The Impact of Parental Behaviors on the Experience of Stress in Adolescent Offspring of Depressed Parents

by

Elisa Gabrielle Price Geiss

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Psychology)
in the University of Michigan
2016

Doctoral Committee:

Associate Professor Nestor L. Lopez-Duran, Chair
Associate Professor Shawna J. Lee
Professor Christopher S. Monk
Professor Sheryl L. Olson

Dedication

This dissertation is dedicated to my husband, Bradford, and parents, John and Teri, for always supporting me. Thank you for always being there.

Acknowledgements

This research was supported by a Student Award Program grant from the Blue Cross and Blue Shield of Michigan Foundation, Rackham Graduate School One Term Dissertation Fellowship, Rackham Graduate School Student Research Grant, and the University of Michigan Department of Psychology Dissertation Grant.

Thank you, Dr. Nestor Lopez-Duran, for being my supervisor and mentor throughout graduate school. The wisdom and advice you have given throughout the years has helped shaped me not only as a student, but as a person.

This work would not have been possible without the MPAL Lab. Thank you to graduate students who have talked about and read through this dissertation (Dr. Kate Kuhlman, Dr. Ivan Vargas, Steffi Mayer, Ellen Waxler, and Andrea Roberts), or worked as a clinician on the project. The research assistants provided invaluable help by running studies every weekend and coding hours of parent-child interactions.

I appreciate all of the guidance, comments and feedback from my dissertation committee, Dr. Sheryl Olson, Dr. Christopher Monk, and Dr. Shawna Lee. I also want to thank my mentors, Dr. Shelly Schreier, and Dr. Bob Gunn, for supporting my career development.

Thank you to friends and colleagues who diligently worked with me (thanks Dr. Lisa O'Donnell and Katie Foster) and celebrated accomplishments. Finally, I am grateful for the ever present and unwavering support from my family.

Table of Contents

List of Tables

List of Figures

List of Appendices

List of Acronyms

ACTH - Adrenocorticotropic hormone

CDI – Child Depression Inventory

CRH – Corticotropin Releasing Hormone

HPA-axis – Hypothalamic Pituitary Adrenal Axis

MSPSS – Multidimensional Scale of Perceived Social Support

NA – Negative Affect

PA – Positive Affect

TSST – Trier Social Stress Task

VAS – Visual Analog Scale

Abstract

Adolescents who have a parent with a history of depression are at increased risk for depression. The intergenerational transmission of risk may be due, at least in part, to deficits in stress regulation related to ineffective parent-child regulatory processes. While parents play a key role in facilitating children's emotion regulation, it is currently unknown how parents with a history of depression impact their adolescent's stress regulation. The current study aims to characterize the nature of supportive behaviors that parents give to their adolescents, and examine how this may influence the adolescent's emotional and neuroendocrine stress regulation to a laboratory stressor. 63 adolescents aged 12-16 years (41 at high and 22 at low familial risk) participated in a socially evaluated speech task and were randomized into having their parents with them or being alone for 10 minutes after the task. Observed parental supportive and unsupportive behaviors were coded. Subjective and objective measures of stress were obtained through adolescent's report of their emotional distress, and by collecting salivary cortisol to index neuroendocrine stress reactivity. Adolescents reported decreases in happiness, increases in negative affect, and showed increases in cortisol during the stress task. In response to this distress, parents provided supportive behaviors to their adolescent, yet there was less support given by parents with a history of depression. When adolescents had more depressive symptoms, parents without depression provided greater support, whereas parents with a history of depression provided greater unsupportive behaviors. Greater support related to faster up-regulation of positive affect after the stress task, yet the influence on cortisol was dependent

upon risk status. Higher parental support was associated with lower peak cortisol in high risk adolescents, and higher peak cortisol in adolescents at low familial risk. Although this link was surprising, greater support in the context of high cortisol levels may reflect sensitivity of non-affected parents to the child's distress. Overall, while adolescents at high familial risk for depression do not receive as much supportive emotion socialization in response to stressors, higher levels of parental support aids in stress regulation and may be protective against future depression.

Chapter 1: Introduction

Specific Aims

Parental depression confers a two- to four-fold increased risk for developing depression in adolescents (Beardslee, Gladstone, & O'Connor, 2011; Williamson, Birmaher, Axelson, Ryan, & Dahl, 2004). Key pathways to the intergenerational transmission of depression are stress exposure and the family environment (Hammen, 2015). In particular, greater psychosocial stress exposure, increased stress sensitivity, and poor stress regulation have been implicated in the increased risk for depression observed in these children (Gotlib, Joormann, & Foland-Ross, 2014). Parents play a key role in helping to regulate their children's distress (Diamond & Aspinwall, 2003), yet depression influences parenting skills that may impact their ability to serve as regulatory partners (e.g., more negative affect, less positive affect; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Thus parental behaviors may play a critical role in exacerbating or mitigating the effect of stress in youth at high familial risk. This study aims to examine how parents with and without a history of depression influence stress regulation in their adolescent offspring.

How parents react to their child's distress influences the child's emotion regulation skills (A. S. Morris, Silk, Steinberg, Myers, & Robinson, 2007). Yet, most of this research has focused on young children and little is known about how parents impact stress regulation during adolescence (A. S. Morris et al., 2007). There is a greater prevalence of depression during adolescence (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015), which has been linked to poor emotion regulation skills in the face of stress (Yap, Allen, & Sheeber, 2007). Adolescents continue to utilize their parents for support during stressful times (Laursen & Collins, 2009; Smetana, Campione-Barr, & Metzger, 2006), and parents influence emotion

regulation even into adolescence (Yap et al., 2007). Thus, understanding how parents help to externally regulate stress during adolescence may be one potential factor in understanding the prevalence of depression in this age group.

Parents with depression are thought to have difficulty in parenting and to negatively influence the co-regulation process (Goodman & Gotlib, 1999). Indeed, parents with a history of depression have been found to suggest less coping strategies to children (Monti, Rudolph, & Abaied, 2014), and ignore or exhibit more negativity in response to their young children's negative affect (Silk et al., 2011). However, how this translates to adolescence is unknown. Only one study has examined parental support in this context, finding that depressed mothers are perceived by their adolescent as being less supportive (Kutcher et al., 2004), yet the actual behaviors that they display is currently unknown. Thus, the first aim of this study will be to characterize group differences (i.e., in parents with and without a history of depression) in supportive and unsupportive parental behaviors to their adolescent's distress, and determine whether these behaviors are influenced by concurrent parental or child depressive symptoms.

Offspring of parents with a history of depression may have poor emotion regulation skills and cognitive vulnerabilities that confer a greater risk for depression (Hankin & Abramson, 2001). Indeed, children at familial risk for depression have worse emotion regulation capabilities (Gotlib et al., 2014), resulting in globally less positive affect and more negative affect across development (Kovacs & Lopez-Duran, 2010; Kovacs & Yaroslavsky, 2014). In the face of stressors, cognitive vulnerabilities, such as rumination and negative schemas (Gotlib & Joormann, 2010), may influence the appraisal of stressful events in a manner that increases negative affect to stress (Hankin & Abramson, 2001). Thus, adolescents at high familial risk for depression may experience more negative affect in the face of stressors, which may confer a

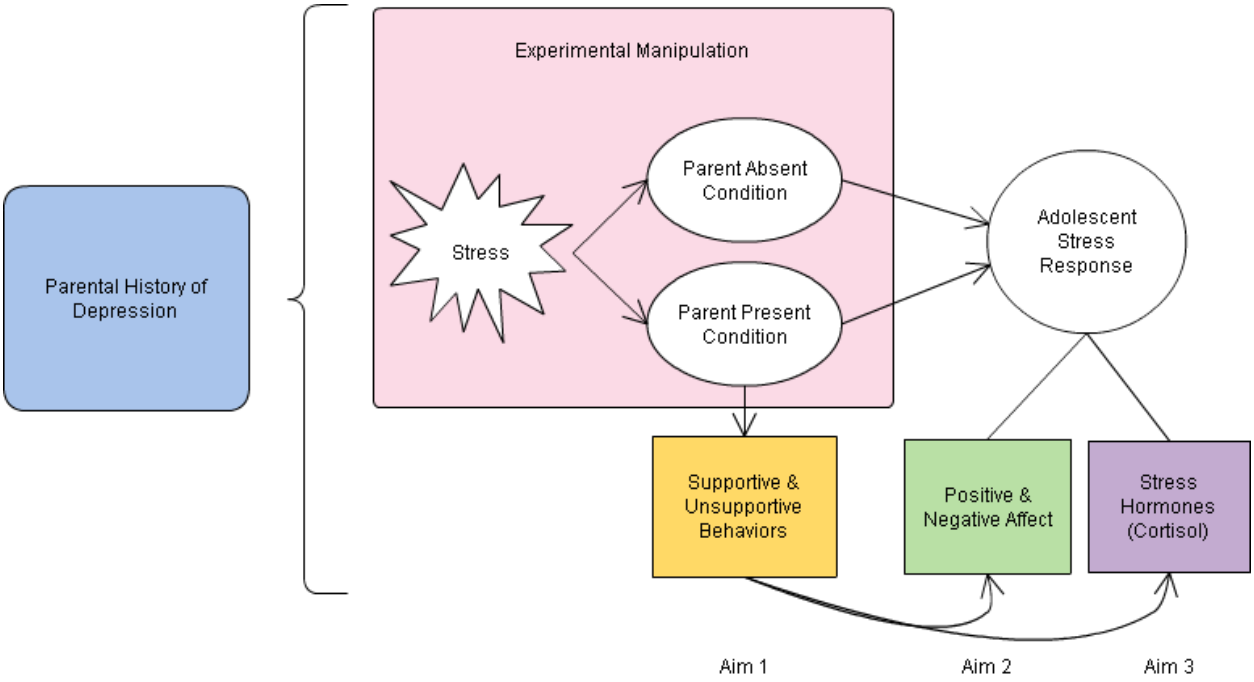
greater risk for a depressive episode (Hankin & Abramson, 2001). Yet, parental support can function to improve emotion regulation by changing the appraisal of the event, or influencing the emotional experience of stress (Power, 2004). For example, when parents give suggestions of cognitive reframing or distraction for a disappointment task, children express less intensity of sadness or anger (A. S. Morris et al., 2011). Alternatively, parents may negatively influence this process as parents with a history of depression reported that they suggest less coping strategies to their children (Monti et al., 2014), and unsupportive responses have been related to emotion dysregulation and depressive symptoms (Sanders, Zeman, Poon, & Miller, 2015). However, it is currently unclear how parents with a history of depression influence their *adolescent's* emotion regulation. The second aim of this study will determine whether the presence of a parent with a history of depression influences the adolescents' subjective mood rating to a stressor and how this may be related to parental support behaviors.

Moreover, parental support has the ability to impact stress regulation at the biological level by modulating the Hypothalamic Pituitary Adrenal Axis (HPA-axis) response to psychosocial stressors (Hostinar & Gunnar, 2013). The HPA-axis is a neuroendocrine stress response system that is critical to the regulation of stress (Tsigos & Chrousos, 2002). Recent studies indicate that parents may reduce the output of cortisol (the end product of the HPA-axis) in youth when they were present either before or after a laboratory stress task, which is commonly called the social buffering effect (Hostinar, Johnson, & Gunnar, 2015; Seltzer, Ziegler, & Pollak, 2010). This social buffering effect may be influenced by multiple factors, such as dyadic coping (Meuwly et al., 2012) and quality of the parent-child relationship (Hostinar, Sullivan, & Gunnar, 2014). Yet, parents with depression and their children engage in greater mutuality of negative affect (Connell, Hughes-Scalise, Klostermann, & Azem, 2011) and co-

rumination (Grimbos, Granic, & Pepler, 2013) during stress tasks. As the social buffering effect has never been extended to families with psychopathology, the third aim of the study will determine if having a parent present after the stress task will influence the HPA-axis response to the stressor in adolescents at high and low familial risk for depression.

Overall, this study will examine the mechanisms of risk for depression among offspring of depressed parents, specifically in the areas of stress experience, stress regulation, and parental support. The objective is to characterize parental behavior to distress; how this influences emotional experience of a lab stressor; and lastly, how parents may buffer stress hormone reactivity. See Figure 1. Given the in-depth examination at multiple levels of analysis, this dissertation will help to understand how parents with a history of depression influence intergenerational transmission of depression in the context of stress.

Figure 1. Overview Diagram of Dissertation Aims



Children at Risk for Depression

Depression is a major public health concern, occurring in approximately 11% of adolescents in the United States (Avenevoli et al., 2015). Depression in youth is associated with social impairment (Weissman et al., 2006), poor academic functioning, lower educational attainment (Dahlen, 2016; Fergusson & Woodward, 2002), and increased risk of committing suicide (Avenevoli et al., 2015). The prevalence of depression spikes during adolescence (Avenevoli et al., 2015), and those affected continue to have strong rates of relapse throughout their lifespan (Birmaher, Williamson, et al., 2004; Kovacs, 1996; Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013). Due to these negative outcomes, it is important to identify factors that may predict depression onset and that can guide the development of more effective preventive interventions.

One of the strongest predictors of depression is having a biological parent with a history of depression (Gotlib et al., 2014). Indeed, offspring with a depressed parent have a 3-fold increased risk for depression compared to the general population (Weissman et al., 2006) and also have increased risk for other forms of psychopathology (Goodman et al., 2011; van Santvoort et al., 2015; Williamson et al., 2004). Those offspring who develop depression have an earlier onset of depression and tend to have more functional impairment than depressed youth without a family history of depression (Weissman et al., 2006). Familial risk even extends past two generations (Weissman et al., 2005), thus the genetic and environmental loading in these families is high. Identifying risk and protective factors with this particularly vulnerable group has been important to understanding the intergenerational transmission of depression and producing early intervention and treatments.

Youth whose parents have a history of depression may be at risk for depression via numerous mechanisms. Many theories follow a diathesis-stress model in which vulnerabilities in the cognitive, social and/or biological domain are activated in response to stressors and contribute to the onset of depression (Hammen, 2009). For example, children may inherit genes that confer risk by having poor biological regulation, or other vulnerabilities, such as negative personality traits (Goodman & Gotlib, 1999) or difficult temperament (Beardslee et al., 2011). Additionally, being exposed in utero to depression increases the likelihood of dysregulation in biological stress response systems, which may contribute to poor emotion regulation skills (Goodman & Gotlib, 1999). Children may also be exposed to and develop maladaptive cognitions or emotion regulation skills by virtue of their parent modeling these behaviors (Goodman & Gotlib, 1999; Goodman, 2007). Then, after experiencing a stressor, these cognitive vulnerabilities such as negative schemas and rumination, may prolong and amplify the activation of negative affect which then may lead to depressive symptoms (Hankin & Abramson, 2001). Furthermore, environmental factors such as a stressful family environment, and increased stress exposure have been tied to intergenerational transmission (Hammen, Hazel, Brennan, & Najman, 2012; Hammen, 2015). In these theories, vulnerabilities operate in tandem and interact with each other to contribute to depression in this group. Overall, there are multiple pathways for the transmission of depression, and no one theory captures all potential risk or protective factors.

This dissertation will examine a subset of the mechanisms of risk, in particular how parental behaviors may impact the experience and regulation of stress in a group of adolescent offspring of parents with a history of depression. Importantly, both stress and parental behaviors have strong links with the transmission of depression and depression onset. The experience and exposure to stress has been identified as an important component of some of the major theories

of the development of depression, such as the cognitive vulnerability hypothesis (e.g., Hankin & Abramson, 2001), and stress generation hypothesis (Hammen, 1991). Furthermore, the interpersonal and environmental impact of the parent with a history of depression has long been implicated as contributing to both a stressful environment and to the development of depression in this group (e.g., Goodman & Gotlib, 1999; Hammen, 1991, 2009; Lovejoy et al., 2000). Importantly, there are theories that posit that social support (Thoits, 2011) and warm responsive parenting (Eisenberg, Cumberland, & Spinrad, 1998) may buffer the effects of stress and have a direct impact on improving mental health. For example, warm parenting during early childhood predicted lower adolescent internalizing symptoms through a pathway of biological stress reactivity (HPA-axis) during middle childhood (Kuhlman, Olson, & Lopez-Duran, 2014). Given that offspring are at risk through both the experience of greater stressors and the interpersonal context of living with a depressed parent, it is important to understand whether parental behaviors may mitigate or exacerbate these risks for depression and how it may influence the experience of stress with children.

Stress in Children at High Familial Risk for Depression

Stress is a particularly salient and important risk factor for offspring of depressed parents. Children at risk for depression are exposed to greater chronic and episodic life stressors compared to those at low familial risk (Adrian & Hammen, 1993; Feurer, Hammen, & Gibb, 2016; Gershon et al., 2011). In particular, high-risk daughters are exposed to more severe chronic interpersonal and non-interpersonal stressors than daughters of non-depressed mothers (Gershon et al., 2011). Children may be exposed to a generally stressful environment by virtue of their parents being depressed (Garber & Cole, 2010; Goodman & Gotlib, 1999; Hammen et al., 2012). For example, parental depression is associated with greater family adversity such as a more

negative mother-child relationship, greater perceived maternal hostility, and greater youth negative life events (Hammen, Brennan, & Shih, 2004). Additionally, children at risk for depression may generate their own stressors within the interpersonal domain (Feurer et al., 2016). Thus, adolescents at high familial risk experience stressful environments to a greater extent than their low risk peers, and may even contribute to more stressors in their lives.

The increased exposure to these types of stressors has been related to greater symptoms of depression in adolescents at familial risk for depression. There is a stronger association between chronic and episodic stress and depressive symptoms in children at high familial risk compared to low risk (Bouma, Ormel, Verhulst, & Oldehinkel, 2008; M. C. Morris, Ciesla, & Garber, 2010). For example, adolescents at risk are more likely to become depressed than peers who had the same level of high conflict in families (Hammen et al., 2004). Thus, this may indicate that there is greater sensitivity to stress, since the negative consequences of stress (e.g., depression) are more likely to occur in this group (Bouma et al., 2008; Gotlib et al., 2014).

Offspring of parents with a history of depression may be at risk for depression in the face of stressors due to cognitive vulnerabilities and emotion regulation skills. Adolescents at high familial risk experienced more negative cognitions, interpersonal stressors and also higher depressive symptoms consistently over a 6 year period (Carter & Garber, 2011). Furthermore, within those offspring that demonstrate maladaptive cognitions such as brooding rumination and negative affect bias, there is increased risk for depression (Gibb, Grassia, Stone, Uhrlass, & McGeary, 2012; Kilford et al., 2015). This is consistent with cognitive vulnerability theory (Hankin & Abramson, 2001), in which these negative cognitive styles increase the risk for depression. Furthermore, offspring who show greater negative cognitive styles, also use more disengagement coping strategies and less active coping (Dunbar et al., 2013), which in turn is

associated with increased risk of depression within this group (Dunbar et al., 2013; Jaser et al., 2005). Thus, while not all offspring at familial risk demonstrate cognitive styles associated with depression, there is evidence that the presence of these cognitions within these children increases the risk for depression in the face of stress.

Another potential reason for the increased sensitivity to stress may be dysfunctional biological stress responses systems (Foland-Ross, Hardin, & Gotlib, 2012; Goodman & Gotlib, 1999; A. Miller, 2007). For example, there is growing evidence for dysregulation in systems such as the Hypothalamic Pituitary Adrenal –Axis (HPA-Axis; Guerry & Hastings, 2011) and for functional and structural anomalies in the brain implicated in emotion regulation (Gotlib et al., 2014) in youth at higher familial risk. This may contribute to increased stress reactivity, or less ability to effectively regulate emotions, which leaves children more vulnerable to negative effects of stress (Gotlib et al., 2014). For example, it is hypothesized that dysregulation in the HPA-axis is associated with poor mood repair due to its effects on reducing the stress threshold, increasing reactivity to the stressor, and facilitating a negative mood bias (Kovacs & Lopez-Duran, 2010). Thus, neurobiological mechanisms may leave these children with greater sensitivity to stress, which in turn confers greater vulnerability to having depression in the face of stressors.

Parenting Behaviors in Parents with a History of Depression

The experience of depression may directly undermine parenting skills and emotional availability of parents to respond appropriately to their children (Dix & Meunier, 2009; Restifo & Bögels, 2009). Indeed, both mothers and fathers who have depression have less positive and more negative and disengaged behaviors toward their children (Lovejoy et al., 2000; Wilson & Durbin, 2010). Within children at risk, parenting style and behavior mediates the transmission of

depression from the parent to child (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007; Goodman & Gotlib, 1999; L. Miller, Warner, Wickramaratne, & Weissman, 1999). For example, the link between maternal depression and internalizing symptoms was exacerbated by the presence of maternal rejection (Zalewski, Thompson, & Lengua, 2015), and parenting that was intrusive and neglectful fully mediated the association between parental depression and childhood depressive symptoms in low income families (Reising et al., 2013). Together, negative parenting may be a mediator of the relationship between depression in parents and children.

However, there is variability in parenting skills in those with a history of depression. In a meta-analysis, while parents with depression on average showed more negative and less positive behaviors during parent-child interactions, not all studies found a difference between parents with and without a history of depression (Lovejoy et al., 2000). Additionally, positive parenting behaviors (e.g., warmth) plays a protective effect against future depressive symptoms in adolescents at familial risk for depression (Birmaher, Bridge, et al., 2004; Brennan, Brocque, & Hammen, 2003). Some of the differences may be attributable to mood state, as parenting behaviors fluctuate around concurrent depressive symptoms (Ewell Foster, Garber, & Durlak, 2008; Lovejoy et al., 2000). However, other studies found that parenting deficits continue to occur even when taking into account concurrent maternal depression (Feng, Shaw, Skuban, & Lane, 2007; Kluczniok et al., 2016). Overall, depression may negatively impact parenting skills, yet there is also evidence of specific parenting skills that may decrease the offspring's vulnerability to depression. It is therefore important to examine under what context parents are able to utilize these positive parenting behaviors and how this may influence their children.

It is important to note that not only does parenting influence children, but children's behavior also impacts parenting, as there are reciprocal influences of children and parents

(Sameroff & Fiese, 2000). In fact, children's behavior may influence the onset of parental depressive symptoms (Elgar, McGrath, Waschbusch, Stewart, & Curtis, 2004). Also, symptoms experienced by children, such as externalizing behaviors, influence parenting behaviors such as maternal criticism two years later even after accounting for maternal depressive symptoms (Frye & Garber, 2005). In dynamic observational studies, parents and children respond to and mirror each other's affect (Butler & Randall, 2013; Lunkenheimer, Olson, Hollenstein, Sameroff, & Winter, 2011), which suggests there are short term reciprocal interactions that occur as well. Thus, it is important to understand the reciprocal nature of the interactions between parent and child within this process.

Diathesis Stress Model: Adolescent Stress and Parenting by Parents with a History of Depression

In the particular model adopted for this dissertation, stressors and parenting do not independently contribute to depression in youth, but interact as risk factors. In particular, a meta-analysis concluded family variables, such as the parent-child relationship, mediate the association between stressors and psychopathology in children (Grant et al., 2006). Additionally, maternal depression leads to offspring depression by virtue of stress exposure, and maternal behaviors (Hammen et al., 2012). Furthermore, in the face of stress, parental socialization of emotions may be important to decrease the likelihood of depression in their children (Abaied & Rudolph, 2010). Thus, there is evidence to suggest that it is the combination of the experience of stress and negative parenting behaviors that is particularly detrimental to the development of depression in this group, and that in the face of stressors positive parenting may decrease the likelihood of negative outcomes.

Overall, parenting may exacerbate or decrease the effects of stressors through what will be broadly defined as stress regulation for the purposes of this dissertation. Although there are multiple terms for the effect that parents have on children when they are distressed (e.g., coping, social support, emotion regulation, dyadic coping, emotion socialization), each describe parents as having an important effect on decreasing the intensity of a stressor (Eisenberg et al., 1998; Meuwly et al., 2012; Power, 2004; Silk et al., 2011). This may be through changing the environment so that the child does not have to experience stressors (Power, 2004), changing how children appraise the stressor (Marroquín, 2011), or in helping to decrease their distress through methods such as comfort and touch (Diamond & Aspinwall, 2003). While this role for parents figures very importantly during the early years of life (for example, infants are reliant on parents to regulate their distress), it also plays a role throughout the lifespan. For example, adolescents continue to use parents as social support (Helsen, Vollebergh, & Meeus, 2000), a lack of social support from family, but not friends predicted increases in depressive symptoms (Stice, Ragan, & Randall, 2004), and supportive others continue to benefit stress regulation into adulthood (Thoits, 2011). Conversely, social relationship can be a source of stress. For example, social negativity and expressed emotion can lead to a recurrence of depression in those who have previously had depression (Ibarra-Rovillard & Kuiper, 2011). Yet, there has been a lack of research regarding what parents do when they encounter distress in their adolescent child, especially within high-risk families.

This dissertation aims to understand the context of how parents with a history of depression are able to respond to their adolescent's distress, and the effect that this has on children's experience of stress. In particular, the first aim is to characterize parenting behaviors after the adolescent has experienced a stressor. The second aim will examine whether and how

parental presence is related to affect regulation to the stressor. The third aim will examine whether parents influence biological stress regulation to the stressor. Thus, adolescents will undergo a laboratory psychosocial stressor (Trier Social Stress Task), and be randomized into whether they will spend the next 10 minutes after the stressor with a parent or not. Interactions will be video recorded and coded for parenting behaviors. Additionally, adolescents will fill out questionnaires about basic emotional experiences throughout the task, and their appraisal of the stressor. Lastly, they will be asked to obtain saliva samples to record levels of cortisol (the end product of the Hypothalamic Pituitary Adrenal Axis; HPA-Axis) in order to obtain information about whether presence of a parent will aid in biological regulation of stress.

Developmental and Gender Influences on Depression

Any study regarding depression in youth must include the influences of age and gender. The point prevalence of depression is relatively low during early and middle childhood (approximately 2%), and rises during adolescence (approximately 5-8% of youth) (Rohde et al., 2013). In early development, rates of depression are about equal for both boys and girls; however, during adolescence, girls have twice the rate of depression than boys (Cicchetti & Toth, 1998; Hankin et al., 1998; Sheeber, Davis, & Hops, 2002), with gender differences being maintained throughout adulthood (Nolen-Hoeksema & Girgus, 1994). Gender differences may be due to girls having more risk factors and experiencing more challenges associated with depression during adolescence than boys (Nolen-Hoeksema & Girgus, 1994). For example, girls experience greater interpersonal difficulties and have greater stress sensitivity (Hankin & Abramson, 2001; Hankin, Mermelstein, & Roesch, 2007), and differ in emotion reactivity, pubertal development, and cognitive style (Hyde, Mezulis, & Abramson, 2008).

Within children at risk for depression, the highest incidence of depression in this group is between 15-20 years of age (Weissman et al., 2006). Not many studies have examined gender differences in prevalence of depression within offspring of depressed parents (Beardslee et al., 2011), yet a meta-analysis suggests that girls are more likely than boys to develop internalizing problems in this group (Goodman et al., 2011). Furthermore, in a 20 year longitudinal study of offspring, girls were found to have a higher rate of depression than boys during adolescence (Weissman et al., 2006). It has been suggested that girls may be more likely to show internalizing symptoms, whereas boys are more likely to show externalizing symptoms in the context of maternal depression (Foster et al., 2008; Sheeber et al., 2002). However, not all studies have found gender differences in first episode of depression in youth (Williamson et al., 2004). Together, there is some evidence to suggest similar developmental and gender effects in children at both low and high risk for depression.

Gender of the parent may also influence intergenerational transmission of depression, yet most studies have examined mothers only. While little is known about fathers, there is evidence for decreased positive and increased negative parenting behaviors (Wilson & Durbin, 2010) similar to what is seen for mothers (Lovejoy et al., 2000), and for paternal depression to be related to future psychopathology in their children even when controlling for maternal depression (Jacobs, Talati, Wickramaratne, & Warner, 2015; Ramchandani, Stein, Evans, & O'Connor, 2005). Furthermore, there may be an interaction with child gender, as paternal depression was related to increases in depression in sons, but not daughters; yet, there were no gender differences for maternal depression (Eberhart, Shih, Hammen, & Brennan, 2006). Results were equivocal as maternal depression predicted increased rates of their daughter's depressive symptoms, whereas paternal depression was related to increased depression in both sons and

daughters (Mason, Chmelka, Trudeau, & Spoth, 2016). Thus, within this dissertation, all analyses will include gender of the child as a covariate, yet, unfortunately, the sample size was not large enough to include the gender of the parent as a covariate.

Aim 1: Observed Parental Behaviors to Adolescent's Distress after a Stress Task

In the previous section, it was argued that parenting behaviors of depressed parents are more negative and less positive than their non-depressed peers (Lovejoy et al., 2000), however, there is known variability in parenting skills (Zalewski et al., 2015). One goal is to clarify under what circumstances parenting skills are more intact, and how this may affect children. Most observational studies that characterize these behaviors are based in parent-adolescent conflict or a positive interaction task (e.g., Dietz et al., 2008; McMakin et al., 2011). However, solely studying this type of context is limiting since this is only one type of stressor that adolescents experience. In particular, transitioning to adolescence includes becoming more independent and spending more time outside of the home and with friends (Steinberg & Morris, 2001), and thus children may experience more stressors outside of the home. As adolescents continue to utilize parents as a secure base during times of stress (Barbot, Heinz, & Luthar, 2014; Rueger, Malecki, & Demaray, 2010), it is fruitful to investigate how parents with a history of depression support their child after the occurrence of an outside stressor.

Multiple experimental studies have examined how parents and children interact in positive and conflict related tasks. Generally these tasks require adolescents and parents to discuss a recent topic of conflict and problem solve toward a resolution. It is either preceded or followed by a positive engagement task, such as planning a family vacation (e.g., Dietz et al., 2008; Sheeber, Allen, Davis, & Sorensen, 2000; Yap, Allen, & Ladouceur, 2008). Parents with a history of depression generally demonstrate more disengagement and lower positive affect, and their children show lower positive affect in interactions across positive and negative tasks (Dietz et al., 2008; Jacob & Johnson, 1997; McMakin et al., 2011). There was less flexibility in emotional expression across positive and negative tasks in both 3 ½ year old children and their

mothers with higher depressive symptoms (Lunkenheimer, Albrecht, & Kemp, 2013). Additionally, the dyad have been shown to engage in greater co-rumination and less problem solving compared to non-depressed dyads (Grimbos et al., 2013). Thus, depressed mothers may model ineffective coping strategies (Grimbos et al., 2013) and also provide less scaffolding during problem solving (Hoffman, Crnic, & Baker, 2006). The consequence of these behaviors during interactions may be higher childhood depressive symptoms, since lower levels of positive maternal behaviors and higher levels of negative behaviors were related to greatest increases in depressive symptomatology in young adolescents over time (Olino et al., 2016). Thus, these tasks indicate that parents with a history of depression and their children are more negative and less positive, that parents provide less affect regulation, and furthermore that this may be associated with future depression, however, all of these studies involve direct conflict.

One difficulty in examining only conflict tasks is that it involves both parent and child in a state of distress. As the task is to engage parents and children in a discussion about a topic that has recently caused a problem, both may be more likely to have negative affect. Indeed, adolescents have greater negative affect in conflict with parents compared to peers (Laursen & Collins, 1994), and the intensity of conflict peaks during mid-adolescence (Laursen, Coy, & Collins, 1998). Adolescents also are more likely to submit and disengage during conflict with their parents (Laursen & Collins, 1994), which may leave the conflict unresolved, and make adolescents more upset. Additionally, parents who experience higher levels of distress to their children's negative affect gave more harsh parental coping strategies to their children (Fabes, Leonard, Kupanoff, & Martin, 2001). Thus, while there are benefits to using conflict tasks to understand parent-child interactions, it does not include the full range of situations where parents may influence their children, and may actually heighten the degree to which parents and children

show negative behaviors. This may not allow for an accurate representation of parenting behaviors in at-risk groups, and other contexts should be examined to decrease this gap in the literature.

Responsiveness to distress, also termed emotion contingent reactions (Howard Sharp, Cohen, Kitzmann, & Parra, 2016) and parental socialization of emotions (Eisenberg et al., 1998), characterizes parent's behavior when a child is upset or distressed (Davidov & Grusec, 2006). On one end of the spectrum are negative, insensitive behaviors such as hostility, dismissing the child's distress, and being overwhelmed by their [parent's] own distress. On the other end are positive, sensitive reactions such as caring and helping (Davidov & Grusec, 2006). These behaviors are hypothesized to play a role in the socialization of emotions, and the effectiveness of coping strategies (Eisenberg et al., 1998). Responding to emotions conveys explicit and implicit information about how to regulate these emotional experiences, such as how emotions should be experienced, how to problem solve, or strategies to manage distress (Silk et al., 2011). For example, parental sensitivity is associated with better affect regulation and better physiological regulation to stress (Davidov & Grusec, 2006; Willemen, Schuengel, & Koot, 2009), whereas perceived unsupportive responses are related to greater emotion dysregulation, poorer emotion coping, and depressive symptoms (Sanders et al., 2015). Thus, parental behaviors to distress influence affect regulation in their children.

The emotion socialization literature has focused on two categories of parental responses to children's distress. *Supportive* responses (also known as *reward* and *emotion coaching*) refer to positive behaviors that encourage the expression of emotions, such as validating feelings, problem solving about emotions, and using comforting behaviors (Malatesta-Magai, 1991; Silk et al., 2011). This may improve emotion regulation skills, and indeed a lack of this type of

response to sadness has been related to greater adolescent internalizing and externalizing behaviors (Klimes-Dougan et al., 2007). Additionally, there are multiple *unsupportive* socialization practices which teach children that emotions are "bad" and should be suppressed (Sanders et al., 2015). For example, parents may dismiss children's emotional experiences through minimizing or distracting from emotions, such as telling the child that "things are not that bad" (Lunkenheimer, Shields, & Cortina, 2007; Silk et al., 2011), punish, or show disapproval of the emotional experience (Silk et al., 2011), magnify the affect, (Klimes-Dougan et al., 2007) or neglect the child's emotional experience (Silk et al., 2011). These negative support practices have been related to poorer outcomes such as internalizing symptoms (Silk et al., 2011), problem behaviors (Lunkenheimer et al., 2007), greater emotion dysregulation (Sanders et al., 2015), and greater psychological distress in young adults (Garside & Klimes-Dougan, 2002). Thus, how parents respond to their children during times of distress influences both immediate emotion regulation outcomes as well as mental health outcomes.

Parental depression may influence the ability of a parent to provide supportive emotion socialization. Parental depressive symptoms when children were in kindergarten was related to more negative responsiveness to distress in first grade, and child's conduct problems during adolescence (Cummings, George, Koss, & Davies, 2013). Additionally, parents with childhood onset depression reported that they were less likely to respond to their 3- 9 year old child's distress with supportive behaviors, and more likely to respond with a magnifying, punitive, or neglectful response (Silk et al., 2011). In turn negative responses to emotions were related to internalizing symptoms both concurrently and one year in the future (Silk et al., 2011). Additionally, in young children 1-8 years of age, mothers with childhood onset depression demonstrated less responsivity to their child's negative emotions, but showed similar levels of

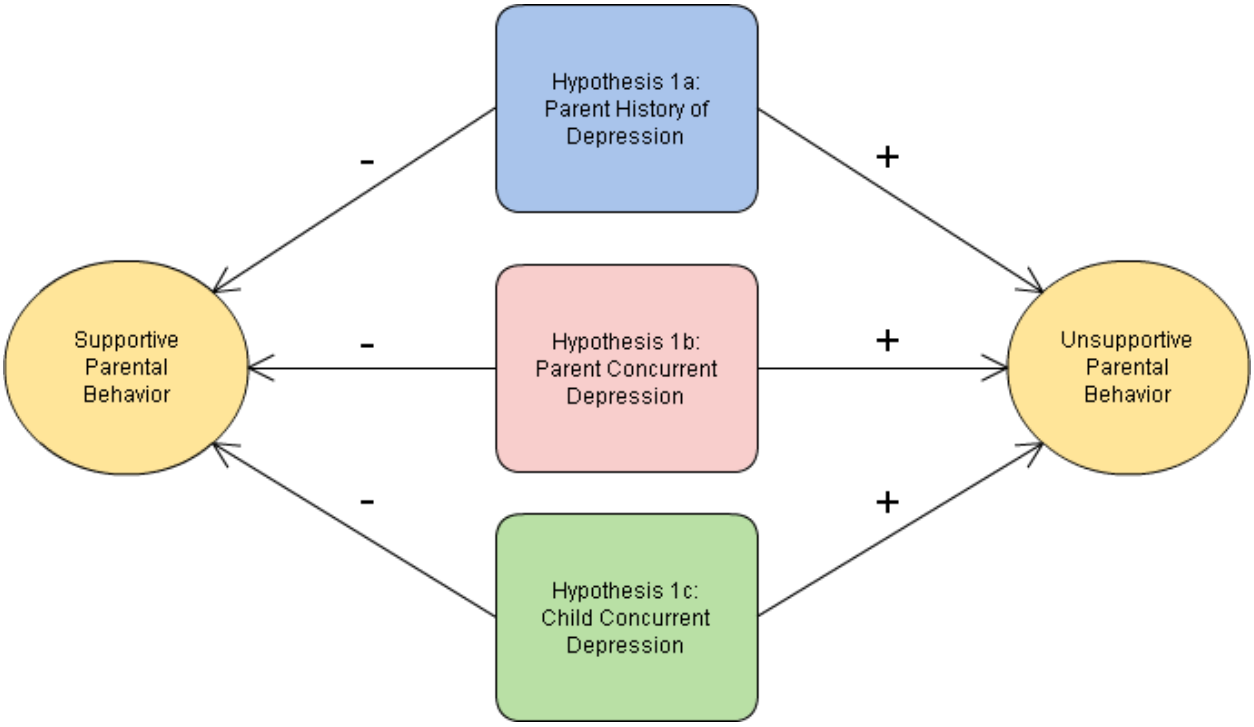
responding to positive behaviors (Shaw et al., 2006). This also extends to strategies that parents give to their children to regulate emotions. Mothers with depressed mood reported that they would give their 2nd grade child less cognitive restructuring and more cognitive avoidance suggestions to peer hostility (Monti et al., 2014). While this suggests that parents with depression are poorer at suggesting coping strategies, all of these observational studies were focused on younger children and little is known about how this extends to adolescence.

In general, few studies have examined emotion socialization during adolescence (Bariola, Gullone, & Hughes, 2011; Miller-Slough & Dunsmore, 2016; A. S. Morris et al., 2007), yet, parents may play a critical role in helping their adolescent navigate this developmental transition of greater stressors (Hankin, 2006). However, adolescents reported that their mothers with a history of depression are less supportive in helping them during stress (Kutcher et al., 2004). Depressive symptoms in both the parent and adolescent were related to lower perceived parental support over the course of 18 months (Kim, Thompson, Walsh, & Schepp, 2015). Yet, it is unclear if these finding reflects differences in the perception of support by the adolescence or actual parental behavior. Furthermore, as noted previously, parental depressive symptoms may moderate their effectiveness in providing support to their children, since parenting skills vary based on level of symptoms (Dietz et al., 2008; Ewell Foster et al., 2008; Lovejoy et al., 2000). Additionally, the child's symptoms of psychopathology also influences the parent's responses to their children (Birmaher, Bridge, et al., 2004; Nelemans, Hale, Branje, Hawk, & Meeus, 2014). Thus, examining whether parental and child depressive symptoms moderate the association between group status and parenting behaviors in the context of distress will be important to understanding the circumstances by which parents may be effective or not in providing aid to their child.

Thus, **Aim 1** is to determine the frequency that parents with and without a history of depression provided supportive and unsupportive behaviors to their adolescent's distress in the context of an outside stressor and determine whether parent and child depressive symptoms influences these parenting behaviors. Adolescents underwent a laboratory stress task, and were randomized to be with their parent or watch a neutral movie for 10 minutes after the stressor. Interactions were video recorded and behaviors were coded for parental responses to children's emotions which include coaching, dismissing, punishing, and magnifying. Parents and adolescents both reported on their current depressive symptoms. Specifically, the hypotheses are:

Hypothesis 1a. Parents with a history of depression will demonstrate less supportive responses and greater unsupportive responses compared to low risk parents. ***Hypothesis 1b.*** Parents experiencing greater current depressive symptoms will demonstrate less supportive responses and greater unsupportive responses compared to those with lower depressive symptoms. ***Hypothesis 1c.*** Children reporting greater depressive symptoms will have parents who respond to their distress with more unsupportive responses, and less supportive responses. See Figure 2 for a diagram depicting each hypothesis.

Figure 2. Diagram of Aim 1 Hypotheses.



Aim 2: Impact of Parent on Adolescent Affect Regulation

The cognitive vulnerability hypothesis (Beck, 2008) states that some people are more vulnerable to developing depression due to the interpretation of stressful life events and subsequent prolonged negative affect after stressors (Carter & Garber, 2011; Lakdawalla, Hankin, & Mermelstein, 2007). Children at risk for depression are more likely to have a negative cognitive styles (Jaenicke et al., 1987), and atypical affectivity (Kovacs & Yaroslavsky, 2014), which may predict sustained negative affect in the face of stressors, and in turn greater depressive symptoms (Espejo, Hammen, & Brennan, 2012). Parents and other support partners play a role in decreasing stress levels through external reappraisals and co-regulation of emotions (Diamond & Aspinwall, 2003), thus, parents play an important role in stress regulation especially for those with stress dysregulation. Unfortunately, how parents affect emotion regulation after stress has never been examined in adolescents at high familial risk for depression, and thus it is unclear whether parents with a history of depression are able to facilitate or hinder affect regulation in children and whether this depends on specific parenting behaviors.

Children at high familial risk for depression have been found to have atypical affectivity characterized by low positive affect and less effective emotion regulation strategies (Kovacs & Lopez-Duran, 2010). High-risk children experience more attenuated positive affect across development (Kovacs & Yaroslavsky, 2014; Olino et al., 2011), which has been theorized to lead to depression. Likewise, in the face of stress, offspring of depressed parents are more likely to use ineffective and passive mood repair strategies (Kovacs & Yaroslavsky, 2014; Silk, Shaw, Skuban, Oland, & Kovacs, 2006), which may be related to prolonged and sustained negative affect (McMakin et al., 2011). Adolescent girls at high familial risk furthermore have an attentional bias for negative mood stimuli (Joormann, Talbot, & Gotlib, 2007), and interpret

ambiguous stimuli more negatively (Dearing & Gotlib, 2009). Thus, children at high familial risk may experience longer sustained negative and less positive emotional experiences when experiencing stress.

While children at familial risk for depression may have impaired mood regulation which may lead to sustained negative affect in the face of stressors, parents influence affect regulation by providing external coping support. Parents use a variety of coping suggestions, such as cognitive reappraisals (encouraging children to think different about themselves or the situation), changing the stressor (problem solving), or disengaging from the stressor or their thoughts (Abaied & Rudolph, 2010; Marroquín, 2011; Monti et al., 2014; Power, 2004; Uchino, Bowen, Carlisle, & Birmingham, 2012). Parental encouragement for their children to cope with or solve the problem that caused the distress is associated with more positive coping strategies (Eisenberg, Fabes, & Murphy, 1996), and cognitive reframe or distraction from a disappointment task is associated with lower levels of anger and sadness (A. S. Morris et al., 2011). Thus, parental support may play a role in modulating negative affect in response to stress.

However, how parents with a history of depression help adolescents to cope with the emotional experience of a stressor has never been investigated using observational methods. It is possible that parents with a history of depression do not engage in the type of supportive behaviors necessary to facilitate their offspring affect regulation in response to stress, since they themselves do not use them, or depression may make them more withdrawn from interactions from children and thus less likely to encourage positive suggestions (Monti et al., 2014). It has also been suggested that parents model depressogenic cognitive, affective, and behavioral styles which may transmit depression to their children (Goodman & Gotlib, 1999; Goodman, 2007). There is some evidence to suggest that parents with depression respond to their child's distress

with negative coping skills. For example, depressed parents are more likely to suggest cognitive avoidance strategies and less active coping, like cognitive restructuring, to their second grade children (Monti et al., 2014). Parents with depression may also engage in more co-rumination with their adolescent children (Grimbos et al., 2013), which may prolong the negative affect. While not examining high-risk children, those adolescents with higher stress levels and parents who provide more disengagement coping strategies had the highest level of depressive symptoms (Abaied & Rudolph, 2010). Thus, there is evidence to suggest that there may be maladaptive responses from parents with depression to their children's distress, which may influence the length of negative affect or depressive symptoms.

However, some parents with a history of depression may engage in supportive behaviors and may as effective as their non-affected peers in helping their children regulate stress. This may partially explain why some children of depressed parents do not develop depression and are considered "resilient". Indeed, positive parenting style in depressed parents has been associated with more resilient adolescents (Brennan et al., 2003). Alternatively, such resiliency may be due to characteristics of the child. For example, low levels of negative appraisals of the event may make the lack of supportive parenting irrelevant given that they may not need external regulatory support. Therefore, clarifying what aspects of parenting and child characteristics may be responsible for variability in affect responses to stress can point to specific areas of risk and resilience that may be target of intervention.

Therefore, **Aim 2** is to determine whether parents with a history of depression affect adolescent's emotion regulation to a stressor and how this is impacted by parental behaviors. Adolescents will report their emotional experience prior to, during, and after the Trier Social

Stress Task for Children (TSST-C). Additionally, in half the sample, parents will be present with their children for 10 minutes after the stress task. The hypotheses are:

Hypothesis 2a. Adolescents at high familial risk for depression will have lower levels of positive affect and higher levels of negative affect than low risk adolescents after a stress task. See Figure 3. ***Hypothesis 2b.*** Presence of a parent will be related to lower negative affect ratings after the stress task in adolescents at low familial risk, but not in those with high familial risk. See Figure 4. ***Hypothesis 2c.*** Parental behavior will moderate the association between group status and affect regulation, so that adolescents of parents with and without a history of depression will not differ among parents displaying high levels of supportive behaviors. See Figure 5.

Figure 3. Diagram of Aim 2 Hypothesis A.

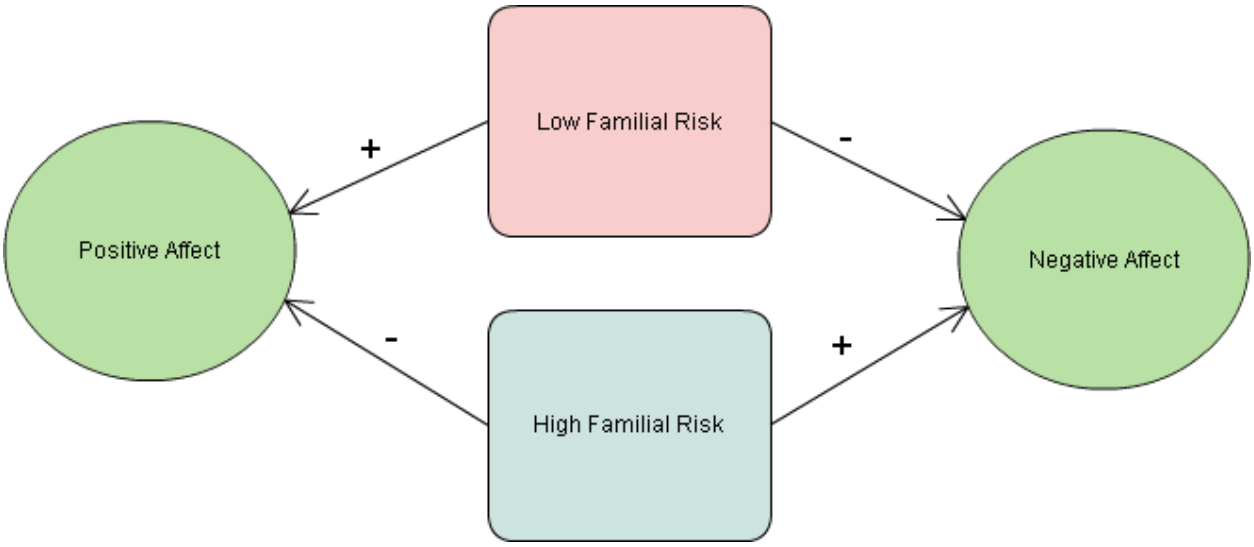


Figure 4. Diagram of Aim 2 Hypothesis B.

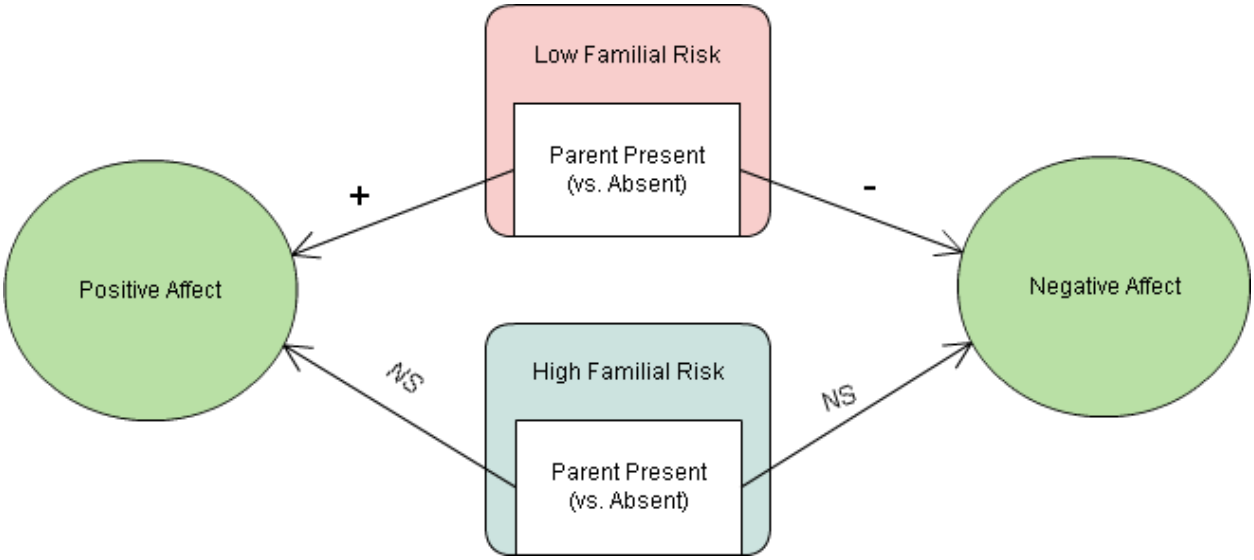
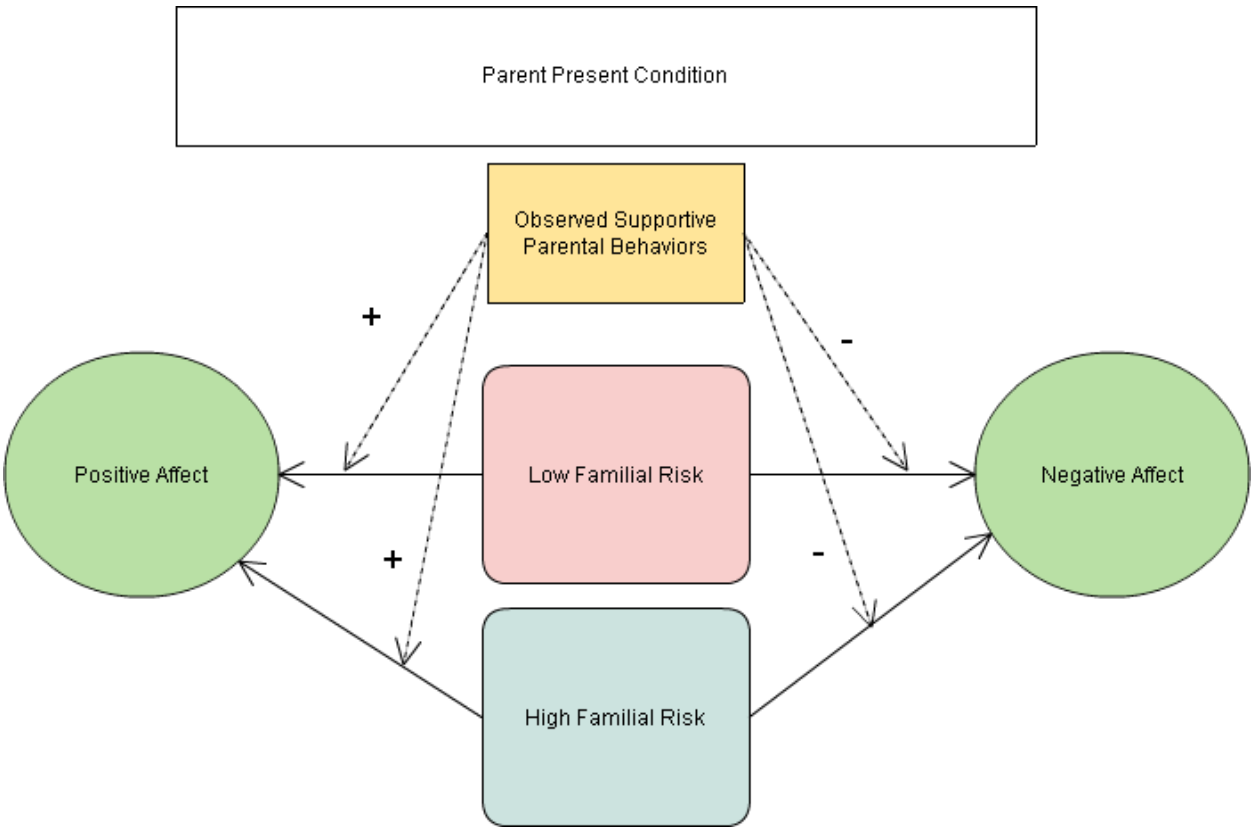


Figure 5. Diagram of Aim 2 Hypothesis C.



Aim 3: Impact of Parent on Adolescent's HPA-Axis Reactivity and Recovery

The Hypothalamic Pituitary Adrenal Axis (HPA-axis) is a neuroendocrine system that is activated in response to stress and is involved with stress regulation. Dysregulation of this system has been implicated in the onset and maintenance of youth depression (Lopez-Duran, Kovacs, & George, 2009) and has also been found in youth at high familial risk (Guerry & Hastings, 2011). Given the strong link to depression, understanding the HPA-axis functioning in those at risk for depression may give a clearer sense of how stress regulation may contribute to depression. Recently, studies have established that social relationships may influence the reactivity and recovery of the HPA-axis to stress (Hostinar & Gunnar, 2013). This stress buffering effect has furthermore been extended to children (Hostinar et al., 2015; Seltzer et al., 2010), however this has not been investigated within a high familial risk group. Given potential dysregulation in the HPA-axis within at-risk adolescents, and the potentially limited ability by depressed parents to help offspring's stress regulation, it is possible that social buffering of neuroendocrine functioning is not present in adolescents at familial risk for depression. Thus, we examined how parents with and without a history of depression differ in their ability to influence neuroendocrine stress regulation in their offspring.

The Hypothalamic Pituitary Adrenal-axis (HPA-axis) is a neuroendocrine system that is activated in response to stress, and modulates physiological changes to help manage that stressor. When a stressor is present, the hypothalamus sends corticotropin-releasing hormone (CRH) to the anterior pituitary gland, which in turn sends adrenocorticotropic hormone (ACTH) to the adrenal gland where cortisol is then secreted (Tsigos & Chrousos, 2002). Cortisol has broad ranging impact on multiple biological systems, for example by changing the metabolic processes to mobilize energy, such as increasing blood glucose levels (Dickerson & Kemeny, 2004; D. B.

Miller & O'Callaghan, 2002) and enhancing cardiovascular output (Fries, Dettenborn, & Kirschbaum, 2009). Furthermore, cortisol acts as its own regulatory agent and decreases activation of the hypothalamus when there is more cortisol, in turn shutting off the stress response through a negative-feedback loop (D. B. Miller & O'Callaghan, 2002). In a healthy organism the HPA-axis activation process is time limited, and acutely activating and then shutting off the system is important to maintaining homeostasis (Tsigos & Chrousos, 2002). Yet, those who suffer from mental health difficulties, may either have an over active stress response system that maintains higher levels of cortisol, termed *hyper-reactivity* (Burke, Davis, Otte, & Mohr, 2005), or alternatively have less reactivity of the system, termed *hypo-reactivity* (Heim, Ehlert, & Hellhammer, 2000). Both over and under activation of the stress response system may have negative consequences, such as immune suppression, and maintaining negative mental health problems (Dickerson & Kemeny, 2004; McEwen, 1998a).

The HPA-axis is initiated in response to stress, and has reliably been activated in laboratory studies (Dickerson & Kemeny, 2004). Tasks that are uncontrollable and socially evaluative are most effective in activating a stress response (Dickerson & Kemeny, 2004). One particular well studied task is the Trier Social Stress Task (TSST), which has demonstrated effectiveness in provoking an HPA-axis response in both children and adults (Gunnar, Talge, & Herrera, 2009; Kudielka, Hellhammer, & Kirschbaum, 2007). In this task, participants give a speech and do mental arithmetic in front of peer judges, which taps into social evaluation and uncontrollability (Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009). Typical responses from participants is to have an increase in cortisol with peaks about 21-40 minutes after the stressor (Dickerson & Kemeny, 2004), and a recovery of the system with decreasing levels to baseline. Given that the modulation of a stress response is important to maintain homeostasis (Tsigos &

Chrousos, 2002), and that prolonged or blunted activation may lead to long term health problems (McEwen, 1998a), it is important to understand how high-risk adolescents react to and recover from these types of stressors.

The HPA-axis has been implicated in the development and maintenance of depression (Guerry & Hastings, 2011; Lopez-Duran et al., 2009). Elevated levels of cortisol (hypercortisolemia) has been hypothesized to be a marker for depression in adults (P. J. Cowen, 2010). In terms of reactivity, a meta-analysis found that depressed adults have higher baseline levels of cortisol, more blunted reactivity than controls, and higher levels of cortisol during recovery, suggesting that the system does not shut down as quickly once activated (Burke et al., 2005). Not as many studies have examined the HPA-axis in depressed children and adolescents, although results are somewhat consistent showing elevated basal cortisol levels (Lopez-Duran et al., 2009), and hypersecretion in response to acute laboratory stressors (Guerry & Hastings, 2011), which may be due to longer duration of activation of the system (Lopez-Duran et al., 2015). There are developmental shifts present, where *hypo*reactivity in children prior to puberty was found to be associated with later depressive symptoms, yet *hyper*reactivity was correlated later in pubertal development (Colich, Kircanski, Foland-Ross, & Gotlib, 2015). Thus, "typical" associations between hypercortisolemia may be present once youth achieve later pubertal development. Less is known about acute stress reactivity in these high-risk children, although this may be an important indicator of how the stress response system is functioning.

Biologically, children at familial risk for depression may inherit similar neuroendocrine functioning as their parents, and in general have been found to be more reactive to stressors (Gotlib et al., 2014; Gotlib, Joormann, Minor, & Hallmayer, 2008; Guerry & Hastings, 2011). There has been recent evidence that depressed mothers and daughters have concordant

hyperactive diurnal cortisol levels (LeMoult, Chen, Foland-Ross, Burley, & Gotlib, 2015). Thus, there may be transmission of atypical patterns of HPA-axis functioning between parents and children. In terms of acute stress reactivity, in a sample of 3rd, 6th, and 9th grade students, the average cortisol levels were heightened when children's mothers had higher concurrent levels of depressive symptoms (Badanes, Watamura, & Hankin, 2011). In a similar sample, only high-risk children who had a negative cognitive style had greater overall cortisol (AUCg) to a stress task (Hayden et al., 2014). In 9-14 year old girls, those high risk children with lower positive affect to the task had heightened and sustained cortisol reactivity (Waugh, Muhtadie, Thompson, Joormann, & Gotlib, 2012). Additionally, maternal postnatal depression was related to enhanced cortisol reactivity and a stronger decline during recovery to a stress task at age 22 (Barry et al., 2014). Results have been equivocal however, as another study found blunted response in 16 year old high-risk girls (Bouma, Riese, Ormel, Verhulst, & Oldehinkel, 2011), and no differences in cortisol reactivity between high-risk and low risk girls 10-14 years of age (Gotlib et al., 2015). Overall, there seems to be greater reactivity to stressors for high-risk youth. However there may be multiple moderators of this effect, such as concurrent parental depression, affect toward the task, and cognitive style.

Social support by parents may be capable of aiding stress hormone regulation, yet, it is currently unknown how this may function in adolescents at familial risk for depression, especially in the context of HPA-axis reactivity dysregulation. In healthy individuals, social support can dampen the HPA-axis response to stress (Hostinar & Gunnar, 2013) by facilitating a faster recovery to the stressor (Kikusui, Winslow, & Mori, 2006). Adult studies suggest that having a socially supportive partner present prior to and after a stress task decreases the stress response (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Kirschbaum, Klauer, Filipp, &

Hellhammer, 1995; Meuwly et al., 2012). Only three studies have examined this in school aged children and adolescents, and show similar findings to adults. In 7-12 year old premenstrual girls, the presence of their mother in the room or speaking to them over the phone after a stress task led to lower post-TSST cortisol compared to girls who were left alone (Seltzer et al., 2010). Presence of parents during the preparation portion of the TSST has been linked to blunted HPA-axis activation to the TSST in children, but not adolescents (Hostinar et al., 2015). These results were further explained by pubertal timing; those adolescents who were farther along in puberty were less likely to have social buffering (Doom, Hostinar, VanZomeren-Dohm, & Gunnar, 2015). One limitation of the previous study was that parents were randomized to be present prior to the stress task, yet every child had their parent present after the stress task. This may only show that the presence of a parent was not able to modulate the HPA-axis *activation* to the stressor, but does not tease apart whether the *recovery* to the stressor is affected in adolescents. It is also known that adolescents demonstrate higher cortisol reactivity to psychosocial stress tasks compared to children (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Stroud et al., 2009), and thus, the presence of a parent may not be enough to mitigate the entire stress response. Given the non-specificity in the previous study, there may be important implications for parents to be able to affect the *recovery* curve of cortisol, even in adolescence. Thus, while it is currently unknown whether this social buffering effect may occur in children at high familial risk (Hostinar & Gunnar, 2013), it may be one mechanism by which parents either help or hinder the stress recovery process.

There are multiple key components that affect the social buffering response, some of which may be absent in families with history of depression. The acceptance of support is influenced by whether the child asks for the support, receives social support that match the needs

of the child, and the relationship quality of the dyad (Uchino, Carlisle, Birmingham, & Vaughn, 2011). Thus, it is predicted that social buffering would be most effective when parents provide encouragement and validation (Hostinar et al., 2014). Furthermore, it is known that positive dyadic coping either before or after a stressor is helpful in allowing for stress buffering (Meuwly et al., 2012). It is questionable whether high-risk adolescents and their parents with a history of depression will experience positive dyadic coping, as parents with high depressive symptoms and their children exhibited greater mutual negative affect and poor mutual regulation in conflict tasks (McMakin et al., 2011). Thus, parents with a history of depression may be able to provide social support that is effective in buffering the response, or they may be impaired in this capacity.

Overall, **Aim 3** examines the differences in high and low familial risk adolescents in social buffering and whether it is influenced by parental behaviors. Adolescents were asked to participate in the Trier Social Stress Task and to collect saliva samples in order to monitor salivary cortisol prior to, during and after the task. Adolescents either had their parent present after the task or watched a calming movie for the first 10 minutes after the task. In particular, it is expected that parents with a history of depression will not affect the recovery slope to the task, whereas control parents will be able to increase the rate of which the curve returns to baseline (thereby improving regulation).

Hypothesis 3a. Adolescents who have a family history of depression will demonstrate higher cortisol reactivity and slower recovery compared to low risk adolescents. See Figure 6. ***Hypothesis 3b.*** Presence of a parent will be related with a lower cortisol response in adolescents without a family history of depression compared to high risk peers. See Figure 7. ***Hypothesis 3c.*** Older adolescents will be less likely to show a parental buffering effect compared to younger

adolescents. ***Hypothesis 3d.*** Parental behavior will moderate the association between risk status and recovery slopes, so that adolescents' cortisol response will not differ among parents who display high levels of support. See Figure 8.

Figure 6. Diagram of Aim 3 Hypothesis A.

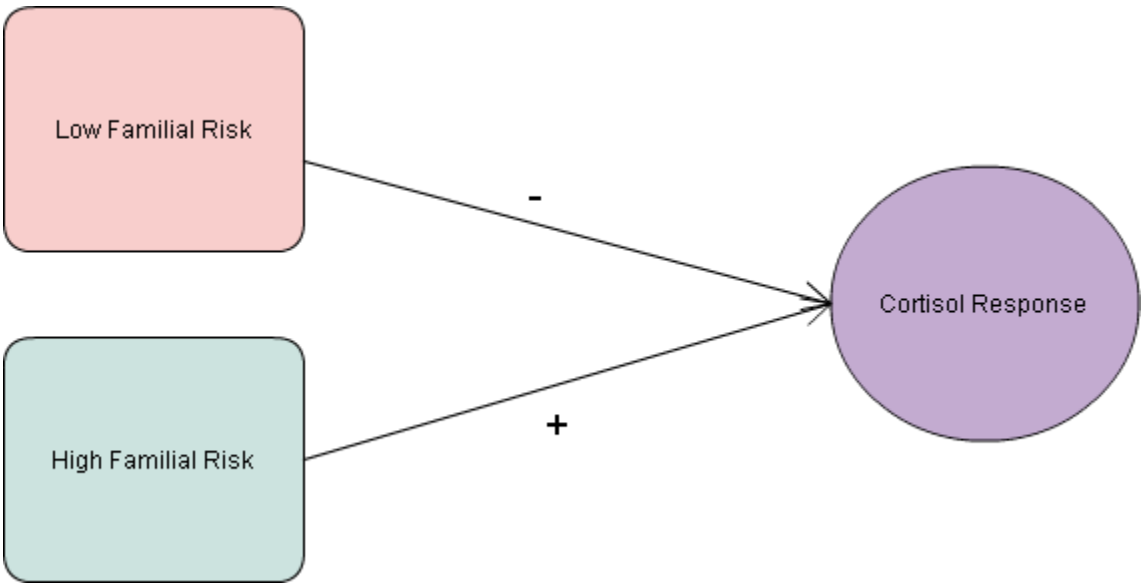


Figure 7. Diagram of Aim 3 Hypothesis B.

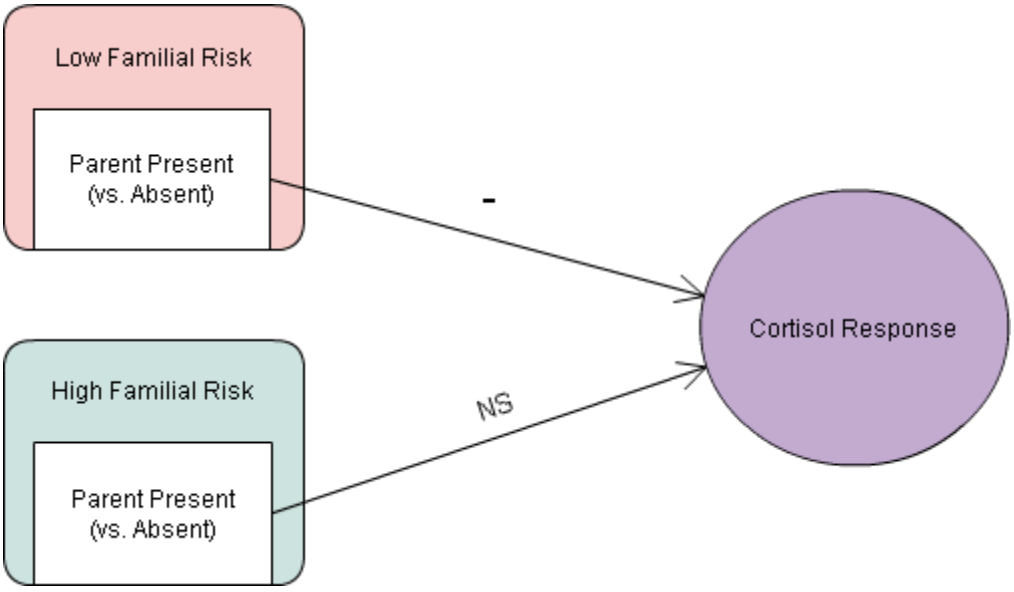
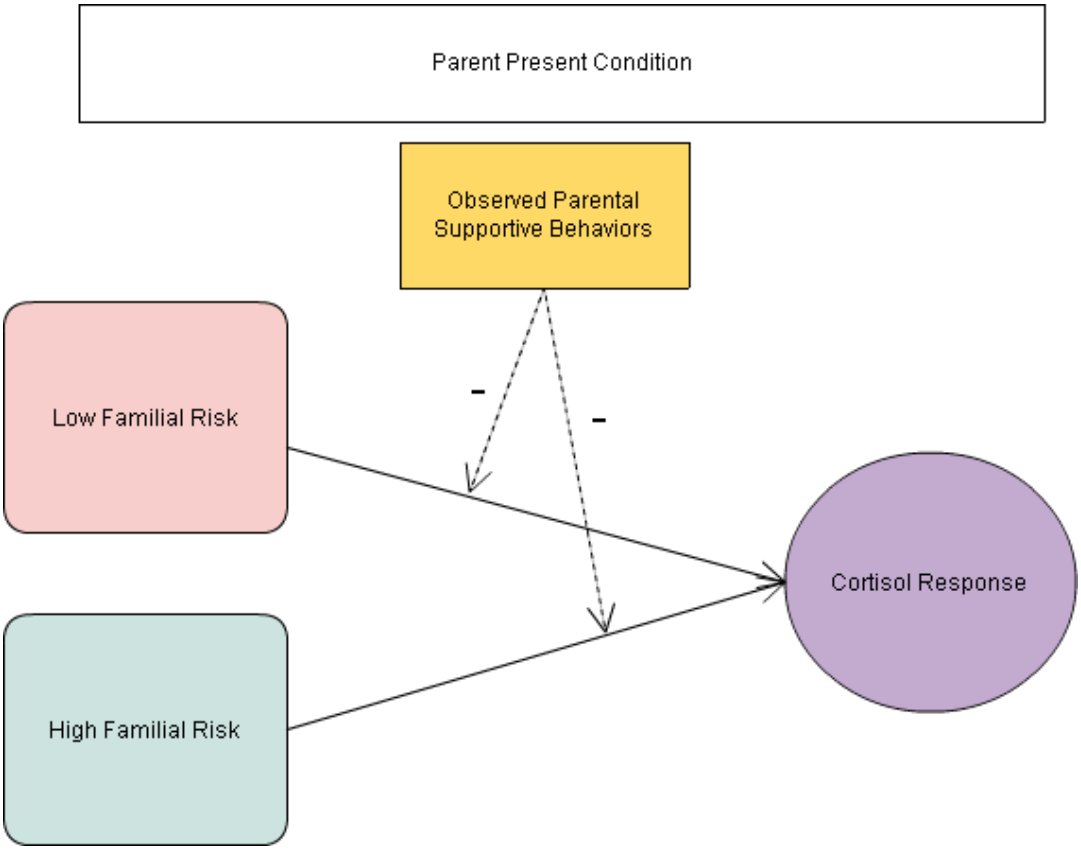


Figure 8. Diagram of Aim 3 Hypothesis D.



Chapter 2: Methods

Participants

This study is part of a larger longitudinal study under Dr. Nestor Lopez-Duran which investigates the effects of stress exposure on adolescents at high familial risk for depression. This dissertation study is conducted during the first day of the larger study, and includes 41 high-risk adolescents and 22 control adolescents. To be included in the high-risk group, adolescents' biological parent must have a history of a depressive disorder, and the adolescent must not be in an active depressive episode. Control adolescents did not have a history of depression, or a first degree relative with depression.

Procedures

Subjects were recruited through advertisements placed throughout a large Midwestern community and online. Interested parents completed a phone screen interview to determine initial eligibility for the study. Those who met eligibility were scheduled to come into the laboratory for a 3-4 hour study. See Figure 9 for the laboratory visit timeline. During the laboratory visit, the parent and adolescent independently underwent clinical interviews regarding the adolescent's current and past symptoms of psychiatric disorders. Parents additionally were interviewed about their own past and current psychiatric diagnoses. Adolescents underwent a socially evaluative stress task (TSST), and provided salivary cortisol samples. Adolescents were randomized into whether their parent was present or not for 10 minutes after the stress task. Families also responded to questionnaires regarding family functioning, social support, attachment, and internalizing and externalizing symptoms. The study was conducted at the

Michigan Psychoneuroendocrinology Affective Laboratory (Michigan PAL) located at the University of Michigan, Department of Psychology.

Figure 9. Timeline of the Study in Minutes

<table>
<tr><td>Minute</td><td>Child</td><td>Parent</td></tr>
<tr><td></td><td colspan="2">Consent</td></tr>
<tr><td>0</td><td rowspan="3">TSST Baseline and Questionnaires</td><td rowspan="4">Diagnostic Interview</td></tr>
<tr><td>10</td></tr>
<tr><td>20</td></tr>
<tr><td>30</td><td rowspan="2">TSST</td></tr>
<tr><td>40</td></tr>
<tr><td>50</td><td rowspan="2">Diagnostic Interview</td></tr>
<tr><td>60</td><td colspan="2">Interaction</td></tr>
<tr><td>70</td><td rowspan="3">TSST Regulation</td><td rowspan="4">Diagnostic Interview</td></tr>
<tr><td>80</td></tr>
<tr><td>90</td></tr>
<tr><td>100</td><td rowspan="2">Questionnaires</td></tr>
<tr><td>110</td><td rowspan="10">Questionnaires</td></tr>
<tr><td>120</td><td rowspan="10">Diagnostic Interview</td></tr>
<tr><td>130</td></tr>
<tr><td>140</td></tr>
<tr><td>150</td></tr>
<tr><td>160</td></tr>
<tr><td>170</td></tr>
<tr><td>180</td></tr>
<tr><td>190</td></tr>
<tr><td>200</td></tr>
</table>

Measures

Phone screen interview. Parents of target participants who respond to advertisements in the community were administered an initial phone screen to determine appropriateness and eligibility for the study.

Inclusion and exclusion criteria for adolescents at high familial risk. To participate in the study, high-risk adolescents were between the ages of 12-16, one of their biological parents

must have had a purported history of clinical depression, and the adolescent was currently not experiencing a depressive episode. Diagnoses of both parent and child were confirmed during the intake visit (see Diagnostic Interview section below).

Inclusion and exclusion criteria for adolescents at low familial risk. Low risk adolescents were between the ages of 12-16, and neither the biological parent nor the adolescent endorsed a history of depression.

Exclusion criteria for all participants. Adolescents were excluded if they had mental retardation, major systemic medical condition, or history of psychosis, schizophrenia, bipolar disorder, or pervasive developmental disorder.

Questionnaires. Adolescents and parents were asked to provide responses to multiple self-report questionnaires online. As the full study is larger in scope than this dissertation, only questionnaires that were relevant to the current dissertation are reported here. See Appendix 1 for all questionnaires filled out by participants during the study.

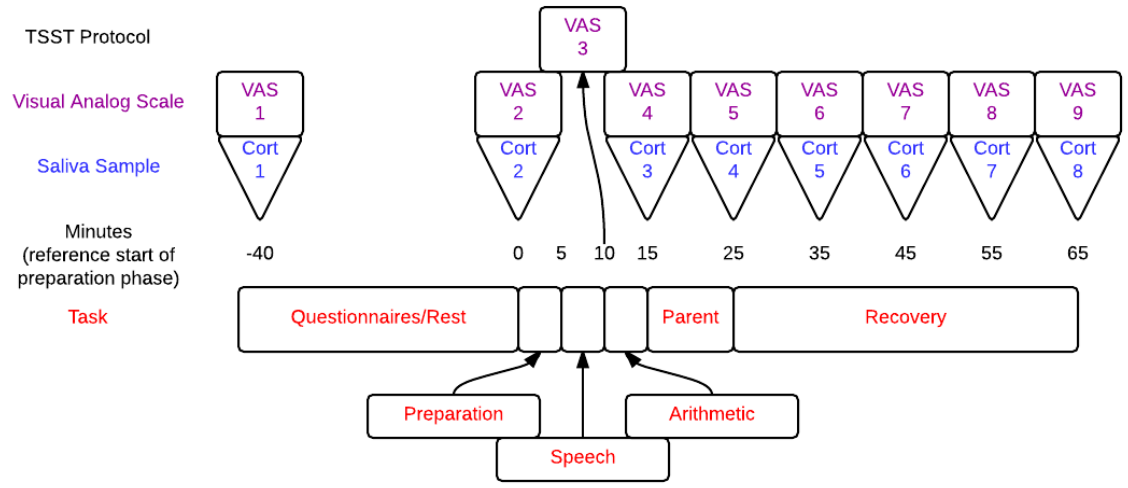
Current parental depressive symptoms. Parents were asked to assess their current depressive symptoms using the Beck Depression Inventory-II (BDI-II; Beck, Steer, Ball, & Ranieri, 1996). The BDI-II is a 21-item questionnaire that assesses for symptoms related to depression including affective and somatic symptoms. The BDI-II has high internal consistency, test-retest reliability, and validity with both psychiatric and community samples (Beck et al., 1996). In the current study, the alpha levels showed excellent internal reliability ($\alpha = .95$).

Current adolescent depressive symptoms. Parents and children reported on the adolescent's depressive symptoms using the Children's Depression Inventory – Parent and Self Report (CDI; Kovacs, 2010). The parent version includes 10 items, whereas the self-report version has 27 items. This is a widely used inventory in adolescent research and has

demonstrated acceptable reliability and validity (Kovacs, 2010). For the current study, Cronbach's alpha was good for parent report (α= .86) and child report (α= .91) of depressive symptoms.

Emotional response to stress task. Participants were asked to provide their emotional response to the stress task using the visual analog scales (VAS; Ahearn, 1997). Questions were asked regarding to what extent they feel 10 different emotions, including sad, confused, and angry. Participants marked their response on a 100 centimeter line on the computer ranging from "not at all" to "extremely". Participants were given this scale immediately after consent, right before the TSST, during the TSST, and every 10 minutes after the TSST. See Appendix 2 for the scale and Figure 10 for the timeline of administration.

Figure 10. Timing of Cortisol Sampling and VAS Ratings during the Trier Social Stress Task Protocol.



Perceived social support. Adolescents reported on how much they perceived their family to be supportive using the Multidimensional Scale of Perceived Social Support (MSPSS; (Zimet, Dahlem, Zimet, & Farley, 1988). This 12-item questionnaires asks on a 1-7 Likert scale how much adolescents agree with statements such as "My family really tries to help me" or "I can

talk about problems with my family". The subscale of family support was used in this dissertation which includes 4 items solely about family support. The MSPSS has been used with adolescents and has been found to have good internal reliability (Zimet, Powell, Farley, Werkman, & Berkoff, 1990). In this study Cronbach's alpha was .88.

If adolescents were in the parent present condition, they additionally reported on how supported they felt during the parent-child interaction. These questions were based on a 0-100 scale similar to the VAS. In this dissertation, we examined the question "To what degree did you feel supported by your parent/guardian?" See Appendix 3 for a description of the scale.

Pubertal timing. The Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) is a 9-item questionnaire designed to assess stage of pubertal development. It is rated on a 1-4 scale, and the composite score is an average of 4 key developmental changes. It has been shown to correlate to other measures of puberty such as Sexual Maturation Scale (Petersen et al., 1988).

Phase of menstrual cycle. Since phase of menstrual cycle may impact cortisol reactivity, a variable was computed to determine phase of cycle. Girls reported on the last day of their most recent period, and on average how many days between their menstrual cycles. Most subjects reported their cycle was about a month long, thus, we calculated the number of days since the start of their last period. Those who were greater than 14 days were defined as being in the luteal phase, whereas those less than 14 days were in the follicular phase. Additionally, if girls reported they were taking birth control, we grouped them with those in the follicular phase, as they theoretically had similar cortisol levels. To increase power, we included boys in this variable as they had similar cortisol response to girls during the luteal phase (Kudielka, Hellhammer, & Wüst, 2009).

Diagnostic Interviews. Adolescents and their parents underwent psychiatric diagnostic interviews separately. All diagnostic interviews were reviewed during weekly diagnostic meetings with the clinicians and Dr. Lopez-Duran who is a licensed clinical psychologist.

Adolescent psychiatric diagnostic interview. Trained master's level clinicians interviewed both the adolescent and parent independently about the child's experiences of current and past psychiatric disorders using a modified version of the Schedule for Affective Disorders and Schizophrenia for School Aged Children (K-SADS-PL; Kaufman et al., 1997). This interview was modified to account for diagnostic criteria changes from DSM-IV-TR to DSM-5. Diagnoses were determined using the best estimate procedure for multiple informants (Leckman, 1982).

Parent psychiatric diagnostic interview. Master's level clinicians interviewed the biological parent using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2012) or the Structured Clinical Interview for DSM-5 Axis I Disorders (First, 2015) to determine whether they have past or current depressive disorder. The DSM-IV version was modified to include DSM-5 criteria, and once the DSM-5 version of the interview was published, that version was adopted.

Diagnostic interview inclusion and exclusion. After conducting the diagnostic interviews, participants were grouped into either the high or low familial risk group. To be included in the high familial risk group, there was a confirmed parental diagnosis of past or current MDD or Dysthymia. To be included in the low risk group, participants and parents were free from depressive disorders.

Modified Trier Social Stress Task for Children (TSST). The TSST is a well-established mild psychosocial stress task designed to reliably elicit a cortisol response in children

and adolescents (Buske-Kirschbaum et al., 1997), and has been used in other social buffering studies (Hostinar & Gunnar, 2015). In this paradigm, participants are asked to make a 5-minute speech and complete 5 minutes of mental arithmetic in front of a panel of judges. The current study used a modified version which asked adolescents to give a speech as if they were running for class president (Ellenbogen & Hodgins, 2009). Modifications of the TSST in youth have been shown to reliably activate stress hormones (Gunnar, Talge, et al., 2009). See Figure 10 (above) for a graphic of the TSST.

Participants were asked to provide their first saliva sample and VAS ratings immediately after consent, and then fill out questionnaires and rest for 40 minutes, to ensure that they have lower levels of cortisol by the start of the task. Subjects provided their second saliva sample and VAS rating immediately before the preparation phase of the task. After taking sample 2, a research assistant told the adolescent that they will be asked to give a speech in front of a video camera and a panel of judges, with their speech being recorded to assess for facial expression and the content of what they say. Adolescents were given 5 minutes to prepare their speech on running for class president (Ellenbogen & Hodgins, 2009) in the room with judges and video camera. Once the preparation time was complete, participants were asked to stand and complete their speech in 5 minutes. The judges are asked to be stoic and not show any signs of approval. If the participant stopped talking before 5 minutes had elapsed, the judges asked the participant to continue, or asked a question from a predetermined question list, such as "What talents would you bring to the presidency?" or "Do you have any weaknesses?". After the speech, subjects were instructed to serially subtract the number 13 from 1023 for 5 minutes. On every failure, subjects were asked to start from the beginning.

After the task, subjects were escorted to a calming room where they provided the third saliva sample and VAS rating. Participants were debriefed and told that the judges were instructed to be stern. Subjects were then randomized into having their parent present or absent for the next 10 minutes (Seltzer et al., 2010). In the parent absent condition, participants were escorted to a room to watch a video (detailed below). In the parent present condition participants were escorted to the room their parent was in, and the dyad was videotaped for 10 minutes in order to code for parental and child behaviors. Parents were told that their child has participated in the TSST, which may be a potentially stressful procedure, and that they are to provide any support they see necessary. After these 10 minutes, adolescent participants were left in the room alone, videotaping was stopped, and they watched a non-emotionally evocative Natural Geographic movie. Additionally, before each of these saliva samples, they were asked to fill out a new VAS.

Cortisol sampling. Saliva samples were collected from adolescents before, during, and after the TSST. All participants began their lab visit during the diurnal nadir in cortisol between 2 and 4 pm in order to standardize the timing of cortisol collection. Additionally, participants were asked to not eat or drink anything other than water 1 hour prior to collect of saliva, as glucose influences cortisol values. Immediately after consent, subjects were asked to spit into a salivette in order to measure baseline levels of cortisol. Salivary cortisol was then collected immediately before and after the TSST. After the TSST, saliva samples were collected every ten minutes until 65 minutes was reached. See Figure 10 (above) for more detailed graphic of saliva sampling. The samples were frozen and stored in a secured room in preparation for analysis. Samples were assayed at a University of Michigan Core Assay Facility within 6 months of collection in duplicate and averaged using a commercial enzyme immunoassay kit (Salimetrics).

To decrease interassay variability, all samples from the same child were assayed in the same batch. There were 3 samples in the entire sample that had duplicates varying more than 15%, and were re-assayed.

Coding of parent-child interaction. Parental behavior during their 10 minutes of interaction was coded using a modified version of the Emotion Socialization Strategies (Bosler, Morris, & Criss, 2012), which is based off an emotional discussion coding system and previously used with adolescent children and their parents (Bosler et al., 2012). Considering that the original manual was designed to code for parental behaviors in the context of explicitly discussing emotions and the current study did not have explicit instructions to do so, the manual had to be changed to fit the current context. In order to modify the manual for the current purposes, a team of 6 research assistants and the author watched 5 randomly selected parent-child videos from this study. Each member of the team coded the videos separately using the Emotion Socialization Manual as originally prepared. In a group, each video was watched, and any discrepancies between different coders were discussed. Furthermore, the group engaged in brainstorming of codes that may be added in or taken out of the manual for the current purposes. For example, given that parents and children were not given specific instructions to discuss the stress task in the current study, there was variability in the topics for discussion. Codes were included for being "on topic" which encompassed discussing any part of the study (e.g., questionnaires, the stress task) and "off topic" if they talked about other issues. Furthermore, additional examples of codes were generated that fit the current study (e.g., under *Comforting* we included encouragement for future performance, considering that parents used this strategy often). We also collapsed across different codes of *Teaching* and *Problem Solving*, since these caused excessive disagreement among coders.

Once the final manual was generated based off the initial 5 videos, coders were given randomly selected videos to code. Since coders were involved with data collection (for example by being the visit leader or TSST confederate), care was taken to not assign videos to the coders in which they participated in the data collection. All videos were double coded by two coders and pairing of coders was changed so that all individual coders coded at least one video with each of the other coders. After coders independently coded videos, they discussed the video with a partner and created a master code.

Parental responses to adolescent emotions were coded continuously during "on topic" times and grouped into 2 different categories encompassing global supportive and unsupportive interactions. Specifically supportive interactions included *Comforting* (e.g., touching the child, comforting statements such as "I'm sure you did great"), *Validation of feelings* (e.g., labeling emotions, indicating understanding of the emotional experience), and *Problem solving or Teaching about emotions* (e.g., discussing coping strategies, utilizing past experiences to discuss current distress). Unsupportive interactions included *Dismissing* (i.e., discourages the expression of emotions through minimizing or distraction), *Punishing* (i.e., discourages emotion through punishing or expressing disapproval of emotion, such as invalidating emotions and making fun of or teasing) and *Magnifying* (i.e., increasing the emotion, or expanding on express emotion).

Interrater reliability was computed by splitting the videos into 20 second increments, and noting whether each coder and the master code had recorded a behavior within those increments (either yes/no). For supportive parental behaviors, there was 91% agreement between the coders and master code, $\kappa = .80$, and 81% agreement between the two coders, $\kappa = .58$. There was 95% agreement between the master code and individual coders for unsupportive behaviors, $\kappa = .68$, and 90% agreement between the two coders, $\kappa = .21$. The lower agreement for the unsupportive

behaviors may have been due to relative infrequency of these behaviors that occurred across videos.

We additionally coded for the adolescent's *Expression of Emotion* in regards to the task. This was coded on a 1-3 scale of intensity indicating 1) whether they brought up the task (e.g., "I had to give a speech and do an arithmetic task"), 2) whether they described the task and feelings about it (e.g., "I was really nervous to give the speech") or 3) whether they explicitly reported distress due to the task (e.g., "I'm really stressed", crying). For the purposes of these analyses, we collapsed across the three intensity codes and obtained a global frequency of how many times the child brought up the stress task or their emotions. For a copy of the scale used in this study, see Appendix 4. These constructs used in this coding system have been investigated theoretically at length (Malatesta-Magai, 1991). There was 92% agreement between the coders and master code, $\kappa = .74$, and 85% agreement between the two coders, $\kappa = .43$.

Videos were recorded via overhead surveillance camera or a web camera attached to a computer. Videos were coded using the Behavioral Observation Research Interactive Software (v. 2.6 or later) (Friard & Gamba, 2012). This software is used for event logging in real time and allowed for a timestamp of each behavior and frequency to be computed.

Data Analysis

A Priori Power Analysis. Prior to conducting the study, we examined how many participants would be needed to have adequate power for our analyses. Given the large effect size ($d = .83$) between stranger and romantic partner support in adults on cortisol responses (Kirschbaum et al., 1995), we calculated the sample size to be 22 participants in each cell (2 Risk Groups x 2 Parent Conditions). This was a similar number of participants in other social buffering studies in children (Hostinar et al., 2015; Seltzer et al., 2010). The computation of

effect size was completed using G*Power, version 3.1.9.2 (Faul, Erdfelder, Lang, & Buchner, 2007).

Aim 1: Observed Parental Behaviors to Adolescent's Distress after a Stress Task. Using SPSS software, version 23, we first examined whether covariates of age and gender predicted supportive and unsupportive behaviors as coded for using the Emotion Socialization Strategies System (Bosler et al., 2012) (see Appendix 4). In order to increase power, frequency of parental behaviors were summed into global supportive (i.e., comforting, validation, teaching/problem solving) and unsupportive (i.e., punishing, dismissing, magnifying) codes for each participant. Supportive behaviors followed a normal distribution, thus, we conducted linear regressions with this data. Considering that unsupportive behaviors did not follow a normal distribution, we partitioned the outcome into 3 categories (none, low and high unsupportive behaviors), which approximately captured a 1/3 of the sample within each category. We then used ordinal logistic regression or multinomial logistic regressions to predict category membership from our independent variables. For the first hypothesis we examined the impact of group membership (high vs. low familial risk) on parental behaviors (supportive vs. unsupportive). Age and gender along with interactions of these terms with group membership were included separately as covariates. For the second hypothesis, parental depressive symptoms were included as a moderator of the effect of group membership on parenting behaviors, and for the third hypothesis, this analysis was repeated with child reported depressive symptoms being included as the moderator.

Aim 2: Impact of Parent on Adolescent Affect Regulation. For the first hypothesis we examined the impact of group membership (high vs. low familial risk), parental presence after the stressor (present vs. absent), and their interaction as predictors of child's positive and

negative affect after the stress. We conducted Growth Curve Analyses utilizing SAS version 9.4. Since we were interested in regulation of affect, we conducted linear modeling starting at the highest level of negative affect (time point 3) or lowest level of positive affect (time point 4) until time point 6, which is 10 minutes after the parent left the room or 20 minutes after the conclusion of the TSST. We averaged the reported levels of tenseness and fear into a composite variable of negative affect, which was negatively skewed, thus we log transformed the data. Levels of happiness generally conformed to a normal distribution.

For the second hypothesis we examined the impact of familial risk group, parental supportive behaviors, and their interaction as predictors of child's negative affect after the stressor. For analyses conducted with observed parental behaviors, we used data obtained from Aim 1, and include only those children who had their parent with them after the stress task (as that is the only observed data we obtained). Additionally, we examined the impact of the child's perceived family support, using the family subscale of the Multidimensional Scale of Perceived Social Support, on linear trajectories of affect. With this perceived support analysis, we were able to utilize the entire sample since all adolescents reported on this questionnaire.

Aim 3: Impact of Presence of Parent on Adolescent's HPA-Axis Reactivity and Recovery. In order to test the hypothesis that parent support will impact cortisol recovery, we ran Growth Curve Analysis utilizing landmark registration (Lopez-Duran, Mayer, & Abelson, 2014) in SAS version 9.4. Landmark registration identifies each individual's peak in the cortisol curve and anchors each individual's cortisol reactivity curve to that landmark. Peaks were identified as the first point in the upward activation slope that was a 20% increase from baseline, and was followed by a decrease in cortisol values. If it was followed by a plateau, then it was tested whether any other sample was 10% higher, and if so, this was identified as the new peak.

The mode peak occurred 25 minutes after the TSST began. Those individuals who did not have a peak that fit these criteria (in the non-responder group) were assigned the mode peak time as their peak. Utilizing this model, we are able to center the intercept to be peak levels of cortisol and model cortisol reactivity and recovery slopes (Lopez-Duran et al., 2014). Given that cortisol values were skewed (skewness overall = 2.02; Shapiro-Wilk = .83, p<.001), results were transformed using Box-Cox transformations. Specifically, we used the following formula as defined by Miller & Plessow (2013), $X' = \frac{(X^{0.26})-1}{0.26}$, as this gives superior results for salivary cortisol data in transforming for normality compared to traditional log transformations. We first modeled covariates of gender, age, pubertal status, phase of menstrual cycle, and time since waking on the impact of the cortisol curves and peak values. There are known effects of women having lower cortisol compared to men, and for it to be impacted by menstrual cycle; specifically women in the follicular phase or on contraceptives have flatter cortisol reactivity curves compared to men and women in the luteal phase of their cycle (Kudielka & Kirschbaum, 2005). Furthermore, age and pubertal status were included since there are known increases in the strength of cortisol when children age, and may be a product of puberty (Gunnar, Wewerka, et al., 2009; Stroud et al., 2009). Time since waking up in the morning was included since it has been shown that time of day impacts cortisol reactivity (Broderick, Arnold, Kudielka, & Kirschbaum, 2004; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004).

For the first hypotheses, the model included the following predictors: group (control vs. high-risk), condition (presence or absence of parent), any significant covariates and baseline cortisol values. Baseline levels were computed as an average of cortisol immediately after coming into the laboratory (40 minutes prior to the TSST) and the sample immediately before the TSST.

For the last hypothesis, parental behaviors (supportive and unsupportive behaviors) were included as a moderator in the model. Supportive and unsupportive behaviors were taken from the coded behaviors in Aim 1. Only those in the parent present condition were included in the analysis.

Chapter 3: Results

Sample Characteristics

A total of 63 participants completed the study for this dissertation. Overall, there were 36 girls and 27 boys accompanied by 54 mothers and 9 fathers. There were a total of 9 families who participated with siblings. The majority of parents reported their children were Caucasian (85.7%). The rest were reported as being biracial (7.9%), Hispanic/Latino (6.3%), Asian/Asian Pacific Islander (3.1%), and African American descent (3.1%) (the total percentage is greater than 100%, since families were allowed to check as many boxes as they see fit). The majority of parents who participated in the study were college graduates (42.3%) or obtained graduate training (40.6%), with the remaining having high school degrees (3.4%) or partial college (10.6%). The majority of families had annual household incomes of $50,000 – 100,000 (43.8%) or greater than $100,000 (43.8%). 10.4% of parents made between $25,000 - 50,000 and the remaining 1.7% had income less than $12,000 per year. Most parents were married (81.4%), fewer were divorced or separated (15.1%), or never married (3.4%).

We examined group level differences in variables of interest. See Table 1. Adolescents at high and low familial risk did not differ based on age, gender, condition, pubertal status, or parental education. Yet, families with a history of depression had lower income levels. As expected, high risk adolescents and their parents reported higher depression scores and moderately lower perceived support from family compared to low risk adolescents. Group comparisons between the parent present and absent condition did not see any differences in amount of participants, gender, age, risk status, nor on any depression or support measures.

Furthermore, there were a few gender differences, with girls reported being farther along in puberty as well as having higher depressive symptoms.

Table 1. Demographic Differences in the Full Sample

	High Familial Risk	Low Familial Risk	Difference
Count	N = 41	N = 22	
Condition	Parent Present = 18 Parent Absent = 23	Parent Present = 13 Parent Absent = 9	χ^2 (1) = 1.32, p = .25
Gender	Girls = 25 Boys = 16	Girls = 11 Boys = 11	χ^2 (1) = .70, p = .40
Age	M = 13.9, SD = 1.3	M = 14.1, SD = 1.2	t (61) = .43, p = .67
Income [a]	M rank = 25.0	M rank = 37.6	U = 195.5, p = .005*
Primary Caregiver Education Level [a]	M rank = 30.5	M rank = 29.1	U = 372.0, p = .76
Pubertal Development (PDS)	M = 2.7, SD = .53	M = 2.8, SD = .61	t (61) = .43, p = .67
Child Depressive Symptoms Self Report (CDI Self)	M = 9.7, SD = 8.4	M = 5.5, SD = 4.8	t (61) = -2.2, p = .03*
Child Depressive Symptoms Parent Report (CDI Parent)	M = 13.4, SD = 6.4	M = 7.6, SD = 5.4	t(58)= -3.5, p =.001**
Parent Depressive Symptoms (BDI)	M = 3.2, SD = 6.4	M = .10, SD = .29	t (59) = -2.3, p = .03*
Child Perceived Family Support (MSPSS Family)	M = 4.1, SD = .79	M = 4.4, SD = .62	t (61) = 1.7, p = .10[t]
Child Reported Anxiety Symptoms (SCARED)	M = 23.9, SD = 16.7	M = 14.1, SD = 10.9	t (59) = 2.4, p = .02*

[a] For income and parental education, we ran non-parametric Mann Whitney U tests, since levels of these variables were ordinal and not normally distributed.
** p <.01, * p <.05, [t] p <.10

	Parent Present	Parent Absent	Difference [b]
Count	N = 31	N = 32	
Gender	Girls = 17 Boys = 14	Girls = 19 Boys = 13	χ2 (1) = .13, p = .72
Age	M = 13.9, SD = 1.2	M = 14.0, SD = 1.3	t (61) = .31, p = .76

[b] In order to save space, we only included measures that were significantly different between condition and gender.

	Girls	Boys	Difference [b]
Count	N = 36	N = 27	
Age	*M* = 14.1, *SD* = 1.3	*M* = 13.8, *SD* = 1.1	*t* (61) = .96, *p* = .34
Pubertal Development (PDS)	*M* = 2.9, *SD* = .32	*M* = 2.5, *SD* = .70	*t*(61) = 3.2, *p*= .002**
Child Depressive Symptoms Self Report (CDI Self)	*M* = 10.1, *SD* = 7.9	*M* = 5.7, *SD* = 6.5	*t* (61) = 2.3, *p* = .02*
Child Reported Anxiety Symptoms (SCARED)	*M* = 26.9, *SD* = 15.1	*M* = 12.5, *SD* = 12.2	*t* (59) = 4.0, *p* <.001**

Aim 1: Observed Parental Behaviors to Adolescent's Distress after a Stress Task

In these analyses, we only considered those adolescents who were randomized into the parent present condition (i.e., had their parent with them after the stress task). Thus, there were 18 high familial risk and 12 low risk adolescents and their parents involved in this aim. One adolescent's video (high risk girl) was not coded since the video was recorded without sound (experimenter error), thus there were only 17 adolescents at high familial risk in this analysis. Given the smaller sample size, we again tested differences in the high and low risk group on variables of interest, see table 2, and included the correlation table, see table 3. Results were similar to the full sample.

Table 2. Demographic Differences between High and Low Familial Risk Adolescents in the Parent Present Condition

	High Familial Risk	Low Familial Risk	Difference
Gender	Girls = 10 Boys = 7	Girls = 5 Boys = 7	$\chi 2$ (1) = .83, p =.36
Age	M = 13.88, SD = 1.32	M = 13.92, SD = 1.17	t (27) = -.07, p = .94
Child Self-Report Depressive Symptoms (CDI-Self)	M = 11.82, SD = 9.43	M = 4.25, SD = 4.97	t (27) = 2.54, p = .02*
Parent-Reported Child Depressive Symptoms (CDI-P)	M = 14.18, SD = 5.84	M = 8.18, SD = 6.68	t (26) = 2.51, p = .02*
Parental Depressive Symptoms (BDI)	M = 4.0, SD = 6.07	M = .08, SD = .29	t (27) = 2.22, p = .04*

Table 3. Correlations of Demographics, Depressive Symptoms, and Parental Behaviors in Parent Present Condition

	Age	CDI P	CDI S	BDI	Supportive	Unsupportive
Child's Age	1					
CDI Parent	0.13	1				
CDI Self	0.27	0.48**	1			
BDI	-0.24	0.19	0.01	1		
Supportive Parent Behaviors	-0.21	-0.08	-0.26	-0.14	1	
Unsupportive Parent Behaviors	0.43	0.06	0.26	-0.22	-0.09	1
Child's Expressed Emotion	0.00	-0.02	0.06	-0.17	0.36^{t}	0.31^{t}

** p <.01, * p <.05, ^{t} p <.10; CDI Parent = Child Depression Inventory Parent Report; CDI Self = Child Depression Inventory Self Report; BDI = Beck Depression Inventory

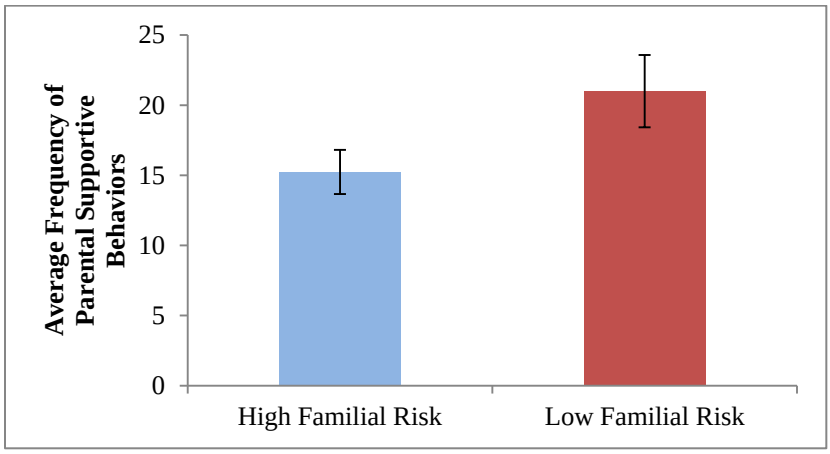
Parental Supportive Behaviors. For the analyses with supportive parental emotion socialization behaviors, we utilized t-tests and regressions. As frequency of supportive parental behaviors were normally distributed across this sample (range = 5 – 35, skewness = .74, Shapiro-Wilk = .93, p =.06), and the mean was over 10 (M = 17.6, SE = 1.48), assumptions of normality were not violated and regression analyses were appropriate (Coxe, West, & Aiken, 2009).

Initially, we examined the impact of age and gender on supportive parental behaviors in order to determine if we would include them as covariates to the analysis. Neither age nor gender emerged as statistically significant covariates, thus they were not included in further models.

Hypothesis 1a: Group Differences in Supportive Parental Behaviors. In order to test whether supportive parental behaviors differed between the high and low familial risks groups, a t-test was conducted. Parents without a history of depression showed a moderately greater frequency of supportive behaviors during the interaction task compared to parents with a history

of depression, t (27) = 2.02, p =.054, (High Familial Risk: M = 15.2, SD = 6.5; Low Familial Risk: M = 21.0, SD = 8.9). See Figure 11.

Figure 11. Group Differences in Frequency of Supportive Parental Behaviors



Note: Error bars reflect standard error of the mean.

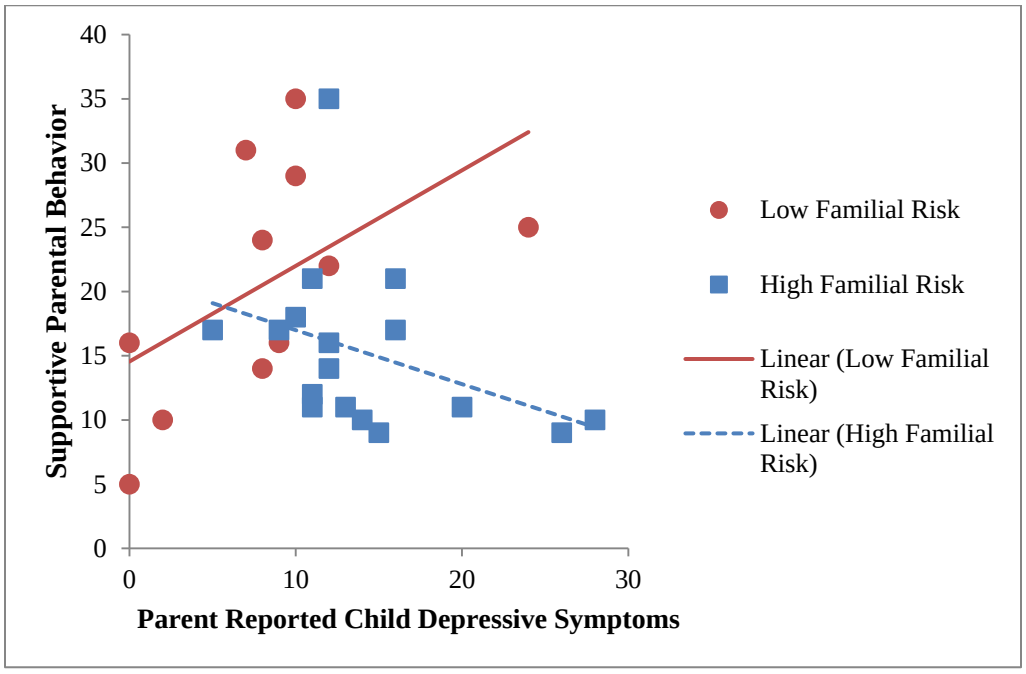
Hypothesis 1b: Impact of Parental Depressive Symptoms on Supportive Behaviors. We tested whether concurrent parental depressive symptoms predicted amount of supportive parental reactions using a linear regression. There was no effect of symptoms on supportive behaviors, F (1, 27) = .55, p =.47. We included group risk status and its interaction with depressive symptoms to determine if there was a different association for high and low familial risk groups, however no predictors were significant. Thus, there was no association between current parental depressive symptoms and the amount of supportive behaviors parents showed during the parent-child interaction task.

Hypothesis 1c: Impact of Adolescent's Depressive Symptoms on Parental Supportive Behaviors. We also tested whether the adolescent's depressive symptoms were related to parental behaviors. Both parent and child reported on the level of child's depressive symptoms, thus, regressions were conducted independently for parent and child reported symptoms. For child reported depressive symptoms (CDI Self), there was no association between symptoms and

parental supportive behaviors, F (1, 27) = 2.0, p = .17, even when considering the moderator of risk group.

For parent-reported child depressive symptoms (CDI Parent), there was no main effect of symptoms on supportive behaviors, F (1, 26) = .16, p = .70. However, we examined the moderator of group status, and found that the association between parent-reported child symptoms and supportive parental behaviors differed by familial risk membership, *CDI-P x Risk*, F (1, 24) = 6.72, p =.02. Specifically, for the low familial risk group, the greater the parent-reported child depressive symptoms, the more supportive parental behaviors they showed after the TSST, b = .74, t (27) = 2.23, p = .04. However, there was no association for child depressive symptoms and positive behaviors in the high risk group, b = -.42, t (27) = -1.40, p =.18. Therefore, this may suggest that in the low familial risk group, the greater the parent perceives depressive symptoms in their child, the more supportive behaviors they show to their children after a stressor, yet the same association was not found in families with a parental history of depression. See Figure 12.

Figure 12. Group Differences in the Association between Parent-Reported Child Depressive Symptoms and Supportive Parental Interactions.



Exploratory Analysis: Impact of Adolescent's Distress on Parental Supportive Behaviors. In order to explore the results further and determine whether the association between depressive symptoms and risk status were attributable to levels of distress the adolescent expressed during the interaction, we ran follow up analyses that predicted parental supportive behaviors from the adolescent's own expression of emotion (frequency of verbal mentions of the task or distress during their time with their parent). The mean frequency of expressing emotion was 6 times over the 10 minute task (SEM = 0.98), with a range of 0 – 23 times. Examining outliers, it was found that one child had an expressed emotion Z score greater than 3, and could potentially skew results (Skewness = 1.5, Shapiro-Wilk = .87, p =.002). Thus, we winsorized the outlier value to reflect the highest score in the dataset (Erceg-Hurn & Mirosevich, 2008), and used these values to run statistical analyses. The new expressed emotion frequency ranged from 0-14. Adolescents at low familial risk demonstrated greater expressed emotion (M = 7.83, SD = 4.2) compared to high risk adolescents (M = 3.88, SD = 3.8), t (27) = 2.66, p = .01.

We first examined whether expressed emotion predicted supportive parental behaviors. We found that the greater the expressed emotion, the more supportive behaviors were demonstrated, *Expressed Emotion*, $b = .82$, $F\ (1, 27) = 6.64$, $p = .02$. We then examined whether the effect of expressed emotion on parental support varied as a function of familial risk, yet did not find any different associations. Furthermore, we examined whether the results from Hypothesis 1c (i.e., low risk parents provided greater supportive behaviors when they perceived higher depressive symptoms in their child) were influenced by child's expressed emotion. However, adolescent's distress during the task did not explain the association between low risk parents giving more support to their adolescent who showed more depressive symptoms. Thus, the current analysis suggests that the effect of depressive symptoms and risk status is relatively independent of how much distress the child expresses during the interaction, although there is a positive association between expressing emotion and supportive behaviors.

Exploratory Analysis: Adolescent's Perception of Support from Parent. After the parent-child interaction, we asked adolescents to report on a scale from 0-100 how supported they felt by their parent. Overall, adolescents reported relatively high levels of support ($M = 83.3$, $SD = 22.5$). We then conducted a t-test to determine whether adolescents reported different levels of support dependent upon their familial risk group. Adolescents at high familial risk for depression reported less support than their low risk counterparts, $t\ (14.4) = -2.55$, $p = .02$, (High Familial Risk: $M = 81.8$, $SD = 18.7$; Low Familial Risk: $M = 94.8$, $SD = 4.1$).

Exploratory Analysis: Impact of Adolescent's Anxiety Symptoms on Parental Support. The association between expressed distress and parental support may be due in part to adolescent's anxiety symptoms, thus we explored this in these additional analyses. First, levels of anxiety symptoms did not predict parental supportive behaviors, *SCARED*, $F\ (1, 27) = .02$, $p =$

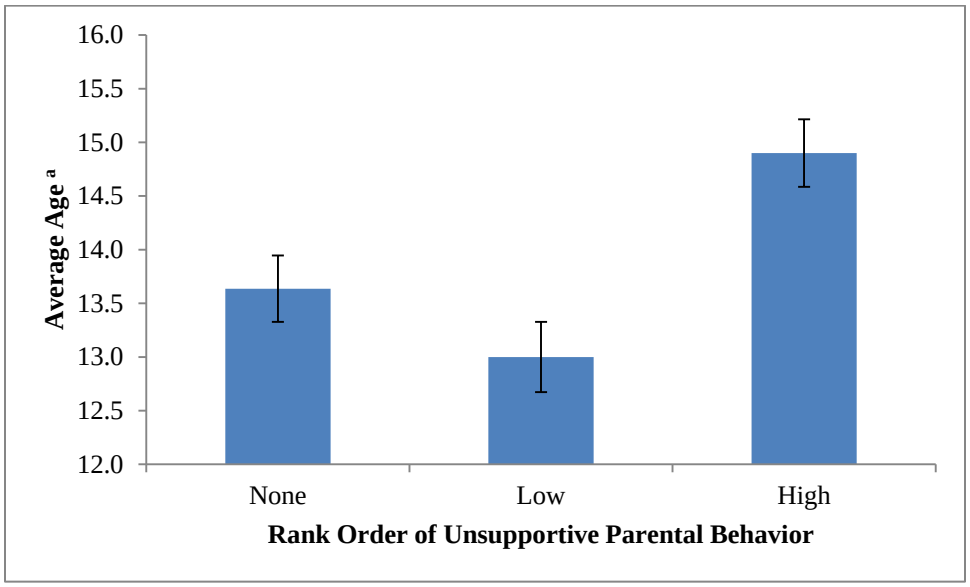
.90. We then examined whether the effect of anxiety on parental support varied as a function of familial risk, yet did not find any different associations.

Parental Unsupportive Behaviors. Unsupportive parent behaviors ranged from 0-11 times during the 10-minute interaction, and the variable was positively skewed (skew = 2.2, Shapiro-Wilk = .74, p <.001). Given that parents showed a low range of these behaviors, we rank ordered the frequency of unsupportive behaviors and combined them to create three categories. The categories were defined as followed: *none*- did not demonstrate any unsupportive behaviors (N = 11), *low*- demonstrated one to two unsupportive behaviors (N = 8) and *high*- demonstrated three or four unsupportive behaviors, except for one outlier who demonstrated 11 (N = 10). From this data, we first ran ordinal logistic regressions, however, if assumptions were violated, we conducted multinomial logistic regression (UCLA: Statistical Consulting Group., 2016).

As in the analyses with supportive behaviors, we first examined age and gender as covariates in the prediction of unsupportive behaviors. In the ordinal logistic regressions, there was a violation of assumption with unequal coefficients across levels of the outcome, thus, we conducted a multinomial logistic regression, with the dependent variable reference category of *none*. In the first model, there was no effect of gender, χ^2 (2) = .46, p = .80. In the model with age, the overall model was significant, χ^2 (2) = 14.32, p = .001. As children aged, they were more likely to be in the *high* unsupportive category compared to those in the *no*, b = 1.28, *Wald* χ^2 (1) = 4.87, p = .03, and *low* unsupportive category, b= 2.05, *Wald* χ^2 (1) = 7.46, p = .006. However there was not a statistically significant difference in age between those that were in the *none* versus *low* categories, b = -.77, *Wald* χ^2 (1) = 1.88, p = .17. Thus, as adolescents age, their parents were more likely to give more unsupportive interactions after the adolescent experienced

a laboratory stressor. Thus, we retained the effect of age in all subsequent analyses. See Figure 13.

Figure 13. Mean Age for Each Category of Unsupportive Parental Behaviors



[a] Error bars reflect Standard Error of the Mean

Hypothesis 1a: Group Differences in Unsupportive Parental Behaviors. We examined whether risk group predicted amount of unsupportive behaviors using ordinal logistic regression. Parents with a history of depression showed on average more unsupportive behaviors (M = 1.88, SD = 1.6) compared to unaffected parents (M = 1.25, SD = 1.6), yet this difference was not statistically significant, b = -.82, *Wald* χ^2 (1) = 1.32, p = .25. See Table 4 for how many participants were in each unsupportive behavior category based on risk group.

Table 4. Cross Tabulation of Membership in Each Unsupportive Behaviors Category by Risk Group

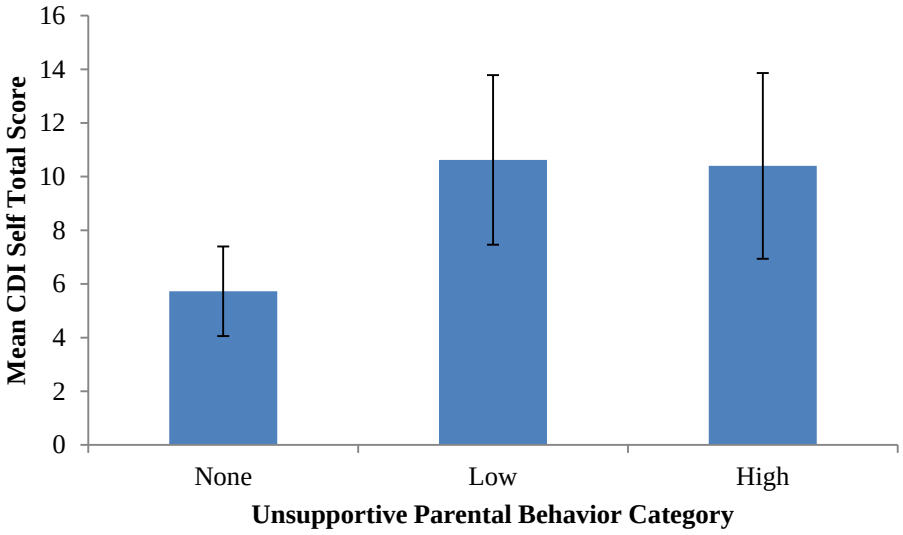
		Unsupportive Behavior Category			
		None	**Low**	**High**	**Total**
Group	**Low Risk**	6	3	3	12
	High Risk	5	5	7	17
	Total	11	8	10	29

Hypothesis 1b: Impact of Parental Depressive Symptoms on Unsupportive Behaviors. In order to determine whether parental depressive symptoms were related to amount of unsupportive behaviors, we ran multinomial logistic regression predicting unsupportive behaviors from parental depressive symptoms on the Beck Depression Inventory including the covariate of child's age. There was no impact of symptoms on level of unsupportive behaviors. We were unable to include the interaction of BDI scores and risk status, due to low variability in BDI scores for the low risk group (almost all reported zero symptoms), and the cell count was too low for the model to be run.

Hypothesis 1c: Impact of Adolescent's Depressive Symptoms on Unsupportive Parental Behaviors. We examined whether adolescent depressive symptoms predicted unsupportive parental interactions. Depressive symptoms reported by parents and children were run in separate analyses. First, we ran an analysis including child reported depressive symptoms with the covariate of age. Secondly, we computed whether the association between child depressive symptoms was moderated by risk status.

For child reported depressive symptoms (CDI self-report), there was an association between symptoms and level of unsupportive parental behaviors, χ^2 (4) = 19.6, p = .001. Specifically, greater child reported depressive symptoms were associated with being more likely to have parents who showed *low* versus *no* unsupportive behaviors, b = .18, *Wald* χ^2 (1) = 4.32, p = .04. However, there was no difference in the prediction of *high* unsupportive behaviors from either of the other categories. See Figure 14.

Figure 14. Mean CDI Self Total Scores Based on Categories of Unsupportive Parental Behavior



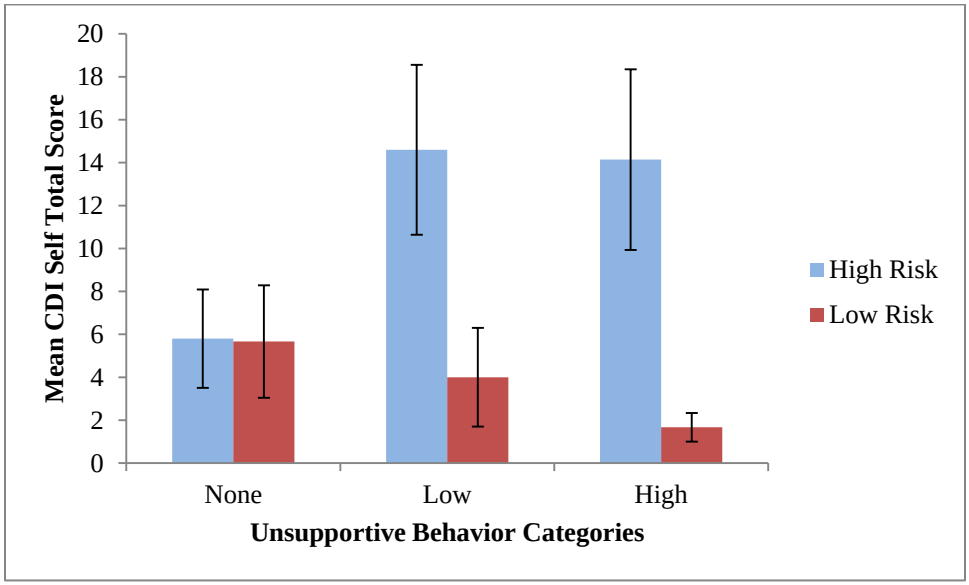
Note: Error bars represent Standard Error of the Mean.

Given that we hypothesized that higher depressive symptoms would predict unsupportive behaviors, and in previous analyses we found a significant association with age and unsupportive behaviors, we conducted an analysis to see if age may influence the association between depressive symptoms and parental behaviors. Indeed, there was a marginally significant effect, in which higher CDI scores and being older predicted a greater likelihood of having parents who showed *high* versus *no* unsupportive behaviors, $b = .60$, *Wald* χ^2 (1) = 2.93, $p = .09$, yet there was no difference between those demonstrating *high* and *low* unsupportive behaviors. Therefore, parents showed the highest levels of unsupportive behaviors when adolescents were older and had more depressive symptoms.

Lastly, we examined whether the impact of depressive symptoms on unsupportive behavior was a function of risk status. We found that this effect was only observed in the high risk parents. Specifically, being in the high risk group and having higher depressive symptoms was associated with being in the *low* unsupportive compared to *none* category, $b = .50$, *Wald* χ^2

(1) = 4.39, p = .04. Yet, there was no effect of depression symptoms among the low risk group. See Figure 15.

Figure 15. Mean Child Depressive Symptoms by Unsupportive Behavior Categories and Risk Status



Note: Error bars reflect Standard Error of the Mean

Using the predictor of parent-reported child depressive symptoms (CDI parent), there was no association between depressive symptoms and parental unsupportive behaviors when controlling for age or risk.

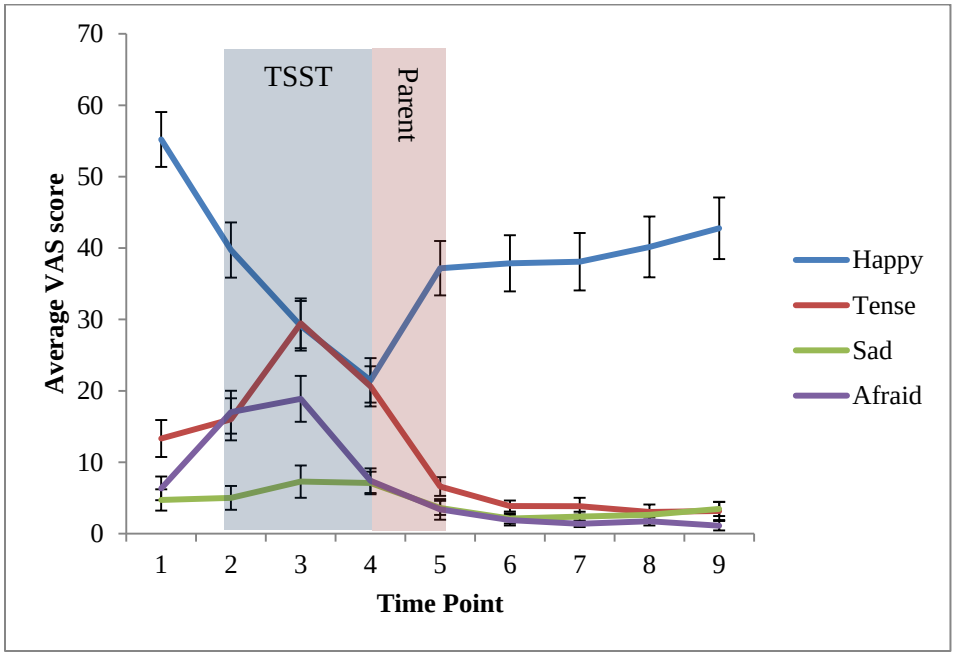
Exploratory Analysis: Impact of Adolescent's Distress on Parental Unsupportive Behaviors. Furthermore to explore this result and determine if parents are unsupportively responding to their child's distress, we ran an ordered logistic regression with adolescent expressed emotion predicting unsupportive behaviors. In a first model including only expressed emotion, there was no association between adolescent's expressed emotion and parental unsupportive behaviors. When including risk and the interaction with adolescent's expressed emotion, there was no interaction or main effect. Overall therefore, the adolescent's expressed emotion to the stress task was not associated with the amount of parental unsupportive behaviors.

Aim 2: Impact of Parent on Adolescent Affect Regulation

For this aim of the dissertation the full sample was utilized since all children had reported their affect during the laboratory visit using the Visual Analog Scale (VAS). See Table 1 in Sample Characteristics section (above) to see group level differences on variables.

First, we examined average levels of affect across the stress task. As evidenced in Figure 16, levels of happiness started generally high (*Time Point 1, M* = 55.2, *SE* = 3.8), then decreased during the stress task (*Time Point 4, M* = 21.5, *SE*= 3.1), and gradually increased after recovery (*Time Point 9, M* = 42.8, *SE*= 4.3). Fear, tenseness, and sadness started lower, increased during the stress task and decreased afterward. Adolescents reported higher levels of happiness (*M* = 40.2, *SD* = 31.0) than tenseness (*M* = 16.4, *SD* = 23.6) right before the TSST, *Time Point 2*, *t* (60) = 4.36, *p* <.001, and right after the parent left, (Happy: *M* = 37.4, *SD* = 30.7; Tense: *M* = 6.8, *SD* =10.5), *Time Point 5, t* (60)= 7.48, *p*<.001, but there was no difference at time 3 or 4. This suggests that mean levels of happiness decrease from the start to the end of the TSST, whereas the opposite was true of tenseness.

Figure 16. Average Levels of Affect Reported Before and After Stress Task



Note: Error bars reflect standard error of the mean. See Table 5 for Description of the Time Points.

Table 5. Description of Time Points for Self-Report Measures of Affect

Time point	Time (min)	Occurrence
1	0	Right after consent
2	40	Right before TSST
3	50	Right after speech portion of TSST
4	55	Right after math portion of TSST (end of task)
5	65	Right after parent
6	75	Regulation
7	85	Regulation
8	95	Regulation
9	105	Regulation

Positive Affect. We used the VAS measure of happiness to capture positive affect over time. Considering that levels of happiness were normally distributed, no transformations were applied to the data.

We ran multiple linear mixed models with predictors of age, gender, condition, and risk on levels of happiness. We constrained our model to only examine the linear slope from time point 4 (at the end of the TSST) to time point 6 (10 minutes after the parent was present) since

we were interested in the change in levels of distress before the parent arrived to after they left. There was a linear increase in happiness from the end of the Trier Social Stress Task to 20 minutes afterward, *Time*, $b = .78$, $t\ (62) = 5.2$, $p < .001$.

We then tested multiple covariates to the model. In the unadjusted model with age, older adolescents had less steep slopes than those who were younger, *Time x Age*, $b = -.23$, $t\ (60) = -1.94$, $p = .06$, indicating that younger children increased positive affect faster. We then included the effect of gender in a separate model, but gender did not influence levels of happiness over time. Thus, we included age as a covariate in all models.

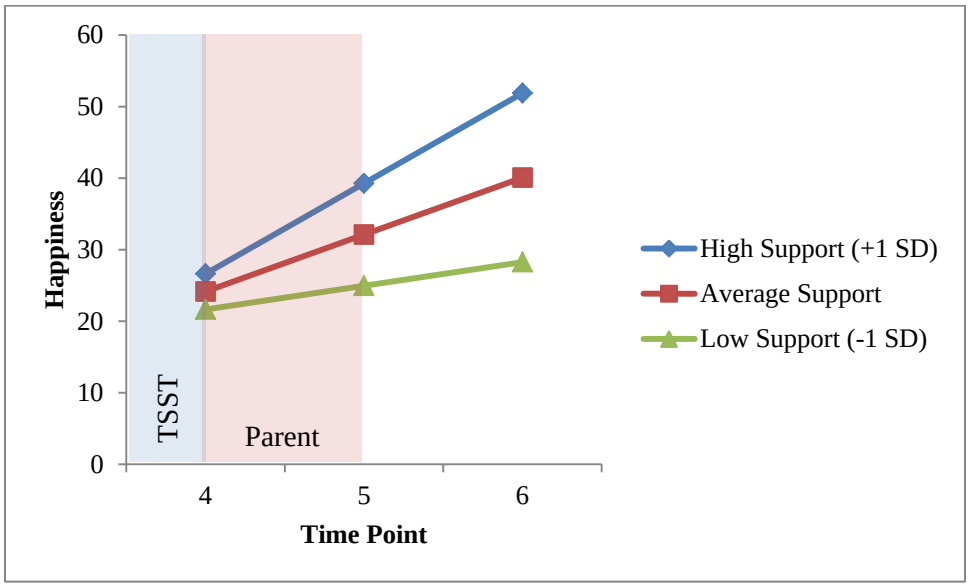
Hypothesis 2a: Group Differences in Happiness. We examined whether adolescents who are at risk for depression experienced lower positive affect after the stress task. There were no significant differences in starting levels of happiness or increases in happiness levels after the stressor between the high and low risk groups, and it was not moderated by age.

Hypothesis 2b: Group Differences in the Impact of Parents on Happiness. For this hypothesis, we first examined whether having a parent present after the stress task influenced happiness levels in adolescents. In the whole sample, there was no difference in the start of happiness or how quickly happiness recovered when parents were present or absent. In the second step of this hypothesis, we examined whether having a parent present after the stress task impacted adolescents at high and low familial risk differently, yet did not find any difference in how parents impacted high and low risk adolescents.

Hypothesis 2c: Impact of Support on Happiness. We examined whether observed parental supportive behaviors (Aim 1) when participants were in the parent present condition would influence self-reported happiness scores. This analysis only included those adolescents who were randomized into the parent present condition (N=29). We found that higher levels of

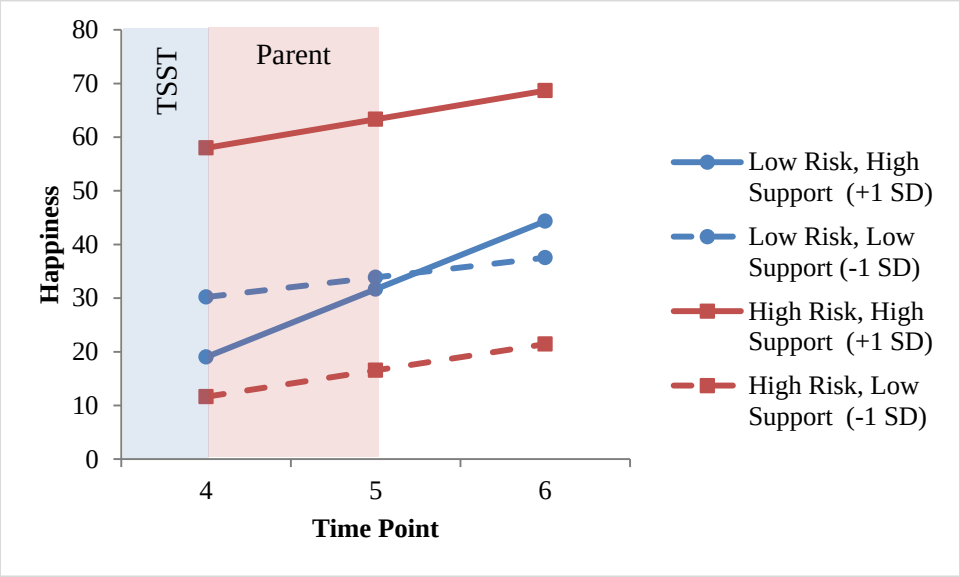
observed parental support was related to faster increases in happiness after the TSST, *Support*, *b* = .06, *t* (27) = 2.06, *p* = .05. See Figure 17.

Figure 17. Influence of Parental Support Behaviors on Happiness after the Stress Task



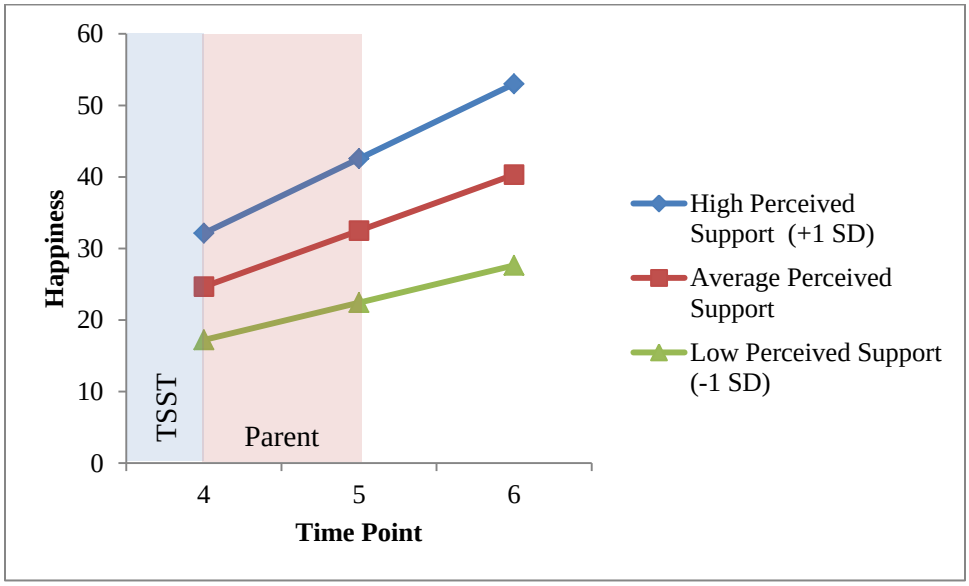
We then included the moderator of risk to see if this association differed between groups. Levels of happiness right after the TSST was higher for adolescents in the high familial risk group who had high levels of support, compared to their high risk counterparts with lower support levels, *Support*, *b* = 2.96, *t* (27) = 2.89, *p* = .008, and compared to those in the low risk group, *Support x Risk*, *b*= 3.68, *t* (27) = 2.97, *p* = .006. Yet, there was no difference between those who received high or low support in the low risk group. This effect disappeared when controlling for happiness experienced during the TSST (time point 3). Thus, it may be that overall levels of happiness, and not just immediately after the stress task, are higher for adolescents at familial risk for depression when parents show higher levels of supportive behaviors. See Figure 18.

Figure 18. Impact of Risk Group and Parental Support on Happiness



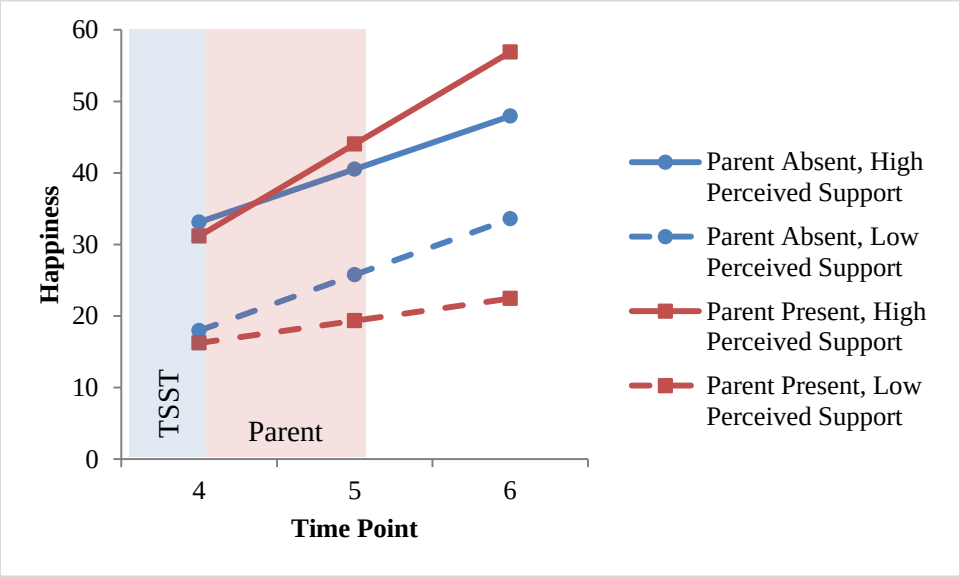
In order to investigate the impact of support further, and increase power by using the full sample, we included the adolescent's self-report of how supported they feel their family is (as measured by the family subscale of Multidimensional Scale of Perceived Social Support) as a predictor of levels of happiness after the TSST. In the first model, we examined whether family support impacted the starting level of happiness and the linear increase in happiness. Those who felt their family was more supportive had higher levels of happiness right after the TSST, *MSPSS Support*, $b = 9.96$, $t\ (60) = 2.49$, $p = .02$, and had moderately faster increases in happiness afterward, *Time x MSPSS Support*, $b = .35$, $t\ (60) = 1.74$, $p = .09$. Thus, this suggests that higher perceived support is associated with better up-regulation of positive affect after a stressor. See Figure 19.

Figure 19. Impact of Perceived Family Support on Levels of Happiness



We then determined whether the effect was different between the parent present and absent condition. As in the previous model, those who had greater support started moderately higher in happiness immediately after the TSST, *MSPSS Support*, $b = 9.97$, $t\,(60) = 1.86$, $p = .06$. There was a moderately significant difference in how having parents present impacted those who perceived high and low support, *Time x MSPSS Support x Condition*, $F(1,60) = 2.94$, $p = .09$. In the parent present condition, those who perceived greater support had faster increases in happiness compared to those who had lower support, *Time x MSPSS Support*, $b = .65$, $t\,(60) = 2.47$, $p = .02$, however there was no difference in slopes between those who had high and low support in the parent absent condition. See Figure 20. Thus, perceiving greater social support was associated with higher levels of happiness and faster up-regulation of happiness when adolescents were able to have their parent present after the stress task.

Figure 20. Effect of Condition and Perceived Family Support on Happiness



We then examined if perceived family support was differentially associated with happiness based on familial risk group, however there were no differences. There additionally was no moderation of risk on the effect of perceived support and parental condition on levels of happiness.

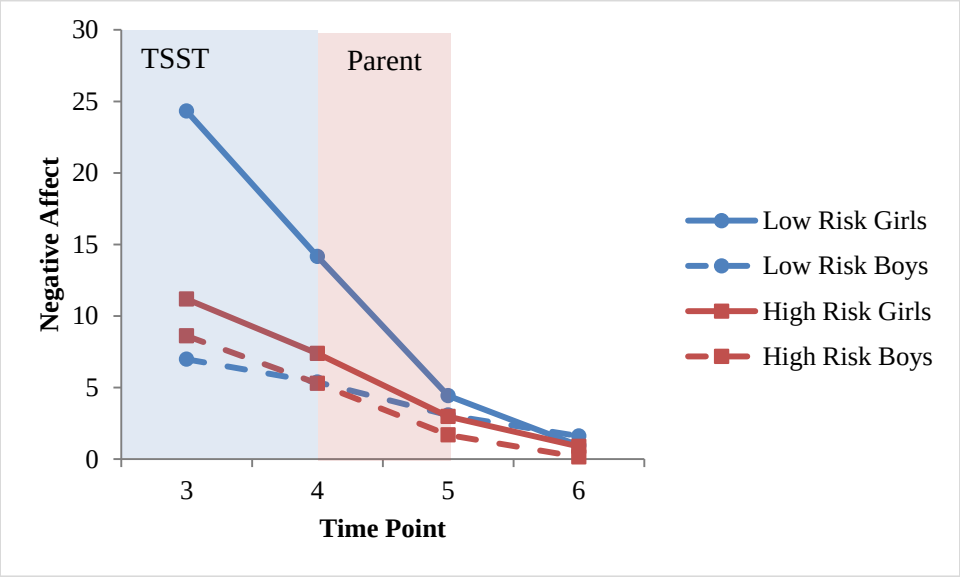
Negative Affect. There were multiple constructs that were conceptualized as negative affect including sadness, fear, and tenseness. We first examined the time course of the three emotions, and found that levels of fear and tenseness peaked on average at time point 3, whereas sadness peaked at time point 4. There also seemed to be low variability in sadness compared to the other emotional ratings. Due to the different time course of peak sadness and that theories suggest that social buffering affects the fear response (Gunnar, Hostinar, Sanchez, Tottenham, & Sullivan, 2015), we decided to examine only fear and tenseness as a report of negative affect. The adolescent's report of fear and tenseness were significant correlated over time, *r*'s ranged from .41 to .47, thus we averaged the two constructs to create a composite of fear and tense, which was termed negative affect. Average levels of negative affect started low, (*Time Point 1*,

M = 9.8, *SE* = 1.8), increased during the stress task, (*Time Point 3, M* = 24.2, *SE* = 3.0) and then decreased (*Time Point 9, M* = 2.1, *SE* = .9).

Since the construct was positively skewed across time, we log transformed the variable. We then ran a mixed model predicting negative affect from time. We set the intercept to be the highest level of negative affect, time point 3, which corresponds to the report immediately after the speech portion of the stress task (during the middle of the TSST). There was a significant linear decrease in negative affect from time 3 to 6, *Time, b* = -.03, *t* (62) = -11.2, *p* <.001. We then included multiple covariates to the data in separate unadjusted models. There was no effect of age, but girls had moderately more negative affect during stress task compared to males, *Gender, b* = 0.24, *t* (122) = 1.75, *p* = .08. Thus, we retained gender effects in the next models.

Hypothesis 2a: Group Differences in Negative Affect. It was hypothesized that adolescents in the high familial risk group would have greater levels of negative affect than those in the low risk group. However, there was no group difference in either initial levels of negative affect or rates of change after the stress task. When including the effect of gender, there was a significant difference in how boys and girls experienced decreases in negative affect dependent upon what familial risk group they were in, *Time x Risk x Gender, F*(1, 122)= 6.23, *p* = .01. Specifically, in the low familial risk group, girls had higher negative affect than boys during the TSST, *Gender, b* = .50, *t* (122) = 2.30, *p* =.02, and decreased faster in negative affect than boys, *Gender x Time, b* = -.03, *t* (122) = -2.66, *p* =.01. However in the high risk group there were no gender differences in the start or slope of negative affect. Girls in the high risk group had a moderately lower starting point, *Risk, b* = -.32, *t* (122) = -1.71, *p* =.09, than girls in the low risk group. Thus, it seems that girls in the low risk group started higher and decreased at a faster rate compared to all other groups. See Figure 21.

Figure 21. Impact of Group and Gender on Recovery of Negative Affect After the TSST



Hypothesis 2b: Group Differences in the Impact of Parent on Negative Affect. In order to determine if there were group differences in how parents impacted the slopes of negative affect, we first examined the overall effect of the parent condition. There were no differences in those in the parent present or absent condition.

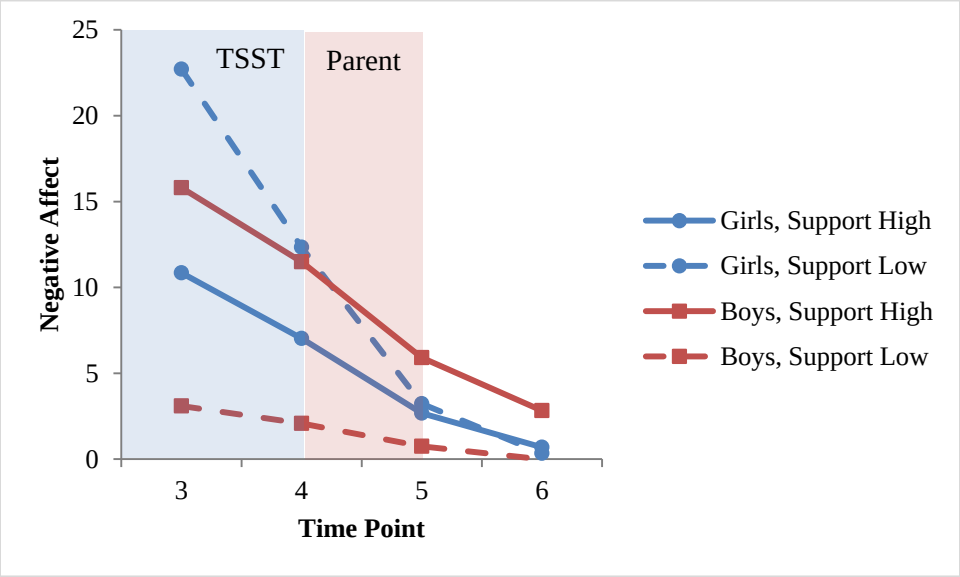
We then examined the effect of familial risk on the association between parent presence and negative affect. There was no significant difference between familial risk groups in how they experienced negative affect after the stressor. Given the significant effect of gender in the previous models, there may be gender differences in the association between risk status and parental condition on negative affect. However, the cell sizes were too small to conduct a model with a three-way interaction (see Table 6), thus, we can only speculate that there may have been a gender difference in this association.

Table 6. Cross Tabulation for Group, Condition, and Gender

		Group	
Gender	**Condition**	High Risk	Low Risk
Girls	Parent Absent	14	5
	Parent Present	11	6
Boys	Parent Absent	9	4
	Parent Present	7	7

Hypothesis 2c: Impact of Support on Negative Affect. We first tested whether supportive parental behaviors as observed in the lab (Aim 1) were related to negative affect regulation. We conducted a mixed model with supportive behaviors and their association with time as predictors of negative affect. We did not find an overall effect of supportive behaviors on either the intercept or slope of negative affect. We then examined whether any support effect differed by gender. Indeed, support impacted the negative affect ratings during the stress task differently for boys and girls, *Support x Gender, F* (1, 55) = 7.87, $p = .007$. At average levels of supportive behaviors, girls started higher in negative affect, *Gender, b* = .31, *t* (55) = 1.9, $p = .06$, and decreased faster than boys, *Time x Gender, b* = -.02, *t* (55) = -2.01, $p = .05$. For boys, higher supportive behaviors was related to higher levels of negative affect to begin with, *Support, b* = .04, *t* (55) = 2.96, $p = .005$. Yet, for girls, there was no impact of support on the intercept or slopes. Thus, boys who started higher in negative affect had parents who provided more support, and this was different from females. See Figure 22.

Figure 22. Impact of Gender and Parental Supportive Behaviors on Negative Affect



Next we examined whether the risk groups differed in the association between supportive behaviors and negative affect, but did not find an overall group effect. Due to lower sample size, we were unable to see if there was an interaction between gender and risk group on levels of support and negative affect.

For consistency, we examined the impact of the adolescent's perceived family support (MSPSS) on the regulation of negative affect. This analysis included all participants since they all reported their perceived support. Higher levels of perceived family support was associated with reporting more negative affect during the stress task, *MSPSS Support*, $b = .17$, $t\ (122) = 1.86$, $p = .07$, but did not affect regulation of affect. The effect of perceived family support was not different based on age, gender, risk group, parent condition, or any of these interactions.

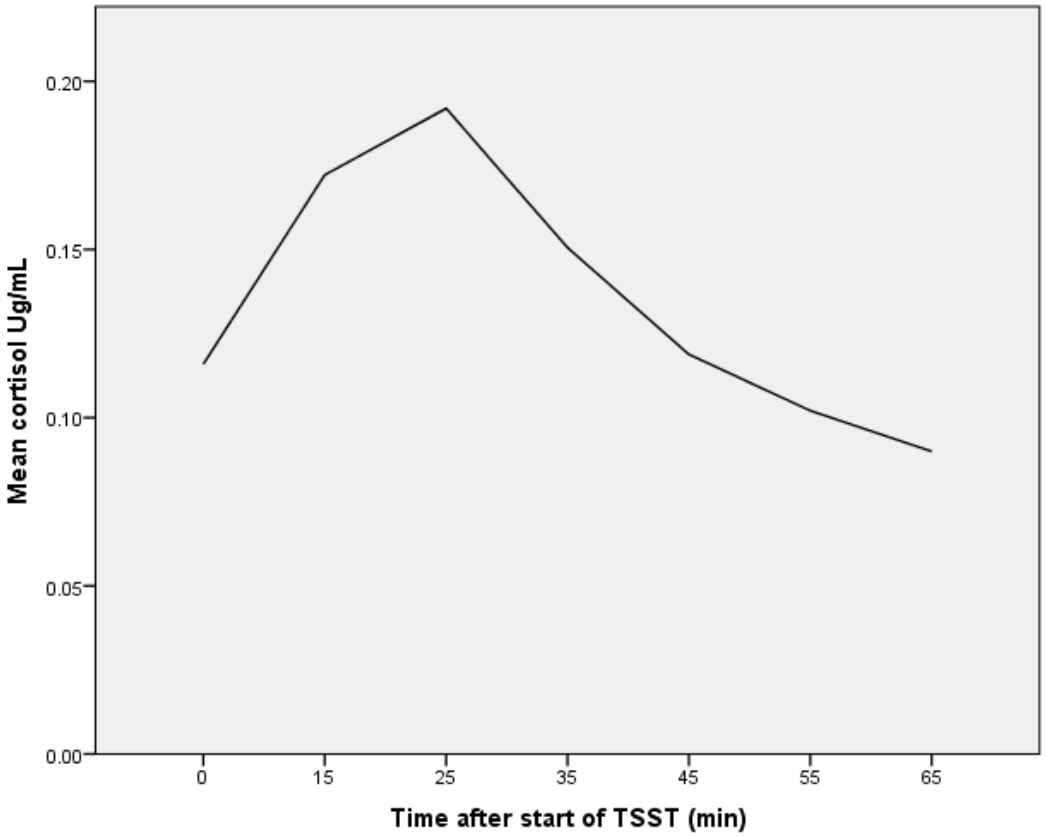
Aim 3. Impact of Parent on Adolescent's HPA-Axis Reactivity and Recovery

For this aim of the dissertation, a total of 56 children had cortisol data that was assayed. Two children were excluded from the analyses; one high risk girl had cortisol values that were extreme (Z-scores between 1.6 and 13.8), and one high risk boy was ill with pneumonia and did not complete multiple cortisol samples. Thus, there were 36 adolescents at high familial risk and 18 at low familial risk in this analysis.

The Trier Social Stress Test was successful in activating the HPA-axis in 80% of the sample. Participants were identified as responders to the task if they exhibited a 20% increase in cortisol from baseline to peak levels. Due to the high success rate of the task, we included all participants with cortisol data in these analyses.

Multiple linear mixed models were conducted. We first modeled the cortisol response (activation slope, peak, recovery slope) without any additional predictors. In the linear model (AIC = 118.4) there was an increase in cortisol towards the peak, *Activation Slope*, $b = .01$, $t(317) = 5.20$, $p < .001$, and a decrease in cortisol after peak, *Recovery Slope*, $b = -.01$, $t(317) = -7.87$, $p < .001$. Thus, we see the typical activation of a rise and fall in cortisol over the course of the task. See Figure 23.

Figure 23. Mean Cortisol Response for the Trier Social Stress Task

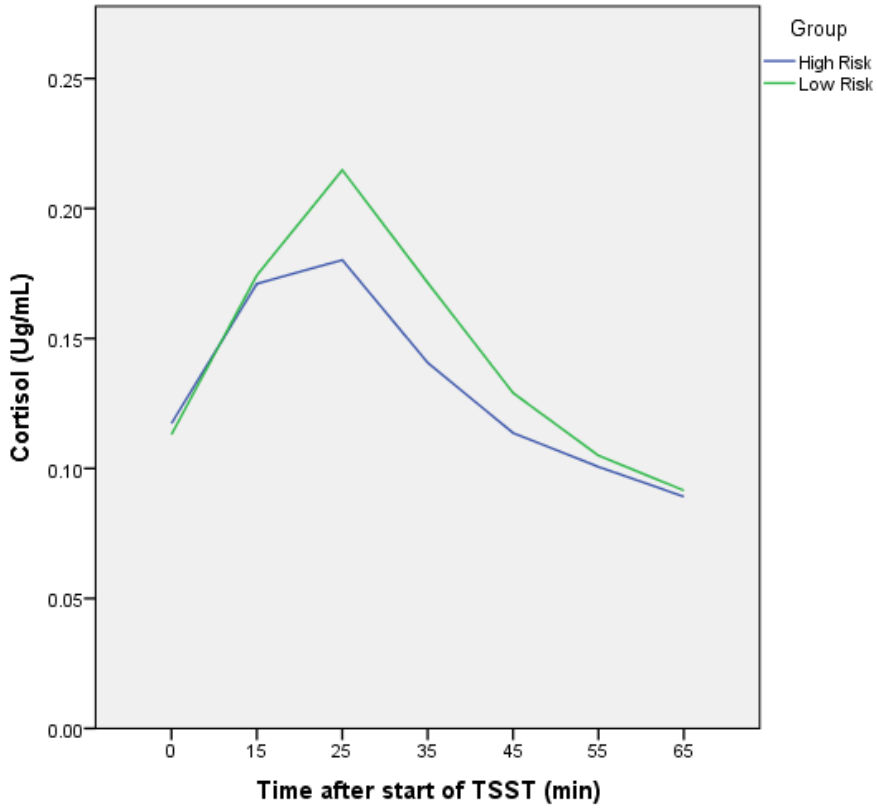


We then conducted unadjusted models to examine the effects of age, gender (Ji, Negriff, Kim, & Susman, 2016), pubertal status, phase of menstrual cycle (Bouma, Riese, Ormel, Verhulst, & Oldehinkel, 2009; Kudielka & Kirschbaum, 2005), time since waking up in the morning, and eating prior to the task (Gonzalez-Bono, Rohleder, Hellhammer, Salvador, & Kirschbaum, 2002). There was no effect of age, gender, puberty or eating prior to the task. As expected (Bouma et al., 2009; Kudielka & Kirschbaum, 2005), when girls were in the follicular phase of their menstrual cycle or were on birth control, there was a lower peak activation of cortisol, *Menstrual Phase x Peak, b* = -.16, $F(1, 51) = 10.05$, $p = .002$, and a moderately significant flatter activation slope, *Menstrual Phase x Activation Slope, b* = -.007, $F(1, 315) = 2.8$, $p = .095$, compared to boys and to girls in the luteal phase. Considering that boys were also

included in this analysis and coded as if they were in the luteal phase, we conducted this analysis again without boys included, and found the same effect on peak, *Menstrual Phase x Peak, b* = -.22, *F* (1, 26) = 10.8, *p* =.003, although not on the activation slope. Therefore, we retained the effect of menstrual phase on the peak values. There was also a significant impact of time since awakening on peak values, *Time Since Awakening x Peak, b* = -.00001, *F* (1, 51) = 12.5, *p*<.001, and a moderately significant impact on activation slope, *Time Since Awakening x Activation Slope, b* = -6.1 x10^{7}, *F* (1,315) = 3.14, *p* = .08. Thus, we retained both the impact of menstrual phase and time since awakening on peak cortisol levels in all subsequent analyses.

Hypothesis 3a: Effect of Risk Group on Cortisol Slopes. Given that previous studies have examined baseline cortisol differences in high and low familial risk groups (Guerry & Hastings, 2011), we first ran a t-test to determine whether there were any differences in cortisol averaged across the first two samples (i.e., right when they came in and before the TSST). There were no group differences in baseline cortisol levels, *t* (52) = .07, *p* = .96. We then conducted growth curve models with risk status predicting cortisol values, while controlling for baseline, menstrual phase, and time since awakening. There was a significant association between group status and peak levels of cortisol, *Risk x Peak, F* (1, 49) = 9.12, *p* = .004. In particular, the high risk group had lower peak levels of cortisol compared to the low risk group, *Risk x Peak, b* = -.14, *t* (49) = -3.02, *p* = .004. See Figure 24. Additionally, we re-ran the model with only those in the control (parent absent) condition in order to verify that this effect occurred without the effect of parent. There continued to be a significant effect, *Risk x Peak, b* = -.14, *t* (21) = -2.05, *p* = .05, thus, there was lower peak cortisol for adolescents in the high familial risk group across conditions.

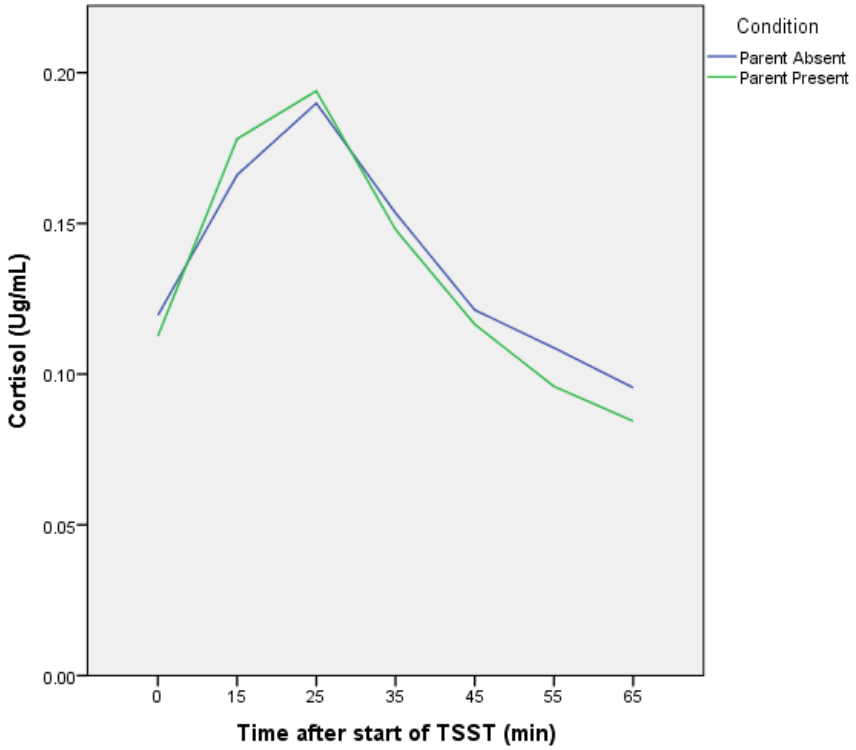
Figure 24. Group Differences in Cortisol



Hypothesis 3b. Group Differences in the Impact of Presence of a Parent on Cortisol.

It was expected that overall, parents would have a buffering effect on cortisol curves (e.g., lower neuroendocrine response). In order to test this hypothesis, we conducted a model with condition (parent present or absent) predicting peak cortisol values and their activation and recovery slopes, while controlling for menstrual cycle, time since awakening, and baseline levels of cortisol. Results of the model indicated that there was no association between parent condition and cortisol response. See Figure 25.

Figure 25. Cortisol Curves in Parent Present and Absent Conditions



In order to determine whether the effect of parent differed based on whether a child had a parent with a history of depression, we included familial risk status and the interaction with parent condition as predictors of the cortisol response. There were no significant differences between risk groups on how the parent condition affected cortisol response, *Risk x Condition x Peak*, F (1, 47) = .12, p = .74, *Risk x Condition x Recovery Slope*, F (1, 311) = .07, p = .80.

Hypothesis 3c: Impact of Age and Parent Presence on Cortisol. Given that a previous study found an effect of age and puberty on the parental buffering of the cortisol response (Doom et al., 2015), we included the effects of age centered at the youngest adolescents in our sample (12 years of age) and condition in the next model. There was a significant difference in how parental condition impacted younger and older adolescent's peak cortisol response, *Condition x Age x Peak*, F (1, 47) = 14.1, p = .0005. At age 12, being in the parent present condition was associated with *higher* peak cortisol response, *Condition*, b = .27, t (47) = 3.24, p = .002,

whereas at age 16, it was associated with *lower* peak response compared to the parent absent (control) group, *Condition b* = -.29, *t* (47) = -3.19, *p* = .003. Thus, we see the parental buffering effect at age 16, but not at age 12. See Figures 26 and 27.

Figure 26. Impact of Age and Condition on Cortisol Response

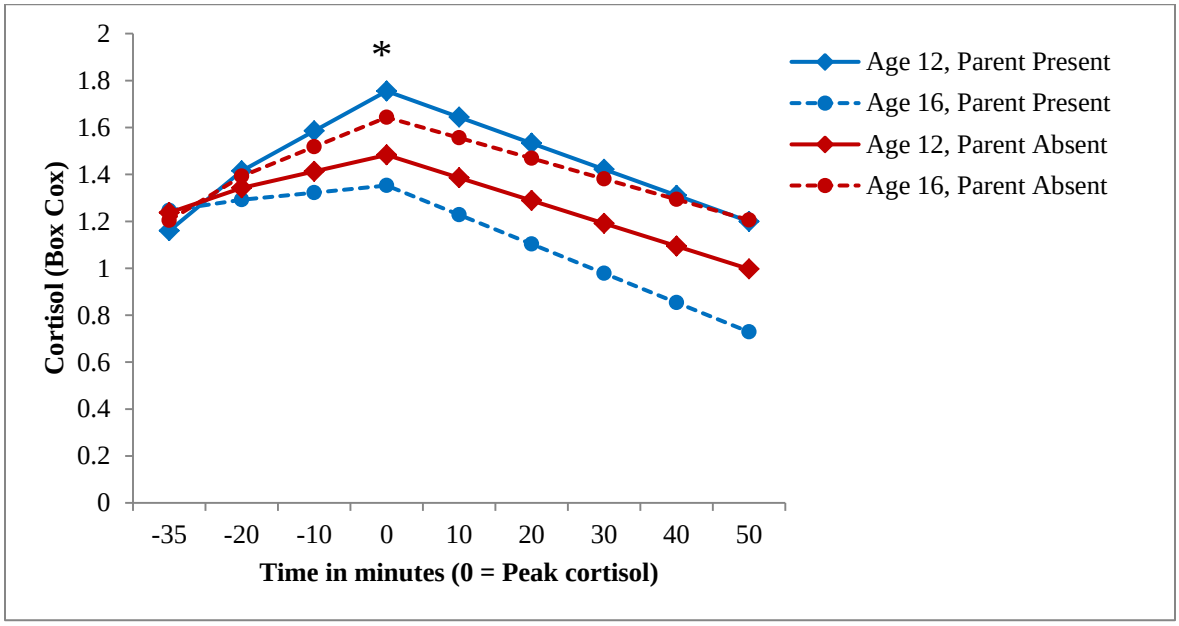
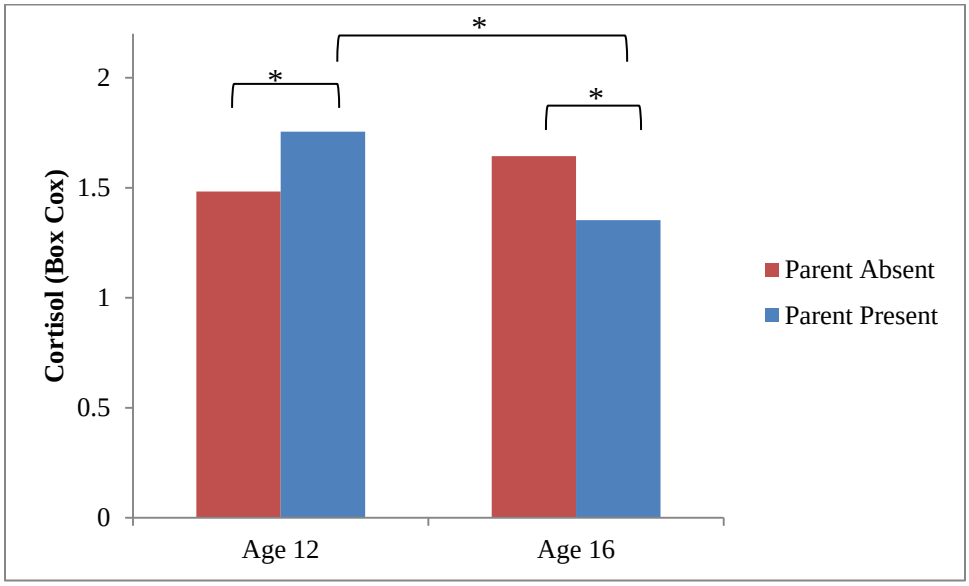


Figure 27. Impact of Age and Parental Condition on Peak Responses of Cortisol



We then examined whether pubertal status moderated the association between parent condition and cortisol response. There was no significant interaction of puberty and parent condition, nor any main effects of puberty in the model. This suggests that the parental buffering of the peak cortisol response was associated with age and not pubertal status.

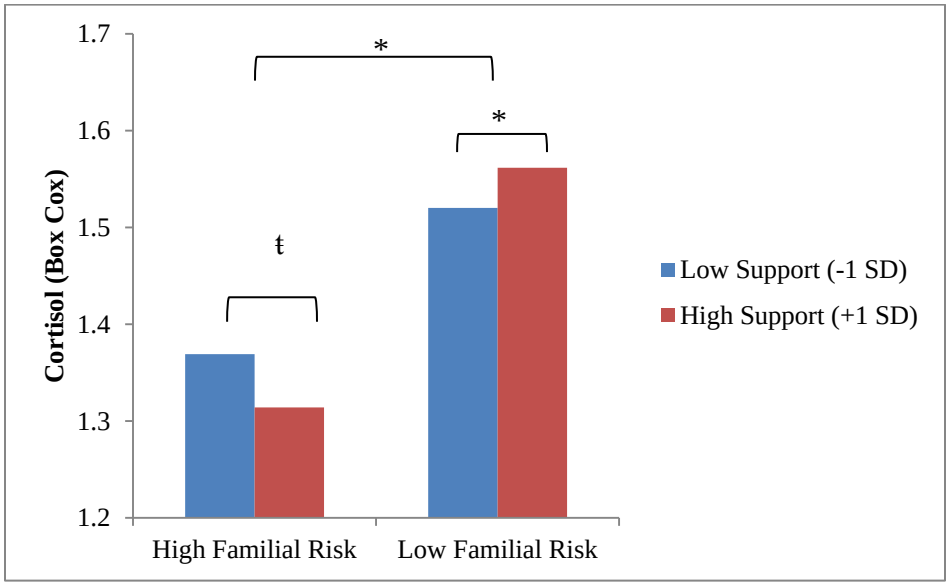
Hypothesis 3d: Impact of Observed Parental Supportive Behaviors on Cortisol Response. For the last hypothesis, we predicted that higher levels of supportive behaviors in both the low and high familial risk group would be related to greater social buffering of the cortisol response. In order to test this, we used the observed parental behaviors obtained in Aim 1 to determine if supportive behaviors moderated the association between group status and recovery slopes. Considering that only half the sample had their parent present, these analyses included 15 high familial risk and 12 low risk participants. The model predicted cortisol values and slopes from supportive behaviors, while controlling for baseline cortisol, menstrual cycle, and time since awakening. Overall, there was an effect of supportive behaviors on peak response of cortisol, *Support x Peak, F* (1, 21) = 6.62, $p = .02$. Specifically, greater levels of supportive behaviors by parents was associated with higher cortisol peak response, *Support x Peak, b* = .01, *t* (21) = 2.57, $p = .02$.

Since there was a significant effect of age in the previous hypothesis, we included the effect of age in the model with parental supportive behaviors. There was no interaction between support and age, yet there was a significant age effect on peak response, with older adolescents having lower peak cortisol, *Age x Peak, b* = -.07, *t* (20) = -2.35, $p = .03$, which was consistent with the previous hypothesis. However, the effect of support was diminished as to no longer be significant on peak levels while controlling for age, *Support x Peak, b* = .009, *t* (20) = 1.32, *p* =

.20. Thus, age may account for some variance in supportive behaviors, and we controlled for this in all additional analyses.

We then examined whether risk status moderated the effect of supportive behaviors on peak values. There was a significant interaction of support and risk on peak values, F (1, 19) = 9.34, p = .007. In the low familial risk group, higher supportive behaviors were associated with higher peak cortisol levels, *Support x Peak*, b = .02, t (19) = 2.56, p = .02, whereas in the high familial risk group, higher support was associated with moderately lower peak cortisol, *Support x Peak*, b = -.02, t (19) = -2.03, p = .06. Thus, parental support was associated with a greater response in low risk adolescents, but a lower response in high risk adolescents. See Figure 28.

Figure 28. Peak Cortisol Response in Low and High Risk Groups with Lower and Higher Supportive Behaviors from Parents

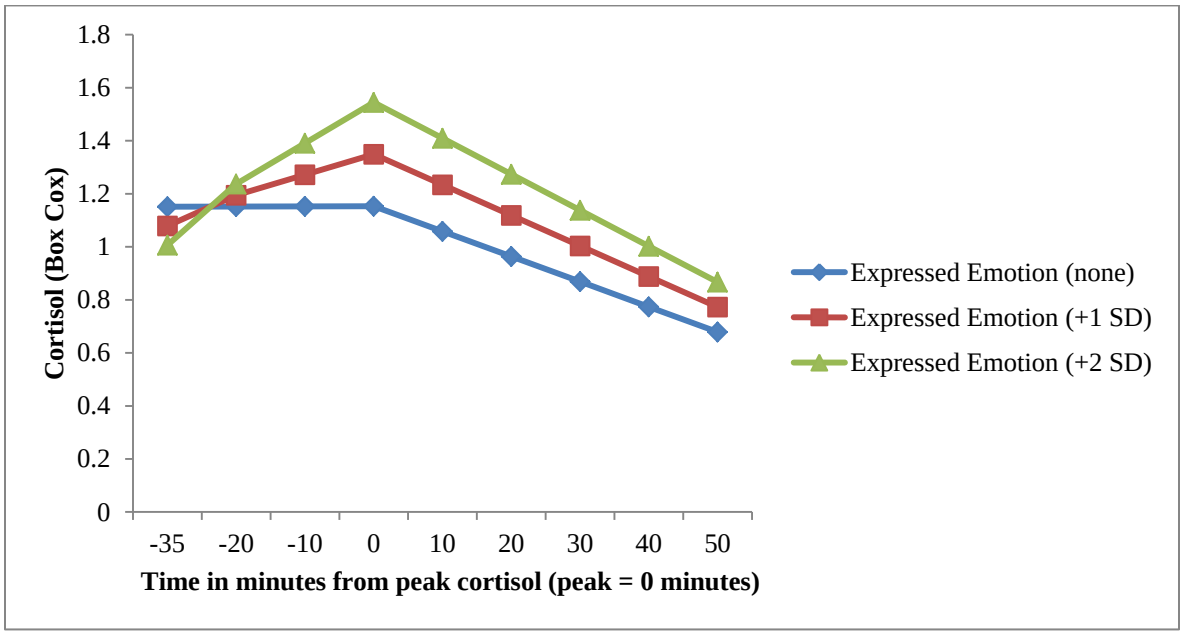


* p<.05, ŧ p<.10.

Exploratory Analysis: Impact of Adolescent's Distress on the Association between Parental Support and Cortisol. We were curious as to whether results of the support of parents may be driven by the child's own distress levels. Level of distress was defined by adolescent's expression of emotions about the stress task or bringing up the stress task with parents (as coded

for in Aim 1). In models examining expressed emotions alone, higher emotional distress was associated with a faster activation slope, *Expressed Emotion x Activation Slope*, $b = .002$, t (149) = 2.96, $p = .004$, and greater peak responses of cortisol, *Expressed Emotion x Peak*, $b = .05$, t (21) = 6.07, $p < .0001$. Thus, children with more distress had a stronger and faster cortisol response than those with lower levels of distress. See Figure 29.

Figure 29. Impact of Expressed Emotion on Cortisol Curve



Next, we included the effect of both parental support and adolescent's expressed emotions in the model of cortisol activation, peak, and recovery while controlling for age, menstrual cycle and time since awakening. We did not examine the interaction between support and expressed emotion since we were interested in whether the effect of parental support was mediated by expressed emotions. We found that there remained a significant effect of expressed emotion on peak response of cortisol, F (1, 20) = 31.17, $p < .0001$, and on activation slope, F (1, 152) = 7.13, $p = .01$, however the effect of support on peak response was non-significant. This indicates that the variance attributable to support on peak response was due to adolescent's own expressed emotion.

Exploratory Analysis: Impact of Anxiety Symptoms on Cortisol. Considering that expression of distress was highly related to cortisol response, it may be that the amount of anxiety adolescents are experiencing may account for this association. Thus, we first examined the impact of the adolescent's overall anxiety symptom level on cortisol response and found that adolescents with higher levels of anxiety had *lower* peak cortisol compared to those with lower anxiety, *SCARED x Peak, b* = -.004, *t* (51) = 3.02, *p* = .004. While this was in the opposite direction expected, those who were high risk had higher scores on the SCARED compared to those at low familial risk (see table 1 above), which is consistent with high risk participants showing lower levels of cortisol overall. However, there was no interaction between levels of anxiety and risk status on cortisol peaks, *SCARED x Risk x Peak, F* (1, 47) = .06, *p* = .81. Anxiety symptoms did not influence the impact of parental condition (present or absent) on cortisol values, *SCARED x Parent Condition x Peak, F* (1, 47) = 0.0, *p* = .98.

We then examined whether level of anxiety symptoms would impact the association between support and cortisol peak. Indeed, anxiety symptoms had an effect, *SCARED x Support x Peak, F* (1, 20) = 4.46, *p* = .05, specifically that the higher the anxiety symptoms, the greater the cortisol peak response when support was highest, *SCARED x Support x Peak, b* = .0007, *t* (20) = 2.11, *p* = .05. Furthermore, anxiety levels influenced the association between expressed distress and cortisol responses in both the activation slope, *SCARED x Expressed Emotion x Activation Slope, F* (1, 150) = 3.27, *p* = .07, and peak levels, *SCARED x Expressed Emotion x Peak, F* (1, 20) = 5.54, *p* = .03. Specifically, the higher the levels of anxiety and expressed emotion, there was a trend for a faster activation slope, *b* = .0001, *t* (150) = 1.81, *p* = .07, and for higher cortisol peak response, *b* = .002, *t* (20) = 2.35, *p* = .03. Together this suggests that higher anxiety levels overall were associated with lower peak cortisol, but when higher anxiety levels

were coupled with greater expressed emotion and supportive parental behaviors, peak cortisol responses were higher.

Chapter 4: Discussion

This dissertation was a preliminary investigation of how parents with and without a history of depression impacted adolescent stress regulation to a laboratory stress task. We found that parents provided greater support to their adolescent when adolescents experienced greater distress. This support in turn was related to greater up-regulation of positive affect after the stressor, indicating that parental support aided in emotional stress regulation. Parents who did not have a history of depression gave more supportive behaviors during the parent-child interaction when adolescents had higher depressive symptoms and greater peak cortisol. This may demonstrate that these parents are particularly sensitive and responsive to distress, which may be protective in these families and mitigate future depression risk in their adolescent. However, there was no modulation of behaviors in parents with a history of depression based on their adolescent's distress, and in fact, parents showed higher unsupportive behaviors when adolescents had greater depressive symptoms. This may be detrimental to the adolescent's ability to regulate their distress. Yet, when parents with a history of depression provided higher support, adolescents demonstrated faster up-regulation of positive affect and had lower peak cortisol responses. Thus, while on average, parents with a history of depression did not demonstrate as many supportive emotion socialization behaviors, when they did, it aided adolescents' stress regulation. As parents were able to influence their adolescent's stress regulation, it would be important to develop treatments to aid parents with a history of depression in increasing responsive support to their children in times of distress.

Aim 1: Observed Parental Behaviors to Adolescent's Distress after a Stress Task

The overall objective for this aim was to examine group differences in the frequency of parental supportive and unsupportive behaviors when parents were with their child after the adolescent experienced the Trier Social Stress Test. Parent provided many instances of supportive (e.g., comforting, validating feelings, problem solving about emotions), and relatively few unsupportive (e.g., punishing, magnifying, dismissing) emotion socialization behaviors. Parents with a history of depression provided less support to their children, although this may have been due in part to these adolescents expressing less distress to their parents compared to families without depression history. Adolescent depressive symptoms were associated with more supportive parental behaviors in adolescents at low familial risk, yet, were related to more unsupportive parental behaviors in those at high familial risk. Overall, there were group differences in parental supportive and unsupportive emotion socialization behaviors, yet the association differed based upon age and child depressive symptoms.

In response to the laboratory stress task, adolescents expressed emotional distress to their parents, and parents generally responded with supportive emotion socialization strategies and relatively few unsupportive behaviors. While not much has been investigated regarding emotion socialization in adolescence (Klimes-Dougan et al., 2007), this study demonstrates that adolescents continue to use their parents in times of distress and that parents continue their role as a support system. Furthermore, it confirms that parents show greater supportive than unsupportive responses to adolescents' distress (Miller-Slough & Dunsmore, 2016). However, in our sample, the older the adolescent was, the greater the amount of unsupportive behaviors parents showed. Consistent with this, parents of older adolescents have been shown to be less supportive and more punitive toward emotional displays based on both child- and parent-report

of behaviors (Klimes-Dougan et al., 2007). While this extends the finding to observational methodology, the result must be tempered by the fact that there were relatively few unsupportive behaviors given to adolescents (range: 0 – 4 instances). Together, there is a general indication that parents are supportive to youth in times of distress, that there are low levels of unsupportive behaviors, yet there was an association between being older and receiving more unsupportive behaviors.

For hypothesis 1a, it was predicted that parents with a history of depression would demonstrate fewer supportive parental behaviors after the adolescent completed the TSST compared to parents without a history of depression. The analyses supported the conclusion at the trend level. Consistent with a meta-analysis of observed parent-child interactions (Lovejoy et al., 2000), depressed parents had fewer positive parental interactions compared to healthy parents, yet, the effect size was small. In terms of emotion socialization, parents with a history of depression report less positive socialization to young children (Silk et al., 2011), and when adolescents are depressed, parents provided fewer positive emotion socialization strategies compared to healthy adolescents (Schwartz, Sheeber, Dudgeon, & Allen, 2012). The results here show a very similar trend of fewer displays of supportive emotion socialization strategies in the high risk group, and is one of the first studies to demonstrate this in an adolescent sample.

It was also expected that parents with a history of depression would demonstrate greater unsupportive emotion socialization. This hypothesis was not supported by the data. While mean levels of unsupportive behaviors were slightly higher for parents with a history of depression compared to non-depressed parents, there was no significant difference between groups. Previous studies suggest that parents with a history of depression are more likely to show negative and disengaged behaviors to their children (Lovejoy et al., 2000), report that they are

more likely to respond to their child's negative emotions with a punishing, magnifying, or neglectful response (Silk et al., 2011), and have a lack of responsiveness to their child's displays of negative affect (Shaw et al., 2006). Yet, there is also data to suggest that parents with a history of depression demonstrate more negativity when they have longer histories of depression or concurrent depressive symptoms (Ewell Foster et al., 2008), yet our sample had relatively low levels of concurrent depression. Furthermore, in our study, the low variability in parental unsupportive behaviors may have decreased our ability to detect any differences seen between groups. Thus, there is some evidence for slight impairment in emotion socialization with parents who have a history of depression, but it may be driven by a lack of supportive interactions rather than greater unsupportive interactions.

For hypothesis 1b, it was predicted that higher parental depressive symptoms would be associated with fewer parental supportive behaviors and more parental unsupportive behaviors. This association was not found in the current data. This was surprising, since higher depressive symptoms predicted lower maternal positivity (Dietz et al., 2008), higher criticism (Tarullo, DeMulder, Martinez, & Radke-Yarrow, 1994), less positive affect and greater negative affect during problem solving tasks (Ewell Foster et al., 2008). Yet, the parents in our sample had relatively low levels of concurrent depression. For example, only one parent with a history of depression met the clinical cutoff for probable depression based off of the Beck Depression Inventory. Despite having relatively depression-free parents who were in our high risk group, they continued to provide less supportive behaviors, as noted in the previous hypothesis. Indeed, depressed parents who were in remission continued to show less emotional availability to their school aged children in a parent-child interaction (Kluczniok et al., 2016), and were less responsive to their preschooler's level of distress (Shaw et al., 2006). Together this suggests that

the lack of supportive parental behaviors shown in the high risk group may be more longstanding deficits rather than based solely on depressive symptoms in the moment.

For hypothesis 1c, it was expected that adolescent's depressive symptoms would be associated with lower supportive parental behaviors and higher unsupportive parental behaviors. The opposite effect was found with supportive behaviors, but only among the low risk participants. When parents in the low familial risk group perceived higher levels of depressive symptoms in their children, they provided more supportive emotion socialization behaviors, yet this was not found in high risk dyads. Further analyses indicated that this was independent of the adolescent's expressed distress and level of anxiety symptoms. This may mean that parents in the low risk group changed their interactions with their children based on their perception of their adolescent's overall depressive symptoms rather than the distress adolescents showed in the moment. This may be a protective factor within this group, as more positive maternal engagement and support was associated with lower depressive symptoms longitudinally (Olino et al., 2016; Stice et al., 2004). Yet, when examining cross sectional studies, parents gave fewer supportive behaviors when adolescents meet clinical cutoff for depression (Lougheed et al., 2015; Pineda, Cole, & Bruce, 2007), which may indicate a more negative parent-child interaction style when children have higher depressive symptoms. However, our low risk adolescents did not meet clinical cutoff for depression, thus, these parents may be responding with greater support to milder symptom levels. Future investigations should examine whether the level of supportive behaviors may be different for those who only have mild symptoms versus clinically significant symptoms of depression. Additionally, we did not find the same modulation of parental supportive behaviors in those parents with a history of depression. Given the differences in parenting between those with and without a history of depression (Lovejoy et al., 2000), there

may be different strategies in response to their child's distress. Thus, in a family in which there is a relative lack of depression, when parents noticed depressive symptoms in their adolescent, they provided more support, which may protect against future depression. Yet, in a high risk family, parents did not increase (or decrease) the amount of support, and this lack of responsiveness may be one reason for the high risk adolescent's greater sensitivity to stress.

Adolescent reported depressive symptoms were associated with unsupportive parental interactions in different ways based on risk status. Specifically, in the high familial risk group, having greater depressive symptoms was associated with parents showing low levels of unsupportive behaviors, yet there was no association in the low risk group. Indeed, adolescents reported higher depressive symptoms when they have parents who are more negative in parent-child interactions (Olino et al., 2016; Sanders et al., 2015; Yap, Schwartz, Byrne, Simmons, & Allen, 2010), which may be a function of long standing parent-child interactions that may be more negative. This may be particularly problematic in this high familial risk group as they receive both lower levels of supportive behaviors overall, and more unsupportive behaviors when they have greater depressive symptoms. As unsupportive behaviors are related longitudinally to greater depressive symptoms (Schwartz, Dudgeon, et al., 2012), this may put these adolescents at even more risk for having a depressive episode.

We additionally conducted exploratory analyses to determine how the frequency of the adolescent's expressed distress during the parent-child interaction impacted parental supportive and unsupportive behaviors. Adolescent's distress was measured by how frequently the adolescent verbally brought up the task or their emotional reaction to the task. When distress was higher, parents responded with greater levels of supportive behaviors, yet distress was unrelated to unsupportive behaviors. This was not moderated by group; thus, parents with and

without a history of depression were relatively equivalent in providing support in the context of greater adolescent distress. In contrast, parents with a history of depression provided lower support validation during a conflict task (McMakin et al., 2011), and lower sensitivity during a free play and puzzle task (Kluczniok et al., 2016). One reason for this difference may be the context; here, parents were actively recruited to help their children through a moment of distress rather than being involved in the distressing event itself (such as discussing a topic of conflict). Thus, parents with a history of depression provided support when their child verbally expressed needing support after the adolescent experienced an outside stressor. However, adolescents at risk for depression overall did not display as much distress as their low risk peers. Thus, our findings regarding high risk parents showing less supportive behaviors may be an interaction between the parent and child; the high risk adolescent did not show as much distress, their parent responded with support, yet, overall levels of support were lower since the adolescent's support seeking was lower. We may be capturing long standing interaction patterns, in which these adolescents do not display as much distress because they do not feel as supported by their parent. Indeed, in our study, high risk adolescents reported feeling less parental support compared to low risk adolescents. Together this suggests that adolescents at high familial risk may not be using their parents for interpersonal emotion regulation, despite parents providing support.

Overall, the results of Aim 1 indicate that parents with a history of depression may provide less supportive behaviors to their children, yet this may be a function of how adolescents seek and perceive the support. That is, adolescents in the high familial risk group did not perceive their parents to be as supportive and did not verbally express as much distress to their parent as low risk adolescents, yet parents with and without a history of depression provided support in a similar manner based on level of distress. However, parents provided more

unsupportive responses in the high familial risk group when adolescents had greater depressive symptoms. Additionally, parents in the low risk group provide more supportive responses to their children who have more depressive symptoms, which may be a protective factor. This provides some evidence that parents with a history of depression may provide less supportive emotion socialization and that both parental factors (e.g., history of depression) and child factors (e.g., depressive symptoms, level of distress) may account for the difference in supportive behavior levels.

Aim 2: Impact of Parent on Adolescent Affect Regulation

We examined adolescents' subjective emotional experiences after a stress task and how these experiences changed based on whether the adolescent did or did not have their parent with them. Overall, adolescents responded to the stress task with lower positive affect and greater negative affect, and quickly regulated these emotions after the task was over. While the mere presence of a parent did not affect how adolescents recovered emotionally from the stress task, the amount of support that parents gave their adolescent significantly influenced positive and negative affect. There was quicker up-regulation of positive affect when adolescents perceived and received greater support from parents. Furthermore, boys who had higher negative affect to the stressor had parents who provided the greatest support. Overall, the starting point and regulation of happiness was related to levels of support that adolescents perceived and received, whereas results for negative affect were influenced by gender differences.

As expected, the Trier Social Stress Test decreased happiness and increased levels of negative affect, and adolescents regulated these emotions afterward. In general, younger adolescents had faster increases in happiness during regulation compared to older adolescents. This is mirrored by results from momentary daily diaries, in which younger adolescents report more positive than negative affect compared to those who are older (Larson, Moneta, Richards, & Wilson, 2002). Furthermore, older adolescents were more likely to have stronger stress reactions to the TSST (Stroud et al., 2009), thus, it may be that older adolescents were more reactive, and showed lower levels of happiness after the stressor ceased.

There was also a significant difference between boys and girls in negative affect, especially in the low risk group. Specifically, girls in the low risk group had higher initial negative affect during the TSST and a steeper declining slope throughout regulation compared to

boys and those in the high risk group. This is in contrast to what was expected; both siblings of those with depression (Kovacs et al., 2015) and girls who have parents with a history of depression (Waugh et al., 2012) experienced less positive and more negative affect across laboratory tasks. Yet others have reported reduced positive and negative emotional reactivity in depressed individuals (Bylsma, Morris, & Rottenberg, 2008) or no differences in negative affectivity in children at high familial risk for depression (McMakin et al., 2011; Olino et al., 2011). The group difference in affect may be attributable to blunted negative emotionality, potentially due to higher depressive symptoms or a past history of depression in our group at high familial risk. As for gender differences, girls display more negative affect than boys do in negative contexts (Casey, 1993), and report higher intensity of anxiety and sadness (Silk, Steinberg, & Morris, 2003). Thus, low risk girls starting at a higher level of negative affect and decreasing at a faster rate than boys may be attributable to gender differences in negative affect, and additional blunted negative affectivity in high risk girls.

For hypothesis 2b, we predicted that presence of a parent would impact the recovery slope for both positive and negative affect, and that this may be different between risk groups. However, we did not find any of these relationships. While this may seem that the manipulation of parental presence was not effective, further results from hypothesis 2c, suggest that the lack of overall influence of parents on affect regulation may be due to the frequency of supportive behaviors or levels of support that adolescents feel from families. In fact, when examining those within the parent present condition, greater supportive behaviors that parents gave during the parent-child interaction resulted in faster recovery of happiness levels. Thus, the key ingredient to parental buffering of positive affect may be the types of behaviors that parents showed during the interaction, rather than the mere presence of parent after the stressor. Consistent with our

results, more supportive emotion socialization was beneficial to increasing positive affect and allowed for more effective emotion regulation (Miller-Slough & Dunsmore, 2016; Yap et al., 2008). Additionally, we examined how the impact of supportive behaviors on happiness levels may differ between high and low risk adolescents. When high risk adolescents had greater support, they reported higher happiness levels during the stress task. This may suggest that these adolescents report higher levels of happiness *throughout* the task, and may be more resilient to psychological stress. Indeed, maintaining positive affect in the face of stressors may be protective (Folkman & Moskowitz, 2000), and one mechanism by which this may occur could be through higher parental support. Yet, given that these adolescents benefited from supportive parental behaviors *prior* to seeing their parent, this observed support during the interaction may serve as a proxy for overall levels of family support. Indeed, when we examined adolescent's report of global social support from the family, higher perceived support predicted higher positive affect after the stressor and a faster recovery of positive affect. This was a stronger association when adolescents further had the ability to be with their parent during regulation (i.e., in the parent present condition). Thus, global, trait-like family social support played a role in increasing levels of positive affect in the face of a stressor, and additionally, was beneficial to the recovery of positive affect when adolescents were with their parent and able to receive this support. Conceptually, social support may enhance the ability to cope with a stressor (Lakey & Cohen, 2000), both in the moment due to the type of support given as well as more globally based on the internalization of this support (Eisenberg et al., 1998). We found both the momentary benefit of social support (as seen with the impact of observed supportive parental behaviors on happiness), and the longer lasting effect of perceived social support (as evidenced

by the initial levels of happiness being higher with global social support), thus, both mechanisms may be at play in improving levels of positive affect after the stressor.

Furthermore, there was a significant difference in how observed parental support impacted girls and boys level of negative affect when they were in the parent present condition. Girls reported higher levels of negative affect than boys and decreased rapidly despite the frequency of parental supportive behaviors. This suggests that they were able to use their parents as a support partner regardless of actual parental behaviors. Girls may "tend and befriend" in the face of stress (Taylor et al., 2000), and disclose more emotions (Papini, Farmer, Clark, Micka, & Barnett, 1990), thus may feel better with just the presence of a support partner. Yet, for boys who reported higher negative affect during the TSST, their parents gave more supportive behaviors during the parent-child interaction compared to those who did not report as much negative affect. Thus, parents of boys gave more positive emotion socialization only when boys were more distressed. Parents have been shown to use emotion socialization in the same way with boys and girls (Klimes-Dougan et al., 2007) and adults retrospectively report that mothers responded to levels of fear similarly for men and women (Garside & Klimes-Dougan, 2002). Thus, it may be that parents are differentially responding to their adolescent boys dependent upon the levels of affect that they showed (higher negative affect was associated with higher support), yet for girls, there may not be as much differential response from parents dependent upon affect level.

Additionally, we examined adolescent's perceived global support from family, and found that higher perceived support was related to more negative affect right after the TSST. Yet, this result was not dependent upon gender or whether the adolescent did or did not have their parent present. It may be that higher levels of negative affect may occur with higher perceived social support since emotions may be more accepted. Emotion socialization theory posits that the way

in which parents interact with their children about their emotions provides children with guidelines about whether certain emotions are "allowed" to be expressed or should be suppressed (Eisenberg et al., 1998). Parents who may believe that experiencing and expressing emotions is appropriate may show greater supportive behaviors in response to their child's negative affect and this in turn may allow children to better regulate their negative affect (Eisenberg et al., 1998). On the other hand, suppression of emotions, which may occur when parents dismiss the child's emotion and children internalize that they should not experience said emotion, may increase sympathetic activation and overall lead to greater levels distress (Gross, 2002). Thus, the result of greater perceived family support being associated with increased initial negative affect may be protective since adolescents are able to experience the emotion, potentially decrease sympathetic activation, and regulate negative affect more effectively.

Overall, Aim 2 demonstrated that supportive parental behaviors and global family support may be beneficial to adolescent's regulation of positive affect. In particular, those with higher support (both actual behaviors and perception of support) had greater increases in positive affect after the task, especially when their parent was with them, and higher support was especially beneficial for adolescents at high familial risk. For negative affect, low risk girls started higher and decreased faster than all other groups. Parents responded to their boy's negative affect with higher levels of supportive behaviors, yet, there was not this modulation in behaviors with girls. Thus, parental support is effective in helping adolescents to up-regulate positive affect after stressors, and may influence boys' ability to regulate negative affect.

Aim 3: Impact of Parent on Adolescent's HPA-Axis Reactivity and Recovery

The purpose of this aim was to determine whether parents would act as stress buffers of the HPA-axis after adolescents at high and low familial risk experienced a stress task. Adolescents at high familial risk had shorter duration of cortisol activation compared to those at low risk. Furthermore, older adolescents had lower cortisol responses when parents were present compared to younger adolescents. When in the parent present condition, level of adolescent's expressed distress was related to higher peak cortisol, and parents provided higher supportive behaviors in response. These supportive behaviors did not influence cortisol curves in the low risk group, yet in the high risk group, higher support was related to lower peak cortisol. Thus, we obtained a parental buffering in older adolescents, found differential associations between how parental supportive behaviors impacted peak cortisol in low and high risk adolescents, and lastly found that amount of expressed emotion by the child may account for the relationship between supportive parental behaviors and cortisol response.

The Trier Social Stress Test was successful in activating the HPA-axis in 80% of participants in this study. This result is similar to the 60-70% response rate with adults (Kirschbaum, Pirke, & Hellhammer, 1993; Kudielka et al., 2009) and adolescents (Ji et al., 2016). Furthermore, we found significant covariates of menstrual cycle and time since awakening on the cortisol curves. Specifically, girls in the follicular phase of the menstrual cycle or who took birth control had lower peak cortisol response compared to girls in the luteal phase or boys, which is consistent with previous literature (Kudielka & Kirschbaum, 2005). We also found a significant time since awakening effect on peak cortisol, and a moderately significant impact on the activation slope. This suggests that those who had been awake longer had less steep slopes and lower cortisol levels at peak compared to those who were awake for a

shorter period of time by the start of the study. Although not many studies have examined the impact of time since awakening on cortisol values, time of day seems to be an important factor, and this may be a proxy for time since awakening. Specifically, studies conducted in the morning have shown greater salivary free cortisol compared to those conducted in the afternoon (Kudielka et al., 2004), which may reflect how long these participants have been awake (those awake for short periods of time have higher overall cortisol values). This is consistent with our study, especially considering that we controlled for time of day.

Cortisol peak responses were significantly higher for adolescents with low compared to high familial risk. Considering that peak is a function of baseline levels, slope intensity, and duration of activation, and that there were no differences in activation or baseline levels, the findings reflect differences between groups in duration of activation (Lopez-Duran et al., 2014); that is the low risk group showed longer duration of cortisol activation, with later peaks than the high risk group. In the literature, the nature of reactivity in high risk adolescents have been relatively equivocal; two reported elevated cortisol when there was higher concurrent parental depression (Badanes et al., 2011) and lowered positive affect (Waugh et al., 2012), whereas another found a blunted response to their stress task in high risk girls (Bouma et al., 2011), yet another did not find any differences between those at risk and not (Gotlib et al., 2015, 2008). This conflicting evidence may be due to differences in methodology, such as including parent-child conflict as a stressor (Badanes et al., 2011), and examining only girls without a prior history of depression (Gotlib et al., 2008; Waugh et al., 2012). Our results are in line with one study of high risk adolescents (Bouma et al., 2011) and adults with depression (Burke et al., 2005), which may suggest there is a subgroup of adolescents at risk for depression who suffer from blunted cortisol activation. Theoretically, blunted reactivity may reflect prolonged and

repeated activation of the HPA-axis due to chronic stressful environments (Heim et al., 2000). With increasing exposure to stress, down regulation of the corticotropin- releasing hormone (CRH) receptors in the brain may be adaptive in decreasing the activation of the HPA-axis and protecting against excessive cortisol (Bouma et al., 2011). Children in our risk group may have more chronic stress factors associated with blunted cortisol reactivity such as uncontrollable stress (G. E. Miller, Chen, & Zhou, 2007), or greater chronicity and severity of depression (Booij, Bouma, De Jonge, Ormel, & Oldehinkel, 2013; Harkness, Stewart, & Wynne-Edwards, 2011). Blunted activation of the HPA-axis may lead to negative consequences, such as an increased risk for future internalizing problems (Badanes et al., 2011; Ruttle et al., 2011). An alternative explanation for the blunted activation may be that the adolescents did not find the stress task stressful, potentially due to excessive stressors in their lives outside of the laboratory context (Badanes et al., 2011; Gunnar & Vazquez, 2001). However, as noted in Aim 2, there were significant decreases in positive affect and increases in negative affect over the stress task which was similar between high and low risk adolescents. Therefore, the less prolonged duration of cortisol activation may reflect a specific characteristic of our high risk group related to chronic stress, and understanding the role of these factors in our data will be an important future direction for this research.

For hypothesis 3b, we tested whether there was a parental buffering effect in the high and low familial risk groups. Overall, there was no effect of parental presence on cortisol curves, and this was not moderated by risk status. However, we included age into the model since this has previously been found to impact the stress buffering of parents (Doom et al., 2015), and found a significant interaction with age and peak levels of cortisol. Although not reaching significance, in the parent absent condition, older children had greater cortisol peak activation

compared to younger children. This was an expected result, as the overall amount of cortisol produced in response to laboratory stressors increases as children age, especially after puberty (Gunnar, Wewerka, et al., 2009; Stroud et al., 2009). When examining the parental buffering effect, at older ages there was a decrease in peak levels of cortisol in the parent present compared to absent condition, indicating a robust parental buffering effect in the older adolescents. However, this was the opposite in younger adolescents who showed higher cortisol peak values when their parents were with them. This was a surprising result as the parental stress buffering effect was found in children (Seltzer et al., 2010), yet was not for adolescents (Hostinar et al., 2015), and this difference is due to pubertal timing and age (Doom et al., 2015). One explanation may be that we had different methodology from Gunnar's group (Doom et al., 2015; Hostinar et al., 2015), which examined the impact of parental presence during the preparation of the speech task, whereas ours investigated the randomization during the recovery phase. However, we modeled our protocol after Seltzer's group, who found an impact of parent in younger prepubescent girls aged 9-12. One major difference between our study and the Seltzer study is the pubertal status of our participants. Our youngest adolescents were about midway through puberty, and for girls, 5 of 6 had started menstruation, whereas all girls in the Seltzer paper were pre-menarche. Thus, our sample was farther along in development, which may explain some of the differences between studies.

The age effect found in social buffering may be explained by individual child characteristics and the changing developmental interactions between parents and adolescents. It may be that children in our younger age range were more unfamiliar with giving public speeches, since they would have less opportunity to experience them in school settings. It is known that novel and uncontrollable socially evaluative tasks are those that produce largest increases in

cortisol (Dickerson & Kemeny, 2004), and larger increases may make it more difficult for children to use parents as a support system to down-regulate stress levels. In our sample higher expressed emotion was associated with higher cortisol values, thus, this may be a unique subset of young children who were especially distressed by the task and unable to benefit from parental presence. Older adolescents on the other hand, may also be able to utilize the support their parents give, since they have more practice with speech tasks and potentially a better ability to relate their distress to parents verbally. The relationship between parents and adolescents change over time; while there is less time spent with parents, the amount of time talking increases, especially about interpersonal issues (Larson, Richards, Moneta, Holmbeck, & Duckett, 1996). Thus, older adolescents may be able to relate what happened during the speech task to their parent more effectively and garner the support they need. Additionally, there is more parent-child conflict in early adolescence (10-12 years of age) than mid adolescence (13-16 years) (Laursen et al., 1998), and a corresponding increase in positive affect between those ages (Larson et al., 1996), which may suggest that the relationship at older ages is perhaps more supportive. Lastly, a third variable may account for some of the differences. For example, touch from social support partners (Hostinar et al., 2014), and attachment security (Hostinar & Gunnar, 2013; Meuwly et al., 2012) may be important determinants of social buffering, and perhaps are also correlated with age and parental support. Thus, future research should consider the parent-child relationship quality in analyses of the social buffering effect in order to more fully explain the results.

For hypothesis 3d, we found that higher parental supportive behaviors significantly predicted higher cortisol peak responses in the adolescents who had their parent present. This was an unexpected finding, as we theorized that greater support would lead to a dampened

cortisol response or quicker recovery. However, there have been relatively few studies that have examined the type, quality, or quantity of support during social buffering studies. Only one examined positive versus negative dyadic coping (similar to the construct of support here) and found that positive dyadic coping in romantic couples was associated with faster recovery from stress (Meuwly et al., 2012). Another study with children did examine observational measures of support (Hostinar et al., 2015), although they did not explicitly test whether supportive behaviors were related to social buffering of the HPA-axis. Thus, our study was the first to find an association between high levels of positive support after a stress task and greater cortisol response to the stressor. Although it is possible that high levels of positive support increased the stress response, it is more likely that this finding reflects parental sensitivity to the child's level of distress after the task. That is, the more distressed the adolescent is, the stronger the cortisol response, and the more parents used positive support. In fact, we found that the adolescent's expressed emotion when their parents were present mediated the impact of support on cortisol peak; that is the greater the expressed emotion, the greater the activation slope and peak levels, and the variance accounted for by supportive behaviors was mitigated. Previous studies have found that higher distress, such as perceived stress or lowered positive affect, was related to higher cortisol reactivity in children (Gunnar, Wewerka, et al., 2009; Waugh et al., 2012), which is in line with our study. This suggests that distress may be a more potent factor in explaining the cortisol response than the type of support parents give. Overall, the results indicate that parents responded to their child's distress levels with greater amounts of support and those adolescents with highest amounts of distress had the greatest cortisol activation.

Further dissecting the association between support and cortisol, we found a significant group interaction. In the low risk group, there was an association between higher support

behaviors and greater peak cortisol; however, in the high risk group, higher parental supportive behaviors were associated with lower peak cortisol. Low risk parents may be responding to their child's distress levels with higher support, yet, it is not effective with mitigating or helping to regulate the stress response. However, parents with a history of depression are not giving as much overall level of support and thus, may not be responding as effectively to their adolescent's distress. Consistent with this, in response to their young children's distress, parents with a history of depression reported that they ignore or exhibit more negativity (Silk et al., 2011). This suggests that parents with a history of depression may not be as sensitive as non-affected parents in responding to their adolescent's distress. Yet, when parents did provide greater support, there was a decrease in peak cortisol response. Since the high risk group had blunted cortisol peak values overall, the significant decrease in the response when parents have higher support may be potentially negative. Theoretically, the stress buffering effect is adaptive since it decreases overall cortisol activation and mitigates the effect of stress (Kikusui et al., 2006). However, adaptive functioning of the HPA-axis involves evoking a cortisol response, and then shutting down the axis (McEwen, 1998b). If these adolescents are not modulating an adaptive response to begin with, having parents further decrease the response may be maladaptive, since they are not reacting appropriately to the stressor. Yet, in our study, 83 percent of high risk adolescents had a significant cortisol response to our task. Thus, these adolescents *are* effectively modulating a stress response and the effect of parent may be adaptive in decreasing activation of the HPA-axis. However, it is also noted that adolescents in the high familial risk group received less support than those in the low risk group, thus, the impact on biological stress regulation may only occur with those parents with the highest support. Altogether, low risk adolescents may not

benefit as much from higher supportive behaviors from their parent, and social buffering is effectively seen in high risk adolescents when parents give more support.

This study demonstrates that the social buffering effect occurs in older adolescents, and may depend on the level of distress of the child and quality of support from the parent. High risk adolescents had lower cortisol than those at low familial risk, which indicates some blunting of the response. Adolescent's own level of distress is a more potent indicator of peak cortisol response than supportive behaviors by parents. Furthermore, there is evidence suggesting that parental buffering does occur in those families with a history of depression, but it depends on greater supportive parental behaviors. This may indicate that social support is beneficial in high risk families, especially with biological stress regulation. Further analyses with a larger sample size and taking into account more parent-child relationship variables will be necessary in future directions.

General Discussion

This dissertation aimed to understand the impact of parental support on stress regulation in adolescents who were and were not at risk for depression. Adolescents at high familial risk were less emotionally and biologically reactive to acute stress than low risk adolescents. Parents were able to influence stress regulation, but it depended upon the amount of support that they demonstrated. Higher support from parents was associated with better regulation of positive affect, and higher levels of cortisol, especially in low risk groups. While parents with a history of depression were less likely to give support, when they provided higher levels, high risk adolescents were able to regulate stress more effectively. Thus, there were differential associations between how parents impacted stress regulation with high and low risk adolescents.

It is important to first discuss adolescent's stress reactivity to the Trier Social Stress Test and how parental presence may have influenced it. The TSST was effective in increasing negative affect and decreasing positive affect (Aim 2), and increasing cortisol in the majority of participants (Aim 3). Thus, the task seemed effective in inducing stress, and we were able to measures stress regulation from these adolescents based on both self-report and biological stress measures. However, the experimental manipulation of having a parent present after the TSST did not seem to overall influence this stress regulation. Conceptually, we had predicted differences in stress reactivity between our high and low risk groups, and were interested in the impact of parents on stress regulation. Thus, having the parent present condition *after* the stress task was the best manipulation to answer questions about how adolescents at high and low familial risk experienced stress and about how parents impact stress regulation. Yet, most social buffering studies have examined the impact of social support partners *prior to* the stressor (Heinrichs et al., 2003; Hostinar et al., 2015; Kirschbaum et al., 1995), although see (Meuwly et

al., 2012; Seltzer et al., 2010) for different protocols. Thus, we may not have captured as strong of a stress buffering effect, since the parent may be most helpful *prior* to the stress task. This may be due to the provision of instrumental support (Hostinar & Gunnar, 2013), or the ability of the parent to change the experience to be less stressful (Power, 2004). For example, in our study, fear and tenseness were at highest peak levels during the stress task (time point 3), and adolescents had experienced less negative affect by the time they were with their parent (time point 4) (Aim 2). Thus, parents were interacting with their adolescent children when adolescents were not experiencing as much distress, and there may not have been as much ability for parents to help with regulation. Potentially, in this paradigm, parents may play a stronger role when they are able to modulate the experience rather than just react to their children's emotions or distress, and future research should tease apart the influence of parents both before and after stressors.

We were also interested in overall differences in stress reactivity with our adolescents at high and low risk for depression. Low risk adolescents who were in the parent present condition expressed more distress to their parents than high risk adolescents (Aim 1), and low risk girls reported higher levels of negative affect during the stress task (Aim 2). The distress levels were positively associated with faster increases in cortisol and higher peak responses, and thus low risk adolescents had higher peak cortisol than those at high familial risk (Aim 3). This suggests that there may have been blunting in stress reactivity in high risk adolescents. Based on previous literature, we expected to find greater stress reactivity in the offspring of depressed parents (Bouma et al., 2008; M. C. Morris et al., 2010). Less reactivity to stressors may indicate lower effort by our high risk participants, reduced awareness or perception of stress, or reduced physiological capacity to respond (Phillips, Ginty, & Hughes, 2013). Alternatively, it may have been that adolescents in our high risk group had more chronic difficulties with depression which

may have led to blunted hedonic capacity (Bylsma et al., 2008) or cortisol (Burke et al., 2005). While we ensured that no adolescent was currently in a depressive episode, we allowed for our high risk group to have a prior history of depression, and some of our participants had experienced a depressive episode in the past. Thus, in order to tease apart whether blunted stress reactivity was due to depression or another cause, future directions should control for duration, frequency and onset of depressive episodes and gain more insights into how the adolescent perceived the stress task.

Importantly, this dissertation examined the emotion socialization behaviors that parents gave to their children when they had the opportunity to see their children after the stress task. This showed that parents generally gave supportive behaviors, such as comforting, validation of emotions, and problem solving about emotions, and relatively few unsupportive behaviors such as dismissing and punishing. Parents modulated and gave more support to their adolescent child when there was more distress as evidenced by how frequent adolescents discussed the stress task with their parent (Aim 1), the amount of self-reported negative affect (Aim 2), and cortisol response (Aim 3). Results showed that this supportive behavior was influential in helping adolescents to regulate their distress. For example, higher parental support (either received or perceived) allowed for children to up-regulate their positive affect after the stress task faster than those without as much parental support (Aim 2). Yet, parental support seemed to be less effective for aiding in biological regulation of stress (Aim 3). Thus, subjectively adolescents benefited to some extent by having parents who gave more supportive reactions, yet it may not have impacted biological stress regulation.

Emotion socialization strategies that parents used differed based on risk group and may have impacted groups differently. Adolescents who were at risk for depression had parents who

demonstrated moderately less supportive reactions than those without familial depression (Aim 1). In the context of their own emotional distress, high risk adolescents discussed their emotions less with their parents (Aim 1), which may explain in part why parents showed less supportive behaviors to their children, as adolescents asked less for help. Parental behaviors were consistent across mood state (Aim 1), thus, parents with a history of depression may consistently show less supportive reactions. High risk adolescents reported that they felt less satisfied by the support their parent gave them after the stress task (Aim 1), and also reported moderately lower global perceived social support from their family (Aim 2). Hence, high risk adolescents may not feel as supported when they do receive support from their parents. When parents in the low risk group perceived greater depressive symptoms in their child, they provide more supportive behaviors, regardless of the distress the child was displaying in the moment (Aim 1). Yet, parents with a history of depression did not modulate their supportive behaviors, and demonstrated greater unsupportive reactions when their adolescent has greater depressive symptoms (Aim 1). Therefore, the modulation for the low risk group may be protective against future depressive symptoms, but it may exacerbate high risk depressive symptoms. Together, data from this dissertation suggests that adolescents at familial risk for depression have more negative emotion socialization from their parents in the context of an acute laboratory stressor.

On the other hand, adolescents at risk for depression may especially benefit from higher levels of support that parents give. Despite the overall lower support, when high risk adolescents received higher levels of support, they had more effective stress regulation. These adolescents reported higher levels of happiness after a stress task (Aim 2), which may indicate a better ability to maintain positive affect in the face of stressors. Also, adolescents who perceived higher global family support had increased levels of happiness slopes regardless of parent condition and

risk status (Aim 2). As depressed individuals may be particularly impaired with lowered positive affect (Forbes & Dahl, 2007), this buffering of positive affect may be especially important in decreasing the risk for depression. Also, the high risk group demonstrated lower peak cortisol responses when parents gave them higher support in the parent present condition (Aim 3), which is the expected stress buffering effect. Thus, while parents with a history of depression did not show as many supportive behaviors, those who showed greater support had adolescents who were better able to regulate their stress levels. Consistent with this, positive parenting in a high risk sample has been associated with more resilient outcomes (Brennan et al., 2003), thus one possible mechanism by which this occurs is through stress regulation.

It is also interesting to note that adolescents at low familial risk adolescents in this study appeared very reactive to the stress task. Not only do these children express more distress to their parents (Aim 1), girls in the low risk group reported highest levels of negative affect immediately after the stress task (Aim 2), and they experienced the highest peak cortisol levels overall (Aim 3). Parents, while providing support to their distressed child, were also unable to effectively modulate the cortisol response (Aim 3). Thus, the low risk adolescents in this sample may have been more reactive, and demonstrate greater distress to the stress task. There is also a possibility that outliers in our low risk sample were more influential in driving results, as we had a low sample size in comparison to our high risk sample. Preliminary analyses were conducted to determine whether the low risk sample had higher levels of self-reported anxiety which may drive some of the results, yet, we found that there was less anxiety compared to those in the high risk group. On the other hand, higher levels of anxiety coupled with higher expressed distress predicted greater cortisol activation slopes and peak levels, thus, anxiety may have amplified the cortisol response within the low risk group. Future analyses should control for levels of anxiety,

and include other moderators that may be present in these low risk adolescents which may affect how fearful and tense they may be.

These results should be considered in the context of a few limitations. In particular, this study did not have as many participants as expected in the a priori power analysis, thus, this should be seen as a preliminary investigation, and we may have been under powered to detect some differences. Thus, it will be important to confirm the results here with a larger sample size. We were unable to control for the gender of the parent in our study as only 9 fathers came (only 3 were in the parent present condition). The gender difference in parents may be due to a higher prevalence of depression in women compared to men (Kessler, 2003), thus, it may need to be corrected in the future by oversampling for fathers with a history of depression. Given that in general, there were not as many parent present versus absent differences, it would be fruitful to change the manipulation to have parental support given prior to the stress task, rather than afterward. A comparison between the conditions (before and after the stressor) would be helpful to understand how parents most effectively help adolescents regulate emotional experiences.

With our observed parental supportive behaviors (Aim 1), we only coded for the frequency rather than duration or intensity of parental behaviors and child's expressed distress. Only a few parental behaviors could be coded on a continuum; for example, physical comforting may be extended if a parent hugs their child for a minute rather than just at one moment in time. We controlled for this duration by coding a new "event" of comforting for every minute that the parent continues to provide this behavior. Other behaviors such as verbal statements may also differ in intensity (e.g., "I was so scared by the task" is qualitatively different from "I did a speech task today", yet would be coded equivalently as one instance of expressed emotion).

Future directions should account for duration and intensity of these behaviors, as this may differ significantly across groups.

Furthermore, the scale of affect used in Aim 2 could have been improved by including multiple positive and negative affect terms, which would improve the construct validity. It also may have been helpful to use less discrete emotions in the negative affect construct. For example, adolescents may be less likely to respond that they were "sad" versus a more neutral term such as being "stressed" or "upset" as was used in other studies (Waugh et al., 2012). Examining and testing multiple versions of the VAS may improve future results for this study.

Overall, there were numerous strengths to our study. The TSST itself was an appropriate stressor, and extended findings of parental emotion socialization to a context in which parents were not present during the stressor and did not involve parents and adolescents in direct conflict. Our parent condition manipulation directly tested whether parents may be beneficial in stress regulation, and allowed us to test for group differences in stress reactivity. Additionally, this was one of the first studies to examine the types of parental behaviors that were present in social buffering studies, and found specific benefits to parental supportive behaviors on stress regulation in both self-report of affect and in stress hormones. The statistical analysis conducted in Aim 3 (growth curve modeling with landmark registration) was a relative strength as it is able to model different aspects of the cortisol response which traditional analyses do not capture (Lopez-Duran et al., 2014). Furthermore, we were successful in recruiting many families with a history of depression which may help to accurately understand how parental emotion socialization occurs in adolescents at risk for depression.

Together, this study contributes to the understanding of how parents with a history of depression impact stress regulation in their adolescent child. Overall, high risk children seem to

not be as reactive to the stressors, and that parents with a history of depression do not respond in as supportive of a manner as those who do not have a history of depression. Yet, we also see that in the context of higher supportive behaviors, high risk adolescents had better stress regulation. Thus, support may be particularly beneficial for these children, and may confer less of a risk for future depression. Implications of this study are to help improve supportive emotion socialization behaviors of parents, so that they may be able to respond to their child's distress in a more effective manner, and help the child to develop better emotion regulation skills. Furthermore, this study directly showed that emotion socialization continues into adolescents and aids in the ability for the child to regulate their distress. In sum:

> The importance of parents in adolescents' lives depends less on the physical power of parents and the extent to which they share experiences with their children and more on the emotional and instrumental support the family provides and the psychological bond between parents and children. (Larson et al., 1996)

Appendices

Appendix 1. Questionnaires in Study

Questionnaire	Reporter	Assessment
Beck Anxiety Inventory (Beck, Epstein, Brown, & Steer, 1988)	Parent	Parent's anxiety symptoms during previous two weeks
Beck Depression Inventory-II (Beck et al., 1996)	Parent	Parent's depressive symptoms during previous two weeks
Child Depression Inventory 2 (Kovacs, 2010)	Parent & Adolescent	Child's depressive symptoms during previous two weeks
Childhood Trauma Questionnaire (Bernstein et al., 2003)	Adolescent	Self-report of child abuse and neglect
Demographic form	Parent	SES, race, family composition, etc.
Disruptive Behavior Disorders Rating Scale (Pelham, Gnagy, Greenslade, & Milich, 1992)	Parent	Child symptoms of ODD, CD, ADHD.
DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure—Adult (2013)	Parent	Parent symptoms of substance use, OCD
Inventory of Parent and Peer Attachment (Armsden & Greenberg, 1987)	Adolescent	Adolescent's perspective of attachment to their parents
McMaster Family Assessment Device (Epstein, Baldwin, & Bishop, 1983)	Parent & Adolescent	Family functioning
Multidimensional Scale of Perceived Social Support (Zimet et al., 1988)	Adolescent	Perception of social support system
Perceived Stress Scale (Cohen, 1994)	Adolescent	Self-report of perceived stress over previous month
Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989)	Adolescent	Child's quality of sleep over the previous month

Pubertal Development Scale (Petersen et al., 1988)	Adolescent	Self-report of pubertal development
Screen for Child Anxiety Related Emotional Disorders-Revised (Birmaher et al., 1999)	Adolescent	Child's symptoms of anxiety
Self Report Delinquency Scale (Elliott, Ageton, Huizinga, Knowles, & Canter, 1983)	Adolescent	Frequency of aggressive and delinquent behavior
Visual Analog Scales (Ahearn, 1997)	Adolescent	Assesses self-report of mood/emotions to TSST task

Appendix 2. Visual Analog Scale

Please answer the following questions with reference to how you have felt on average over the past 5 to 10 minutes.

	Not at all	Moderately	Extremely
Sad			
Happy			
Afraid			
Amused			
Disgusted			
Angry			
Energetic			
Tired			
Tense			
Confused			

Appendix 3. Adolescent's Appraisal of Parental Support in Parent Present Condition

Please answer the following questions thinking about how you felt while being together with your parent over the past 10 minutes

To what degree did you feel supported by your parent/guardian?

Not at all Moderately Extremely

To what degree did you feel that being with your parent/guardian was typical or like an everyday interaction?

Not at all Moderately Extremely

How was this today different or the same?

To what degree did being with your parent/guardian change the way you felt or thought about the task you did?

Not at all Moderately Extremely

To what degree did you feel in control when you were with your parent?

Not at all Moderately Extremely

How typical was the support that your parent/guardian gave compared to how they usually support you?

Not at all Moderately Extremely

What does s/he usually do to support you?

Appendix 4. Emotion Socialization Strategies Observational Coding Manual

Emotion Socialization Strategies: Observational Coding Manual
Adapted from:
Cara D. Bosler, Amanda Sheffield-Morris, & Michael M. Criss
Oklahoma State University

Emotion Socialization Strategies
Coding Manual for Detecting Risk of Youth Depression Study
December 15, 2015

OVERVIEW

The following coding system was developed in order to measure how parents respond to their adolescents' emotions. The type of emotion socialization behaviors the parent displays will be coded. These behaviors fall under 5 different categories including coaching, overriding, punishing, magnifying, and moralizing. Coaching involves those behaviors that encourage discussion and regulation of emotion. Parents using an overriding strategy discourage the youth's displays of emotion by suggesting emotions are not important. Punitive behaviors convey disapproval of emotional displays and are indicative of the punishing emotion socialization strategy. Parents who magnify emotions promote the escalation of their child's emotions rather than help them to regulate. Finally, moralizing is characterized by long lectures about emotions.

Coding Overview

In the descriptions of the rating scales that are presented in this code book, the following format will be used for defining each scale.

1. ***Name and abbreviation of scale:*** The scale name indicates the word or combination of words by which a scale is regularly known. The abbreviation is a two-letter referent that stands for the scale.

2. ***Clarification/Examples:*** This section provides more clear examples to illustrate and clarify what types of behaviors constitute the code.

Strategies for Viewing Videos

1. The participant ID# will be ***randomly selected*** for each coder.

2. First, watch the assigned task for a selected family one time through without stopping the tape for a general overview of relationships and behaviors.

3. Then, you will watch the video focusing only on the parent.

4. You probably will need to stop and start the video so you can write down evidence.

5. A task ***begins*** when the research assistant has finished instructions and leaves the room and a family member starts describing the situation that elicited the specified emotion. A task ends when the interviewer ***returns***. You should hear a knock at the door just before.

6. Record in BORIS any indication of a specified behavior. This is continuous recording, thus, you must indicate the letter of the behavior (e.g., "C" refers to "comforting behavior") within a second of the behavior occurring.

7. When you watch the tapes, turn off the theory-making part of your brain. Don't try to understand or diagnose the family or the person. Focus only on the ***specific behaviors***.

8. When you are coding, it is recommended that you do it in a quiet room without any distractions (e.g., music playing, reading while you code). Also, you should wear headphones when coding.

9. You will receive a list of tapes to code that are only identified with identification numbers. These numbers are assigned to each family so that their identity is kept confidential.

10. Make sure that you only code ES when they are discussing the stress task.

Mechanics of Coding

- Each coder needs to watch the tape at least 2 times: once to code for half of the behaviors, and the second time for the other half of behaviors (e.g., code 2 or 3 behaviors the first time and 3 the second time). Videos may be viewed more times if needed or may be paused or replayed to be sure behaviors are coded accurately.
- Multiple viewings are VERY important because the coders may miss some rather subtle behavior while looking at one of the members of the dyad.

- A specific parental response (i.e., laughing) can be coded under more than one response category (e.g., dismissing - minimizing and punishing – making fun of/teasing). However, the subcategories under each response category are mutually exclusive, meaning that a response should only be coded under each category once. If a situation arises in which a response could be coded as more than one subcategory, it should be coded under the one it better fits under.

DROYD Coding Manual for both Parent Interactions after the Stress Task

Responses to Youth Emotion

Comforting

- Touching (arm, hand, shoulder, hug)
- Clear physical gestures (father touching son on leg)
- Statements that are comforting in the situation
    - "It will be okay"
    - "I'm sure you did great"

        Nice gesture (offering a drink)

        Encouragement for Future
    - "You'll do better next time"

Validation of feelings

- Labeling of emotions
    - "So it makes you angry when..."
    - trying to associate a word with the feelings that are being expressed by the child

        Validation of feelings
    - "I can see how that would make you angry"
    - "that must have been hard"
    - "you have every right to feel stressed"
    - Telling the child that it makes sense for them to feel the way they do

        Indication of understanding of emotion stated
    - "yeah, wow, I know, I understand"
    - "uh huh", nodding

        Reflection of emotion, rephrasing what youth says
    - "yeah you look pretty shook up"

        Asking questions to clarify emotions
    - "Were you mad at yourself for not finishing the speech?"
    - "Were you embarrassed?"
    - "Are you okay now?"

        Asking clarifying questions about the speech/arithmetic
    - "Why did you want be class president?"

Problem Solving about Emotion/ Teaching

- Works through emotion and actively involved in discussion about coping
    - "How can I help you with your stress?"

        Active participation about emotions by discussing solutions
    - "What do you think you could do when you get stressed?"

        Asking for more information on how the adolescent coped with emotions
    - "What did you do to relieve yourself from your stress?"
    - "How did you make yourself feel better?"
    - coped implies past events

        Teach strategies for regulating/expressing emotions
    - "take a deep breath"

- "think about something else"

Utilizing one's own or the child's experiences/ life lessons to relate to the emotional state of the other

- "I remember one time when I had to give a speech…"
- "Remember when you were worried about the speech in school, but you did great"
- "Whenever I get stressed, I think about being on a beach"

Teaching information about the study to comfort child

- Reason: Putting child's emotions into context
- "They make you spit in a tube because they want to measure your cortisol levels"
- "They were mean to you to make you stressed"

Dismissing

- Parental responses to emotion that discourage the expression of emotion through minimizing or distracting from emotions
- When child brings up emotion parent does not acknowledge them.
- Minimizing
    - Downplaying or not paying attention to the emotion of the child
        - "you weren't that upset"
        - "that shouldn't make you that stressed"
        - laughing at child
    - Discounting/ dismissing youth's emotion when stated
        - "you weren't angry; you were worried"

Punish

- Parental responses to emotion that discourage the expression of emotion by punishing or expressing disapproval of emotion
    - Invalidating/ derogating emotions
        - "If you are upset about that then that is just stupid"
    - expressed disapproval of feelings or expressions
        - "you should be ashamed"
        - "grow up"
        - "stop crying"
    - making fun of feelings or teasing
        - laughing
        - "You cried? Ha-ha"

Magnify

- Parental responses to emotion that encourage the expression of emotion through parental escalation of emotion, or expanding on expressed emotion
    - Escalation
    - Inappropriate sharing of emotion

Moralizing

- Parental response to emotion characterized by lecturing on how to feel or react to or express emotions (ex: "Oh but you're a numbers guy you should be good at that")

- Extensive monologues or stories that dominates the conversation about emotions
- Dominates conversation with "should" and "should not"
- Telling one how they feel and don't feel
    - "you don't feel that way"
    - "only babies cry and you're not a baby"
    - "you should be happy that I drove you here in the first place"

Changing the Topic

- Valence can either be positive or negative

1) Changing topic to dismiss emotions
    a) Child is bringing up distress/implied distress, parent changes topic to avoid talking about emotion
    b) Cutting off expression of distress
        i) Child: "Oh I want to cry" → Parent: "It's spirit week, what are you wearing"
2) Changing topic to help alleviate stress
    a) "Do you want to talk about something else now?"

On topic/ off topic:

*Note: when you code parent, start coding on/off topic and just make a comment each time it happens whether it is Parent or Child who does it.

We can still code for emotion socialization when the child brings up an unrelated task that is emotionally valenced for the child. (e.g., birthday party they weren't invited to). Code as if it was about the TSST, just make sure it is not coded as "on topic".

- On topic is defined as talking about TSST or the study in general
- Off topic - unrelated to anything with the study.

Child's Expressing Emotion/ Asking for help (E)

1) Bringing up TSST task
    a) "I had to give a speech and do an arithmetic task"
2) Description of task & feelings about it
    a) "I thought it was really difficult"
    b) "I was really nervous to give the speech"
3) Expressing stress in response to task
    a) "I really need a hug after that"
    b) "I'm really stressed"
    c) Crying

References

Abaied, J. L., & Rudolph, K. D. (2010). Mothers as a resource in times of stress: Interactive contributions of socialization of coping and stress to youth psychopathology. *Journal of Abnormal Child Psychology*, *38*(2), 273–89. doi:10.1007/s10802-009-9364-7

Adrian, C., & Hammen, C. L. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology*, *61*(2), 354–9. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8473589

Ahearn, E. P. E. (1997). The use of visual analog scales in mood disorders: a critical review. *Journal of Psychiatric Research*, *31*(5), 569–579. doi:10.1016/S0022-3956(97)00029-0

Armsden, G. C., & Greenberg, M. T. (1987). The inventory of parent and peer attachment: Individual differences and their relationship to psychological well-being in adolescence. *Journal of Youth and Adolescence*, *16*(5), 427–54. doi:10.1007/BF02202939

Avenevoli, S., Swendsen, J., He, J.-P., Burstein, M., & Merikangas, K. R. (2015). Major depression in the National Comorbidity Survey–Adolescent Supplement: Prevalence, correlates, and treatment. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(1), 37–44.e2. doi:10.1016/j.jaac.2014.10.010

Badanes, L. S., Watamura, S. E., & Hankin, B. L. (2011). Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders. *Development and Psychopathology*, *23*(3), 881–96. doi:10.1017/S095457941100037X

Barbot, B., Heinz, S. L., & Luthar, S. S. (2014). Perceived parental reactions to adolescent distress: development and validation of a brief measure. *Attachment & Human Development*, *16*(1), 1–21. doi:10.1080/14616734.2013.804328

Bariola, E., Gullone, E., & Hughes, E. K. (2011). Child and adolescent emotion regulation: The role of parental emotion regulation and expression. *Clinical Child and Family Psychology Review*, *14*(2), 198–212. doi:10.1007/s10567-011-0092-5

Barry, T. J., Murray, L., Fearon, P., Moutsiana, C., Cooper, P., Goodyer, I. M., … Halligan, S. L. (2014). Maternal postnatal depression predicts altered offspring biological stress reactivity in adulthood. *Psychoneuroendocrinology*. doi:10.1016/j.psyneuen.2014.12.003

Beardslee, W. R., Gladstone, T. R. G., & O'Connor, E. E. (2011). Transmission and prevention of mood disorders among children of affectively ill parents: A review. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(11), 1098–109. doi:10.1016/j.jaac.2011.07.020

Beck, A. T. (2008). The evolution of the cognitive model of depression and its neurobiological correlates. *American Journal of Psychiatry*, *165*(8), 969–977. doi:10.1176/appi.ajp.2008.08050721

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, *56*(6), 893–897. doi:10.1037/0022-006X.56.6.893

Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment*, *67*(3), 588–97. doi:10.1207/s15327752jpa6703_13

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., … Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, *27*(2), 169–90. Retrieved from http://www.sciencedirect.com/science/article/pii/S0145213402005410

Birmaher, B., Brent, D. A., Chiappetta, L., Bridge, J., Monga, S., & Baugher, M. (1999). Psychometric properties of the Screen for Child Anxiety Related Emotional Disorders (SCARED): A replication study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*(10), 1230–6. doi:10.1097/00004583-199910000-00011

Birmaher, B., Bridge, J. A., Williamson, D. E., Brent, D. A., Dahl, R. E., Axelson, D. A., … Ryan, N. D. (2004). Psychosocial functioning in youths at high risk to develop major depressive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(7), 839–46. doi:10.1097/01.chi.0000128787.88201.1b

Birmaher, B., Williamson, D. E., Dahl, R. E., Axelson, D. A., Kaufman, J., Dorn, L. D., & Ryan, N. D. (2004). Clinical presentation and course of depression in youth: Does onset in childhood differ from onset in adolescence? *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(1), 63–70. doi:10.1097/00004583-200401000-00015

Booij, S. H., Bouma, E. M. C., De Jonge, P., Ormel, J., & Oldehinkel, A. J. (2013). Chronicity of depressive problems and the cortisol response to psychosocial stress in adolescents: The TRAILS study. *Psychoneuroendocrinology*, *38*(5), 659–666. doi:10.1016/j.psyneuen.2012.08.004

Bosler, C. B., Morris, A. S., & Criss, M. M. (2012). Emotion socialization strategies: Observational coding manual. *Unpublished Coding Manual*.

Bouma, E. M. C., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2008). Stressful life events and depressive problems in early adolescent boys and girls: The influence of parental depression, temperament and family environment. *Journal of Affective Disorders*, *105*(1-3), 185–93. doi:10.1016/j.jad.2007.05.007

Bouma, E. M. C., 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*(6), 884–93. doi:10.1016/j.psyneuen.2009.01.003

Bouma, E. M. C., Riese, H., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2011). Self-assessed parental depressive problems are associated with blunted cortisol responses to a social stress test in daughters. The TRAILS study. *Psychoneuroendocrinology*, *36*(6), 854–863. doi:10.1016/j.psyneuen.2010.11.008

Brennan, P. A., Brocque, R. Le, & Hammen, C. L. (2003). Maternal depression, parent–child

relationships, and resilient outcomes in adolescence. *J. Am. Acad. Child Adolesc. Psychiatry*, *42*(12), 1469–1477. doi:10.1097/01.CHI.0000091509.46853.7c

Broderick, J. E., Arnold, D., Kudielka, B. M., & Kirschbaum, C. (2004). Salivary cortisol sampling compliance: comparison of patients and healthy volunteers. *Psychoneuroendocrinology*, *29*(5), 636–650. doi:10.1016/S0306-4530(03)00093-3

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: A meta-analysis. *Psychoneuroendocrinology*, *30*(9), 846–56. doi:10.1016/j.psyneuen.2005.02.010

Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine*, *59*(4), 419–26. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9251162

Butler, E. A., & Randall, A. K. (2013). Emotional coregulation in close relationships. *Emotion Review*, *5*(2), 202–210. doi:10.1177/1754073912451630

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. doi:10.1016/0165-1781(89)90047-4

Bylsma, L. M., Morris, B. H., & Rottenberg, J. (2008). A meta-analysis of emotional reactivity in major depressive disorder. *Clinical Psychology Review*, *28*(4), 676–691. doi:10.1016/j.cpr.2007.10.001

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), 779–96. doi:10.1037/a0025441

Casey, R. J. (1993). Children's emotional experience: Relations among expression, self-report, and understanding. *Developmental Psychology*, *29*(1), 119–129. doi:10.1037/0012-1649.29.1.119

Cicchetti, D., & Toth, S. L. (1998). The development of depression in children and adolescents. *American Psychologist*, *53*(2), 221–241. doi:10.1037/0003-066X.53.2.221

Cohen, S. (1994). Perceived stress scale. *Psychology*, (February), 1–3. Retrieved from http://www.mindgarden.com/products/pss.htm

Colich, N. L., Kircanski, K., Foland-Ross, L. C., & Gotlib, I. H. (2015). HPA-axis reactivity interacts with stage of pubertal development to predict the onset of depression. *Psychoneuroendocrinology*, *55*, 94–101. doi:10.1016/j.psyneuen.2015.02.004

Connell, A. M., Hughes-Scalise, A., Klostermann, S., & Azem, T. (2011). Maternal depression and the heart of parenting: Respiratory sinus arrhythmia and affective dynamics during parent-adolescent interactions. *Journal of Family Psychology*, *25*(5), 653–62. doi:10.1037/a0025225

Cowen, P. J. (2010). Not fade away: The HPA axis and depression. *Psychological Medicine*, *40*(01), 1–4. doi:10.1017/S0033291709005558

Coxe, S., West, S. G., & Aiken, L. S. (2009). The analysis of count data: A gentle introduction to poisson regression and its alternatives. *Journal of Personality Assessment*, *91*(2), 121–136. doi:10.1080/00223890802634175

Cummings, E. M., George, M. R. W., Koss, K. J., & Davies, P. T. (2013). Parental depressive symptoms and adolescent adjustment: Responses to children's distress and representations of attachment as explanatory mechanisms. *Parenting, Science and Practice*, *13*(4), 37–41. doi:10.1080/15295192.2013.832568

Dahlen, H. M. (2016). The impact of maternal depression on child academic and socioemotional outcomes. *Economics of Education Review*, *000*, 1–14. doi:10.1016/j.econedurev.2016.01.006

Davidov, M., & Grusec, J. E. (2006). Untangling the links of parental responsiveness to distress and warmth to child outcomes. *Child Development*, *77*(1), 44–58. doi:10.1111/j.1467-8624.2006.00855.x

Dearing, K. F., & Gotlib, I. H. (2009). Interpretation of ambiguous information in girls at risk for depression. *Journal of Abnormal Child Psychology*, *37*(1), 79–91. doi:10.1007/s10802-008-9259-z

Diamond, L. M., & Aspinwall, L. G. (2003). Emotion regulation across the life span: An integrative perspective emphasizing self-regulation, positive affect, and dyadic processes. *Motivation and Emotion*, *27*(2), 125–156. doi:10.1023/A:1024521920068

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–91. doi:10.1037/0033-2909.130.3.355

Dietz, L. J., Birmaher, B., Williamson, D. E., Silk, J. S., Dahl, R. E., Axelson, D. A., … Ryan, N. D. (2008). Mother-child interactions in depressed children and children at high risk and low risk for future depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(5), 574–82. doi:10.1097/CHI.0b013e3181676595

Dix, T., & Meunier, L. N. (2009). Depressive symptoms and parenting competence: An analysis of 13 regulatory processes. *Developmental Review*, *29*(1), 45–68. doi:10.1016/j.dr.2008.11.002

Doom, J. R., Hostinar, C. E., VanZomeren-Dohm, A. A., & Gunnar, M. R. (2015). The roles of puberty and age in explaining the diminished effectiveness of parental buffering of HPA reactivity and recovery in adolescence. *Psychoneuroendocrinology*, *59*, 102–111. doi:10.1016/j.psyneuen.2015.04.024

DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure—Adult. (2013). *American Psychiatric Association*.

Dunbar, J. P., McKee, L., Rakow, A., Watson, K. H., Forehand, R., & Compas, B. E. (2013). Coping, negative cognitive style and depressive symptoms in children of depressed parents. *Cognitive Therapy and Research*, *37*(1), 18–28. doi:10.1007/s10608-012-9437-8

Eberhart, N. K., Shih, J. H., Hammen, C. L., & Brennan, P. A. (2006). Understanding the sex difference in vulnerability to adolescent depression: An examination of child and parent

characteristics. *Journal of Abnormal Child Psychology*, *34*(4), 495–508. doi:10.1007/s10802-006-9020-4

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, *9*(4), 241–273. doi:10.1207/s15327965pli0904_1

Eisenberg, N., Fabes, R. A., & Murphy, B. C. (1996). Parents' reactions to children's negative emotions: Relations to children's social competence and comforting behavior. *Child Development*, *67*(5), 2227–2247. doi:10.1111/j.1467-8624.1996.tb01854.x

Elgar, F. J., McGrath, P. J., Waschbusch, D. A., Stewart, S. H., & Curtis, L. J. (2004). Mutual influences on maternal depression and child adjustment problems. *Clinical Psychology Review*, *24*(4), 441–459. doi:10.1016/j.cpr.2004.02.002

Elgar, F. J., Mills, R. S. L., McGrath, P. J., Waschbusch, D. A., & Brownridge, D. A. (2007). Maternal and paternal depressive symptoms and child maladjustment: The mediating role of parental behavior. *Journal of Abnormal Child Psychology*, *35*(6), 943–955. doi:10.1007/s10802-007-9145-0

Ellenbogen, M. A., & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology*, *34*(5), 773–785. doi:10.1016/j.psyneuen.2008.12.011

Elliott, D. S., Ageton, S. S., Huizinga, D., Knowles, B. A., & Canter, R. J. (1983). The prevalence and incidence of delinquent behavior: 1976–1980. *Boulder, CO: Behavioral …*. Retrieved from https://scholar.google.com/scholar?hl=en&q=Elliott%2C+Ageton%2C+Huizanga%2C+Knowles%2C+%26+Canter%2C+1983&btnG=&as_sdt=1%2C23&as_sdtp=#0

Epstein, N. B., Baldwin, L. M., & Bishop, D. S. (1983). The McMaster Family Assessment Device. *Journal of Marital and Family Therapy*, *9*(2), 171–180. doi:10.1111/j.1752-0606.1983.tb01497.x

Erceg-Hurn, D. M., & Mirosevich, V. M. (2008). Modern robust statistical methods: An easy way to maximize the accuracy and power of your research. *American Psychologist*, *63*(7), 591–601. doi:10.1037/0003-066X.63.7.591

Espejo, E. P., Hammen, C. L., & Brennan, P. a. (2012). Elevated appraisals of the negative impact of naturally occurring life events: A risk factor for depressive and anxiety disorders. *Journal of Abnormal Child Psychology*, *40*(2), 303–315. doi:10.1007/s10802-011-9552-0

Ewell Foster, C., Garber, J., & Durlak, J. A. (2008). Current and past maternal depression, maternal interaction behaviors, and children's externalizing and internalizing symptoms. *Journal of Abnormal Child Psychology*, *36*(4), 527–37. doi:10.1007/s10802-007-9197-1

Fabes, R. A., Leonard, S. A., Kupanoff, K., & Martin, C. L. (2001). Parental coping with children's negative emotions: Relations with children's emotional and social responding. *Child Development*, *72*(3), 907–920. doi:10.1111/1467-8624.00323

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power. Retrieved from http://www.gpower.hhu.de/en.html

Feng, X., Shaw, D. S., Skuban, E. M., & Lane, T. (2007). Emotional exchange in mother-child dyads: Stability, mutual influence, and associations with maternal depression and child problem behavior. *Journal of Family Psychology*, *21*(4), 714–25. doi:10.1037/0893-3200.21.4.714

Fergusson, D. M., & Woodward, L. J. (2002). Mental health, educational, and social role outcomes of adolescents with depression. *Archives of General Psychiatry*, *59*(3), 225–231. doi:10.1001/archpsyc.59.3.225

Feurer, C., Hammen, C. L., & Gibb, B. E. (2016). Chronic and episodic stress in children of depressed mothers. *Journal of Clinical Child & Adolescent Psychology*, *45*(3), 270–278. doi:10.1080/15374416.2014.963859

First, M. B. (2015). *User's Guide to Structured Clinical Interview for Dsm-5 Disorders-Scid-5: Clinician Version.* Amer Psychiatric.

First, M. B., Spitzer, R., Gibbon, M., & Williams, J. (2012). *Structured Clinical Interview for DSM-IV® Axis I Disorders (SCID-I), Clinician Version, Administration Booklet*. American Psychiatric Pub. doi:10.1163/_q3_SIM_00374

Foland-Ross, L. C., Hardin, M. G., & Gotlib, I. H. (2012). Neurobiological markers of familial risk for depression. In P. J. Cowen, T. Sharp, & J. Y. F. Lau (Eds.), *Current topics in Behavioral Neurosciences* (Vol. 14, pp. 181–206). Springer Berlin Heidelberg. doi:10.1007/7854_2012_213

Folkman, S., & Moskowitz, J. T. (2000). Positive affect and the other side of coping. *American Psychologist*, *55*(6), 647–654. doi:10.1037/0003-066X.55.6.647

Forbes, E. E., & Dahl, R. E. (2007). Neural systems of positive affect: Relevance to understanding child and adolescent depression? *Development and Psychopathology*, *17*(3), 827–850. doi:10.1017/S095457940505039X

Foster, C. E., Webster, M. C., Weissman, M. M., Pilowsky, D. J., Wickramaratne, P. J., Rush, a. J., … King, C. A. (2008). Course and severity of maternal depression: Associations with family functioning and child adjustment. *Journal of Youth and Adolescence*, *37*(8), 906–916. doi:10.1007/s10964-007-9216-0

Friard, O., & Gamba, M. (2012). Behavioral Observation Research Interactive Software. Retrieved from http://penelope.unito.it/boris/

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology*, *72*(1), 67–73. doi:10.1016/j.ijpsycho.2008.03.014

Frye, A. A., & Garber, J. (2005). The relations among maternal depression, maternal criticism, and adolescents' externalizing and internalizing symptoms. *Journal of Abnormal Child Psychology*, *33*(1), 1–11. doi:10.1007/s10802-005-0929-9

Garber, J., & Cole, D. A. (2010). Intergenerational transmission of depression: A launch and grow model of change across adolescence. *Development and Psychopathology*, *22*(4), 819–30. doi:10.1017/S0954579410000489

Garside, R. B., & Klimes-Dougan, B. (2002). Socialization of discrete negative emotions:

Gender differences and links with psychological distress. *Sex Roles*, *47*(3-4), 115–128. doi:10.1023/A:1021090904785

Gershon, A., Hayward, C., Schraedley-Desmond, P., Rudolph, K. D., Booster, G. D., & Gotlib, I. H. (2011). Life stress and first onset of psychiatric disorders in daughters of depressed mothers. *Journal of Psychiatric Research*, *45*(7), 855–62. doi:10.1016/j.jpsychires.2011.03.016

Gibb, B. E., Grassia, M., Stone, L. B., Uhrlass, D. J., & McGeary, J. E. (2012). Brooding rumination and risk for depressive disorders in children of depressed mothers. *Journal of Abnormal Child Psychology*, *40*(2), 317–26. doi:10.1007/s10802-011-9554-y

Gonzalez-Bono, E., Rohleder, N., Hellhammer, D. H., Salvador, A., & Kirschbaum, C. (2002). Glucose but not protein or fat load amplifies the cortisol response to psychosocial stress. *Hormones and Behavior*, *41*(3), 328–33. doi:10.1006/hbeh.2002.1766

Goodman, S. H. (2007). Depression in mothers. *Annual Review of Clinical Psychology*, *3*, 107–35. doi:10.1146/annurev.clinpsy.3.022806.091401

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, *106*(3), 458–490. doi:10.1037/0033-295X.106.3.458

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child and Family Psychology Review*, *14*(1), 1–27. doi:10.1007/s10567-010-0080-1

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, *6*, 285–312. doi:10.1146/annurev.clinpsy.121208.131305

Gotlib, I. H., Joormann, J., & Foland-Ross, L. C. (2014). Understanding familial risk for depression: A 25-year perspective. *Perspectives on Psychological Science*, *9*(1), 94–108. doi:10.1177/1745691613513469

Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA Axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry*, *63*(9), 847–51. doi:10.1016/j.biopsych.2007.10.008

Gotlib, I. H., LeMoult, J., Colich, N. L., Foland-Ross, L. C., Hallmayer, J., Joormann, J., … Wolkowitz, O. M. (2015). Telomere length and cortisol reactivity in children of depressed mothers. *Molecular Psychiatry*, *20*(5), 615–620. doi:10.1038/mp.2014.119

Grant, K. E., Compas, B. E., Thurm, A. E., McMahon, S. D., Gipson, P. Y., Campbell, A. J., … Westerholm, R. I. (2006). Stressors and child and adolescent psychopathology: Evidence of moderating and mediating effects. *Clinical Psychology Review*, *26*(3), 257–83. doi:10.1016/j.cpr.2005.06.011

Grimbos, T., Granic, I., & Pepler, D. (2013). The relation between co-rumination, maternal depressive symptoms and child psychopathology. *Journal of Psychopathology and Behavioral Assessment*, *35*(3), 335–345. doi:10.1007/s10862-013-9342-9

Gross, J. J. (2002). Emotion regulation: affective, cognitive, and social consequences.

Psychophysiology, *39*(3), 281–91. doi:10.1017.S0048577201393198

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), 135–60. doi:10.1007/s10567-011-0084-5

Gunnar, M. R., Hostinar, C. E., Sanchez, M. M., Tottenham, N., & Sullivan, R. M. (2015). Parental buffering of fear and stress neurobiology: Reviewing parallels across rodent, monkey, and human models. *Social Neuroscience*, *10*(5), 474–478. doi:10.1080/17470919.2015.1070198

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

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: potential indices of risk in human development. *Dev Psychopathol*, *13*(3), 515–538. doi:10.1017/S0954579401003066

Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009). Developmental changes in hypothalamus-pituitary-adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, *21*(1), 69–85. doi:10.1017/S0954579409000054

Hammen, C. L. (1991). *Depression Runs in Families*. New York, NY: Springer US. doi:10.1007/978-1-4684-6410-8

Hammen, C. L. (2009). Adolescent depression: Stressful interpersonal contexts and risk for recurrence. *Current Directions in Psychological Science*, *18*(4), 200–204. doi:10.1111/j.1467-8721.2009.01636.x

Hammen, C. L. (2015). Stress and depression: Old questions, new approaches. *Current Opinion in Psychology*, *4*, 80–85. doi:10.1016/j.copsyc.2014.12.024

Hammen, C. L., Brennan, P. A., & Shih, J. H. (2004). Family discord and stress predictors of depression and other disorders in adolescent children of depressed and nondepressed women. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(8), 994–1002. doi:10.1097/01.chi.0000127588.57468.f6

Hammen, C. L., Hazel, N. A., Brennan, P. A., & Najman, J. (2012). Intergenerational transmission and continuity of stress and depression: Depressed women and their offspring in 20 years of follow-up. *Psychological Medicine*, *42*(5), 931–42. doi:10.1017/S0033291711001978

Hankin, B. L. (2006). Adolescent depression: Description, causes, and interventions. *Epilepsy & Behavior : E&B*, *8*(1), 102–14. doi:10.1016/j.yebeh.2005.10.012

Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin*, *127*(6), 773–796. doi:10.1037/0033-2909.127.6.773

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender

differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*(1), 128–140. doi:10.1037/0021-843X.107.1.128

Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: Stress exposure and reactivity models. *Child Development*, *78*(1), 279–95. doi:10.1111/j.1467-8624.2007.00997.x

Harkness, K. L., Stewart, J. G., & Wynne-Edwards, K. E. (2011). Cortisol reactivity to social stress in adolescents: Role of depression severity and child maltreatment. *Psychoneuroendocrinology*, *36*(2), 173–181. doi:10.1016/j.psyneuen.2010.07.006

Hayden, E. P., Hankin, B. L., Mackrell, S. V. M., Sheikh, H. I., Jordan, P. L., Dozois, D. J. A., … Badanes, L. S. (2014). Parental depression and child cognitive vulnerability predict children's cortisol reactivity. *Development and Psychopathology*, *26*(4 pt 2), 1445–1460. doi:10.1017/S0954579414001138

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, *25*(1), 1–35. doi:10.1016/S0306-4530(99)00035-9

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry*, *54*(12), 1389–1398. doi:10.1016/S0006-3223(03)00465-7

Helsen, M., Vollebergh, W., & Meeus, W. (2000). Social support from parents and friends and emotional problems in adolescence. *Journal of Youth and Adolescence*, *29*(3), 319–335. doi:10.1023/A:1005147708827

Het, S., Rohleder, N., Schoofs, D., Kirschbaum, C., & Wolf, O. T. (2009). Neuroendocrine and psychometric evaluation of a placebo version of the "Trier Social Stress Test". *Psychoneuroendocrinology*, *34*(7), 1075–86. doi:10.1016/j.psyneuen.2009.02.008

Hoffman, C., Crnic, K. A., & Baker, J. K. (2006). Maternal depression and parenting: Implications for children's emergent emotion regulation and behavioral functioning. *Parenting*, *6*(4), 271–295. doi:10.1207/s15327922par0604_1

Hostinar, C. E., & Gunnar, M. R. (2013). Future directions in the study of social relationships as regulators of the HPA axis across development. *Journal of Clinical Child and Adolescent Psychology*, *42*(4), 564–75. doi:10.1080/15374416.2013.804387

Hostinar, C. E., & Gunnar, M. R. (2015). Social support can buffer against stress and shape brain activity. *AJOB Neuroscience*, *6*(3), 34–42. doi:10.1080/21507740.2015.1047054

Hostinar, C. E., Johnson, A. E., & Gunnar, M. R. (2015). Parent support is less effective in buffering cortisol stress reactivity for adolescents compared to children. *Developmental Science*, *18*(2), 281–297. doi:10.1111/desc.12195

Hostinar, C. E., Sullivan, R. M., & Gunnar, M. R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: A review of animal models and human studies across development. *Psychological Bulletin*, *140*(1), 256–82. doi:10.1037/a0032671

Howard Sharp, K. M., Cohen, R., Kitzmann, K. M., & Parra, G. R. (2016). Mechanisms

mediating children's perceived maternal nonsupportive reactions to sadness and children's social and emotional functioning. *Journal of Child and Family Studies*, *25*(2), 367–380. doi:10.1007/s10826-015-0240-5

Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: Integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological Review*, *115*(2), 291–313. doi:10.1037/0033-295X.115.2.291

Ibarra-Rovillard, M. S., & Kuiper, N. A. (2011). Social support and social negativity findings in depression: Perceived responsiveness to basic psychological needs. *Clinical Psychology Review*, *31*(3), 342–52. doi:10.1016/j.cpr.2011.01.005

Jacob, T., & Johnson, S. L. (1997). Parent–child interaction among depressed fathers and mothers: Impact on child functioning. *Journal of Family Psychology*, *11*(4), 391–409. doi:10.1037/0893-3200.11.4.391

Jacobs, R. H., Talati, A., Wickramaratne, P., & Warner, V. (2015). The influence of paternal and maternal major depressive disorder on offspring psychiatric disorders. *Journal of Child and Family Studies*, *24*(8), 2345–2351. doi:10.1007/s10826-014-0037-y

Jaenicke, C., Hammen, C. L., Zupan, B., Hiroto, D., Gordon, D., Adrian, C., & Burge, D. (1987). Cognitive vulnerability in children at risk for depression. *Journal of Abnormal Child Psychology*, *15*(4), 559–572. doi:10.1007/BF00917241

Jaser, S. S., Langrock, A. M., Keller, G., Merchant, M. J., Benson, M. A., Reeslund, K., … Compas, B. E. (2005). Coping with the stress of parental depression II: Adolescent and parent reports of coping and adjustment. *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 193–205. doi:10.1207/s15374424jccp3401_18

Ji, J., Negriff, S., Kim, H., & Susman, E. J. (2016). A study of cortisol reactivity and recovery among young adolescents: Heterogeneity and longitudinal stability and change. *Developmental Psychobiology*, *58*(3), 283–302. doi:10.1002/dev.21369

Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of Abnormal Psychology*, *116*(1), 135–43. doi:10.1037/0021-843X.116.1.135

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., … Ryan, N. (1997). Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(7), 980–8. doi:10.1097/00004583-199707000-00021

Kessler, R. C. (2003). Epidemiology of women and depression. *Journal of Affective Disorders*, *74*(1), 5–13. doi:10.1016/S0165-0327(02)00426-3

Kikusui, T., Winslow, J. T., & Mori, Y. (2006). Social buffering: Relief from stress and anxiety. *Philosophical Transactions of the Royal Society of London.*, *361*(1476), 2215–28. doi:10.1098/rstb.2006.1941

Kilford, E. J., Foulkes, L., Potter, R., Collishaw, S., Thapar, A., & Rice, F. (2015). Affective bias

and current , past and future adolescent depression : A familial high risk study. *Journal of Affective Disorders*, *174*, 265–271. doi:10.1016/j.jad.2014.11.046

Kim, J., Thompson, E. A., Walsh, E. M., & Schepp, K. G. (2015). Trajectories of parent-adolescent relationship quality among at-risk youth: Parental depression and adolescent developmental outcomes. *Archives of Psychiatric Nursing*, *29*(6), 434–440. doi:10.1016/j.apnu.2015.07.001

Kirschbaum, C., Klauer, T., Filipp, S.-H., & Hellhammer, D. H. (1995). Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. *Psychosomatic Medicine*, *57*(1), 23–31. doi:10.1097/00006842-199501000-00004

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test" – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1-2), 76–81. doi:10.1159/000119004

Klimes-Dougan, B., Brand, A. E., Zahn-Waxler, C., Usher, B., Hastings, P. D., Kendziora, K., & Garside, R. B. (2007). Parental emotion socialization in adolescence: Differences in sex, age and problem status. *Social Development*, *16*(2), 326–342. doi:10.1111/j.1467-9507.2007.00387.x

Kluczniok, D., Boedeker, K., Fuchs, A., Hindi Attar, C., Fydrich, T., Fuehrer, D., … Bermpohl, F. (2016). Emotional availability in mother-child interaction: The effects of maternal depression in remission and additional history of childhood abuse. *Depression and Anxiety*, *33*(7), 648–657. doi:10.1002/da.22462

Kovacs, M. (1996). Presentation and course of major depressive disorder during childhood and later years of the life span. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*(6), 705–15. doi:10.1097/00004583-199606000-00010

Kovacs, M. (2010). *Children's Depression Inventory 2*. Pearson.

Kovacs, M., Bylsma, L. M., Yaroslavsky, I., Rottenberg, J., George, C. J., Kiss, E., … Kapornai, K. (2015). Positive affectivity is dampened in youths with histories of major depression and their never-depressed adolescent siblings. *Clinical Psychological Science*, 1–14. doi:10.1177/2167702615607182

Kovacs, M., & Lopez-Duran, N. (2010). Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: Implications for prevention. *Journal of Child Psychology and Psychiatry*, *51*(4), 472–496. doi:10.1111/j.1469-7610.2010.02230.x

Kovacs, M., & Yaroslavsky, I. (2014). Practitioner review: Dysphoria and its regulation in child and adolescent depression. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *55*(7), 741–57. doi:10.1111/jcpp.12172

Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology*, *34*(1), 2–18. doi:10.1016/j.psyneuen.2008.10.004

Kudielka, B. M., Hellhammer, H., & Kirschbaum, C. (2007). Ten years of research with the Trier Social Stress Test. In E. Harmon-Jones & P. Winkielman (Eds.), *Social Neuroscience: Integrating biological and psychological explanations of social behavior.* (pp. 56–83). New

York, NY: Guilford Press.

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology*, *69*(1), 113–132. doi:10.1016/j.biopsycho.2004.11.009

Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology*, *29*(8), 983–992. doi:10.1016/j.psyneuen.2003.08.009

Kuhlman, K. R., Olson, S. L., & Lopez-Duran, N. L. (2014). Predicting developmental changes in internalizing symptoms: Examining the interplay between parenting and neuroendocrine stress reactivity. *Developmental Psychobiology*, *56*(5), 908–923. doi:10.1002/dev.21166

Kutcher, S., Kusumakar, V., LeBlanc, J., Santor, D., Lagace, D., & Morehouse, R. (2004). The characteristics of asymptomatic female adolescents at high risk for depression: the baseline assessment from a prospective 8-year study. *Journal of Affective Disorders*, *79*(1-3), 177–85. doi:10.1016/S0165-0327(02)00458-5

Lakdawalla, Z., Hankin, B. L., & Mermelstein, R. (2007). Cognitive theories of depression in children and adolescents: A conceptual and quantitative review. *Clinical Child and Family Psychology Review*, *10*(1), 1–24. doi:10.1007/s10567-006-0013-1

Lakey, B., & Cohen, S. (2000). Social support theory and measurement. In S. Cohen, L. Underwood, & B. Gottlieb (Eds.), *Social Support Measurement and Interventions: A Guide for Health and Social Scientists* (pp. 29–52). Oxford, UK: Oxford University Press. Retrieved from http://mirlyn.lib.umich.edu/Record/010335377

Larson, R. W., Moneta, G., Richards, M. H., & Wilson, S. (2002). Continuity, stability, and change in daily emotional experience across adolescence. *Child Development*, *73*(4), 1151–1165. doi:10.1111/1467-8624.00464

Larson, R. W., Richards, M. H., Moneta, G., Holmbeck, G., & Duckett, E. (1996). Changes in adolescents' daily interactions with their families from ages 10 to 18: Disengagement and transformation. *Developmental Psychology*, *32*(4), 744–754. doi:10.1037/0012-1649.32.4.744

Laursen, B., & Collins, W. (2009). Parent—child relationships during adolescence. In *Handbook of adolescent psychology* (pp. 3–xx). John Wiley & Sons, Inc. doi:10.1002/9780470479193.adlpsy002002

Laursen, B., & Collins, W. A. (1994). Interpersonal conflict during adolescence. *Psychological Bulletin*, *115*(2), 197–209. doi:10.1037//0033-2909.115.2.197

Laursen, B., Coy, K. C., & Collins, W. A. (1998). Reconsidering changes in parent-child conflict across adolescence: A meta-analysis. *Child Development*, *69*(3), 817–832. doi:10.1111/j.1467-8624.1998.tb06245.x

Leckman, J. F. (1982). Best estimate of lifetime psychiatric diagnosis. *Archives of General Psychiatry*, *39*(8), 879. doi:10.1001/archpsyc.1982.04290080001001

LeMoult, J., Chen, M. C., Foland-Ross, L. C., Burley, H. W., & Gotlib, I. H. (2015). Concordance of mother–daughter diurnal cortisol production: Understanding the

intergenerational transmission of risk for depression. *Biological Psychology*, *108*, 98–104. doi:10.1016/j.biopsycho.2015.03.019

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, *34*(9), 1272–83. doi:10.1016/j.psyneuen.2009.03.016

Lopez-Duran, N. L., Mayer, S. E., & Abelson, J. L. (2014). Modeling neuroendocrine stress reactivity in salivary cortisol: Adjusting for peak latency variability. *Stress*, *17*(4), 285–295. doi:10.3109/10253890.2014.915517

Lopez-Duran, N. L., McGinnis, E., Kuhlman, K., Geiss, E., Vargas, I., & Mayer, S. (2015). HPA-axis stress reactivity in youth depression: Evidence of impaired regulatory processes in depressed boys. *Stress*, *18*(5), 545–553. doi:10.3109/10253890.2015.1053455

Lougheed, J. P., Craig, W. M., Pepler, D., Connolly, J., O'Hara, A., Granic, I., & Hollenstein, T. (2015). Maternal and peer regulation of adolescent emotion: Associations with depressive symptoms. *Journal of Abnormal Child Psychology*. doi:10.1007/s10802-015-0084-x

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior. *Clinical Psychology Review*, *20*(5), 561–592. doi:10.1016/S0272-7358(98)00100-7

Lunkenheimer, E. S., Albrecht, E. C., & Kemp, C. J. (2013). Dyadic flexibility in early parent-child interactions: Relations with maternal depressive symptoms and child negativity and behaviour problems. *Infant and Child Development*, *22*(3), 250–269. doi:10.1002/icd.1783

Lunkenheimer, E. S., Olson, S. L., Hollenstein, T., Sameroff, A. J., & Winter, C. (2011). Dyadic flexibility and positive affect in parent–child coregulation and the development of child behavior problems. *Development and Psychopathology*, *23*(02), 577–591. doi:10.1017/S095457941100006X

Lunkenheimer, E. S., Shields, A. M., & Cortina, K. S. (2007). Parental emotion coaching and dismissing in family interaction. *Social Development*, *16*(2), 232–248. doi:10.1111/j.1467-9507.2007.00382.x

Malatesta-Magai, C. (1991). Emotional socialization: Its role in personality and developmental psychopathology. In D. Cicchetti & S. L. Toth (Eds.), *Internalizing and Externalizing Expressions of Dysfunction*. Psychology Press. Retrieved from https://books.google.com/books?hl=en&lr=&id=CYcTkAwcclUC&oi=fnd&pg=PA203&ots=E84VT1QX5W&sig=yf6i4VM_3Z9RNco9RmqPGIhZEIs

Marroquín, B. (2011). Interpersonal emotion regulation as a mechanism of social support in depression. *Clinical Psychology Review*, *31*(8), 1276–90. doi:10.1016/j.cpr.2011.09.005

Mason, W. A., Chmelka, M. B., Trudeau, L., & Spoth, R. L. (2016). Gender moderation of the intergenerational transmission and stability of depressive symptoms from early adolescence to early adulthood. *Journal of Youth and Adolescence*. doi:10.1007/s10964-016-0480-8

McEwen, B. S. (1998a). Protective and damaging effects of stress mediators. *The New England Journal of Medicine*, *338*(3), 171–9. doi:10.1056/NEJM199801153380307

McEwen, B. S. (1998b). Stress, adaptation, and disease. Allostasis and allostatic load. *Annals of*

the New York Academy of Sciences, *840*, 33–44. doi:10.1111/j.1749-6632.1998.tb09546.x

McMakin, D. L., Burkhouse, K. L., Olino, T. M., Siegle, G. J., Dahl, R. E., & Silk, J. S. (2011). Affective functioning among early adolescents at high and low familial risk for depression and their mothers: A focus on individual and transactional processes across contexts. *Journal of Abnormal Child Psychology*, *39*(8), 1213–1225. doi:10.1007/s10802-011-9540-4

Meuwly, N., Bodenmann, G., Germann, J., Bradbury, T. N., Ditzen, B., & Heinrichs, M. (2012). Dyadic coping, insecure attachment, and cortisol stress recovery following experimentally induced stress. *Journal of Family Psychology*, *26*(6), 937–47. doi:10.1037/a0030356

Miller, A. (2007). Social neuroscience of child and adolescent depression. *Brain and Cognition*, *65*(1), 47–68. doi:10.1016/j.bandc.2006.02.008

Miller, D. B., & O'Callaghan, J. P. (2002). Neuroendocrine aspects of the response to stress. *Metabolism*, *51*(6), 5–10. doi:10.1053/meta.2002.33184

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*(1), 25–45. doi:10.1037/0033-2909.133.1.25

Miller, L., Warner, V., Wickramaratne, P., & Weissman, M. (1999). Self-esteem and depression: Ten year follow-up of mothers and offspring. *Journal of Affective Disorders*, *52*(1-3), 41–49. doi:10.1016/S0165-0327(98)00042-1

Miller, R., & Plessow, F. (2013). Transformation techniques for cross-sectional and longitudinal endocrine data: Application to salivary cortisol concentrations. *Psychoneuroendocrinology*, *38*(6), 941–946. doi:10.1016/j.psyneuen.2012.09.013

Miller-Slough, R. L., & Dunsmore, J. C. (2016). Parent and friend emotion socialization in adolescence: Associations with psychological adjustment. *Adolescent Research Review*. doi:10.1007/s40894-016-0026-z

Monti, J. D., Rudolph, K. D., & Abaied, J. L. (2014). Contributions of maternal emotional functioning to socialization of coping. *Journal of Social and Personal Relationships*, *31*(2), 247–269. doi:10.1177/0265407513492304

Morris, A. S., Silk, J. S., Morris, M. D. S., Steinberg, L., Aucoin, K. J., & Keyes, A. W. (2011). The influence of mother-child emotion regulation strategies on children's expression of anger and sadness. *Developmental Psychology*, *47*(1), 213–225. doi:10.1037/a0021021

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Soc Dev.*, *16*(2), 361–388. doi:10.1111/j.1467-9507.2007.00389.x

Morris, M. C., Ciesla, J. A., & Garber, J. (2010). A prospective study of stress autonomy versus stress sensitization in adolescents at varied risk for depression. *Journal of Abnormal Psychology*, *119*(2), 341–54. doi:10.1037/a0019036

Nelemans, S. A., Hale, W. W., Branje, S. J. T., Hawk, S. T., & Meeus, W. H. J. (2014). Maternal criticism and adolescent depressive and generalized anxiety disorder symptoms: A 6-year longitudinal community study. *Journal of Abnormal Child Psychology*, *42*(5), 755–766. doi:10.1007/s10802-013-9817-x

Nolen-Hoeksema, S., & Girgus, J. S. J. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin*, *115*(3), 424–443. doi:10.1037/0033-2909.115.3.424

Olino, T. M., Lopez-Duran, N. L., Kovacs, M., George, C. J. C., Gentzler, A. A. L., & Shaw, D. S. D. (2011). Developmental trajectories of positive and negative affect in children at high and low familial risk for depressive disorder. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *52*(7), 792–9. doi:10.1111/j.1469-7610.2010.02331.x

Olino, T. M., McMakin, D. L., Nicely, T. A., Forbes, E. E., Dahl, R. E., & Silk, J. S. (2016). Maternal depression, parenting, and youth depressive symptoms: Mediation and moderation in a short-term longitudinal study. *Journal of Clinical Child & Adolescent Psychology*, *45*(3), 279–290. doi:10.1080/15374416.2014.971456

Papini, D. R., Farmer, F. F., Clark, S. M., Micka, J. C., & Barnett, J. K. (1990). Early adolescent age and gender differences in patterns of emotional self-disclosure to parents and friends. *Adolescence*, *25*(100), 959–976.

Pelham, W. E., Gnagy, E. M., Greenslade, K. E., & Milich, R. (1992). Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*(2), 210–8. doi:10.1097/00004583-199203000-00006

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, *17*(2), 117–33. doi:10.1007/BF01537962

Phillips, A. C., Ginty, A. T., & Hughes, B. M. (2013). The other side of the coin: Blunted cardiovascular and cortisol reactivity are associated with negative health outcomes. *International Journal of Psychophysiology*, *90*(1), 1–7. doi:10.1016/j.ijpsycho.2013.02.002

Pineda, A. Q., Cole, D. A., & Bruce, A. E. (2007). Mother-adolescent interactions and adolescent depressive symptoms: A sequential analysis. *Journal of Social and Personal Relationships*, *24*(1), 5–19. doi:10.1177/0265407507072564

Power, T. G. (2004). Stress and coping in childhood: The parents' role. *Parenting*, *4*(4), 271–317. doi:10.1207/s15327922par0404_1

Ramchandani, P., Stein, A., Evans, J., & O'Connor, T. G. (2005). Paternal depression in the postnatal period and child development: A prospective population study. *Lancet*, *365*(9478), 2201–2205. doi:10.1016/S0140-6736(05)66778-5

Reising, M. M., Watson, K. H., Hardcastle, E. J., Merchant, M. J., Roberts, L., Forehand, R., & Compas, B. E. (2013). Parental depression and economic disadvantage: The role of parenting in associations with internalizing and externalizing symptoms in children and adolescents. *Journal of Child and Family Studies*, *22*(3), 335–343. doi:10.1007/s10826-012-9582-4

Restifo, K., & Bögels, S. (2009). Family processes in the development of youth depression: translating the evidence to treatment. *Clinical Psychology Review*, *29*(4), 294–316. doi:10.1016/j.cpr.2009.02.005

Rohde, P., Lewinsohn, P. M., Klein, D. N., Seeley, J. R., & Gau, J. M. (2013). Key

characteristics of major depressive disorder occurring in childhood, adolescence, emerging adulthood, adulthood. *Clinical Psychological Science*, *1*(1), 1–21. doi:10.1177/2167702612457599

Rueger, S. Y., Malecki, C. K., & Demaray, M. K. (2010). Relationship between multiple sources of perceived social support and psychological and academic adjustment in early adolescence: comparisons across gender. *Journal of Youth and Adolescence*, *39*(1), 47–61. doi:10.1007/s10964-008-9368-6

Ruttle, P. L., Shirtcliff, E. A., Serbin, L. A., Fisher, D. B.-D., Stack, D. M., & Schwartzman, A. E. (2011). Disentangling psychobiological mechanisms underlying internalizing and externalizing behaviors in youth: longitudinal and concurrent associations with cortisol. *Hormones and Behavior*, *59*(1), 123–32. doi:10.1016/j.yhbeh.2010.10.015

Sameroff, A. J., & Fiese, B. (2000). Transactional regulation: The developmental ecology of early intervention. In J. P. Shonkoff & S. J. Meisels (Eds.), *Handbook of Early Childhood Intervention* (2nd ed., Vol. 2, pp. 135–159). New York, NY US: Cambridge University Press.

Sanders, W., Zeman, J., Poon, J., & Miller, R. (2015). Child regulation of negative emotions and depressive symptoms: The moderating role of parental emotion socialization. *Journal of Child and Family Studies*, *24*(2), 402–415. doi:10.1007/s10826-013-9850-y

Schwartz, O. S., Dudgeon, P., Sheeber, L. B., Yap, M. B. H., Simmons, J. G., & Allen, N. B. (2012). Parental behaviors during family interactions predict changes in depression and anxiety symptoms during adolescence. *Journal of Abnormal Child Psychology*, *40*(1), 59–71. doi:10.1007/s10802-011-9542-2

Schwartz, O. S., Sheeber, L. B., Dudgeon, P., & Allen, N. B. (2012). Emotion socialization within the family environment and adolescent depression. *Clinical Psychology Review*, *32*(6), 447–453. doi:10.1016/j.cpr.2012.05.002

Seltzer, L. J., Ziegler, T. E., & Pollak, S. D. (2010). Social vocalizations can release oxytocin in humans. *Proceedings. Biological Sciences / The Royal Society*, *277*(1694), 2661–6. doi:10.1098/rspb.2010.0567

Shaw, D. S., Schonberg, M., Sherrill, J., Huffman, D., Lukon, J., Obrosky, D., & Kovacs, M. (2006). Responsivity to offspring's expression of emotion among childhood-onset depressed mothers. *Journal of Clinical Child & Adolescent Psychology*, *35*(4), 490–503. doi:10.1207/s15374424jccp3504_1

Sheeber, L. B., Allen, N., Davis, B., & Sorensen, E. (2000). Regulation of negative affect during mother–child problem-solving interactions: Adolescent depressive status and family processes. *Journal of Abnormal Child Psychology*, *28*(5), 467–479. doi:10.1023/A:1005135706799

Sheeber, L. B., Davis, B., & Hops, H. (2002). Gender-specific vulnerability to depression in children of depressed mothers. *Children of Depressed Parents: Mechanisms of Risk and Implications for Treatment*, (September 2015), 253–276. doi:10.1037/10449-010

Silk, J. S., Shaw, D. S., Prout, J. T., O'Rourke, F., Lane, T. J., & Kovacs, M. (2011). Socialization of emotion and offspring internalizing symptoms in mothers with childhood-

onset depression. *Journal of Applied Developmental Psychology*, *32*(3), 127–136. doi:10.1016/j.appdev.2011.02.001

Silk, J. S., Shaw, D. S., Skuban, E. M., Oland, A. A., & Kovacs, M. (2006). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *47*(1), 69–78. doi:10.1111/j.1469-7610.2005.01440.x

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child Development*, *74*(6), 1869–1880. doi:10.1046/j.1467-8624.2003.00643.x

Smetana, J. G., Campione-Barr, N., & Metzger, A. (2006). Adolescent development in interpersonal and societal contexts. *Annual Review of Psychology*, *57*, 255–84. doi:10.1146/annurev.psych.57.102904.190124

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Annual Review of Psychology*, *52*, 83–110. doi:10.1146/annurev.psych.52.1.83

Stice, E., Ragan, J., & Randall, P. (2004). Prospective relations between social support and depression: Differential direction of effects for parent and peer support? *Journal of Abnormal Psychology*, *113*(1), 155–159. doi:10.1037/0021-843X.113.1.155

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*(01), 47. doi:10.1017/S0954579409000042

Tarullo, L. B., DeMulder, E. K., Martinez, P. E., & Radke-Yarrow, M. (1994). Dialogues with preadolescents and adolescents: Mother-child interaction patterns in affectively ill and well dyads. *Journal of Abnormal Child Psychology*, *22*(1), 33–51. doi:10.1007/BF02169255

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. a, & Updegraff, J. a. (2000). Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychological Review*, *107*(3), 411–429. doi:10.1037/0033-295X.107.3.411

Thoits, P. A. (2011). Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior*, *52*(2), 145–61. doi:10.1177/0022146510395592

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, *53*(4), 865–871. doi:10.1016/S0022-3999(02)00429-4

Uchino, B. N., Bowen, K., Carlisle, M., & Birmingham, W. (2012). Psychological pathways linking social support to health outcomes: A visit with the "ghosts" of research past, present, and future. *Social Science & Medicine (1982)*, *74*(7), 949–57. doi:10.1016/j.socscimed.2011.11.023

Uchino, B. N., Carlisle, M., Birmingham, W., & Vaughn, A. A. (2011). Social support and the reactivity hypothesis: Conceptual issues in examining the efficacy of received support during acute psychological stress. *Biological Psychology*, *86*(2), 137–42. doi:10.1016/j.biopsycho.2010.04.003

UCLA: Statistical Consulting Group. (2016). Ordered Logistic Regression. Retrieved June 4, 2016, from http://www.ats.ucla.edu/stat/spss/dae/ologit.htm

van Santvoort, F., Hosman, C. M. H., Janssens, J. M. A. M., van Doesum, K. T. M., Reupert, A., & van Loon, L. M. A. (2015). The impact of various parental mental disorders on children's diagnoses: A systematic review. *Clinical Child and Family Psychology Review*, *18*(4), 281–299. doi:10.1007/s10567-015-0191-9

Waugh, C. E., Muhtadie, L., Thompson, R. J., Joormann, J., & Gotlib, I. H. (2012). Affective and physiological responses to stress in girls at elevated risk for depression. *Development and Psychopathology*, *24*(2), 661–75. doi:10.1017/S0954579412000235

Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Pilowsky, D., & Verdeli, H. (2006). Offspring of depressed parents: 20 years later. *The American Journal of Psychiatry*, *163*(6), 1001–8. doi:10.1176/appi.ajp.163.6.1001

Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Verdeli, H., Pilowsky, D. J., … Bruder, G. (2005). Families at high and low risk for depression: A 3-generation study. *Archives of General Psychiatry*, *62*(1), 29–36. doi:10.1001/archpsyc.62.1.29

Willemen, A. M., Schuengel, C., & Koot, H. M. (2009). Physiological regulation of stress in referred adolescents: The role of the parent-adolescent relationship. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *50*(4), 482–90. doi:10.1111/j.1469-7610.2008.01982.x

Williamson, D. E., Birmaher, B., Axelson, D. A., Ryan, N. D., & Dahl, R. E. (2004). First episode of depression in children at low and high familial risk for depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(3), 291–7. doi:10.1097/00004583-200403000-00010

Wilson, S., & Durbin, C. E. (2010). Effects of paternal depression on fathers' parenting behaviors: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 167–180. doi:10.1016/j.cpr.2009.10.007

Yap, M. B. H., Allen, N. B., & Ladouceur, C. D. (2008). Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development*, *79*(5), 1415–31. doi:10.1111/j.1467-8624.2008.01196.x

Yap, M. B. H., Allen, N. B., & Sheeber, L. B. (2007). Using an emotion regulation framework to understand the role of temperament and family processes in risk for adolescent depressive disorders. *Clinical Child and Family Psychology Review*, *10*(2), 180–96. doi:10.1007/s10567-006-0014-0

Yap, M. B. H., Schwartz, O. S., Byrne, M. L., Simmons, J. G., & Allen, N. B. (2010). Maternal positive and negative interaction behaviors and early adolescents' depressive symptoms: Adolescent emotion regulation as a mediator. *Journal of Research on Adolescence*, *20*(4), 1014–1043. doi:10.1111/j.1532-7795.2010.00665.x

Zalewski, M., Thompson, S. F., & Lengua, L. J. (2015). Parenting as a moderator of the effects of maternal depressive symptoms on preadolescent adjustment. *Journal of Clinical Child & Adolescent Psychology*, *4416*(April), 1–10. doi:10.1080/15374416.2015.1030752

Zimet, G. D., Dahlem, N. W., Zimet, S. G., & Farley, G. K. (1988). The Multidimensional Scale of Perceived Social Support. *Journal of Personality Assessment*, *52*(1), 30–41. doi:10.1207/s15327752jpa5201_2

Zimet, G. D., Powell, S. S., Farley, G. K., Werkman, S., & Berkoff, K. A. (1990). Psychometric characteristics of the Multidimensional Scale of Perceived Social Support. *Journal of Personality Assessment*, *55*(3-4), 610–617. doi:10.1207/s15327752jpa5503&4_17