stress response dysregulation in childhood is associated with cognitive dysfunction, behavioral problems, and

mood disorders (Lupien, McEwen, Gunnar, & Heim, 2009). However, unlike the consistent finding regarding HPA hyperactivity and depression in adults, directionality has been less clear in children and adolescents. For example, while Luby et al. (2003) found that depressed preschoolers demonstrated increased cortisol response to stressors, Dorn & Chrousos (1997) found that depressed adolescents demonstrated significantly lower, or hypoactive, cortisol profiles compared to non-depressed controls. On the other hand, Susman et al. (1999) found no association between cortisol and depression in a group of adolescent girls. One factor that may contribute to mixed findings is the use of parent versus child reported symptoms of depression.

Lakdawalla, Hankin, and Mermelstein (2007) argue for the use of multiple informants and multiple methods when assessing youth depressive symptoms as depressed adolescents, similar to depressed adults, tend to have a more pessimistic outlook which may cause them to overestimate their symptom severity. Therefore, parent reported symptoms may offer a more conservative estimate of youth's depressive symptoms. Further research is needed over the adolescent transition in order to understand whether stress response (dys)regulation may represent an important candidate risk process in adolescent depression.

More recently, research has started to focus on examining stress response in adolescent populations at high risk for depression (based on family history of MDD). There is now evidence that youth at risk for depression vary from their same age peers with respect to their HPA axis

functioning. Compared to adolescents without depression, adolescents at high-risk for depression have been found to exhibit elevated cortisol response to stress. For example, Gotlib et al. (2006) exposed adolescent girls at risk for depression (based on maternal history of MDD) to a 15- minute stress session and found the at-risk girls showed increased cortisol response to the stressor compared to the control group. These findings lead the authors to conclude that stress may activate the HPA axis in high-risk adolescents to a greater extent than their less vulnerable peers. Other studies have shown at-risk adolescents exhibit higher morning cortisol levels than expected, which may reflect an overproduction of circulating cortisol acting as a risk factor for onset of depression (Goodyer, Herbert, Tamplin, & Altham, 2000; Halligan, Herbert, Goodyer, & Murray, 2007; Mannie, Harmer, & Cowen, 2007). Overall, findings suggest HPA axis functioning may represent a trait like vulnerability factor for depression as well as a mechanism by which stress may play a role in the onset of the disorder.

Negative Cognitive Style and Biological Stress Response

It is likely that both biological and cognitive factors influence depression in adolescents. However, few studies have examined how these constructs interact by including both variables in the same models. This may be an important next step considering findings by Gaab, Rohleder, Nater, & Ehlert (2005) showing cortisol changes in response to stressors are closely tied to cognitive appraisals of the events. This study assessed cognitive processes involved in neuroendocrine responses to acute stress in 81 healthy

males and found that anticipatory cognitive appraisal explained 35% of the variance in salivary cortisol response to the TSST. Moreover, anticipatory cognitive appraisal was a better predictor of cortisol stress response than retrospective stress appraisal or general personality factors. A meta-analysis of acute laboratory-based stressors and emotion indices by Denson, Spanovic, & Miller (2009) demonstrated a significant positive relationship between rumination and cortisol reactivity, suggesting cognitive appraisals and emotional responses predict cortisol response to stress. Thus, there is preliminary evidence to suggest that there may be moderating mechanisms of joint influence on depression.

A moderating model suggests that individuals high in both cognitive vulnerability and biological vulnerability will have the greatest risk for depressive symptoms. Individuals differ not only in the number of stressors they are exposed to, but also in how they modulate their stress response with psychological variables (e.g. cognitive styles) and physiological variables (cortisol response). Therefore, there is reason to believe that at similar levels of stress, individuals who exhibit more maladaptive responses (excessive cortisol secretion + negative cognitive style) will be more likely to develop symptoms of depression than individuals with more adaptive responses. This model is supported by Bouhuy et al.'s (2006) study of a remitted sample of depressed adults, showing that although neurobiological (e.g cortisol) and psychosocial (e.g. fear perception) measures were individually somewhat predictive of course of depression, the combination of these measures was the

most powerful predictor of relapse. Moderation analyses showed that remitted patients with high levels of cortisol secretion and high levels of fear perception were at higher risk of becoming depressed again as well as remitted patients with low levels of cortisol secretion and low levels of fear perception.

Rationale

While there are numerous investigations examining cognitive vulnerability and biological stress response as independent risk factors for depression, research has neglected to examine how these two risk factors interact. Greater understanding of how these known risk factors interact may increase the predictive power of risk factors and help identify mechanisms for intervention. Therefore, the present sought to examine moderating mechanisms of joint influence on adolescent depression by measuring links between negative cognitive style, cortisol response to stress, and prospective changes in depressive symptoms in a diverse community sample of urban adolescents. This population is of particular interest given adolescence represents a period of development associated with increases in both number of stressors and rates of depression (Avenevoli et al., 2015; Thapar et al., 2012). Drawing on previous research findings, we also aimed to test whether the impact of cortisol reactivity on negative cognitive style and depressive symptoms varied by sex or life stress exposure.

Statement of Research Questions & Hypotheses

The current study will examine the following hypotheses and research questions:

Hypothesis I: Decreased cortisol response to stress (T1) will be associated with increased symptoms of depression (T2).

Hypothesis II: Adolescents with higher levels of negative cognitive style (T1) will have increased symptoms of depression (T2).

Hypothesis III: The interaction between negative cognitive style (T1) and cortisol response to stress (T1) will significantly predict symptoms of depression (T2), such that adolescents with high levels of negative cognitive style (T1) and low cortisol reactivity (T1) will develop the greatest number of depressive symptoms (T2).

Research Question IV: Does the impact of cortisol reactivity on negative cognitive style and depression differ by sex?

Research Question V: Does the impact of cortisol reactivity on negative cognitive style and depression differ by previous stress exposure?

Method

Research Participants

Participants were taken from a larger study of stress and coping in urban youth. The current study included 187 adolescents from a larger study stress and coping in urban youth: 52% female, 84% minority, and on average

14.40 years old ($SD = 1.9$ years). Those in the in the larger study were excluded from the current study based on factors known to influence cortisol reactivity, including use of oral contraceptives, thyroid medications, steroids, and psychotropic medications (Granger, Hibel, Fortunato, & Kapelewski, 2009; Hibel, Granger, Kivlighan, & Blair, 2006; Rohleder & Nater, 2009). Participants were also excluded from the current study if they endorsed smoking or regular alcohol and/or drug use, given links between substance use and alterations in stress response (Kirschbaum & Hellhammer, 1994).

Procedures

All measures and protocols were approved by IRB at DePaul University and Northwestern University. Consent and assent forms were collected for all participants. Each participant attended an all-day data collection event at DePaul University on one of five consecutive Saturdays during the fall of 2012. Participants were randomly assigned to one of three groups, which determined the order of tasks they completed. All groups started with check-in, orientation, and breakfast. Next, each group completed either life stress interviews, the Group Public Speaking task for Adolescents (GPST-A), health and executive functioning measures, surveys, a campus tour, or watched short films. Lunch was served after the initial activity and then the groups went on to complete the remaining two tasks they had not yet completed.

The stress paradigm in the current study, the Group Public Speaking task for Adolescents (GPST-A; (Hostinar, McQuillan, Mirous, Grant, &

Adam, 2014), is based on an adaptation of the Trier Social Stress Test for children (TSST-C; Buske-Kirschbaum et al., 1997), as well as a recently created version in group format (TSST-G; von Dawans, Kirschbaum, & Heinrichs, 2011). The GPST-A session began with a baseline mood survey. Research assistants then demonstrated saliva sampling using the passive drool method. Participants were instructed to complete the first saliva sample (approximately 15 minutes before the start of the GPST-A) and then either completed a form with their contact information (for a random subsample participating in a diary study) or a brief positive mood induction (for those not in the diary study). Participants were told to prepare a 1.25-min speech to introduce themselves to a hypothetical classroom of students and were asked to discuss positive and negative aspects of themselves in their introduction. Participants were informed that judges trained to evaluate speech content and body language would enter the room and call participants in random order to begin their speech and may return to ask them further questions at any point. Adolescents had 3 minutes to prepare their speech before the judges entered the room. Participants then provided their second saliva sample (0 minutes before the start of the GPST-A) at the end of their speech preparation period. The two judges, always one male and one female, entered the room dressed in professional business attire and sat at the front of the room with a conspicuous video camera that was set up to record the participants' speeches. Judges called on participants in a random order by their divider number to begin their speech and retained a neutral expression throughout each speech. Judges also

prompted participants to continue speaking if their speech ended early or they were quiet for more than 20 seconds.

Immediately following the group speech task, the judges left the room and participants provided the third saliva sample (approximately 15 minutes after the start of the GPST-A). The participants then completed a post-task mood survey, provided the fourth saliva sample (approximately 30 minutes after the start of the GPST-A), and moved to a different room for debriefing and recovery. During the debriefing and recovery session, all participants were informed about the goals of the study, told their speeches were not actually evaluated for content, and were reassured about their performance. A fifth saliva sample (approximately 40 minutes after the start of the GPST-A) was collected after completion of the debriefing. Lastly, adolescents provided their sixth and final saliva sample (approximately 50 minutes after the start of the GPST-A) before moving to another room for the next set of assessments.

Adolescents returned for a follow-up data collection session during the spring of 2013. This data collection mirrored the fall session.

Measures

Adolescent Depressive Symptoms. Symptoms of depression were assessed at Time 1 and Time 2 using the Child Behavior Checklist for Ages 6–18 (CBCL/6–18; Achenbach & Rescorla, 2001). The CBCL is a norm-referenced behavior rating scale that asks parents use a three-point rating scale to rate 113 behavioral and emotional problems that have occurred during the

past six months. The subscale of Anxious/Depressed behaviors was used in the current analysis (Time 1 $\alpha = .82$, Time 2 $\alpha = .78$).

Negative Cognitive Style. The Adolescent Cognitive Style Questionnaire (ACSQ) is a self-report measure of cognitive vulnerability in youth as conceptualized by the Hopelessness Theory of Depression (Hankin & Abramson, 2002). The ACSQ contains 12 negative hypothetical events in achievement, interpersonal, and appearance domains and asks the adolescent to make inferences about the causes (internal-external, stable-unstable, global-specific), consequences, and self-worth implications of the hypothetical event. The child uses a 7-point Likert scale to rate the internality, stability, and globality of the cause of the hypothetical event, as well as the consequences and potential self-worth implications of each event. The ACSQ has demonstrated excellent internal consistency (α s ranging from 0.81 to 0.93; Hankin & Abramson, 2002), good validity (Alloy et al. 2012), and a factor structure consistent with the hopelessness theory (Hankin & Abramson, 2002). An overall score is obtained by computing the mean of all items across hypothetical events, with higher scores indicating a more negative cognitive style. Internal consistency was $\alpha = .93$ in the current study.

Adolescent Stressful Life Experiences. The Urban Adolescent Life Experiences Scale (UALES) was used to measure adolescents' major life events and daily hassles prior to Time 1. The UALES is a self-report questionnaire developed to measure stressful life events among urban adolescents (Allison et al., 1999). The UALES items were generated by low-

income, urban, predominantly African American, youth. Respondents are asked to rate the frequency with which they have been exposed to each of the stressful life experiences on a Likert scale ranging from never (1) to everyday (5), with higher values indicating greater frequency of exposure to stressors. The UALES has demonstrated high test–retest reliability among urban adolescents. Internal consistency was $\alpha = .85$ in the current sample.

Cortisol Response to Stress. Salivary cortisol is a reliable and valid measure of unbound, or free cortisol levels in plasma (Kirschbaum & Hellhammer, 1989; Kirschbaum & Hellhammer, 1994). It is a particularly useful measure for assessing acute changes in cortisol due to stress and has been used successfully in numerous studies with children and adolescents (Gunnar, Talge, & Herrera, 2009). Six saliva samples were collected from each participant over the course of the stress session. Participants expelled saliva through a straw into 5 ml tubes. Following collection, samples were frozen at -80°C until being shipped on dry ice to the University of Trier, Germany where free saliva cortisol concentrations were assayed using a time-resolved fluorescence immunoassay (dissociation-enhanced lanthanide fluorescent immunoassay [DELFIA]). Intra- and inter-assay coefficients of variation were below 10%. All samples were assayed in duplicate and averaged. Participants with unlikely cortisol values ($> 3SD$) were excluded from analyses. Consistent with prior studies assessing cortisol reactivity, the data were log transformed prior to analyses to assure normal distribution. Cortisol response to the GPSTA was calculated using Area Under the Curve

Increase (AUC_I) according to the method by Preessner and colleagues (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) which is derived from the trapezoid formula.

Results

Descriptive Statistics

The sample included a total of 187 adolescents (52% female; 48% male). The average age of participants was 14.4 years, ($SD=1.9$). The sample included 84% ethnic minority participants (32% Hispanic, 32% African-American, 12% Asian, 8% other) and 16% Non-Hispanic Caucasian adolescents. Average total family income fell in the \$50,001 to \$80,000 range, with over 46% of participants having a combined family income less than \$50,001. Demographic differences (age, gender, income, and race) were explored using independent samples t tests and analysis of variance on other main study variables of interest (negative cognitive style, total stress, and cortisol response to stress). Simple correlations between all study variables are reported in Table 1. Total stress measured by the UALES at T1 was positively associated with depressive symptoms at T1 but not T2. Only T1 depressive symptoms was associated with depressive symptoms at T2. Age and negative cognitive style were also associated with total stress at T1.

Table 1. Bivariate correlations between main study variables

Variable	1	2	3	4	5	6
1. T1 CBCL Anx/Dep	-					
2. T2 CBCL Anx/Dep	.51**	-				
3. T1 ACSQ	.44**	.12	-			
4. T1 UALES	.18*	.06	.33**	-		
5. Sex	.08	0.2	.02	-.02	-	
6. T1 Log Cortisol Reactivity	.03	.02	.02	.03	-.01	-
7. Age	.05	-.08	.07	.35**	.16	.02

Note: * $p < .05$, ** $p < .01$.

Preliminary Analyses

Before beginning substantive analyses, potentially relevant covariates were tested for significant associations with the outcome variable - depressive symptoms. Tested covariates included: previous stress exposure (UALES total stress), minority status, family income, sex, and age. These covariates were tested using independent samples t tests and analysis of variance. Age and previous stress exposure emerged as significant predictors of depressive symptoms at Time 1 and were therefore included as covariates in subsequent analyses. Additionally, given that the study focused on predicting change in depressive symptoms over time, Baseline (T1) levels of depressive symptoms were included as a priori covariates in all analyses predicting Time 2 depressive symptoms.

Hierarchical Multiple Regression

Main Effects. Individual hierarchical multiple regressions were performed to examine the independent contributions of negative cognitive style and cortisol response to stress. Regression statistics are reported in Table 2. In the first model, negative cognitive style was entered in the second step of

the analysis after entering age, stress, and T1 depressive symptoms in the first. In step one, T1 depressive symptoms made the only significant contribution to the regression model ($\beta = .55, p < .001$). Age, stress, and T1 depressive symptoms accounted for 33.7% of the variation in T2 depressive symptoms. In step two, Negative Cognitive Style did not make a significant contribution to the model ($\beta = .14, p = .22$) when controlling for age, stress, and T1 depressive symptoms and only accounted for an additional 1.8% of the variation in T2 Depressive symptoms. The overall model was significant, $F(4, 55) = 7.58, p < .001$.

In the next model, cortisol response was entered in the second step of the analysis after entering age, stress, and T1 depressive symptoms in the first. In step one, T1 depressive symptoms made the only significant contribution to the regression model ($\beta = .55, p < .001$). Age, stress, and T1 depressive symptoms accounted for 34.8% of the variation in T2 depressive symptoms. Including cortisol response did not make a significant contribution to the model ($\beta = -.06, p = .56$) when controlling for age, stress, and T1 depressive symptoms and only accounted for an additional 0.3% of the variation in T2 Depressive symptoms. The overall model was significant, $F(4, 56) = 7.59, p < .001$. Regression statistics are reported in Table 3.

Table 2. Negative cognitive style at baseline predicting depressive symptoms at follow-up.

Regression Variable	β	t	R^2	ΔR^2
Step 1			0.34	0.34***
Age	-0.09	-0.78		
UALES	0.10	0.84		
T1 CBCL Anx/Dep	0.55***	5		
Step 2			0.36	0.02
ACSQ	0.14	1.26		

* $p < .05$, ** $p < .01$, *** $p < .001$. Note: T1= Time 1 (baseline); 2= Time 2 (follow-up); CBCL Anx/Dep= Child Behavior Checklist Anxious/Depressed Subscale; ACSQ= Adolescent Cognitive Style Questionnaire; UALES= Urban Adolescents Life Events Scale.

Table 3. Cortisol stress response at baseline predicting depressive symptoms at follow-up.

Regression Variable	β	t	R^2	ΔR^2
Step 1			0.348	0.348***
Age	-0.10	-0.82		
UALES	0.14	1.15		
T1 CBCL Anx/Dep	0.55***	5.10		
Step 2			0.35	0.003
Cortisol	-0.06	-0.54		

* $p < .05$, ** $p < .01$, *** $p < .001$. Note: T1= Time 1 (baseline); 2= Time 2 (follow-up); CBCL Anx/Dep= Child Behavior Checklist Anxious/Depressed Subscale; ACSQ= Adolescent Cognitive Style Questionnaire; UALES= Urban Adolescents Life Events Scale; Cortisol =Log cortisol response to stress T1.

Interactive Effects. In order to test hypothesis 3, the two-way interaction between negative cognitive style and cortisol response to stress was created and entered in the third step of a regression equation. Control variables were entered in the first step and the main effects of negative cognitive style and cortisol response were entered in the second step. Consistent with our hypothesis, there was a significant interaction between negative cognitive style and cortisol response to stress at T1 in predicting depressive symptoms at T2, controlling for T1 depressive symptoms ($\beta = -$

1.54, $p = .02$, R^2 change = .061). Both T1 depression symptoms ($\beta = .58$, $p < .001$) and Negative cognitive style ($\beta = 1.51$, $p = .03$) were significant predictors in this model. The overall model was significant [$F(6, 50) = 6.09$, $p < .001$] and explained 42.2 % of the variation in T2 depressive symptoms. Regression statistics are reported in Table 4.

Simple slopes analysis using the PROCESS macro for SPSS (Hayes & Little, 2017) were conducted to probe the nature of this interaction and test the conditional effects of negative cognitive style at two levels of cortisol response to stress. The association between negative cognitive style and T2 depressive symptoms was significant at lower, but not higher, levels of cortisol response to stress (conditional effect = 1.26, $t = 2.78$, 95% CI = 0.31-2.16, $p < .01$) such that those with more negative cognitive styles had higher levels of depressive symptoms ($b = 1.23$, $p < .01$). At high levels of cortisol response to stress (+1 SD above mean), negative cognitive style was negatively associated with T2 depressive symptoms ($b = -.69$, $p = .24$), though this relationship did not reach statistical significance.

Table 4. Interaction between negative cognitive style and cortisol stress response at baseline predicting depressive symptoms at follow-up.

Variable	β	t	R^2	ΔR^2
Step 1			0.35	0.35***
Age	-0.08	-0.66		
UALES	0.14	1.10		
T1 CBCL Anx/Dep	0.58***	4.95		
Step 2			0.38	0.02
ACSQ	1.51*	0.15		
Cortisol	-0.03	-0.30		
Step 3			0.42	0.04*
ACSQ x Cortisol	-1.54*	-2.02		0.06

* $p < .05$, ** $p < .01$, *** $p < .001$. Note: T1= Time 1 (baseline); CBCL Anx/Dep= Child Behavior Checklist Anxious/Depressed Subscale; ACSQ= Adolescent Cognitive Style Questionnaire; UALES= Urban Adolescents Life Events Scale; Cortisol= Log cortisol response to stress T1.

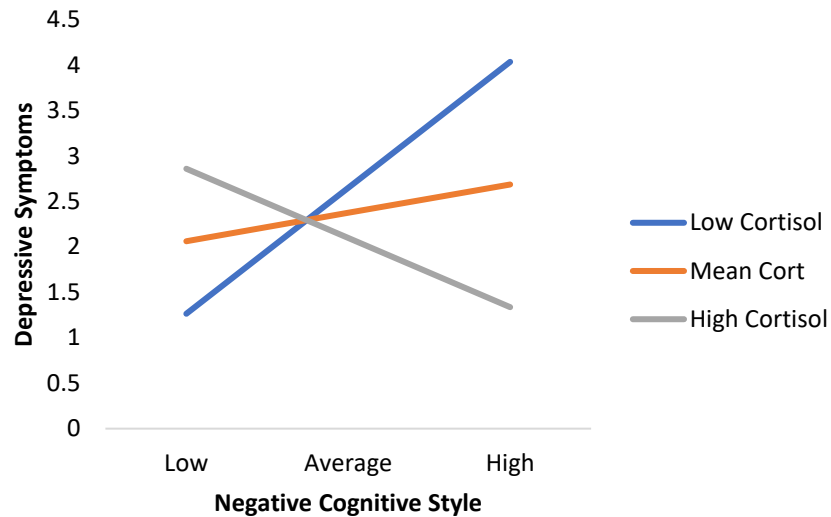
Based on our exploratory research questions, additional moderation analyses were conducted to test whether the impact of cortisol reactivity on negative cognitive style and depressive symptoms varied by sex or life stress exposure. Separate regression models were run using PROCESS (Model 2; Hayes, 2017) to test the conditional effect of Negative Cognitive Style on T2 Depressive Symptoms as a function of sex or total stress and Cortisol Reactivity from an additive multiple moderation model. For the gender model, we regressed T2 Depressive Symptoms (Y) on Negative Cognitive Style (X), Sex (W), Cortisol Reactivity (Z), the product of Negative cognitive style and Sex (XW), and the product of Negative Cognitive Style and Cortisol Reactivity (XZ). As in previous analyses, T1 depressive symptoms, age, and total stress were included as covariates.

Findings indicated both sex [$b = 1.58, t(48) = 2.94, p = .005$] and cortisol reactivity [$b = -2.17, t(48) = -2.22, p = .03$] functioned as moderators of the effect of negative cognitive style on T2 depressive symptoms. Specifically, among boys, negative cognitive style was a significant [$b = -1.46, t(48) = -2.42, p = .02$] predictor of T2 depressive symptoms only at high (1 *SD* above mean), but not low or mean levels of cortisol response to stress. Among girls, negative cognitive style predicted T2 depressive symptoms at low [$b = 1.90, t(48) = 2.73, p < .001$] and average [$b = 1.01, t(48) = -2.42, p =$

.01] but not high levels of cortisol response to stress. Tests of highest order unconditional interactions showed the moderation of the effect of negative cognitive style by sex uniquely accounts for 8.2% of the variance [$F(1,48) = 8.63, p < .01$], whereas the moderation by cortisol response uniquely accounts for 4.7% of the variance, [$F(1,48) = 4.95, p = .03$]. The overall model was significant [$F(8, 48) = 7.22, p < .001$] and explained 54.6 % of the variation in T2 depressive symptoms.

A similar model was built to test the influence of total stress exposure. For this model we regressed T2 Depressive Symptoms (Y) on Negative Cognitive Style (X), Total Stress (W), Cortisol Reactivity (Z), the product of Negative cognitive style and Total Stress (XW), and the product of Negative Cognitive Style and Cortisol Reactivity (XZ). Consistent with previous analyses, T1 depressive symptoms, age, and total stress were included as covariates. Only the interaction between Negative Cognitive Style and Cortisol Response was significantly [$b = -2.26, t(49) = -2.05, p = .05$] associated with T2 depressive symptoms in this model. The effect of negative cognitive style on depression did not vary by level of previous stress exposure [$b = -.01, t(49) = -.44, p = .66$].

Figure 1. Interaction between negative cognitive style and cortisol response predicting prospective depressive symptoms.



Discussion

The purpose of this study was to determine whether cognitive vulnerability and physiological stress response prospectively predicted depressive symptoms in adolescence. Using a diverse sample of adolescents, the study hypothesized that youth with a more negative cognitive style and more stress reactivity would experience the most depressive symptoms. We also expected that adolescents' cortisol response to stress would moderate the association between negative cognitive style and depressive symptoms by changing the direction of the relationship. Study hypotheses were partially supported. Neither negative cognitive style nor cortisol response to stress at were statistically significant independent predictors of adolescents' depressive symptoms at T2, when controlling for T1 depression, age, and life stress. However, as hypothesized, a significant interaction effect between cortisol response to stress and negative cognitive style emerged. At lower levels of

physiological reactivity to stress, a more negative cognitive style predicted more depressive symptoms, but this relation was not found at higher levels of physiological reactivity. Additionally, sex, but not previous stress exposure further moderated this relationship such that the effect was present for girls, but not for boys.

Cognitive Vulnerability and Cortisol Moderation

The present study first focused on the cognitive-vulnerability stress component derived from the Hopelessness Theory of depression. The finding of negative cognitive style assessed at baseline predicting prospective increases in depressive symptoms over time, after controlling for stress exposure, supports the cognitive vulnerability- stress component from HT. Given girls' increased incidence of depression over adolescence, we also examined sex as a potential moderator. We found differential effects of sex on the relationship between negative cognitive style and stress predicting change in depressive symptoms. This is in line with other findings of stronger cognitive vulnerability-stress interactions for girls compared to boys. For example, Prinstein & Aikins (2004) found a three-way interaction between 16-year old's attributional style, peer rejection stress, and sex such that the combination of a negative attributional style and peer stress at Time 1 predicted depressive symptoms at Time 2 for girls but not boys.

In order to incorporate known biological risk factors (i.e., HPA-axis dysregulation) and sex differences in adolescent depression, we also explored the Affective, Biological, Cognitive (ABC) model proposed by Hyde,

Mezulis, & Abramson (2008). This model of depression integrates affective, biological, and cognitive models to explain the emergence of sex differences in depression. According to this model, depressogenic vulnerability is characterized by the confluence of biological (i.e., genes, hormones, and pubertal timing), affective (i.e., temperament) and cognitive (i.e., negative cognitive style and rumination) vulnerabilities- which emerge or intensify in early adolescence- and are hypothesized to interact with negative life events to cause depression. Our finding that at differential levels of physiological reactivity to stress, negative cognitive style predicts depressive symptoms for adolescent girls in a different pattern than adolescent boys offers additional support for this model. The ABC model applies the concept of *equifinality*- the principle that individuals with a common outcome (e.g., adolescent depression) can be reached by many different pathways or trajectories (Cicchetti & Rogosch, 1996). The integrative framework of this model is important as it generates testable hypothesis to explain the emergence of sex differences in depression observed over adolescence. Additional longitudinal studies are needed to explore this model in greater detail.

Sex Differences

Sex, but not previous stress exposure, emerged as a significant moderator in the current study. These results are consistent with previous research showing no differences between girls and boys exposure to stressors (Maciejewski, Prigerson, & Mazure, 2001), and instead suggests girls' differential perceptions and increased sensitivity to stress may be related to

their higher rates of depression. Our finding that negative cognitive style interacted with blunted cortisol stress response to prospectively predict depressive symptoms for adolescent girls but not boys is also consistent with work by Hankin & Abramson (2002) demonstrating gender-related differences in processing stressful experiences may be related to observed rates of depression. For example, the study above found that cognitive style- in addition to attributional style and negative inferences for self, mediated high school girls' greater levels of depressive symptoms compared to boys. On the other hand, stress is an integral part of the diathesis- stress models adopted in this study; therefore, the lack of support for moderation by previous stress exposure is surprising. We suspect stress exposure failed to reach significance as a moderator due to the way it was measured in the current study, in addition to the inherent difficulty in establishing 3-way interactions. Based on previous research and relationships among the data, we hypothesize stress exposure is also implicated in this relationship; however, fuller tests of mediation with more frequent time points are needed to detect this relationship.

While models examined in the current study were particularly effective in predicting depressive symptoms for girls, they were less effective in predicting depressive symptoms for boys. Several possibilities may explain this finding. First, it is possible that sex hormones associated with the onset of puberty contributed to sex differences in cortisol response to stress. Empirical evidence showing the shifts in HPA-axis activity around the onset of puberty

is well established (Gunnar, Wewerka, et al., 2009). However, because the majority of adolescents in our sample were post-pubertal, as they were 14 years old during the beginning of the study, pubertal stage was less likely to contribute to sex differences in the current study. Another possible explanation could be related to the nature of the stress paradigm we used, or the value adolescents assigned to the stressor. The Group Public Speaking task for Adolescents (GPST-A) is a social stressor and was conducted in front of same age peers. Based on research demonstrating adolescent girls are particularly sensitive to social stress, it is possible they assigned more value to the stressor, thus influencing physiological responses to the stressor. Future research should further explore sex differences across multiple domains of stress.

Strengths and Limitations

The current study has several strengths. First, the prospective design is notable because it allowed us to examine change in depressive symptoms over time. Adding the dimension of time builds upon previous related cross-sectional studies which were limited by their inability to draw conclusions on risk-factors for depression. Second, the inclusion of biological and cognitive risk-factors for depression in the same study allowed us to test for interactive effects of joint influence across multiple systems. There is support for the inclusion of both physiology and cognition in intervention and prevention efforts using biofeedback. For example, Siepmann, Aykac, Unterdörfer, Petrowski, & Mueck-Weymann (2008) assessed the feasibility of using heart

rate variability feedback to treat moderate to severe depression and found a significant reduction in participants' depression symptoms and heart rate after only six sessions of biofeedback. Additionally, our use of multiple informants (parent and child report) to assess stress, cognitions, and symptoms of depression is a relative strength and minimizes observed effects of adolescents overestimating levels of distress on self-report measures of depression. Lastly, our study focused on a diverse, predominately low-income sample of urban youth. Therefore, results provide a more nuanced understanding of the role of cognitive and biological risk-factors in this high-risk, traditionally understudied adolescent population. Implications may help inform targets for prevention and intervention. For example, findings from the current study suggest cognitive therapeutic interventions such as cognitive restructuring may be particularly effective when paired with biofeedback skills aimed at identifying and regulating physiological responses to stress.

Results should be interpreted in the context of several limitations. First, adolescents' stressful life experiences were only assessed at Time 1. It is possible that additional stressors may have influenced the development of depressive symptoms at Time 2 independent from Time 1. Additionally, the total stress score on the UALES was used to measure overall levels of stress exposure. Future studies should investigate whether these effects are related to specific types of stress exposure (e.g. social stressors vs community violence). For example, Peckins et al. (2012) found previous exposure to violence was predictive of cortisol response to the TSST 12 months later in boys but not

girls, while exposure to violence was associated with symptoms of anxiety and depression in both boys and girls. Another study found that interpersonal stress (i.e., low maternal attachment) was associated with blunted cortisol response to the TSST in adolescent girls but not boys (Cameron et al., 2017). Next, our study focused on a community sample of low-income primarily minority youth; therefore, it may be important to replicate findings in other community samples. Given lower SES is commonly associated with greater cumulative stress, and some evidence of diminished cortisol reactivity to laboratory stressors for minority youth (Hajat et al., 2010), results may not be generalizable. Replication is also needed in samples selected for different risk factors, such as family history of depression. Future studies using clinical samples, as well as longitudinal designs following children over the entire adolescent transition are needed to provide further insight into the developmental antecedents of depression and shed light on periods when interventions may be particularly effective. Finally, HPA dysregulation and negative cognitions may have similar predictors (Alloy et al., 2001; Oskis, Loveday, Hucklebridge, Thorn, & Clow, 2011), but this study did not collect the data to examine their reciprocal influences over time.

Summary

In sum, paralleling the emergence of sex differences in rates of depression over adolescence, we found evidence of sex differences in the joint contribution of biological and cognitive risk factors on prospective changes in depressive symptoms among urban adolescents. Negative cognitive style

combined with blunted cortisol response to stress is associated with increased depressive symptoms for girls but not boys. Synthesizing prior independent bodies of literature documenting biological stress response and negative cognitions independently contribute to the way environmental stressors amplify risk for depression, results suggest moderating mechanisms of joint influence for adolescent girls. Findings have important implications for identifying risk factors for and etiological foundations of depression in adolescence as well as developing interventions to reduce adolescent girls' increased rates of depression.

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Appendix A. Additional Mediation Analyses

There was a nonsignificant indirect effect of negative cognitive style on T2 depressive symptoms through cortisol reactivity, $b = .0002$, 95% CI $[-.0047, .0046]$.

Appendix B. Measures

Urban Adolescents Life Experiences Survey (UALES)

WE WANT TO KNOW ABOUT THINGS THAT MAY OR MAY NOT HAVE HAPPENED TO YOU. PLEASE READ EACH OF THE SENTENCES BELOW AND FILL IN THE CIRCLE TO SHOW **HOW OFTEN** IT HAS HAPPENED TO YOU.

1. I get bad grades.

	Has Happened	Happens	Happens	Happens
Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

2. I change schools.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

3. I understand classwork.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

4. I have good school supplies.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

5. I have bad teachers.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

6. I flunk a grade.

Never	Has Happened Once or Twice	Happens Once a Month	Happens Once a Week	Happens Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

7. I do as well at school as my parents would like.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

8. A friend has died.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

9. Friends get in trouble.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

10. Friends get drunk.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

11. Friends use drugs.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

12. I have problems getting dates.

Never	Has Happened Once or Twice	Happens Once a Month	Happens Once a Week	Happens Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

13. I break up with a boyfriend or girlfriend.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

14. I fight with a boyfriend or girlfriend.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

15. A boyfriend or girlfriend cheats on me.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

16. A boyfriend or girlfriend uses drugs.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

17. A boyfriend or girlfriend sells drugs.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

18. A friend goes to jail.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

19. I see friends using drugs.

Never	Has Happened Once or Twice	Happens Once a Month	Happens Once a Week	Happens Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

20. I see friends drinking alcohol.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

21. I get beat up by a boyfriend or girlfriend.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

22. I have chores at home.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

23. A parent or family member is sick.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

24. I take care of younger family members.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

25. My parents get upset or worried.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

26. A parent or family member dies.

Never	Has Happened Once or Twice	Happens Once a Month	Happens Once a Week	Happens Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

27. Family gets on my nerves.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

28. Family gets along.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

29. A family member gets pregnant.

Never	Once or Twice	Once a Month	Once a Week	Once a Day
-------	---------------	--------------	-------------	------------

-
- 30. A parent dates someone new.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 31. I dislike who my parent dates.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 32. I do not see or have contact with a parent.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 33. A parent gets attacked, beat up, or injured.**
 Never Has Happened Happens Happens Happens
 Once or Twice Once a Month Once a Week Once a Day
-
- 34. Someone in my family goes to jail.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 35. I get along with my parent or parents.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 36. My parents break up or divorce.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 37. A parent moves out of the house.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 38. The parent I live with breaks up with his/her boyfriend or girlfriend.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 39. My parent's boyfriend or girlfriend moves out.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 40. My parents fight with each other.**
 Never Has Happened Happens Happens Happens
 Once or Twice Once a Month Once a Week Once a Day

41. I get punished.

Never Once or Twice Once a Month Once a Week Once a Day

42. I get pressure from parents or family to do better at school.

Never Once or Twice Once a Month Once a Week Once a Day

43. I have a parent who uses drugs.

Never Once or Twice Once a Month Once a Week Once a Day

44. I have a parent who drinks alcohol.

Never Once or Twice Once a Month Once a Week Once a Day

45. Children are taken from my home.

Never Once or Twice Once a Month Once a Week Once a Day

46. Family members get in trouble.

Never ^{Has Happened} Once or Twice ^{Happens} Once a Month ^{Happens} Once a Week ^{Happens} Once a Day

47. My neighborhood is noisy.

Never Once or Twice Once a Month Once a Week Once a Day

48. I see or hear about crime in my neighborhood.

Never Once or Twice Once a Month Once a Week Once a Day

49. I move to a new neighborhood.

Never Once or Twice Once a Month Once a Week Once a Day

50. I live in a crowded house or apartment.

Never Once or Twice Once a Month Once a Week Once a Day

51. A parent loses a job.

Never Once or Twice Once a Month Once a Week Once a Day

-
- 52. A parent remarries.**
 Never Has Happened Once or Twice Happens Once a Month Happens Once a Week Happens Once a Day
-
- 53. Someone new moves into my house.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 54. I lose my house in a fire.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 55. I visit a parent that doesn't live with me.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 56. A family member has an emotional problem.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 57. I have places to go.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 58. I'm able to do what I want to do.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 59. I lose a job.**
 Never Has Happened Once or Twice Happens Once a Month Happens Once a Week Happens Once a Day
-
- 60. I have trouble getting a job.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 61. I'm treated different because of my race.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 62. I am pressured into sex.**
 Never Once or Twice Once a Month Once a Week Once a Day

-
- 63. I am forced to have sex.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 64. I am touched in a way I do not like.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 65. I find out that I am pregnant.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 66. I find out I got someone pregnant.**

 Never Has Happened Happens Happens Happens
 Once or Twice Once a Month Once a Week Once a Day
- 67. I have a miscarriage.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 68. I have an abortion.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 69. I have a girlfriend who had a miscarriage.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 70. I have a girlfriend who had an abortion.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 71. I am concerned about getting AIDS.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 72. I know someone with AIDS.**
 Never Once or Twice Once a Month Once a Week Once a Day
- 73. I am asked to sell drugs.**
 Never Has Happened Happens Happens Happens
 Once or Twice Once a Month Once a Week Once a Day

-
- 74. People think I sell drugs.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 75. I am pressured to use drugs.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 76. People lie about me.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 77. I am taken advantage of.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 78. I am arrested or in trouble with the police.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 79. I go to jail.**
 Never Has Happened Happens Happens Happens
 Once or Twice Once a Month Once a Week Once a Day
-
- 80. I am placed on probation.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 81. I have enough money.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 82. I have the things I need (food, clothes, etc.)**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 83. Friends and family ask me for money.**
 Never Once or Twice Once a Month Once a Week Once a Day
-
- 84. I have transportation.**
 Never Once or Twice Once a Month Once a Week Once a Day

- | | | | | | |
|--|-----------------------|-------------------------------|-------------------------|------------------------|-----------------------|
| | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 85. I go to the hospital for treatment. | | | | | |
| | Never | Has Happened
Once or Twice | Happens
Once a Month | Happens
Once a Week | Happens
Once a Day |
| | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 86. I get hurt or injured. | | | | | |
| | Never | Once or Twice | Once a Month | Once a Week | Once a Day |
| | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| 87. I get robbed. | | | | | |
| | Never | Once or Twice | Once a Month | Once a Week | Once a Day |
| | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Adolescent Cognitive Style Questionnaire (ACSQ)

Directions

Please try to imagine yourself clearly in each of the situations that follow. Place yourself in each situation and decide what you think would have caused the event if it actually happened to you. We want you to choose only one cause for the event—the main cause if the event actually happened to you. For each situation, you will write down this cause in the blank at the top of the page. Then we will ask you some questions about what it would mean to you if the situation actually happened to you.

It is important to remember there are no right or wrong answers to the questions. The important thing is to answer the questions how you would think and feel if the situations actually were occurring in your life.

1. You want a boyfriend/ girlfriend but you don't have one.

- a. Write down why you think you don't have a boyfriend/ girlfriend.

- b. Do you not have a boyfriend/ girlfriend because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
-------------------------------------	---	---	---	---	---	---	---	--

- c. Do you think the reason you don't have a boyfriend/ girlfriend will cause you to not have a boyfriend/ girlfriend in the future? (Circle one number).

Will never again cause me not to have a boyfriend/ girlfriend	1	2	3	4	5	6	7	Will also cause me not to have a boyfriend girlfriend in the future
--	---	---	---	---	---	---	---	---

- d. Do you think the reason you don't have a boyfriend/ girlfriend will cause problems in other parts of your life? (Circle one number).

Will only cause problems in my love life	1	2	3	4	5	6	7	Will cause problems in all areas of my life
--	---	---	---	---	---	---	---	---

- e. Do you think other bad things will happen to you because you don't have a boyfriend/ girlfriend? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
----------------------------	---	---	---	---	---	---	---	--------------------------------

- f. Do you think there is something wrong with you because you don't have a boyfriend/ girlfriend? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

2. You get a bad report card for the semester.

- a. Write down why you think you got a bad report card.

-
- b. Did you get a bad report card because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
-------------------------------------	---	---	---	---	---	---	---	--

- c. Do you think the reason you got a bad report card will also cause you to get bad report cards in the future? (Circle one number).

Will never again		Will also cause
------------------	--	-----------------

cause me to get bad report cards	1	2	3	4	5	6	7	me to get bad report cards in the future
----------------------------------	---	---	---	---	---	---	---	--

- d. Do you think the reason you got a bad report card will cause problems in other parts of your life? (Circle one number).

Will only cause problems with my report cards	1	2	3	4	5	6	7	Will cause problems in all areas of my life
---	---	---	---	---	---	---	---	---

- e. Do you think other bad things will happen to you because you got a bad report card? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
-------------------------	---	---	---	---	---	---	---	-----------------------------

- f. Do you think there is something wrong with you because got a bad report card? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

3. Your girlfriend/ boyfriend breaks up with you, but you still want to stay together.

- a. Write down why you think you your boyfriend/ girlfriend broke up with you.

- b. Did they break up with you because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
----------------------------------	---	---	---	---	---	---	---	--------------------------------------

- c. Do you think the reason they broke up with you will also cause others to break up with you again in the future? (Circle one number).

Will never again cause others to break up with me	1	2	3	4	5	6	7	Will also cause others to break up with me
---	---	---	---	---	---	---	---	--

- d. Do you think the reason they broke up with you will cause problems in other parts of your life? (Circle one number).

Will only cause problems in my love life	1	2	3	4	5	6	7	Will cause problems in all areas of my life
--	---	---	---	---	---	---	---	---

- e. Do you think other bad things will happen to you because they broke up with you? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
-------------------------	---	---	---	---	---	---	---	-----------------------------

- f. Do you think there is something wrong with you because they broke up with you? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

4. You get in a big fight with your parents.

- a. Write down why you think you got in a big fight with your parents.

- b. Did you get in the fight with your parents because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
----------------------------------	---	---	---	---	---	---	---	--------------------------------------

- c. Do you think the reason you got in the fight will also cause you to get in fights with your parents in the future? (Circle one number).

Will never again cause me to get in a fight with my parents	1	2	3	4	5	6	7	Will also cause me to get in fights with my parents in future
---	---	---	---	---	---	---	---	---

- d. Do you think the reason you got in the fight with your parents will cause problems in other parts of your life? (Circle one number).

Will only cause problems with my parents	1	2	3	4	5	6	7	Will cause problems in all areas of my life
--	---	---	---	---	---	---	---	---

- e. Do you think other bad things will happen to you because you got in the fight with your parents? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
-------------------------	---	---	---	---	---	---	---	-----------------------------

- f. Do you think there is something wrong with you because you got in the fight with your parents? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

5. You don't get chosen for an extracurricular activity (such as sports team, club, play) that you want to be a part of.

- a. Write down why you think you were not chosen for the extracurricular activity.

- b. Did you not get chosen for the activity because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
----------------------------------	---	---	---	---	---	---	---	--------------------------------------

- c. Do you think the reason you didn't get chosen for the activity will also cause you to not get chosen for activities in the future? (Circle one number).

Will never again cause me to not be chosen for activities	1	2	3	4	5	6	7	Will also cause me to not get chosen for future activities
---	---	---	---	---	---	---	---	--

- d. Do you think the reason you didn't get chosen for the activity will cause problems in other parts of your life? (Circle one number).

Will only cause		Will cause
-----------------	--	------------

problems with my activities	1	2	3	4	5	6	7	problems in all areas of my life
-----------------------------	---	---	---	---	---	---	---	----------------------------------

- e. Do you think other bad things will happen to you because you didn't get chosen for the activity? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
-------------------------	---	---	---	---	---	---	---	-----------------------------

- f. Do you think there is something wrong with you because you didn't get chosen for the activity? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

6. You didn't make the honor roll but you wanted to.

- a. Write down why you didn't make the honor roll.

- b. Did you not make the honor roll because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
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- c. Do you think the reason you didn't make the honor roll will also cause you not to make the honor roll in the future? (Circle one number).

Will never again cause me to miss the honor roll	1	2	3	4	5	6	7	Will also cause me to miss honor roll again in the future
--	---	---	---	---	---	---	---	---

- d. Do you think the reason you didn't make the honor roll will cause problems in other parts of your life? (Circle one number).

Will only cause problems with my academics	1	2	3	4	5	6	7	Will cause problems in all areas of my life
--	---	---	---	---	---	---	---	---

- e. Do you think bad things will happen to you because you didn't make the honor roll? (Circle one number).

Nothing bad will happen	1	2	3	4	5	6	7	Very bad things will happen
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- f. Do you think there is something wrong with you because you didn't make the honor roll? (Circle one number).

Doesn't mean anything is wrong with me	1	2	3	4	5	6	7	Definitely means something is wrong with me
--	---	---	---	---	---	---	---	---

7. Someone says something bad about how you look.

- a. Write down why you think they said something bad about your looks.

- b. Did someone say something bad about your looks because of something about you or because of something else? (Circle on number).

Totally caused by something else	1	2	3	4	5	6	7	Totally caused by something about me
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- c. Do you think the reason someone said something bad about your looks will cause people to say bad things about your looks in the future? (Circle one number).

Will never again cause people to say bad things about my looks	1	2	3	4	5	6	7	Will also cause people to say bad things about my looks in the future
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- d. Do you think the reason someone said something bad about your looks will cause problems in other parts of your life? (Circle one number).

Will only cause problems with what	1	2	3	4	5	6	7	Will cause problems in all
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people say about
my looks

areas of my life

- e. Do you think other bad things will happen to you because someone said something bad about your looks? (Circle one number).

Nothing bad
will happen

1

2

3

4

5

6

7

Very bad things
will happen

- f. Do you think there is something wrong with you because someone said something bad about your looks? (Circle one number).

Doesn't mean
anything is wrong
with me

1

2

3

4

5

6

7

Definitely means
something is
wrong with

