Parenting and Family-related Protective Factors for Adolescents at High Risk for Depression and Suicide

by

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Dedication

To my daughter, Ella, who is the most wonderful person that I know. May you always reach for your dreams.

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I strongly believe that the "It takes a village" adage does not only apply to raising children, but that a supportive village continues to be necessary throughout our lives. I am blessed and eternally grateful for the most wonderful of villages. My greatest hope is that the gifts I have received from you all will come back to each of you in a myriad of ways, and that this dissertation honors those gifts.

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Abstract

Adolescence is marked by increases in stress and the onset of stress-related mental health disorders. There is variability in outcomes after stress exposure, which is likely influenced by a variety of factors, including parental and familial characteristics that may serve to mitigate the harmful effects of stress. This dissertation includes studies to examine how parents play a regulatory role in helping their adolescents cope with stress. The studies are drawn from two at-risk samples: one at high risk for depression, and one at high risk for suicidal behavior. The first study examines whether parents and family help to buffer the neuroendocrine stress response in adolescents who have participated in a controlled laboratory stress task. We found that the perception of parent and family support was related to neuroendocrine regulation, but not the presence of the parent. The second study explores parental and familial factors that may help to mitigate the association between stressful life events in the past year and internalizing symptoms in adolescents. We found that stress exposure was related to internalizing symptoms but that parent and family factors did not buffer this association. The third study characterizes levels of parental caregiving stress for parents of hospitalized youth across three months post-discharge, explores factors associated with caregiving stress, and explores the impact of caregiving stress on the parental ability to support their teen post-discharge. We found that caregiving stress declined over time, that risk and protective factors influence this decline, and that both stress and self-efficacy were related to increases in support post-discharge. In sum, this dissertation highlights the, sometimes limited, role of parents in helping their teens regulate.

Keywords: stress, adolescence, depression, anxiety, suicide, parenting, family, cortisol, caregiving stress

Chapter 1 Dissertation Overview

Stress increases during adolescence and can have a negative impact on adolescent mental health outcomes (Roberts & Lopez-Duran, 2019). However, there is significant variability in adolescents' ability to cope with stress, and this variability is likely influenced by a variety of individual and family-level factors. Despite literature that suggests parents lose power as regulatory entities during the teen years as support becomes more peer-focused (Hostinar, Johnson, & Gunnar, 2015; Hostinar, Sullivan, & Gunnar, 2014), there is emerging evidence suggesting that this may not be true for all adolescents – particularly for populations at higher risk for mental health difficulties (Miller, Esposito-Smythers, & Leichtweis, 2015). Because parents can continue to serve as supportive figures in their adolescents' lives, their support may be especially important for helping adolescents at risk for depression and suicide cope with stress.

Stress increases significantly during adolescence as a function of increases in academic (Torsheim & Wold, 2001) and social demands (Nelson, Leibenluft, McClure, & Pine, 2005). Experiences with stress in adolescence are normative and most adolescents successfully navigate this developmental period with few negative outcomes. This is not surprising given that adolescence involves the maturation of the regulatory processes that help teens cope with stress (Roberts & Lopez-Duran, 2019). Nonetheless, many adolescents experience significant stress-related negative impacts on school functioning (Kaplan, Liu, & Kaplan, 2005), mental health (Lopez-Duran, Micol, & Roberts, 2019), and risk for suicide (Miller & Prinstein, 2019). Given

the potential for negative consequences of stress, it is important to understand the protective factors related to stress regulation during the adolescent developmental period.

It is well-documented that parental support plays a protective role in stress regulation for children early in development (Ellenbogen & Hodgins, 2009; Kuhlman, Olson, & Lopez-Duran, 2014; Zhang, Gatzke-Kopp, Fosco, & Bierman, 2020). While for typically developing teens there is a shift in sources of support from parents to peers as they progress into adolescence (Bokhorst, Sumter, & Westenberg, 2010), parents continue to be a source of support for teens with additional needs (Miller et al., 2015). Indeed, parents can help regulate stress for teens in laboratory settings (Doom, Doyle, & Gunnar, 2017), and parental support has even been shown to be more important than peer support in some higher-risk contexts, such as adolescents at high risk for suicidal behavior (Kang et al., 2017; Mackin, Perlman, Davila, Kotov, & Klein, 2017). Thus, studying parenting factors for higher risk adolescents, particularly when risk is conferred from parental characteristics, might be particularly important.

In addition, there is likely variability in the ability of parents to play a regulatory role based on different factors, such as how the adolescent perceives their parent as supportive (Murphy et al., 2020), what types of supportive behaviors the parent displays when their teen is stressed (Yap, Schwartz, Byrne, Simmons, & Allen, 2010), and the context of larger family functioning (Simpson, Vannucci, & Ohannessian, 2018). Additionally, caring for high-risk teens is stressful for parents (Lindqvist, Johansson, & Karlsson, 2008), and parental stress itself likely plays a role in how efficacious parents are in helping their teens regulate (Lee, Lee, & Han, 2019). Thus, research on how parents influence adolescent regulation, particularly in high-stress contexts, is warranted and may provide the basis for therapeutic interventions for high-risk youth and their families.

Stress can be measured physiologically through neuroendocrine markers such as the stress hormone cortisol, the end-product of the hypothalamic-pituitary-adrenal (HPA) axis (Tsigos & Chrousos, 2002). Using cortisol as a measure of stress regulation, childhood research has documented the phenomenon of parental presence providing social buffering to laboratory-induced stress in children (Ahnert, Gunnar, Lamb, & Barthel, 2004; Gunnar, 2006; Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). The social buffering effect of parental presence decreases as children reach puberty (Hostinar et al., 2015, 2014), but little is known about the social buffering effect of parents of high-risk adolescents. Additionally, to our knowledge, no study has examined whether perceived parental support, parental supportive behaviors, and family functioning help to increase the buffering effect to a laboratory stressor in adolescents, and whether or not these factors are more important for teens at high-risk for depression. Therefore, in study 1 of this dissertation, we explore how parental and family support may help to increase the social buffering effect of parental presence in adolescents who have participated in a controlled laboratory stress task, and whether this differs for high and low risk adolescents.

Additionally, exposure to stress has an impact on adolescent mental wellbeing (Roberts & Lopez-Duran, 2019; Tafet & Nemeroff, 2016). Specifically, exposure to stressful life events has been associated with increases in depression and anxiety symptoms (McLaughlin & Hatzenbuehler, 2009; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Waaktaar, Borge, Fundingsrud, Christie, & Torgersen, 2004). Parental support plays a role in helping to buffer this negative impact of life stress on internalizing disorders (Buckholdt, Parra, & Jobe-Shields, 2014; Stocker, Richmond, Rhoades, & Kiang, 2007) and supportive parental behaviors are associated with lower internalizing symptoms (Buckholdt et al., 2014). Despite evidence that perceived support and supportive parenting behaviors are associated with decreases in stress and

stress-related outcomes, to our knowledge, no study has examined whether parental support and *observed* supportive behaviors mitigate the impact of *recent* life stressors on internalizing symptoms in high-risk adolescents. In study 2 of this dissertation, we examine how parental and familial factors (both self-report: perceived social support and family functioning; and observed: video coding of parental behaviors) may help to mitigate the association between stressful life events in the past year and internalizing symptoms in high and low risk adolescents.

Finally, caring for a teen who has been hospitalized for suicide ideation or attempt is very stressful (Ha, Hong, Seltzer, & Greenberg, 2008), however, no quantitative study has documented levels of post-discharge caregiving stress for parents of adolescents who have been psychiatrically hospitalized. Additionally, caregiving stress is related to negative outcomes for parents (Ngwane & van der Wath, 2019) and teens (Lee et al., 2019). The extent to which caregiving stress harms adolescent outcomes is likely influenced by a variety of factors such as adolescents' perceived social support from their parents (Kidd et al., 2006) and parents' own sense of parental self-efficacy (Albanese, Russo, & Geller, 2019). Parents of psychiatrically hospitalized youth also play a unique role in keeping their adolescents safe post-discharge as they are charged with providing emotional and safety-related support to their adolescents (King, Ewell-Foster, & Rogalski, 2013; Nock & Ferriter, 2005; O'Brien, Crickard, Lee, & Holmes, 2013). Given that parents play an important role in keeping their adolescents safe after discharge from psychiatric hospital admission, and caregiving stress in that period is high, an understanding of how caregiving stress affects parental ability to implement safety recommendations and which supportive factors might mitigate that association is warranted. This understanding may help to inform the development of supportive interventions for this population. Therefore, study 3 characterizes levels of parental caregiving stress across three

months post-discharge and examines parent- and adolescent-level characteristics that are associated with caregiving stress in parents of hospitalized youth. We also explore the impact of caregiving stress on the parental ability to support their teen post-discharge, and whether parental efficacy may help to mitigate the negative effects of caregiving stress.

In summary, this dissertation examines parental and family factors as regulatory agents for stress in high-risk samples of adolescents. These three studies are drawn from two different research samples across three contexts: a controlled acute stressor (regulating after a laboratory stressor), less acute, naturalistic stressors (stressful life events over the past year), and an acute stressful naturalistic situation (coping after a suicide attempt). Study 1 and Study 2 are drawn from a sample where participants were oversampled for parental depression risk. This higher risk sample provides the benefit of increased variability in depression and anxiety symptoms and typical and atypical coping since high-risk kids have a greater risk for psychopathology. This sample also allows us to explore whether familial risk for depression differentially impact these parental and familial regulatory processes. Finally, Study 3 is drawn from a sample of parent and adolescent participants for whom the adolescent had been hospitalized for a suicide attempt or ideation. This study sample allows us to examine parental stress in a high-risk context, where parents are charged with helping their teens to regulate post-discharge. Therefore, this dissertation is a unique exploration of parental factors that help adolescents regulate in stressful contexts in two unique samples and across three distinct stressful contexts.

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Chapter 2 Parents as Regulators of the Adolescent Neuroendocrine Response to Laboratory Stress

Abstract

Parent support has a positive influence on the development of stress regulation for children. However, adolescence is associated with decreased utilization of parents as regulators. We examined how parent and family-level support factors were related to endocrine responses to a laboratory stressor in a sample over-represented by adolescents with a family history of depression. We also examined whether parental presence post-stress was associated with a social buffering effect for this sample and examined whether this association increased under the condition of greater perceived parental support, observed supportive parental behaviors, and perceived positive family functioning. We also examined whether there were differences in these associations between adolescents at high and low risk for depression. Our sample was 145 adolescents aged 12-16 years and one of their parents, of which over half (n=85) had a history of depression. Adolescents completed a laboratory stress task (TSST) followed by randomization to parent present or absent conditions where parents were to provide support. Videos were coded for positive and negative parenting behaviors. Salivary cortisol samples were also collected throughout the visit. Adolescents completed self-report questionnaires regarding their perception of positive family functioning and parent support. We found that perceived parent support and parental supportive behaviors were associated with regulation of the HPA axis post-stressor. However, we failed to find a stress-buffering effect of parenting presence in our sample, except in the high-risk adolescents. Further, the buffering effect remained non-existent even in the

context of greater parenting/family supportive factors. Our findings suggest that more positive parenting is associated with HPA regulation, but that it is the perception of this support, not the presence of the parent that has an impact on most of the adolescents in our sample.

Keywords: stress, adolescence, depression, parenting, family, cortisol

Introduction

Parenting support and behaviors have an important influence on the development of stress regulation (Ellenbogen & Hodgins, 2009; Kuhlman, Olson, & Lopez-Duran, 2014; Zhang, Gatzke-Kopp, Fosco, & Bierman, 2020). Parenting socialization of adolescent emotion and their own emotionality helps to facilitate and scaffold regulatory behavior in children (Eisenberg, Cumberland, & Spinrad, 1998) but can also exacerbate the stress response (e.g., upregulation of negative affect; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). However, parents vary in their effectiveness as co-regulators (Lunkenheimer, Tiberio, Skoranski, Buss, & Cole, 2018). Although factors that contribute to such variability have been studied in childhood, much less is known about how these processes continue during adolescence and, in particular, for high-risk teens.

Stress regulation in adolescence

Adolescence is a unique period in human development when the capacity for stress regulation is especially important. Adolescence involves physiological changes and advancement of cognitive functioning (Blakemore, Burnett, & Dahl, 2010; Juraska & Willing, 2017) as well as changes in the social environment, such as individuation from the family unit (Erikson, 1968). Additionally, stress greatly increases during this developmental period (Collins & Steinberg, 2007) and this can lead to mental health-related sequelae (Roberts & Lopez-Duran, 2019; Tafet & Nemeroff, 2016). In fact, stress has been implicated as a causal and maintaining factor in psychopathology (Lopez-Duran, Micol, & Roberts, 2019), and stress-related psychopathology peaks during adolescence (Costello, Copeland, & Angold, 2011; Kessler et al., 2012).

Stress regulation research has largely focused on the measurement of stress through biological methods, such as neuroendocrine factors. Specifically, the hypothalamic-pituitary-

adrenal axis (HPA axis) is a critical component of the stress response and measuring its end-product, the hormone cortisol, is an established method for measuring stress regulation in laboratory settings (Tsigos & Chrousos, 2002). For instance, measuring cortisol output, either through saliva or blood, in response to laboratory stress tasks can provide information about how the HPA axis may be responding to stress in the real world. Additionally, this methodology can provide a basis for research on protective effects in the development of stress regulation. For example, having a parent present after a laboratory stressor may facilitate support for children. Specifically, parental social buffering against stress, whereby parental presence decreases the stress response, has been reliably demonstrated in early childhood (Ahnert, Gunnar, Lamb, & Barthel, 2004; Gunnar, 2006; Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). However, this effect declines as children enter adolescence (Doom, Hostinar, VanZomeren-Dohm, & Gunnar, 2015; Hostinar, Johnson, & Gunnar, 2015; Hostinar, Sullivan, & Gunnar, 2014). For example, a more advanced pubertal stage is associated with diminished parental buffering of initial cortisol reactivity, and older age is associated with diminished parental buffering of cortisol recovery (Doom et al., 2015). The general speculation is that this decline is due to adolescents' increased reliance on peer support for social buffering (Hostinar et al., 2014). However, it is also possible that the quality of the parent-child relationship becomes especially important in adolescence as this appears to be a potent moderator for the social buffering effect (Ahnert et al., 2004; Smith, Loving, Crockett, & Campbell, 2009).

Parental support and stress regulation

Few have examined whether parental presence post-stressor may serve to regulate adolescents (Doom et al., 2015; Hostinar et al., 2015; Seltzer, Ziegler, & Pollak, 2010), with none to our knowledge, looking at the supportive quality of the parent-adolescent relationship.

For example, Seltzer and colleagues (2010) found that the physical presence of a mother and even speaking with her over the phone after a laboratory stress task reduced the cortisol response in early adolescent girls. However, Doom and colleagues (2015) examined the buffering effect of parental presence on cortisol trajectory in a group of male and female children and later adolescents and found that the buffering effect seemed to diminish with age. However, neither of these studies included measurement of relationship quality. It could be that the variability in findings is attributed to variability in perceived social support or supportive behaviors exhibited during the stress regulation. For example, the degree to which a child is attached to their mother (evidence of the quality of the relationship) is related to the social buffering effect in infants (Ahnert et al., 2004). Likewise, in adults, relationship factors related to closeness are necessary for social buffering (Smith et al., 2009). More generally, the presence of a perceived supportive person post-stress has been shown to have stress-buffering effects on cortisol response in adults (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; McQuaid et al., 2016). Further, perceived parental support is a strong protective factor across adolescence and is associated with protection from stress-related psychopathology (Gariépy, Honkaniemi, & Quesnel-Vallée, 2016). Given that quality of the parent-child relationship in adolescence serves as protection against harmful outcomes of stress exposure (Hazel, Oppenheimer, Technow, Young, & Hankin, 2014), variability in whether parental presence provides social buffering for adolescents could be a function of the quality of parental support.

Family functioning and stress regulation

Parental effectiveness in facilitating stress regulation may be enhanced by an adolescent's perception of a good family relationship. Positive family functioning, which includes healthy decision-making, expression of feelings, communication, acceptance, and support during crises,

has been studied as a protective factor in the context of high-stress environments. Specifically, poorer family functioning has been shown to have an impact on negative outcomes in adolescents such as greater substance use (Hummel, Shelton, Heron, Moore, & van den Bree, 2013), externalizing problems (Henderson, Dakof, Schwartz, & Liddle, 2006), and internalizing symptoms (Simpson, Vannucci, & Ohannessian, 2018). Additionally, greater family cohesion is a protective factor in reducing the negative impact of early childhood adversity on adolescent mental and physical wellbeing (Balistreri & Alvira-Hammond, 2016), reducing the impact of community violence exposure to violence perpetration later in life (Gorman-Smith, Henry, & Tolan, 2004), and creating a greater quality of life for adolescents with internalizing disorders (Jozefiak & Wallander, 2016). Given the protective nature of family functioning across domains of stress exposure, it is likely that parental presence may facilitate regulation for adolescents under the circumstance of greater perceived family functioning. However, to our knowledge, no studies have examined how family functioning might enhance the social buffering impact of parental presence on stress regulation in adolescents.

Parenting behaviors during stress regulation

In addition to perceived parental support and family functioning, how parents are interacting with their adolescent post-stressor may also influence the stress-buffering impact of parental presence. Supportive parent behaviors and parental warmth have been shown to impact the HPA axis and buffer from internalizing symptoms (Kuhlman et al., 2014). Similarly, greater maternal supportive behaviors and less unsupportive behaviors are related to greater emotion regulation ability and fewer depressive symptoms in adolescents (Yap, Schwartz, Byrne, Simmons, & Allen, 2010). Given these associations, it is likely that parental social buffering in

the context of acute stress would be heavily influenced by the degree to which a parent is exhibiting in-the-moment positive parenting behaviors.

Impact of parental psychopathology on stress regulation

Conversely, parental effectiveness in helping their teens regulate may be negatively impacted by certain parental experiences or attributes. For example, parental history of depression is a known risk factor for adolescent depression (Gotlib, Joormann, & Foland-Ross, 2014) and there are different purported mechanisms for this relationship including greater exposure to life stress (Feurer, Hammen, & Gibb, 2016) as well as an alteration of stress sensitivity itself (Bale, 2006). In fact, adolescents who have a family history of depression are not only more likely to have depression themselves, but also show alterations in cortisol secretion (Lopez-Duran, N. L. et al., 2015; Mannie, Harmer, & Cowen, 2007). Additionally, parental depression can impact in-the-moment behavioral support of an adolescent's stress response. Specifically, a parental depressive state in mothers and fathers increases negative parenting behaviors and decreases positive behaviors (Lovejoy et al., 2000; Wilson & Durbin, 2010). Despite literature supporting these mechanisms of risk, the link between parental depression and negative outcomes in teens is variable and is likely associated with a complex interplay of risk and protective factors across development (Collishaw et al., 2016). A clearer understanding of parental supportive factors in adolescent cortisol regulation, particularly for teens with parents struggling with their own mental health, is of particular importance for understanding intergenerational transmission of risk for depression. It is likely that for adolescents who face greater stress due to parental psychopathology, the stress-buffering effect of parental presence and support may be more salient (Ellenbogen & Hodgins, 2009) and these findings have implications for family and parental supportive interventions.

Conclusion and aims

This study aims to understand how parental presence during a post-laboratory stress task might facilitate the HPA-axis regulation in adolescents at high and low risk for depression. We also propose to explore specific parental and family-related factors that might serve to increase regulation (family functioning, parent support, parental supportive behaviors) or decrease regulation (family history of depression). We also aim to explore how supportive parent/family factors are related to stress regulation as a function of parental history of depression.

Aim 1

In Aim 1, we will examine how parenting and family factors are associated with adolescent regulation of the HPA axis after a laboratory stressor. Specifically, we will examine how parent and family factors such as parental supportive behaviors post-stressor, perceived family functioning, and perceived parental support will impact the cortisol trajectory. We hypothesize that parent and family factors of greater supportive parent behaviors, more cohesive family functioning and greater perceived parental support will be associated with more adaptive regulation of the HPA axis (e.g. lower cortisol peaks and steeper recovery slopes).

Aim 2

In Aim 2, we will examine whether the parenting/family factors of family functioning and parental social support moderate the association between parental presence post-stressor and cortisol trajectory. We hypothesize that family functioning and parent social support will moderate the association between parental presence and cortisol trajectory such that in the context of greater family functioning and greater parental support, parental presence will be associated with greater HPA axis regulation: a further decrease in peak values and steeper recovery slopes.

Aim 3 (Exploratory)

Our third aim is exploratory. In Aim 3, we will also examine whether depression risk status will moderate the Aim 2 associations to examine how a history of parental depression impacts the association of supportive parenting and the social buffering effect. We hypothesize that familial risk status will moderate Aim 2 associations (i.e. moderation of moderation) such that high-risk teens with their parent present and more positive family factors (parental positive behaviors, greater family functioning, higher parental support), would show similar cortisol trajectory as the low-risk teens in this same condition (parent present and high supportive factors). However, high-risk teens with their parents present and lower family support factors would show a more elevated cortisol peak and a flatter recovery slope. These findings would suggest that differences in the impact of risk status on cortisol trajectory are more strongly associated with parenting and family functioning factors than it is with a characteristic risk for depression.

Methods

Participants

Participants are 146 adolescents and one of their parents participating in a study of familial risk for depression (PI: Nestor Lopez-Duran). This study used community-based recruitment strategies to recruit two different groups, those with and without a parental history of depression. Over half, 58.22% (n = 85) of the participants had a parent with a history of depression. The majority of the parents who participated in this study with their teen were mothers (88%). The average participant age was 13.94 (*SD* = 1.35, Range 12-16). Participants were excluded from the study if they or their parents had a history of psychosis or diagnosis of Autism, Down's Syndrome, or significant neurological or medical disorders that may impact

cortisol secretion (e.g., cerebral palsy, cancer, endocrine disorder). 74.29% identified as White/Caucasian, 10.00% identified as African American/Black, 10.00% identified as Asian/Pacific Islander, 0.71% identified as Native American, and 2.86% identified as Biracial or Other. 2.14% of participants identified as Latino/Hispanic. 71.01% of adolescents reported that their parents were married or living together. The majority of participants (81.2%) reported an annual household income of $50,000 or more, including 48.12% earning greater than $100,000 annually.

This study was approved by the University Institutional Review Board and informed consent and assent were obtained from all adolescent participants and their parents. Participants were compensated $50 for their laboratory visit participation.

Procedure

Adolescents and their parents participated in an approximately 4-hour laboratory assessment. The start time of the laboratory visit was standardized (1400h) across all participants to control for diurnal variability in cortisol levels. The laboratory assessment included: parent and adolescent self-report questionnaires, adolescent stress task, and parent and adolescent clinical interviews. Adolescents were told not to eat or drink anything one hour before their visit.

Upon arrival at the laboratory, adolescents and their parents completed the informed consent/assent process before the adolescent provided their first saliva sample. They then completed self-report questionnaires in a room alone for 40 minutes to facilitate accommodation to the lab space. During this time, parents participated in clinical interviews that included a diagnostic assessment of the child and the parent, as well as a contextual stress assessment interview. After the 40-minute acclimation phase, adolescents participated in the in-lab stress task.

Saliva samples were collected at various time points throughout the process as detailed below. Directly after the stress task, adolescents were randomized to one of two conditions: 1) Parent present: teens spend the first 10 minutes of the regulation phase alone with their parents in the room, or 2) Parent absent: teens go directly to watching a neutral movie after the task and do not interact with their parents. The parent/teen interaction was video-recorded and used for behavioral coding of parental emotion socialization. All adolescents then watched the neutral movie, until the regulation period had completed. Following the regulation phase, teens participated in their own clinical interview and contextual stress interview.

Stress Task

We used the Trier Social Stress Test (TSST), a well-established social evaluative stress task designed to elicit a cortisol response (Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009). We used a modified version designed to be more applicable to adolescents (Ellenbogen & Hodgins, 2009). In this task, teens are told that they will be giving a speech to judges who are trained to analyze facial expressions and that the speech will be recorded. The teen is then given five minutes to prepare a speech on why they would be a good class president. They are then instructed to stand before the judges and a video camera to give the speech to a panel of judges. The speech itself lasts for five minutes and the judges are undergraduate confederates who are trained to keep their facial expressions neutral throughout the task and to ask questions to keep the participant speaking for the entire five minutes. Following the speech, the participants are instructed to do an arithmetic task for five minutes in front of the judges. This is a serial subtraction task where they are to start from the beginning if they make a mistake. Directly after the task, teens watched a neutral movie for 40 minutes to standardize and facilitate regulation of

the cortisol response to the task. They were then debriefed and made aware of the purpose of the task.

Cortisol Sampling

Saliva samples were collected via passive drool and were assayed using a commercially available enzyme immunoassay kit (Salimetrics). Samples were frozen at -20 degrees Celcius and were assayed within six months of collection. Participants provided samples at eight time points during the lab visit including 40 minutes before the start of TSST (directly after assent, -40m sample), right before the start of TSST (0m sample), directly after TSST (15m sample), and then every 10 minutes until 65 minutes after the task (25m, 35m, 45m, 55m, 65m). Participants were asked to refrain from eating and drinking (other than water) for one hour before the lab visit and each visit was at the same time of day on a weekend to account for diurnal variability in cortisol. The cortisol samples had an interassay variability coefficient of 7.67% and an intraassay variability coefficient of 6.34%. We performed Winsorization at 98% to minimize the potential effect of extreme values. We also used a Box-Cox power transformation for time series to transform the salivary cortisol values to normalize the distribution. Baseline cortisol values were created by averaging the values of the -40m and 0m samples.

Behavioral Coding of Parent-Adolescent Interaction

The teens who were randomized into the parent present condition spent the first 10 minutes of the regulation phase with their parent in the room alone. Each parent was told that the teen had just undergone a stressful task and that the parents should feel free to provide any support they want to their child. These interactions were video recorded and the recordings were used to code positive and negative parental emotional socialization responses to the teen's display of emotion. Each video was double coded and then the two students who had

independently coded the video met to discuss any discrepancies and created a best-estimate code. See Appendix A for the full coding manual that was used. Seventy-one participants were randomized to the parent present condition, however, due to technical errors, only 58 had a codable video (82% of the possible videos were codable). A previous graduate student in the lab (EG) had trained a group of advanced undergraduate students on this coding system, and two additional groups of advanced undergraduates were subsequently trained by VM. For each group of video coders, care was made to be sure that coding was done in the same way (e.g., review of previous codes, recoding previously coded videos). Videos coded by undergraduates trained by both graduate students did not vary in mean levels of positive $t(56) = -0.17$, $p = 0.87$, and negative $t(56) = 0.21$, $p = 0.83$ behaviors per minute.

Measures

Lab Visit Questionnaire

Parent participants completed a lab visit questionnaire which included demographic information including, age, gender (male or female), race/ethnicity, family income, family structure, when the adolescent woke up (used to calculate time from awakening to the start of the task), date of last menstrual cycle, as well as other health and demographic information not of central importance to the current study.

Pubertal Status

The Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) was used to measure the current stage of puberty. This measure is a non-invasive, nine-item self-report questionnaire that asks participants to report on physical characteristics of puberty, including hair growth, growth spurts, and menarche. Parents completed this measure for their adolescents. Total summed scores are based on gender and participants are placed into one of

five categories of pubertal development: Prepubertal, Early Pubertal, Midpubertal, Late Pubertal, and Postpubertal. This measure of puberty is a reliable, non-invasive measure of pubertal development that correlates with interview ratings of development (Petersen et al., 1988). In our sample, Cronbach's alpha was 0.85 for males and 0.76 for females, indicating acceptable internal consistency.

Parental Diagnostic Interview

To determine familial risk status, parental history of depression was assessed using the Structured Clinical Interview of DSM-5 Axis 1 Disorders (SCID-5; First, Williams, Karg, & Spitzer, 2015). The interview was conducted by graduate students in clinical psychology who were trained by the principal investigator of the study, who also leads the clinical diagnostic training for the doctoral program in clinical psychology at the university where the study was conducted. The SCID-5 is a semi-structured interview with probes for symptoms and anchor points for criteria of diagnosis of DSM-5 disorders. Clinical research staff and the PI reviewed 100% of all cases during clinical diagnostic consensus meetings. The final decision on diagnosis was reached via consensus using the best estimate procedures (Maziade et al., 1992). Based on parental lifetime history of major depression, participants were placed in High Risk (HR; parental depression history present) or Low Risk (LR; no parental depression history) groups. Adolescents who have a parent with lifetime history of depression were considered “High Risk” for this study, because the data were taken from a larger study on risk for depression. Other diagnoses were not included. For example, a parent could have experienced anxiety in the past, but if they had not experienced a depressive disorder, the participant would be considered Low Risk for the purposes of this study.

Parenting Behaviors

We used a modified version of the Emotional Socialization coding system developed by Bosler and colleagues to code the videos (Bosler, Morris, & Criss, 2012). See Appendix A for the full video coding manual. Videos were recorded via overhead cameras or web cameras attached to a computer in the room where parents/teens interacted. Video interactions were coded using the Behavioral Observation Research Interactive Software (v. 2.6 and later; Friard & Gamba, 2016) which allows the recording of timestamped behaviors. The emotion socialization manual was created to measure parental responses to emotion during a task that specifically asked the dyad to discuss emotion. This manual was modified by a previous graduate student (Geiss, 2016) to fit the context of the post-stress task instructions. In response to youth display of emotion, parental supportive behaviors (e.g., Comforting, Validation, or Problem-solving/Teaching about emotions) and unsupportive behaviors (e.g., Dismissing, Punishing, Magnifying) are indicated and summed. This coding system allows for analyses of the individual or collective level of supportive or unsupportive behaviors. Additionally, to measure overall parental supportive behaviors, we created a summary variable by averaging the number of supportive parental behaviors per minute, averaging the number of unsupportive parental behaviors per minute, and creating a difference score of supportive minus unsupportive.

Interrater reliability was computed by dissecting the videos into 20-second increments and then noting whether a behavior was coded as present or absent during each interval (yes/no). This was done for each coder and the best-estimate code and was then used to calculate percent agreement and the kappa coefficient of each type of behavior. For positive parenting behaviors, there was 84% agreement between the two coders ($\kappa = 0.62$, substantial agreement) and 92% agreement between the coders and the best-estimate code ($\kappa = 0.82$, almost perfect agreement).

For unsupportive behaviors, there was 93% agreement between the two coders (κ = 0.33, fair agreement) and 97% agreement between the coders and the best-estimate code (κ = 0.73, substantial agreement). The slightly lower interrater reliability (κ) for the unsupportive behaviors may have been due to the relative infrequency of these types of behaviors. Parent supportive behaviors were positively correlated with parental support post-task, $r(52)= 0.29$, $p = 0.03$, and parent unsupportive behaviors were negatively correlated with parental support post-task, $r(52)= -0.47$, $p = 0.0005$, suggesting that these behaviors are a valid representation of support in this sample.

Family Functioning

Adolescents reported their perceived family functioning by completing the McMaster Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983), which is a 53-item self-report questionnaire. This scale has been widely used and shows good reliability and validity (Miller, Epstein, Bishop, & Keitner, 1985). The questionnaire yields an overall general functioning subscale score as well as subscale scores for family problem solving, communication, roles, affective responsiveness, affective involvement, and behavioral control. For this study, we used the general functioning subscale as our operationalization of positive family functioning. The Cronbach's alpha for the general functioning subscale in our sample was 0.88, indicating good internal consistency.

Parental Social Support – Parent present condition

Participants who were randomized to the 10-minute parent interaction post-stressor were asked about how supportive they felt their parent was directly after the 10-minute task was complete. Participants were asked, "To what degree did you feel supported by your

parent/guardian?" and were to select the level of agreement on a line that ranged from "Not at all" (0) to "Extremely" (100). Higher scores were indicative of greater perceived support.

Parental Social Support – Full Sample

Because not all participants were randomized to the 10-minute parent interaction post-stressor, we computed a composite score of perceived parental support for the full sample. We summed the z-scores from the mother and father attachment totals from the Inventory for Parent and Peer Attachment scale (IPPA; Armsden & Greenberg, 1987) as well as the family total score in the Multidimensional Scale for Perceived Social Support (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988). The IPPA mother and father attachment subscales each consist of 25-items on a five-point Likert scale that assess the degree of mutual trust, quality of communication, and extent of anger and alienation. The IPPA was developed in adolescents and was shown to have appropriate validity (Armsden & Greenberg, 1987). The MSPSS family subscale includes items that measure the degree to which adolescents perceive their family as being supportive on a seven-point scale. The MSPSS has demonstrated acceptable validity and reliability (Zimet, Powell, Farley, Werkman, & Berkoff, 1990). These three summed z-scores represent parental support (referred to as "parent support composite") for the entire sample with greater levels being indicative of greater perceived support. Cronbach's alpha of the parent support composite was 0.76 for our sample, indicating acceptable internal consistency. The parent support composite score was strongly correlated with the support question after the task, $R = 0.65$, $p < 0.001$, indicating that the composite is a good representation of parental support.

Analyses

Modeling cortisol response

This study examines factors associated with the post-stress cortisol trajectory. To model this cortisol response, we used a two-piece multi-level growth curve model with landmark registration with random slopes and intercepts (Lopez-Duran, Mayer, & Abelson, 2014). This approach allows us to examine post-task activation slopes, peak, and recovery slopes simultaneously. It also controls for individual variation in peak times by adjusting for individual differences in peak latency.

To this end, each participant's peak value and the corresponding peak time are identified by visually examining each response curve and selecting the time point for each individual where the upward cortisol trajectory ends – either before a plateau or before a downward trajectory. If the first identified peak was followed by a plateau, we tested whether another point was 10% greater than this original peak and selected that as the peak value. Individuals who did not have a cortisol response, defined as peak levels at least 20% higher than baseline, were considered "non-responders" and given a peak time of 25 min post-task, which was the mode peak time for the responders.

Next, we created an adjusted time variable that is centered at the peak time for all individuals. To do this, we first created a "Minutes-from-Peak" variable for each individual. Samples before peak have a negative "Minutes-from-Peak" value (e.g., -10 "Minutes-from-peak") and samples after peak have a positive value (e.g., 10 "Minutes-from-peak"). Next, we used this "Minutes-from-peak" variable to create two spline variables to account for Time Before (activation slope) and Time After (recovery slope) peak response. We did this by using the following formula:

If Min-from-peak > 0 then TimeBeforePeak = Min-from-peak else TimeBeforePeak= 0

If Min-from-peak > 0 then TimeAfterPeak = Min-from-peak else TimeAfterPeak = 0

Using SAS statistical software, we next used a multilevel random effects model to model the cortisol response. The unconditional model is represented by the following equation:

$$\boldsymbol{Cortisol} = \boldsymbol{\beta_o} + (\boldsymbol{\beta_1} \times \boldsymbol{TimeBeforePeak}) + (\boldsymbol{\beta_2} \times \boldsymbol{TimeAfterPeak}) + \boldsymbol{E}$$

Where β_o is the intercept (peak cortisol), β_1 represents activation slope, and β_2 is recovery slope, plus E error. Using this approach, the intercept is interpreted as peak cortisol because it is the value at which Time Before Peak and Time After Peak equals zero. This model was then used as the base model to predict cortisol trajectory by key variables of interest.

Planned Analyses

All analyses were conducted on SAS statistical software (SAS Studio v. 5.2). We began with covariate analyses to test whether covariates that might impact the cortisol response have a significant impact on any part of the cortisol trajectory. These covariates include age, gender, pubertal status, menstrual cycle status, and time from awakening to the start of the task. We subsequently controlled for all covariates that predicted the cortisol trajectory in subsequent models. Then, to address the possibility that the overall findings were impacted by the reduced power due to a large number of covariates, we conducted additional single-variable sensitivity analyses, where the main effects were examined when controlling for each covariate at a time. We conducted post-hoc power analysis, based on the sample size and number of parameters in the most complex model, to determine whether we were sufficiently powered to detect moderate effect sizes (0.35) at 80% power. This power analysis achieved a power of 0.98, which is above the minimum threshold value of 0.80. Thus, for these parameters and sample size, there is sufficient power to support the analyses results.

Aim 1. In Aim 1, we examined parenting and family factors related to adolescent regulation of the cortisol response to an in-lab stressor. We used multilevel random effects mixed-models to model cortisol activation, peak, and recovery as predicted by our variables of interest. Specifically, we used unadjusted models of perceived parental support post-task, parent support composite scores, observed parental supportive behaviors post-task, and family functioning individually predicting cortisol trajectory. We then used a fully adjusted model with all variables of interest predicting cortisol trajectory without interactions.

Aim 2. In Aim 2, we examined whether the parenting and family factors of perceived family support via the parent support composite score and family functioning moderate the association between parent presence post-stressor and cortisol trajectory. First, we tested whether the social buffering effect is present in our sample by predicting cortisol trajectory from parent presence or absence. We then used two-way interaction models to look at how variables of interest interact with parent presence to predict cortisol trajectory and to test whether these factors influence the social buffering of parental presence post-stressor. We were unable to explore how parent behaviors and parent support post-task in these models because of the limited sample size given that half of the sample does not have data for these factors.

Aim 3 (Exploratory).In Aim 3, we examined whether familial risk for depression status moderates the Aim 2 two-way associations. First, we tested whether familial risk for depression moderates the social buffering effect of parent presence post-task. Then, we conducted three-way interaction models predicting cortisol trajectory to examine whether even in the context of familial risk, there is variability in cortisol trajectory that may be explained by these family factors. We were unable to explore how parent behaviors and parent support post-task in these

models because of the limited sample size given that half of the sample does not have data for these factors.

Results

Sample Characteristics

See Table 2.1 for means and standard deviations of key variables. In our sample, high-risk and low-risk adolescents did not differ in mean levels of supportive parenting behaviors post-task $t(56) = 1.31$, $p = 0.20$. Low-risk adolescents reported greater parent support post-task $t(66) = 2.35$, $p = 0.02$, and greater perceived parent support composite scores $t(142) = 2.74$, $p = 0.001$. High-risk adolescents reported greater levels of family functioning than low-risk adolescents $t(133) = -3.78$, $p < 0.0001$, which is opposite than what would be expected. Adolescents in the parent present and parent absent conditions did not differ in their mean levels of family functioning $t(133) = -1.44$, $p = 0.15$ and perceived parent support composite scores $t(142) = -0.13$, $p = 0.89$.

Table 2.1: Sample Characteristics

Characteristic	*M, SD*
Parent Present Sample	
Parental Support Post-Task	*M* = 79.03, *SD* = 25.27
Positive Parenting behaviors	*M* = 2.24, *SD* = 1.28
Negative Parenting behaviors	*M* = 0.31, *SD* = 0.46
Parent behavior summary	*M* = 1.92, *SD* = 1.42
Full Sample	
Family Functioning (FAD)	*M* = 19.24, *SD* = 5.64
Parent Support Composite Score	*M* = -5.07E-10, *SD* = 2.44

Modeling Cortisol Response

We found an initial increase in cortisol after the task, Activation Slope $b = 0.01$, $t = 8.99$, $p < 0.0001$, followed by a significant decrease after the cortisol peak, Recovery Slope $b = -0.01$, $t = -11.15$, $p < 0.0001$, suggesting the expected increase and decrease of cortisol secretion after the stressor. We then tested common covariates that may impact cortisol trajectory, including pubertal status, gender, age, menstrual cycle status, and time from awakening to the task. We found that pubertal status, $b = 0.11$, $t = 3.76$, $p = 0.0002$, age, $b = 0.03$, $t = 3.05$, $p = 0.003$, and menstrual cycle, $b = -0.11$, $t = -2.86$, $p = 0.005$, all had an impact on peak cortisol levels, while time from wake had an impact on both peak, $b = -0.04$, $t = -4.09$, $p < 0.0001$, and activation slope, $b = -0.002$, $t = -2.42$, $p = 0.02$. Gender did not have an impact on the cortisol trajectory. In subsequent analyses, we controlled for all covariates that predicted the cortisol trajectory. Then, we conducted additional single-variable sensitivity analyses, where main effects were examined controlling for each covariate at a time.

Aim 1: Impact of Supportive Factors on Adolescent Endocrine Response to In-Lab Stressor

We found that adolescent perceived parental support post-stressor, which was only applicable to the parent present condition, impacted peak cortisol levels at trending level, $b = 0.002$, $t = 1.86$, $p = 0.07$, such that greater perceived parent support was associated with *increases* in peak levels of cortisol. Perceived parental support post-task did not significantly impact either activation or recovery slopes of the cortisol trajectory. Further, single variable sensitivity analyses found that these findings remained when individually controlling for age, pubertal status, and menstrual cycle status, and the effect on peak levels became more significant when controlling for the time from awakening to the task, $b = 0.002$, $t = 2.01$, $p = 0.049$. Additionally, to test whether parent support impacted the cortisol trajectory for the entire sample,

we tested whether perceived parent support composite scores impacted the cortisol trajectory. We found that increases in support were also associated with *increased* peak levels of cortisol, $b = 0.03$, $t = 5.27$, $p < 0.001$ using this composite version of parent support. Additionally, parent support was associated with a significant increase in the activation slope of cortisol such that as parent support increased, the activation slope was steeper, $b = 0.001$, $t = 2.51$, $p = 0.01$. There was no impact on the recovery slope. Single variable sensitivity analyses found that these findings remained when individually controlling for age, pubertal status, menstrual cycle status, and time from awakening to the task. In sum, perceived support seems to be associated with increases in cortisol peak levels, which is the opposite of what we had hypothesized. See Table 2.2.

Table 2.2: Impact of Parent Support on Adolescent Cortisol Trajectory Post-stressor (Controlling for covariates)

	b	*SE*	*df*	*t* value	*p* value
Support Post-Task Model					
Intercept	-1.020	0.214	59	-4.760	<.0001
Age	0.007	0.013	59	0.510	0.615
Pubertal Status	0.018	0.035	59	0.520	0.605
Menstrual Cycle	-0.126	0.035	59	-3.620	0.001
Time From Wake	-0.005	0.010	59	-0.470	0.642
ActivationSlope*TimeFromWake	0.001	0.001	453	0.330	0.741
Baseline Cort	0.446	0.021	59	21.520	<.0001
Activation Slope	0.001	0.003	453	0.210	0.830
Recovery Slope	-0.006	0.003	453	-1.870	0.062
Parent Support Post-Task	0.002	0.001	59	1.860	0.067
ActivationSlope*ParentSupportPost-Task	0.001	0.001	453	1.390	0.164
RecoverySlope*ParentSupportPost-Task	0.001	0.001	453	-0.820	0.414
Support Composite Model					
Intercept	-0.660	0.175	130	-3.760	0.000
Age	0.004	0.011	130	0.330	0.741
Pubertal Status	0.119	0.030	130	3.910	0.000
Menstrual Cycle	-0.124	0.032	130	-3.930	0.000
Time From Wake	-0.034	0.008	130	-4.170	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	802	-1.830	0.067

Baseline Cort	0.445	0.023	130	19.710	<.0001
Activation Slope	0.017	0.004	802	4.650	<.0001
Recovery Slope	-0.009	0.001	802	-11.340	<.0001
Parent Support Composite	0.033	0.006	130	5.270	<.0001
ActivationSlope*ParentSupportComposite	0.001	0.001	802	2.510	0.012
RecoverySlope*ParentSupportComposite	-0.001	0.001	802	-1.530	0.127

Observed parental supportive behaviors, which were only recorded for those in the parent present condition, predicted decreases in cortisol peak levels post-stressor, such that an increase in parental supportive behaviors was associated with lower cortisol peak values, b = -0.08, t = -2.05, p = 0.047. This is the opposite of what was found for the perceived support variables above, which both were associated with greater cortisol peaks. Observed parental supportive behaviors did not significantly impact either activation or recovery slopes of the cortisol trajectory. Further, single variable sensitivity analyses found that these findings remained when individually controlling for age, pubertal status, and menstrual cycle status. However, the impact of supportive behaviors on peak levels became trend level significant when controlling from time from awakening to the task, b = -0.08, t = -1.97, p = 0.06. See Table 2.3.

Table 2.3: Impact of Positive Parenting Behaviors on Adolescent Cortisol Trajectory Post-stressor (Controlling for covariates)

	b	*SE*	*df*	*t* value	*p* value
Intercept	-0.009	0.274	40	-0.030	0.973
Age	-0.032	0.017	40	-1.830	0.074
Pubertal Status	0.062	0.042	40	1.470	0.148
Menstrual Cycle	-0.141	0.047	40	-3.010	0.005
Time From Wake	-0.072	0.015	40	-4.880	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	320	-0.770	0.440
Baseline Cort	0.411	0.033	40	12.410	<.0001
Activation Slope	0.008	0.004	320	2.170	0.031
Recovery Slope	-0.010	0.001	320	-7.510	<.0001
Positive Parent Behaviors	-0.082	0.040	40	-2.050	0.047
ActivationSlope*PositiveParentBehaviors	-0.001	0.002	320	-0.550	0.582
RecoverySlope*PositiveParentBehaviors	0.001	0.002	320	0.150	0.879

Family functioning, which was recorded for the entire sample, was associated with decreases in cortisol peak levels post-stressor, such that an increase in family functioning was associated with lower cortisol peaks, b = -0.15, t = -4.35, $p < 0.0001$. Additionally, greater family functioning was associated with less steep activation slopes, b = -0.006, t = -2.50, p = 0.01 (there was no impact on the recovery slope). This is the opposite of what was found for the perceived support variables above, which were associated with increases in cortisol peaks, but is in line with the observed parenting supportive behaviors which were also associated with decreases in peak values. See Table 2.4. Single variable sensitivity analyses found that these findings remained when individually controlling for pubertal status, menstrual cycle status, and time from awakening to the task.

Table 2.4: Impact of Family Functioning on Adolescent Cortisol Trajectory Post-stressor (Controlling for covariates)

	b	*SE*	*df*	*t* value	*p* value
Intercept	-0.410	0.179	123	-2.290	0.024
Age	-0.003	0.012	123	-0.270	0.791
Pubertal Status	0.172	0.034	123	5.100	<.0001
Menstrual Cycle	-0.138	0.032	123	-4.250	<.0001
Time From Wake	-0.039	0.008	123	-4.690	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	762	-2.020	0.044
Baseline Cort	0.451	0.023	123	19.990	<.0001
Activation Slope	0.023	0.004	762	5.270	<.0001
Recovery Slope	-0.011	0.002	762	-6.480	<.0001
Family Functioning	-0.146	0.033	123	-4.350	<.0001
ActivationSlope*FamilyFunctioning	-0.006	0.003	762	-2.500	0.013
RecoverySlope*FamilyFunctioning	0.003	0.002	762	1.400	0.162

Finally, in an adjusted model for those in the parent present condition, which explored the impact of these parenting-level variables on cortisol trajectory in the same model, we found that when controlling for the conditional effect of each other, perceived parental support post-stressor b = -0.006, t = -3.22, p = 0.003, parental supportive behaviors b = -0.09, t = -2.27, p = 0.03, and family functioning b = -0.25, t = -2.80, p = 0.009 all had a buffering impact on cortisol

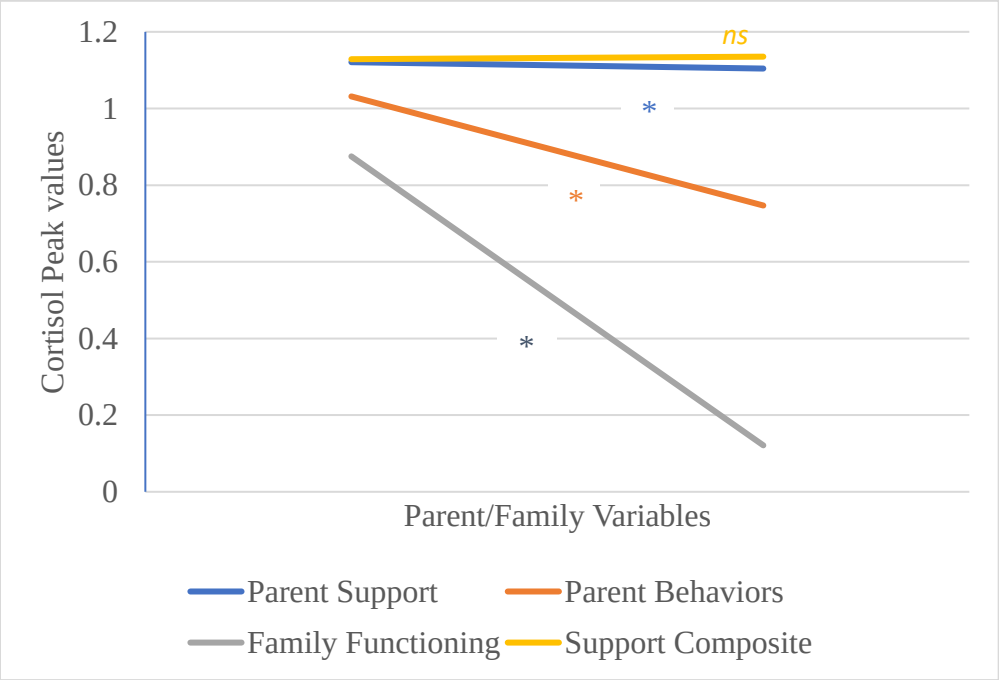
trajectories, such that higher levels of any of these factors were associated with decreased cortisol peak values. In this model, perceived parent support composite scores did not impact peak values $b = 0.002$, $t = 0.11$, $p = 0.91$, and none of these variables impacted activation or recovery slopes. See Table 2.5. Single variable sensitivity analyses resulted in the findings remaining when controlling for the time from awakening to the task. However, parenting behaviors became non-significant in models that controlled for age, pubertal status, and menstrual cycle status. Notably, in this adjusted model, the direction of the effect for perceived support post-stress changed. In models where perceived support post-task was predicting cortisol trajectories by itself, greater support was associated with increases in peak levels (at trend level on its own and significantly when controlling for the time from awakening). However, in this model, when controlling for the conditional effect of the other factors, it is associated with significant decreases in cortisol peak. See Figure 2.1, which shows the changes in peak cortisol for each of the support variables in the adjusted model, while controlling for covariates.

Table 2.5: Impact of Parent/Family Supportive Factors on Adolescent Cortisol Trajectory Post-stressor – Fully Adjusted Model (Controlling for covariates)

	b	*SE*	*df*	*t* value	*p* value
Intercept	1.126	0.358	32	3.150	0.004
Age	-0.054	0.019	32	-2.900	0.007
Pubertal Status	0.188	0.057	32	3.310	0.002
Menstrual Cycle	-0.132	0.049	32	-2.720	0.011
Time From Wake	-0.111	0.016	32	-6.870	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	281	-1.280	0.203
Baseline Cort	0.381	0.034	32	11.260	<.0001
Activation Slope	0.024	0.009	281	2.580	0.011
Recovery Slope	-0.018	0.009	281	-1.920	0.056
Family Functioning	-0.251	0.090	32	-2.800	0.009
Parent Support Composite	0.002	0.020	32	0.110	0.915
Positive Parent Behaviors	-0.095	0.042	32	-2.270	0.030
Parent Support Post-Task	-0.006	0.002	32	-3.220	0.003
ActivationSlope*FamilyFunctioning	-0.006	0.004	281	-1.510	0.132
RecoverySlope*FamilyFunctioning	0.001	0.005	281	0.320	0.753

ActivationSlope*ParentSupportComposite	0.001	0.001	281	-0.510	0.609
RecoverySlope*ParentSupportComposite	0.001	0.001	281	-0.120	0.905
ActivationSlope*PositiveParentBehaviors	-0.001	0.002	281	-0.560	0.574
RecoverySlope*PositiveParentBehaviors	0.001	0.002	281	-0.080	0.937
ActivationSlope*ParentSupportPost-Task	0.001	0.001	281	-1.260	0.209
RecoverySlope*ParentSupportPost-Task	0.001	0.001	281	0.970	0.333

Figure 2.1: Change in Peak Cortisol Values as Parent/Family Factors Increase



Aim 2: Social Buffering Effect of Parental Presence and Moderation by Supportive Factors

We found that parental presence did not have a significant impact on the cortisol peak, *b* = -0.02, *t* = -0.70, *p* = 0.48, activation *b* = 0.0001, *t* = 0.05, *p* = 0.96, or recovery *b* = 0.001, *t* = 0.59, *p* = 0.56, suggesting our sample does not exhibit a social buffering effect solely based on the presence of their parents. This remained non-significant in our single-variable sensitivity analyses of covariates. Further, we tested whether family functioning and parent support composite moderated the buffering effect. Family functioning did not moderate the association

between parental presence and cortisol trajectory, ParentPresent*FamilyFunctioning, peak b = 0.03, t = 0.49, p = 0.62. See Table 2.6. Additionally, the parent support composite did not moderate the association between parental presence and cortisol trajectory peak, ParentPresent*FamilySupport peak b = 0.01, t = 1.11, p = 0.27. See Table 2.7. The effects of family functioning and parent support composite remained non-significant in single variable sensitivity analyses of covariates.

Table 2.6: Moderation of Family Functioning on the Relationship between Parental Presence and Adolescent Cortisol Trajectory Post-stressor (Controlling for covariates)

	Parental Presence	*b*	*SE*	*df*	*t* value	*p* value
Intercept		-0.363	0.185	121	-1.960	0.052
Age		-0.004	0.012	121	-0.310	0.759
Pubertal Status		0.176	0.034	121	5.180	<.0001
Menstrual Cycle		-0.142	0.033	121	-4.340	<.0001
Time From Wake		-0.041	0.008	121	-4.840	<.0001
ActivationSlope*TimeFromWake		-0.001	0.001	758	-2.100	0.036
Baseline Cort		0.443	0.023	121	19.040	<.0001
Activation Slope		0.025	0.005	758	4.780	<.0001
Recovery Slope		-0.011	0.003	758	-4.460	<.0001
Parental Presence Post-Task (PP)	Absent	-0.069	0.066	121	-1.040	0.298
Parental Presence Post-Task (PP)	Present	0.000	.	.	.	.
Family Functioning (FF)		-0.169	0.045	121	-3.770	0.000
ActivationSlope*PP	Absent	-0.003	0.005	758	-0.560	0.578
ActivationSlope*PP	Present	0.000	.	.	.	.
RecoverySlope*PP	Absent	-0.001	0.004	758	-0.200	0.839
RecoverySlope*PP	Present	0.000	.	.	.	.
ActivationSlope*FF		-0.008	0.003	758	-2.220	0.027
RecoverySlope*FF		0.002	0.002	758	0.780	0.436
FF*PP	Absent	0.033	0.068	121	0.490	0.627
FF*PP	Present	0.000	.	.	.	.
ActivationSlope*FF*PP	Absent	0.002	0.005	758	0.470	0.640
ActivationSlope*FF*PP	Present	0.000	.	.	.	.
RecoverySlope*FF*PP	Absent	0.002	0.004	758	0.620	0.537
RecoverySlope*FF*PP	Present	0.000	.	.	.	.

Table 2.7: Moderation of Support Composite on the Relationship between Parental Presence and Adolescent Cortisol Trajectory Post-stressor (Controlling for covariates)

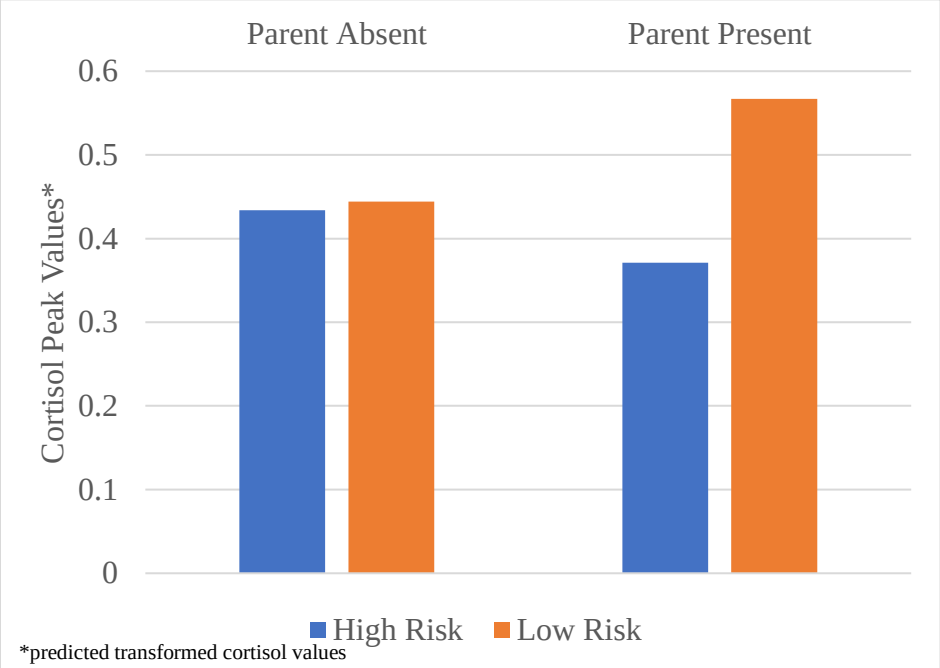
	b	*SE*	*df*	*t* value	*p* value
Intercept	-0.652	0.177	128	-3.690	0.000
Age	0.004	0.011	128	0.330	0.745
Pubertal Status	0.118	0.031	128	3.800	0.000
Menstrual Cycle	-0.127	0.032	128	-3.950	0.000
Time From Wake	-0.034	0.008	128	-3.990	0.000
ActivationSlope*TimeFromWake	-0.001	0.001	798	-1.860	0.063
Baseline Cort	0.443	0.023	128	19.220	<.0001
Activation Slope	0.017	0.004	798	4.420	<.0001
Recovery Slope	-0.010	0.001	798	-8.420	<.0001
Parental Presence Post-Task (PP) -Absent	-0.027	0.030	128	-0.890	0.377
Parental Presence Post-Task (PP) - Present	0.000	.	.	.	.
Parent Support Composite (ParentSupport)	0.026	0.009	128	2.970	0.004
ActivationSlope*PP - Absent	0.001	0.002	798	-0.060	0.951
ActivationSlope*PP - Present	0.000	.	.	.	.
RecoverySlope*PP - Absent	0.001	0.002	798	0.650	0.514
RecoverySlope*PP - Present	0.000	.	.	.	.
ActivationSlope*ParentSupport	0.001	0.001	798	1.530	0.127
RecoverySlope*ParentSupport	0.001	0.001	798	-0.330	0.741
ParentSupport*PP - Absent	0.014	0.013	128	1.110	0.268
ParentSupport*PP - Present	0.000	.	.	.	.
ActivationSlope*ParentSupport*PP - Absent	0.001	0.001	798	0.360	0.716
ActivationSlope*ParentSupport*PP - Present	0.000	.	.	.	.
RecoverySlope*ParentSupport*PP - Absent	-0.001	0.001	798	-1.100	0.273
RecoverySlope*ParentSupport*PP - Present	0.000	.	.	.	.

Aim 3 (Exploratory): Impact of familial risk for depression on the social buffering effect and the role of parenting and family-level factors

The purpose of our exploratory Aim 3 was to test whether risk for depression status moderated our Aim 2 two-way associations. We first tested whether risk status moderated the

association between parental presence post-stressor and cortisol trajectory. We found that risk status moderated the association between parental presence and cortisol trajectory. Specifically, in low-risk participants, parent presence was associated with higher peak levels, Risk*Presence peak $b = 0.19$, $t = 3.06$, $p = 0.003$, and steeper activation slopes, Activation*Risk*Presence $b = 0.01$, $t = 2.78$, $p = 0.006$. This effect was not present in high-risk participants. These findings remained significant in single variable sensitivity analyses. In sum, See Figure 2.2 which shows cortisol peak values.

Figure 2.2: Cortisol Peak values in High and Low-Risk Adolescents under both conditions



Next, we tested whether risk status moderated the two-way association between family functioning and parental presence post-stressor, and found that this was not the case in our sample. We found that this three-way interaction was not significant on cortisol peak levels $b = -0.22$, $t = -1.44$, $p = 0.15$, activation slope $b = -0.001$, $t = -0.08$, $p = 0.94$, and recovery slope $b = 0.004$, $t = 0.51$, $p = 0.61$. Further, this remained non-significant in single variable sensitivity

analyses, except in the model that included time from awakening to the task. In this model, the three-way interaction was significant on peak values b = -0.31, t =-2.01, p = 0.047. In this model, high-risk teens in the parent present condition displayed increased peak cortisol values as family functioning increased. See Table 2.8.

Table 2.8: Risk Status moderation of the association between parental presence and family functioning on adolescent cortisol trajectory (Controlling for Time from Awakening only)

	b	*SE*	*df*	*t* value	*p* value
Intercept	-0.432	0.088	122	-4.910	<.0001
Time From Wake	-0.037	0.009	122	-4.240	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	762	-1.620	0.107
Baseline Cort	0.468	0.024	122	19.310	<.0001
Activation Slope	0.021	0.006	762	3.330	0.001
Recovery Slope	-0.013	0.004	762	-3.330	0.001
Parental Presence Post-Task (PP) - Absent	-0.132	0.088	122	-1.490	0.138
Parental Presence Post-Task (PP) - Present	0.000	.	.	.	.
Family Functioning (FF)	-0.044	0.088	122	-0.500	0.620
Familial Risk (Risk) - High	-0.073	0.098	122	-0.750	0.456
Familial Risk (Risk) - Low	0.000	.	.	.	.
ActivationSlope*PP - Absent	-0.004	0.007	762	-0.660	0.510
ActivationSlope*PP - Present	0.000	.	.	.	.
RecoverySlope*PP - Absent	0.001	0.005	762	0.140	0.886
RecoverySlope*PP - Present	0.000	.	.	.	.
ActivationSlope*FF	0.001	0.007	762	0.150	0.884
RecoverySlope*FF	0.002	0.005	762	0.420	0.674
ActivationSlope*Risk - High	0.001	0.008	762	0.060	0.950
ActivationSlope*Risk - Low	0.000	.	.	.	.
RecoverySlope*Risk - High	0.003	0.005	762	0.550	0.581
RecoverySlope*Risk - Low	0.000	.	.	.	.
FF*PP - Absent	0.081	0.113	122	0.720	0.475
FF*PP - Present	0.000	.	.	.	.
PP*Risk – Absent, High	0.356	0.137	122	2.610	0.010
PP*Risk – Absent, Low	0.000	.	.	.	.
PP*Risk – Present, High	0.000	.	.	.	.
PP*Risk – Present, Low	0.000	.	.	.	.
FF*Risk - High	-0.090	0.105	122	-0.860	0.394
FF*Risk - Low	0.000	.	.	.	.
ActivationSlope*FF*PP - Absent	-0.004	0.009	762	-0.420	0.672
ActivationSlope*FF*PP - Present	0.000	.	.	.	.
RecoverySlope*FF*PP - Absent	0.001	0.006	762	0.130	0.896
RecoverySlope*FF*PP - Present	0.000	.	.	.	.
ActivationSlope*PP*Risk – Absent, High	0.011	0.011	762	1.010	0.314

ActivationSlope*PP*Risk – Absent, Low	0.000	.	.	.	.
ActivationSlope*PP*Risk – Present, High	0.000	.	.	.	.
ActivationSlope*PP*Risk – Present, Low	0.000	.	.	.	.
RecoverySlope*PP*Risk – Absent, High	-0.004	0.008	762	-0.540	0.587
RecoverySlope*PP*Risk – Absent, Low	0.000	.	.	.	.
RecoverySlope*PP*Risk – Present, High	0.000	.	.	.	.
RecoverySlope*PP*Risk – Present, Low	0.000	.	.	.	.
ActivationSlope*FF*Risk - High	-0.009	0.008	762	-1.080	0.279
ActivationSlope*FF*Risk - Low	0.000	.	.	.	.
RecoverySlope*FF*Risk - High	-0.001	0.006	762	-0.210	0.835
RecoverySlope*FF*Risk - Low	0.000	.	.	.	.
FF*PP*Risk – Absent, High	-0.305	0.152	122	-2.010	0.047
FF*PP*Risk – Absent, Low	0.000	.	.	.	.
FF*PP*Risk – Present, High	0.000	.	.	.	.
FF*PP*Risk – Present, Low	0.000	.	.	.	.
ActivationSlope*FF*PP*Risk – Absent, High	0.001	0.012	762	-0.020	0.984
ActivationSlope*FF*PP*Risk – Absent, Low	0.000	.	.	.	.
ActivationSlope*FF*PP*Risk – Present, High	0.000	.	.	.	.
ActivationSlope*FF*PP*Risk – Present, Low	0.000	.	.	.	.
RecoverySlope*FF*PP*Risk – Absent, High	0.004	0.009	762	0.450	0.654
RecoverySlope*FF*PP*Risk – Absent, Low	0.000	.	.	.	.
RecoverySlope*FF*PP*Risk – Present, High	0.000	.	.	.	.
RecoverySlope*FF*PP*Risk – Present, Low	0.000	.	.	.	.

We also tested whether risk status moderated the two-way association between parent support composite scores and parental presence and found that the three-way interaction was not significant for peak values $b = 0.02$, $t = 0.65$, $p = 0.52$, activation slope $b = -0.003$, $t = -2.14$, $p = 0.03$, or recovery slope $b = -0.0001$, $t = -0.05$, $p = 0.96$, suggesting that there is not more variability in the high-risk vs. low-risk social buffering effect in the context of greater family support in our sample. This effect remained non-significant in single variable sensitivity analyses. See Table 2.9.

Table 2.9: Risk Status moderation of the association between parental presence and parent support composite on adolescent cortisol trajectory (Controlling for covariates)

	b	*SE*	*df*	*t* value	*p* value
Intercept	-0.553	0.178	124	-3.110	0.002

Age	0.007	0.011	124	0.620	0.536
Pubertal Status	0.105	0.032	124	3.320	0.001
Menstrual Cycle	-0.130	0.032	124	-4.050	<.0001
Time From Wake	-0.035	0.008	124	-4.160	<.0001
ActivationSlope*TimeFromWake	-0.001	0.001	792	-2.090	0.037
Baseline Cort	0.457	0.023	124	19.810	<.0001
Activation Slope	0.021	0.005	792	4.600	<.0001
Recovery Slope	-0.011	0.002	792	-7.020	<.0001
Parental Presence Post-Task (PP) - Absent	-0.114	0.043	124	-2.670	0.009
Parental Presence Post-Task (PP) - Present	0.000	.	.	.	.
Parent Support Composite (ParentSupport)	-0.025	0.015	124	-1.690	0.094
Familial Risk (Risk) - High	-0.148	0.042	124	-3.490	0.001
Familial Risk (Risk) - Low	0.000	.	.	.	.
ActivationSlope*PP – Absent	-0.002	0.002	792	-0.640	0.525
ActivationSlope*PP – Present	0.000	.	.	.	.
RecoverySlope*PP – Absent	0.001	0.002	792	0.650	0.513
RecoverySlope*PP – Present	0.000	.	.	.	.
ActivationSlope*ParentSupport	-0.001	0.001	792	-0.680	0.497
RecoverySlope*ParentSupport	0.001	0.001	792	0.320	0.746
ActivationSlope*Risk - High	-0.002	0.002	792	-0.890	0.371
ActivationSlope*Risk - Low	0.000	.	.	.	.
RecoverySlope*Risk - High	0.002	0.002	792	1.300	0.195
RecoverySlope*Risk - Low	0.000	.	.	.	.
ParentSupport*PP - Absent	0.030	0.021	124	1.390	0.167
ParentSupport*PP - Present	0.000	.	.	.	.
PP*Risk – Absent, High	0.112	0.051	124	2.200	0.030
PP*Risk – Absent, Low	0.000	.	.	.	.
PP*Risk – Present, High	0.000	.	.	.	.
PP*Risk – Present, Low	0.000	.	.	.	.
ParentSupport*Risk - High	0.066	0.019	124	3.460	0.001
ParentSupport*Risk - Low	0.000	.	.	.	.
ActivationSlope*ParentSupport*PP - Absent	0.002	0.002	792	0.970	0.331
ActivationSlope*ParentSupport*PP – Present	0.000	.	.	.	.
RecoverySlope*ParentSupport*PP - Absent	-0.001	0.001	792	-0.730	0.464
RecoverySlope*ParentSupport*PP - Present	0.000	.	.	.	.
ActivationSlope*ParentSupport*Risk - High	0.003	0.002	792	1.820	0.070
ActivationSlope*ParentSupport*Risk – Low	0.000	.	.	.	.
RecoverySlope*ParentSupport*Risk – High	0.001	0.001	792	-0.390	0.693
RecoverySlope*ParentSupport*Risk – Low	0.000	.	.	.	.
ParentSupport*PP*Risk – Absent, High	-0.014	0.027	124	-0.520	0.606
ParentSupport*PP*Risk – Absent, Low	0.000	.	.	.	.
ParentSupport*PP*Risk – Present, High	0.000	.	.	.	.

ParentSupport*PP*Risk – Present, Low	0.000	.	.	.	.
ActivationSlope*ParentSupport*PP*Risk – Absent, High	-0.002	0.002	792	-0.920	0.356
ActivationSlope*ParentSupport*PP*Risk – Absent, Low	0.000	.	.	.	.
ActivationSlope*ParentSupport*PP*Risk – Present, High	0.000	.	.	.	.
ActivationSlope*ParentSupport*PP*Risk – Present, Low	0.000	.	.	.	.
RecoverySlope*ParentSupport*PP*Risk – Absent, High	0.001	0.001	792	-0.010	0.989
RecoverySlope*ParentSupport*PP*Risk – Absent, Low	0.000	.	.	.	.
RecoverySlope*ParentSupport*PP*Risk – Present, High	0.000	.	.	.	.
RecoverySlope*ParentSupport*PP*Risk – Present, Low	0.000	.	.	.	.

Discussion

We examined whether parental and family supportive factors impacted the regulation of the HPA axis after a laboratory stressor among adolescence at high and low familial risk for depression. We found evidence for our hypotheses that some of the parent and family-level factors were associated with changes in the HPA axis response to stress, sometimes in opposite directions. For instance, we found that parent support composite scores and perceived parent support post-stressor were associated with a sensitizing effect on the axis such that greater levels of perceived parent support was associated with significantly greater peak cortisol levels and steeper activation slopes. However, when controlling for parental behaviors and family functioning, the direction of these effects reversed suggesting a regulatory role for perceived parent support. Likewise, positive parental behaviors post-stress and family functioning were associated with a buffering effect on the axis such that increases in either of these factors were associated with decreases in peak cortisol values. In addition, we found that parental presence did not have the buffering effect expected based on the findings of the impact of parental

behaviors and perceived support. In contrast, parental presence appears to disrupt the regulation of the stress response but only in the low-risk participants. Overall, our findings suggest a complex dynamic in the relationship between parent and family-level factors and stress regulation that likely highlights the role of trait and state-level factors that are not often considered in stress regulation research.

In our first aim, we examined the impact of perceived parental support, parental behaviors, and family function on neuroendocrine stress regulation. In non-adjusted models, we found that perceived parental support, both in response to parental presence and for the entire sample, was linked to increases in cortisol peak levels. In contrast, positive parental behaviors during the task and family function were linked to lower cortisol peaks. One potential explanation of the link between perceived parental support and greater peak levels is that youth with a greater stress response to the task elicited greater efforts of support from the parents and thus greater perceived support. However, when considering these parent/family factors together in a fully adjusted model, only three significant findings remained, all in the direction of down-regulating the HPA response. In this model, higher perceived parental support post-stressor, positive parent behaviors, and greater family functioning were all associated with decreases in cortisol peak values. Parent support via composite scores no longer predicted cortisol trajectory. Notably, the direction of the effect of perceived parent support post-stress changed in the fully adjusted model, such that it was now associated with decreases in cortisol peak. While this may seem conflicting, the fully adjusted model controlled for the correlation between parental behaviors post-stress, perceived support post-stress, and trait-level perceived parental support. Thus, the relationship between perceived post-stress support and cortisol response may be capturing two phenomena: the actual role of support on cortisol response and the fact that youth

with greater stress elicits greater supportive response among highly sensitive parents (Edelstein et al., 2004). The fully adjusted model may parse these two components suggesting that perceived support may have a regulatory role on the HPA axis after controlling for factors that lead to greater efforts of support among highly distressed kids (i.e., trait level perceived parental support).

While, to our knowledge, this is the first study to explore these family factors in the context of a laboratory stressor in this age group, family functioning, and support are protective against the harmful effects of stressful experiences in adolescents (Balistreri & Alvira-Hammond, 2016; Henderson et al., 2006; Simpson et al., 2018) and are related to greater emotion regulation in adolescents (Yap et al., 2010). Additionally, it has been proposed that parenting plays a role in the development of internalizing disorders in later childhood partially through a biological mechanism, through its impact on the HPA axis (Kuhlman et al., 2014). Our findings suggest that parenting may also affect HPA axis regulation for adolescents after an acute stressor.

In our second aim, we examined whether the positive parent behaviors, family functioning, and parent support, moderated the social buffering effect on the cortisol trajectory. Overall, we did not find that our sample displayed the social buffering effect solely based on parental presence. In addition, we found that these parent/family support variables did not impact the parental presence effect, suggesting that the social buffering effect from parental presence is non-significant in our sample, even in the context of adolescents' greater perceived support and greater observed positive parenting behaviors. The fact that we did not find the social buffering effect from parental presence in the overall sample is in line with literature that suggests that social buffering decreases in adolescence (Doom et al., 2015). While the mechanisms are not

completely understood, some evidence suggests that social buffering declines in adolescence due to neurobiological changes associated with advancing through puberty (Doom et al., 2015). Additionally, our study suggests that this buffering effect may be less in this age group even under the condition of improved family factors (perceived support). While it might seem contradictory that we found an impact of the parent/family factors on HPA regulation but did not find the stress-buffering effect from parental presence, the results from aim one suggest that parental behaviors and perceived support can impact the cortisol response, but our sample may have been too small to identify those effects in the context of a parent presence-absence interaction, or another factor, such as a parental history of depression, obscured the effect.

In fact, in Aim 3 we examined whether familial risk for depression impacted the social buffering effect and the role of parenting and family-level factors. We found that for low-risk participants, parent presence was associated with higher peak levels and steeper activation slopes, suggesting that the presence of their parents disrupted the regulation process (i.e., the opposite of the buffering effect). Perhaps other factors not measured, such as conflict or frustration with parents, which are also increasing in adolescence (Collins, Laursen, Mortensen, Luebker, & Ferreira, 1997) may be the cause of this disruption in regulation and may explain why parental presence alone provides less of a buffering effect for teens (Doom et al., 2015). This may be especially true as increases in conflict are not always associated with negative parenting attributes or less positive relationships, and are also a normative experience in adolescence as they navigate autonomy from parents (Collins et al., 1997). Notably, the effect in the low-risk participants likely explained why we did not find an overall buffering effect of parent presence while we found a buffering effect of parent behaviors and perceived support. Surprisingly, this contra-intuitive sensitizing effect of parent presence was not observed in the

high-risk participants. It is possible that our high-risk teens may benefit more from their parental support due to alterations in HPA axis functioning related to greater exposure to stress. In fact, children of parents with a history of depression tend to experience greater levels of stress over their lifetime in comparison to their low-risk peers (Bouma, Ormel, Verhulst, & Oldehinkel, 2008) and display a prolonged HPA axis response to stress (Lopez-Duran et al., 2015). Thus, these high-risk teens may be utilizing their parents as regulators to a greater degree given this history of greater stress exposure, which may have eliminated the sensitizing effect seen in their low-risk peers. It is also possible that the lack of effect of parental presence in high-risk participants is a byproduct of sampling bias. Our participants were recruited from community advertisements and thus parents with a history of depression who responded to study advertisements may be unique in ways that eliminated the sensitizing effect of parental presence. For example, these parents may be more attuned to the needs of their teens preventing the type of interactions that may have led to disruptions in stress regulation.

Our results should be taken in the context of several limitations. First, the fact that we only had behavioral data for half or our sample, created difficulties in being able to test how parenting behaviors may impact the social buffering effect. However, we were able to still examine perceived support and family functioning in those aims. The behavioral data itself was also summarized into one variable which included positive minus negative parenting behaviors. Using this type of summary variable creates heterogeneity in the definition of "High" levels of parenting support behaviors as it may include people with different levels of negative parenting behaviors. Second, our sample was considered "High Risk" based on familial risk for depression only, but did not include risk for other commonly co-occurring psychiatric disorders, such as anxiety disorders. Additionally, while the current study has many strengths, including

psychosocial and neuroendocrine data, it was a relatively homogeneous sample that does not represent the larger population from which it was drawn. It is possible that in a larger, more diverse sample, there would be greater variability in our factors which may result in different results. Additionally, given that the majority of our participant parents were mothers, a larger sample may also allow for additional examination of differences in these associations that may arise when parents and adolescents are participating with the same gender parent versus participating with their opposite gender parent. Additionally, for models where we did not find a significant result, it is possible that these relationships have smaller effect sizes and therefore we may not have been powered to detect such a small effect. In these cases, a larger sample may provide power to detect relationships with a smaller effect. Finally, our study did not include measurements of teen level factors that may influence these bidirectional relationships. For instance, teen irritability, their propensity to accept support, or their preferred style of support were not included and may have had an impact on how the parents were behaving in the interactions.

In conclusion, we found strong evidence that specific parenting behaviors, perceived parenting support, and high levels of positive family functioning were all linked to greater neuroendocrine stress regulation after a laboratory stressor. In contrast, the mere presence of a parent did not result in a buffering effect and instead was found to disrupt the stress regulation in low-risk participants. Our study adds important nuance to the understanding of how parent and family supportive factors may impact adolescent regulation to acute stressors by highlighting the critical role of measuring actual behaviors as well as trait parental characteristics that may obscure the impact of parents as regulatory agents for their adolescents.

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Chapter 3 Parental and Familial Factors as Regulators of Adolescent Internalizing Response to Recent Life Stress

Abstract

Adolescence is a stressful developmental period and is also when stress-related psychopathology peaks. Parent and family support may be a protective factor that buffers the impact of stress on internalizing symptoms. We examined how stress exposure to recent life events was related to internalizing symptoms in a sample over-represented by adolescents with a family history of depression. We also examined whether perceived parental support, observed supportive parental behaviors, and perceived positive family functioning might buffer the effects of stress on internalizing symptoms in this population. Our sample included 145 adolescents aged 12-16 years and one of their parents, of which over half (n = 85) had a history of depression. Adolescents completed a laboratory stress task followed by randomization to parent present or absent conditions where parents were to provide support. Videos were coded for positive and negative parenting behaviors. Adolescents also completed a stressful life events interview and self-report questionnaires regarding their perception of positive family functioning and parent support. We found that stress exposure was related to greater internalizing symptoms. However, we consistently failed to find a stress-buffering effect of the positive parent and family factors examined. Further, the buffering effect was non-existent in both high and low risk for depression teens. Our findings suggest that parenting support, as measured in this study, may have limited utility in minimizing the effect of stressful life events in adolescents.

Keywords: stress, adolescence, depression, anxiety, parenting, family, internalizing

Introduction

Adolescence is a uniquely stressful time in human development. During the pubertal transition, adolescents face many changes in brain maturation and advancement in cognitive functioning (Blakemore, Burnett, & Dahl, 2010; Juraska & Willing, 2017) as well as changes in their social environment, including individuation from the family unit (Erikson, 1968). During these changes, adolescent stress also rises as school (Torsheim & Wold, 2001) and social demands increase (Nelson, Leibenluft, McClure, & Pine, 2005). Increases in stress lead to mental health-related sequelae (Roberts & Lopez-Duran, 2019; Tafet & Nemeroff, 2016). In fact, stress has been implicated as a causal and maintaining factor in psychopathology (Lopez-Duran, Micol, & Roberts, 2019), and much of this stress-related psychopathology is at its peak during adolescence (Costello, Copeland, & Angold, 2011; Kessler et al., 2012). Identifying factors that mitigate the impact of stress in teens is of significant public health importance. While parental support has been associated with helping youth cope with stress (Doom, Hostinar, VanZomeren-Dohm, & Gunnar, 2015; Hostinar, Johnson, & Gunnar, 2015; Seltzer, Ziegler, & Pollak, 2010), few have explored how both perceived parental support and observed parental behaviors might buffer the effect of exposure to stress on internalizing symptoms in adolescence.

The impact of stress on internalizing symptoms in adolescence

Stress is a potent risk factor for the development of internalizing symptoms. Recent life stress is related to increases in depression in adolescents (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Waaktaar, Borge, Fundingsrud, Christie, & Torgersen, 2004). For example, negative life events predict depressive symptoms and hospitalizations (Korkeila et al., 2010). Likewise, community-based studies have found a link between life events and increases in depression symptoms (Waaktaar et al., 2004). In addition, stressful life events also predict

anxiety in adolescents (McLaughlin & Hatzenbuehler, 2009). For example, in nationally representative samples in high-income countries, exposure to early life stressors was related to a greater risk of experiencing anxiety disorders across different development groups, including adolescents (Kessler et al., 2010). Further, in a community sample of adolescents, stressful life events predicted greater anxiety symptoms and anxiety sensitivity (McLaughlin & Hatzenbuehler, 2009). However, effect sizes remain low to moderate, suggesting great variability in the degree to which stress impacts internalizing symptoms (Grant, Compas, Thurm, McMahon, & Gipson, 2004). This variability may likely be in part due to various risk and protective factors, including parenting and family-related supports.

Parental and family factors as protective agents in the stress to internalizing relationship

Perceived parental support and supportive behaviors may serve as protective factors to help regulate adolescents during stressful times and thereby help to reduce internalizing symptoms. Specifically, throughout childhood and adolescence, parents serve a regulatory role through several mechanisms, including modeling (Liga, Inguglia, Gugliandolo, Ingoglia, & Costa, 2020) and sources of support (Bokhorst, Sumter, & Westenberg, 2010) that help youth regulate their responses to stress. While parental presence during laboratory stressors has been shown to have a stress-buffering effect on adolescents (Doom et al., 2015; Hostinar, Sullivan, & Gunnar, 2014), it is unclear what specific types of parental supportive behaviors may help to reduce the negative outcomes of stress in real-life settings. For example, parental emotion coaching, a type of supportive behavior, has been shown to reduce adolescent internalizing symptoms (Stocker, Richmond, Rhoades, & Kiang, 2007). Further, parental validation behaviors have been associated with adolescents' ability to regulate their own emotions, which was in turn associated with fewer internalizing and externalizing symptoms (Buckholdt, Parra, & Jobe-

Shields, 2014). There is some evidence that these parenting behaviors might mitigate the effects of stress. For example, perceived parental support has been shown to reduce the impact of stress on suicidal ideation in adolescents (Kang et al., 2017; Mackin, Perlman, Davila, Kotov, & Klein, 2017), and has linked to lower levels of internalizing symptoms in adolescents and young adults living in areas with high level of community violence (Donnelly & Holzer, 2018). Additionally, positive family functioning, as indexed by the self-reported perception of supportive family factors such as cohesion, support, and communication, is related to a reduction in internalizing symptoms in youth living in poverty (Sheidow, Henry, Tolan, & Strachan, 2014). Despite significant support for perceived support and supportive parenting behaviors being associated with decreases in stress and stress-related outcomes, to our knowledge, no study has yet examined how *perceived* support and *observed* supportive behaviors mitigate the impact of *recent* life stressors on internalizing symptoms in high-risk adolescents.

Aside from parental social support and behaviors, higher levels of perceived family functioning may be protective against internalizing disorders. More cohesive family functioning, which includes warmth, relationship satisfaction, and communication, is a protective factor in the context of high-stress environments. Specifically, poorer family functioning has been shown to have an impact on negative outcomes in adolescents, such as greater substance use (Hummel, Shelton, Heron, Moore, & van den Bree, 2013), externalizing problems (Henderson, Dakof, Schwartz, & Liddle, 2006), and internalizing symptoms (Simpson, Vannucci, & Ohannessian, 2018). Additionally, poor family functioning prospectively predicted greater depression and anxiety symptoms in a large cohort of adolescent students (Simpson et al., 2018). In contrast, greater family cohesion is a protective factor in reducing the negative impact of early childhood adversity on adolescent mental and physical wellbeing (Balistreri & Alvira-Hammond, 2016),

such that greater family cohesion attenuated the negative association between early adverse events and later overall mental well-being. Further, for adolescents who are experiencing internalizing disorders, family cohesion is associated with a greater quality of life (Jozefiak & Wallander, 2016). Family support is thus a potential protective factor associated with decreases in the impact of stress-related outcomes for adolescents. However, to our knowledge, no study has examined how positive family functioning, particularly in high-risk for depression adolescents, might buffer the impact of *recent* life stressors on internalizing symptoms.

The impact of parental psychopathology

On the other hand, parental experiences with their own internalizing symptoms may impact their ability to help their teens regulate. Adolescents of parents with depression are at greater risk for depression (Gotlib, Joormann, & Foland-Ross, 2014) and are more likely to experience the depressogenic effects of stress than those without a parent with depression (Bouma, Ormel, Verhulst, & Oldehinkel, 2008). A variety of purported mechanisms for this relationship exist, including greater exposure to life stress (Feurer, Hammen, & Gibb, 2016) as well as an impact on in-the-moment behavioral support of adolescents' stress response. Specifically, both depressive *state* and *history* of depression in mothers increase negative parenting behaviors (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Additionally, a depressive state has also been shown to decrease positive parenting behaviors in fathers as well (Lovejoy et al., 2000; Wilson & Durbin, 2010).

Despite decades of literature on the mechanisms of risk for depression, the link between parental depression and negative outcomes in teens is variable and is likely associated with a complex interplay of risk and protective factors across development (Collishaw et al., 2016). Some of this variability in outcomes may be related to how different experiences of positive

family functioning, perceived support and observed supportive parental behaviors might buffer the impact of recent life stressors on internalizing symptoms, particularly in adolescents at high risk for depression. Yet to our knowledge, no study has yet examined both perceived support and supportive family functioning alongside observed parental behaviors in adolescence at high familial risk for depression, and tested whether these parent/family factors serve to buffer that stress to internalizing relationship.

Use of Laboratory Stressors and Measurement of Stressful Life Events

While many studies on parental support use self-reported measures of support, there is great value in using laboratory observations of parenting behaviors. Specifically, using in-lab stress tasks for adolescents, followed by a period in which parents are there to support their adolescent, allows for a close examination of parental behaviors in moderately stressful and controlled environments. The current study uses a modified Trier Social Stress Task (TSST; Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009), which is a public speaking task designed to elucidate a stress response in adolescence followed by a period in which parents are instructed to give their teen support. Observing how parents respond to their teen's displays of distress in this controlled environment along with the teen's perception of support allows us to examine how each of these perceived and observed parental support might buffer the effect of recent life events on internalizing symptoms.

There is also variety in how researchers have operationalized and measured stress in examining the relationship between stress and psychopathology. One common approach is to administer self-reported stress exposure checklists or measures of perceived stress (Byrne, Davenport, & Mazanov, 2007; Gray, Litz, Hsu, & Lombardo, 2004; Lee, 2012). While these types of measures are relatively efficient to collect, there is a growing need to operationalize

stress exposure in a way that takes into account contextual factors of the stressful experience (Goodyer, Croudace, Dunn, Herbert, & Jones, 2010). For example, while a checklist may ask whether or not a parent was unemployed, it does not provide more contextual information about the family's finances before unemployment, and thus those who are unemployed but had a large amount of savings may experience this stressor differently than those who are unemployed and did not have any savings. While more labor-intensive, contextual stress interviews can be used to gather additional information to gain context and can give a measure of the objective nature of stress exposure (e.g., how much the event might have impacted a typical person given the context) and the subjective nature of stress exposure (e.g., how impactful the event was perceived by the person). The Stressful Life Events Schedule for Children and Adolescents is one example of this type of intensive measurement of stress exposure which aims to give more context to and precise information about the stress exposure (Williamson et al., 2003). Despite the potential benefits of these types of interviews, few researchers have included them in studies examining the impact of stressful life events (Grant et al., 2004).

Conclusion and Aims

This study examines how exposure to recent life stress, gathered via a contextual stress interview, might impact internalizing symptoms (anxiety and depression) in adolescents at high and low risk for depression. We also explored specific parental and family-related factors (family functioning, family social support, parental supportive behaviors) that might serve to buffer the association between stress and internalizing symptoms. Our third aim is exploratory in nature and designed to explore if these associations might be further impacted by familial risk for depression. We examined the following aims and hypotheses.

Aim 1

In Aim 1, we examined whether parent-level factors (parental supportive behaviors, perceived social support of parents) moderate the association between stress exposure and internalizing symptoms in adolescents. We hypothesized that greater levels of recent life stress would be associated with higher symptoms of anxiety and depression in this sample of adolescents. Further, we hypothesized that parent supportive behaviors and perceived parent social support would have a moderating impact on the stress to internalizing relationship such that in the context of more positive parent behaviors and greater parental support, the association between stress and internalizing would be reduced.

Aim 2

In Aim 2, we examined whether family functioning will moderate the association between recent life stress and internalizing symptoms in adolescents. As with Aim 1, we tested whether the impact of recent life stress on internalizing symptoms is moderated by family functioning. We hypothesized that family functioning would moderate the association between stress and internalizing such that in the context of greater family functioning, the impact of stress on anxiety and depression would be reduced.

Aim 3 (Exploratory)

In Aim 3 we examined how familial risk for depression might further moderate the associations found in Aim 1 and Aim 2. Given the exploratory nature of this aim, we did not propose any hypotheses. It is possible that protective parenting factors may not have a regulating effect on youth at familial risk for depression compared to low-risk kids. However, it is also possible that the presence of positive parental factors is especially important for these higher-risk

kids, and thus the protective effect of parental and family factors will be stronger for high-risk youth.

Methods

Participants

Participants are 145 adolescents and one of their parents participating in a study of familial risk for depression (PI: Nestor Lopez-Duran). This dataset includes cortisol and psychosocial data in a sample of adolescents, oversampled for familial risk for depression. We used community-based recruitment strategies to recruit two different groups, those with and without a parental history of depression. Over half, 58.22% (n = 85), of the participants had a parent with a history of depression. The majority of the parents who participated in this study with their teen were mothers (88%). The average participant age was 13.94 (SD=1.35, Range 12-16). Participants were excluded from the study if they had a history of psychosis or diagnosis of Autism, Down's Syndrome, or significant neurological or medical disorders that may impact cortisol secretion (e.g., cerebral palsy, cancer, endocrine disorder). 74.29% identified as White/Caucasian, 10.00% identified as African American/Black, 10.00% identified as Asian/Pacific Islander, 0.71% identified as Native American, and 2.86% identified as Biracial or Other. 2.14% of participants identified as Latino/Hispanic. 71.01% of adolescents reported that their parents were married or living together. The majority of participants (81.2%) reported an annual household income of $50,000 or more, including 48.12% earning greater than $100,000 annually.

This study was approved by the University Institutional Review Board and informed consent and assent were obtained from all adolescent participants and their parents. Participants were compensated $50 for their laboratory visit participation.

Procedure

Adolescents and their parents participated in an approximately 4-hour laboratory assessment. The laboratory assessment included: parent and adolescent self-report questionnaires, adolescent stress task, and parent and adolescent clinical interviews. The study's main aims were to examine neuroendocrine markers of stress regulation, and therefore included having participants provide saliva samples throughout the study. For the current study, the cortisol data was not used. Previous work has been published on neuroendocrine markers of stress regulation in this sample (Micol, Roberts, Taylor-Cavelier, Geiss, & Lopez-Duran, 2019; Taylor-Cavelier, Micol, Roberts, & Lopez-Duran, 2020, 2021).

Upon arrival at the laboratory, adolescents and their parents completed the informed consent/assent process before completing self-report questionnaires. Parents participated in clinical interviews that included a diagnostic assessment of the child and the parent, as well as a contextual stress assessment interview.

Adolescents participated in the in-lab stress task. Directly after the stress task, they were randomized to one of two conditions: 1) Parent present: teens spend the first 10 minutes of the regulation phase alone with their parents in the room, or 2) Parent absent: teens go directly to watching a neutral movie after the task and do not interact with their parents. The parent/teen interaction was video-recorded and used for behavioral coding of parental emotion socialization. All adolescents then watched a neutral movie, until the regulation period had been completed at which point in time, the teen participated in their own clinical interview and contextual stress interview.

Stress Task

We used the Trier Social Stress Test (TSST), a well-established social evaluative stress task designed to elicit a cortisol response (Het et al., 2009). We used a modified version designed to be more applicable to adolescents (Ellenbogen & Hodgins, 2009). In this task, teens were told that they would be giving a speech to judges who are trained to analyze facial expressions and that the speech will be recorded. The teen is then given five minutes to prepare a speech on why they would be a good class president. They are then instructed to stand before the judges and a video camera to give the speech to a panel of judges. The speech itself lasts for five minutes and the judges are undergraduate confederates who are trained to keep their facial expressions neutral throughout the task and to ask questions to keep the participant speaking for the entire five minutes. Following the speech, the participants are instructed to do an arithmetic task for five minutes in front of the judges. This is a serial subtraction task where they are instructed to start from the beginning if they make a mistake. Directly after the task, teens watched a neutral movie for 40 minutes to standardize and facilitate regulation of the cortisol response to the task. They were then debriefed and made aware of the purpose of the task.

Behavioral Coding of Parent-Adolescent Interaction

The teens who were randomized into the parent present condition spent the first 10 minutes of the regulation phase with their parent in the room alone. Each parent was told that the teen had just undergone a stressful task and that the parents should feel free to provide any support they want to their child. These interactions were video recorded and the recordings were used to code positive and negative parental emotional socialization responses to the teen's display of emotion. Each video was double coded and then the two students who had independently coded the video met to discuss any discrepancies and created a best-estimate code.

See Appendix A for the full coding manual that was used. Seventy-one participants were randomized to the parent present condition, however, due to technical errors, only 58 had a codable video (82% of the possible videos were codable). A previous graduate student in the lab (EG) had trained a group of advanced undergraduate students on this coding system, and two additional groups of advanced undergraduates were subsequently trained by VM. For each group of video coders, care was made to be sure that coding was done in the same way (e.g., review of previous codes, recoding previously coded videos). Videos coded by undergraduates trained by both graduate students did not vary in mean levels of positive $t(56) = -0.17$, $p = 0.87$, and negative $t(56) = 0.21$, $p = 0.83$ behaviors per minute.

Measures

Lab Visit Questionnaire

Parent participants completed a lab visit questionnaire which included demographic information including age, gender (male or female), race/ethnicity, family income, family structure, as well as other health and demographic information not of central importance to the current study.

Parental Diagnostic Interview

To determine familial risk status, parental history of depression was assessed using the Structured Clinical Interview of DSM-5 Axis 1 Disorders (SCID-5; First, Williams, Karg, & Spitzer, 2015). The interview was conducted by graduate students in clinical psychology who were trained by the principal investigator of the study, who also leads the clinical diagnostic training for the doctoral program in clinical psychology at the university where the study was conducted. The SCID-5 is a semi-structured interview with probes for symptoms and anchor points for criteria of diagnosis of DSM-5 disorders. Clinical research staff and the PI reviewed

100% of all cases during clinical diagnostic consensus meetings. The final decision on diagnosis was reached via consensus using the best estimate procedures (Maziade et al., 1992). Based on parental lifetime history of major depression, participants were placed in High Risk (HR; parental depression history present) or Low Risk (LR; no parental depression history) groups. Adolescents who have a parent with lifetime history of depression were considered "High Risk" for this study, because the data is taken from a larger study on risk for depression. Other diagnoses were not included. For example, a parent could have experienced anxiety in the past, but if they had not experienced a depressive disorder, the participant would be considered Low Risk for the purposes of this study.

Parenting Behaviors

We used a modified version of the Emotional Socialization coding system developed by Bosler and colleagues to code the videos (Bosler, Morris, & Criss, 2012). See Appendix A for the full video coding manual. Videos were recorded via overhead cameras or web cameras attached to a computer in the room where parents/teens interacted. Video interactions were coded using the Behavioral Observation Research Interactive Software (v. 2.6 and later; Friard & Gamba, 2016) which allows the recording of timestamped behaviors. The emotion socialization manual was created to measure parental responses to emotion during a task that specifically asked the dyad to discuss emotion. This manual was modified by a previous graduate student (Geiss, 2016) to fit the context of the post-stress task instructions. In response to youth display of emotion, parental supportive behaviors (e.g., Comforting, Validation, or Problem-solving/Teaching about emotions) and unsupportive behaviors (e.g., Dismissing, Punishing, Magnifying) are indicated and summed. This coding system allows for analyses of the individual or collective level of supportive or unsupportive behaviors. Additionally, to measure overall

parental supportive behaviors, we created a summary variable by averaging the number of supportive parental behaviors per minute, averaging the number of unsupportive parental behaviors per minute, and creating a difference score of supportive minus unsupportive.

Interrater reliability was computed by dissecting the videos into 20-second increments and then noting whether a behavior was coded as present or absent during each interval (yes/no). This was done for each coder and the best-estimate code and was then used to calculate percent agreement and the kappa coefficient of each type of behavior. For positive parenting behaviors, there was 84% agreement between the two coders ($\kappa = 0.62$, substantial agreement) and 92% agreement between the coders and the best-estimate code ($\kappa = 0.82$, almost perfect agreement). For unsupportive behaviors, there was 93% agreement between the two coders ($\kappa = 0.33$, fair agreement) and 97% agreement between the coders and the best-estimate code ($\kappa = 0.73$, substantial agreement). The slightly lower interrater reliability (κ) for the unsupportive behaviors may have been due to the relative infrequency of these types of behaviors. Parent supportive behaviors were positively correlated with parental support post-task, $r(52)= 0.29$, $p = 0.03$, and parent unsupportive behaviors were negatively correlated with parental support post-task, $r(52)= -0.47$, $p = 0.0005$, suggesting that these behaviors are a valid representation of support in this sample.

Adolescent Recent Life Stress

Parents and adolescents separately completed a contextual stress interview about the teen's recent life stress called The Stressful Life Events Schedule for Children and Adolescents (SLESCA; Williamson et al., 2003). This interview involves answering a screener questionnaire which has the respondent indicate whether they are currently experiencing or have experienced over the last 12 months any of the listed 80 stressful events. There is also an opportunity for the

respondent to write in additional events if they were not included in the list of 80 common stressors. The interviewer then asked follow-up questions about each event endorsed to get more information about the context of the stressor. The respondent then provides their subjective response to the stressors by rating how much each event has affected them on a scale of 1 “Not at all” to 4 “A lot”. The research team then uses a consensus rating system (provided by the authors of the interview) to assign objective ratings to each event (i.e., How stressful an event should be for an average adolescent given the context of what occurred). For this study, we operationalized adolescent stress exposure by using the objective measure of stress. However, we used the subjective score in our post-hoc analyses as well.

Adolescent Perceived Stress

Adolescents reported their past-month perceived stress level through the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). This 10-item measure asks adolescents how often they have felt certain experiences of stress in the last month (e.g., “In the last month, how often have you been upset because of something that happened unexpectedly?”) and is rated from a 0 “Never” to 4 “Very Often” scale. The 10 items are summed to create a total score, with higher scores indicative of greater perceived stress over the past month. The total score for our sample indicated good internal consistency with a Cronbach’s alpha of 0.87.

Childhood Trauma

Adolescents completed the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003), a 28-item self-reported measure of adverse experiences in childhood. Adolescents rated their experience of each item on a scale from 1 “Never True” to 5 “Always True”. Reverse scored items of positive childhood experiences are also included. Items are summed to create a

total score of childhood trauma, with greater scores indicating a high endorsement of traumatic experiences. Cronbach's alpha for our sample was 0.81, indicating good internal consistency.

Adolescent Internalizing

Depression was measured via the Children's Depression Inventory (CDI; Kovacs, 2011). This 27-item self-report measure asked adolescents to rate their current depressive symptoms over the past two weeks on a scale from 0 to 2 and yielded a total score ranging from 0 to 54. The scale includes feelings of sadness, irritability, and functional impairment. This measure is commonly used to measure adolescent depression and has been shown to have acceptable reliability and validity (Smucker, Craighead, Craighead, & Green, 1986). Cronbach's alpha for our study was 0.82, indicating good internal consistency.

The Screen for Child Anxiety Related Disorders (SCARED) Child Version (Birmaher et al., 1999) was used to assess current adolescent anxiety symptoms. The SCARED is a 41-item questionnaire that has shown to be a reliable and valid measure for screening for adolescent anxiety (Birmaher et al., 1999). Adolescents rated each of the items on a scale of 0, "Not True or Hardly Ever True" to 2, "Very True or Often True" and a total score of 25 or higher indicates the potential presence of an anxiety disorder. Cronbach's alpha for our study was 0.95, indicating excellent internal consistency.

Total depression and anxiety scores were transformed into z scores and then summed to create a composite measure of adolescent internalizing symptoms, which was used as our outcome of interest ("adolescent internalizing symptoms") in our models. Cronbach's alpha of the internalizing composite was 0.77, indicating acceptable internal consistency.

Family Functioning

Adolescents reported their perceived family functioning by completing the McMaster Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983), which is a 53-item self-report questionnaire. This scale has been widely used and shows good reliability and validity (Miller, Epstein, Bishop, & Keitner, 1985). The questionnaire yields an overall general functioning subscale score as well as subscale scores for family problem solving, communication, roles, affective responsiveness, affective involvement, and behavioral control. For this study, we used the general family functioning subscale as our measure of family functioning. The Cronbach's alpha in our sample was 0.88, indicating good internal consistency.

Parental Social Support – Parent present condition

Participants who were randomized to the 10-minute parent interaction post-stressor were asked about how supportive they felt their parent was directly after the 10-minute task was complete. Participants were asked, "To what degree did you feel supported by your parent/guardian?" and were to select the level of agreement on a line that ranged from "Not at all" (0) to "Extremely" (100). Higher scores were indicative of greater perceived support.

Parental Social Support – Full Sample

Because not all participants were randomized to the 10-minute parent interaction post-stressor, we computed a composite score of perceived parental support for the full sample. We summed the z-scores from the mother and father attachment totals from the Inventory for Parent and Peer Attachment scale (IPPA; Armsden & Greenberg, 1987) as well as the family total score in the Multidimensional Scale for Perceived Social Support (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988). The IPPA mother and father attachment subscales each consist of 25-items on a five-point Likert scale that assess the degree of mutual trust, quality of communication, and

extent of anger and alienation. The IPPA was developed in adolescents and was shown to have appropriate validity (Armsden & Greenberg, 1987). The MSPSS family subscale includes items that measure the degree to which adolescents perceive their family as being supportive on a seven-point scale. The MSPSS has demonstrated acceptable validity and reliability (Zimet, Powell, Farley, Werkman, & Berkoff, 1990). These three summed z-scores represent parental support (referred to as "parent support composite") for the entire sample with greater levels being indicative of greater perceived support. Cronbach's alpha of the parent support composite was 0.76 for our sample, indicating acceptable internal consistency. The parent support composite score was strongly correlated with the support question after the task, $R = 0.65$, $p < 0.001$, indicating that the composite is a good representation of parental support.

Planned Analyses

Sensitivity and interaction analyses

All analyses were conducted on SAS statistical software (SAS Studio v. 5.2). We began with sensitivity analyses to test the impact of covariates on dependent variables. These covariates include age, race/ethnicity, and gender. We subsequently controlled for all covariates that predicted the outcome in subsequent models. Then, to address the possibility that the overall findings were impacted by the reduced power due to a large number of covariates, we conducted additional single-variable sensitivity analyses, where the main effects were examined when controlling for each covariate at a time. We conducted post-hoc power analysis, based on the sample size and number of parameters in the most complicated model, to determine whether we were sufficiently powered to detect moderate effect sizes (0.35) at 80% power. This power analysis achieved a power of 0.99, which is above the minimum threshold value of 0.80. Thus, for these parameters and sample size, there is sufficient power to support the analyses results.

Planned analyses

Aim 1. Aim 1 explored the regulating impact of parent-level factors on the association between teen stress and internalizing symptoms. We used generalized linear models to predict internalizing symptoms by adolescent recent life stress and then tested whether parent-level factors of parent behaviors post-stress task and perceived parent social support moderated this association.

Aim 2. Aim 2 is similar in approach to Aim 1, except we examined the regulating impact of family functioning on the association between teen stress and internalizing symptoms. Specifically, we used generalized linear models to predict internalizing symptoms by adolescents' recent life stress and then tested whether family functioning moderated this association.

Aim 3 (Exploratory). Aim 3 is exploratory in nature and examined whether parents with a history of depression are more or less effective in helping kids regulate. For this aim, we explored whether familial risk for depression moderates the moderations in Aim 1 and Aim 2, using generalized linear models with three-way interactions between risk status, supportive factors, and stress exposure.

Results

Sample Characteristics

High risk and low-risk adolescents did not differ in mean levels of supportive parenting behaviors $t(56) = 1.31$, $p = 0.20$ or perceived parenting support post-task $t(75)= 1.89$, $p = 0.06$. Low-risk adolescents had greater perceived parent support composite scores $t(133) = 2.71$, $p = 0.008$. High-risk adolescents reported greater levels of family functioning than low-risk adolescents $t(133) = -3.78$, $p = 0.0002$, which is the opposite of what would be expected.

Adolescents in the parent present and parent absent conditions did not differ in their mean levels of family functioning $t(133) = -1.44$, $p = 0.15$ and perceived parent support composite scores $t(142) = -0.13$, $p = 0.89$.

Adolescents in our sample displayed moderate levels of depression symptoms ($M = 17.18$, $SD = 3.89$) and anxiety symptoms ($M = 21.55$, $SD = 15.77$). High risk adolescents displayed greater levels of depression than their low risk peers, $t(163) = -2.33$, $p = 0.02$, more anxiety than their low risk peers, $t(162) = -3.23$, $p = 0.002$, and greater internalizing composite scores $t(160) = -3.04$, $p = 0.003$, than their low risk peers. Additionally, high risk adolescents reported greater objective stress exposure $t(152) = -5.32$, $p < 0.0001$, than their low risk peers.

We tested covariates that might impact our outcome of interest, the composite measure of internalizing symptoms. We found that age $b = 0.09$, $t = 0.79$, $p = 0.43$ and race/ethnicity $b = 0.55$, $t = 1.65$, $p = 0.10$ did not significantly predict internalizing symptoms, but that females reported greater internalizing symptoms than males $b = 1.40$, $t = 5.25$, $p < 0.001$. Therefore, we controlled for gender in all subsequent analyses. For each model with significant effects, we also conducted single-variable sensitivity analyses, where main effects were examined controlling for each covariate at a time. All results reported are those that remain in all single-variable sensitivity analyses, except where noted.

Aim 1. Parental Supportive Factors Buffering the Stress to Internalizing Symptoms Relationship

First, we tested whether adolescent stressful life events significantly impacted adolescent internalizing symptoms. We found that increases in stress exposure were associated with significant increases in adolescent psychopathology, $b = 0.04$, $t = 3.58$, $p = 0.001$.

After establishing the relationship between stress and internalizing symptoms, we next tested whether parental supportive factors buffered the impact of life events on internalizing symptoms. First, we looked at the parental support after a laboratory stress (TSST), including perceived parent support post-task and coded parental supportive behaviors. In models controlling for stress, we found that perceived parent support post-task, b = -.01, t = -1.48, p = 0.14 (Table 3.1, Model 1a), and parental supportive behaviors, b = 0.25, t = 0.68, p = 0.50 (Table 3.1, Model 1b), were not related to current internalizing symptoms. Further, neither perceived parent support post-task nor parent supportive behaviors buffered the association between life stress exposure and internalizing symptoms, Support*Stress b = 0.0002, t = 0.43, p = 0.67 (Table 3.1, Model 1c), Behaviors*Stress b = -0.04, t = -0.85, p = 0.40 (Table 3.1, Model 1d).

Table 3.1: Impact of parenting factors on adolescent internalizing symptoms

	b	*SE*	*df*	*t* value	*p* value
Model 1a					
Intercept	0.256	0.681	66	0.380	0.708
Gender	1.000	0.410	66	2.440	0.017
Parent Support Post-Task	-0.011	0.008	66	-1.480	0.142
Objective Stress	0.029	0.016	66	1.730	0.088
Model 1b					
Intercept	-0.669	0.436	46	-1.540	0.132
Gender	1.029	0.521	46	1.970	0.055
Positive Parent Behaviors	0.250	0.367	46	0.680	0.499
Objective Stress	0.030	0.022	46	1.380	0.175
Model 1c					
Intercept	0.362	0.728	65	0.500	0.620
Gender	0.976	0.416	65	2.350	0.022
Parent Support Post-Task	-0.012	0.008	65	-1.540	0.129
Objective Stress	0.011	0.044	65	0.260	0.798
ParentSupport*ObjectiveStress	0.001	0.001	65	0.430	0.667
Model 1d					
Intercept	-0.569	0.453	45	-1.260	0.215
Gender	0.963	0.528	45	1.820	0.075
Positive Parent Behaviors	0.171	0.379	45	0.450	0.654
Objective Stress	0.047	0.029	45	1.600	0.117
PositiveParentBehaviors*ObjectiveStress	-0.036	0.043	45	-0.850	0.398

Next, given that our parent support after a laboratory stress variable was only applicable to the subsample of our sample that was in the parent present condition, we tested whether the perceived parental support composite, which was reported by the entire sample, significantly impacted adolescent internalizing symptoms. In this model, we found that increases in parental support were significantly associated with lower internalizing symptoms b = -0.17, t = -3.20, p = 0.002 while controlling for adolescent life events (Table 3.2, Model 1e). However, we did not find evidence to suggest that perceived parental support buffered the link between stressful life events and internalizing symptoms, Support*Stress b = -0.003, t = -0.72, p = 0.47 (Table 3.2, Model 1f).

Table 3.2: Impact of parent support composite on adolescent internalizing symptoms

	b	*SE*	*df*	*t* value	*p* value
Model 1e					
Intercept	-0.610	0.191	142	-3.200	0.002
Gender	1.010	0.258	142	3.910	<0.001
Parent Support Composite	-0.165	0.052	142	-3.200	0.002
Objective Stress	0.029	0.010	142	2.890	0.004
Model 1f					
Intercept	-0.647	0.198	141	-3.270	0.001
Gender	1.042	0.262	141	3.970	<0.001
Parent Support Composite	-0.159	0.052	141	-3.030	0.003
Objective Stress	0.027	0.010	141	2.630	0.009
ParentSupportComposite*ObjectiveStress	-0.003	0.003	141	-0.720	0.473

Aim 2. Family Supportive Factors Buffering the Stress to Internalizing Symptoms Relationship

Next, we tested whether family functioning is associated with adolescent internalizing symptoms and whether greater family functioning buffers the stress to internalizing relationship. We found that greater family functioning was associated with greater internalizing symptoms, b = 0.89, t = 3.37, p = 0.001 (Table 3.3, Model 2a), which is the opposite direction than expected,

though in line with risk group differences in internalizing symptoms and family functioning levels. Additionally, we did not find that family functioning buffered the stressful life events to internalizing relationship, Stress*FamilyFunctioning $b = 0.009$, $t = 0.48$, $p = 0.63$ (Table 3.3, Model 2b).

Table 3.3: Impact of family functioning on adolescent internalizing symptoms

	b	*SE*	*df*	*t* value	*p* value
Model 2a					
Intercept	-1.406	0.290	136	-4.850	<.0001
Gender	1.150	0.258	136	4.460	<.0001
Family Functioning	0.893	0.265	136	3.370	0.001
Objective Stress	0.031	0.010	136	3.140	0.002
Model 2b					
Intercept	-1.403	0.291	135	-4.820	<.0001
Gender	1.166	0.261	135	4.470	<.0001
Family Functioning	0.868	0.271	135	3.210	0.002
Objective Stress	0.023	0.019	135	1.190	0.236
FamilyFunctioning*ObjectiveStress	0.009	0.019	135	0.480	0.630

Aim 3. (Exploratory) Family/Parent Supportive Factors Buffering the Stress to Internalizing Symptoms Relationship – Moderation by Familial Risk Status

Finally, we tested whether the buffering impact of parent and family support factors examined in Aim 1 and Aim 2, on the life events to internalizing relationship were further moderated by parental history of depression. First, we tested whether familial risk moderated the association between stress and internalizing symptoms and found that this was not the case, Risk*Stress $b = -0.01$, $t = -0.33$, $p = 0.75$. We also found no evidence that familial risk status moderated the stress-buffering associations of Aim 1 parent-level factors of perceived parent support post-task, $b = 0.0005$, $t = 0.15$, $p = 0.88$ (Table 3.4, Model 3a), parental supportive behaviors, $b = -0.09$, $t = -0.63$, $p = 0.53$ (Table 3.4, Model 3b), and perceived parent support via composite scores, $b = 0.002$, $t = 0.19$, $p = 0.85$ (Table 3.4, Model 3c). We also found familial

that risk status didn't moderate the stress-buffering effect of family functioning, b = -0.04, t = -0.75, p = 0.46 (Table 3.4, Model 3d).

Table 3.4: Impact of parent and family factors on adolescent internalizing symptoms – moderated by familial risk for depression.

	b	*SE*	*df*	*t* value	*p* value
Model 3a					
Intercept	0.768	3.375	61	0.230	0.821
Gender - Female	0.958	0.436	61	2.190	0.032
Gender - Male	0.000	-	-	-	-
Familial Risk for Depression (Risk) – High	-0.490	3.457	61	-0.140	0.888
Familial Risk for Depression (Risk) – Low	0.000	-	-	-	-
Parent Support Post-Task	-0.018	0.035	61	-0.510	0.613
Objective Stress	0.044	0.314	61	0.140	0.889
ParentSupportPost-Task*ObjectiveStress	0.000	0.003	61	-0.080	0.935
ParentSupportPost-Task*Risk – High	0.007	0.037	61	0.200	0.841
ParentSupportPost-Task*Risk - Low					
ObjectiveStress*Risk – High	-0.031	0.320	61	-0.100	0.924
Objective Stress*Risk - Low					
ParentSupportPost-Task*ObjectiveStress*Risk – High	0.001	0.004	61	0.150	0.881
ParentSupportPost-Task*ObjectiveStress*Risk – Low					
Model 3b					
Intercept	-1.179	1.264	41	-0.930	0.356
Gender - Female	0.944	0.552	41	1.710	0.095
Gender - Male	0.000	.	.	.	.
Familial Risk for Depression (Risk) - High	0.251	1.408	41	0.180	0.860
Familial Risk for Depression (Risk) - Low	0.000	.	.	.	.
Positive Parent Behaviors	0.561	1.147	41	0.490	0.628
Objective Stress	-0.027	0.120	41	-0.220	0.824
PostiveParentBehaviors*ObjectiveStress	0.016	0.120	41	0.130	0.897
PostiveParentBehaviors*Risk - High	0.089	1.347	41	0.070	0.948
PostiveParentBehaviors*Risk - Low	0.000	.	.	.	.
ObjectiveStress*Risk - High	0.102	0.129	41	0.790	0.432
ObjectiveStress*Risk - Low	0.000	.	.	.	.
PostiveParentBehaviors*ObjectiveStress*Risk - High	-0.089	0.141	41	-0.630	0.533
PostiveParentBehaviors*ObjectiveStress*Risk - Low	0.000	.	.	.	.
Model 3c					
Intercept	-0.628	0.269	137	-2.340	0.021

Gender - Female	1.062	0.267	137	3.970	0.000
Gender - Male	0.000	.	.	.	.
Familial Risk for Depression (Risk) - High	0.095	0.290	137	0.330	0.743
Familial Risk for Depression (Risk) - Low	0.000	.	.	.	.
Parent Support Composite	-0.236	0.097	137	-2.420	0.017
Objective Stress	0.045	0.020	137	2.210	0.029
ParentSupportComposite*ObjectiveStress	-0.006	0.009	137	-0.680	0.497
ParentSupportComposite*Risk - High	0.113	0.123	137	0.920	0.360
ParentSupportComposite*Risk - Low	0.000	.	.	.	.
ObjectiveStress*Risk – High	-0.028	0.024	137	-1.160	0.247
ObjectiveStress*Risk – Low	0.000	.	.	.	.
ParentSupportComposite*ObjectiveStress*Risk – High	0.002	0.010	137	0.190	0.850
ParentSupportComposite*ObjectiveStress*Risk - Low	0.000	.	.	.	.
Model 3d					
Intercept	-1.753	0.408	131	-4.300	<.0001
Gender – Female	1.173	0.259	131	4.530	<.0001
Gender - Male	0.000	.	.	.	.
Familial Risk for Depression (Risk) - High	0.697	0.546	131	1.280	0.205
Familial Risk for Depression (Risk) - Low	0.000	.	.	.	.
Family Functioning	1.391	0.538	131	2.590	0.011
Objective Stress	0.014	0.033	131	0.420	0.672
FamilyFunctioning*ObjectiveStress	0.052	0.046	131	1.140	0.258
FamilyFunctioning*Risk - High	-0.721	0.665	131	-1.080	0.280
FamilyFunctioning*Risk - Low	0.000	.	.	.	.
ObjectiveStress*Risk - High	-0.006	0.042	131	-0.150	0.878
ObjectiveStress*Risk - Low	0.000	.	.	.	.
FamilyFunctioning*ObjectiveStress*Risk – High	-0.039	0.052	131	-0.750	0.455
FamilyFunctioning*ObjectiveStress*Risk - Low	0.000	.	.	.	.

Exploratory Post-hoc analyses

Given that we did not find the associations we hypothesized, additional exploratory analyses were conducted to explore what might be driving these non-effects. First, this study varied from past findings suggesting a role of parents and family (Balistreri & Alvira-Hammond, 2016) in buffering stress in that we used an investigator-rating of objective stress of the reported

life events as opposed to a participant-reported checklist of life events. However, we replicated our analyses using the participant-reported subjective total score from the SLES. While greater subjective stress scores were associated with more internalizing symptoms, $b = 0.02$, $t = 4.70$, $p < 0.0001$, none of the parent/family support factors provided buffering of this relationship.

We also replicated our analyses using a broader adolescent self-report measure of perceived stress, which is not linked to specific life events (PSS). We found that greater perceived stress was associated with greater internalizing symptoms $b = 0.19$, $t = 13.80$, $p < 0.0001$. However, neither perceived parent support post-task nor parent behaviors were associated with a buffering effect. Likewise, neither perceived parental support in the whole sample nor family functioning moderated the link between perceived stress and internalizing symptoms.

Finally, our measure of life events focused on events that occurred in the past 12 months. However, family factors may have a buffering effect on the more long-term exposure to stress. Therefore, we re-ran all of our models with the adolescent report of childhood trauma (CTQ). We found that greater reported childhood trauma was associated with greater internalizing symptoms, $b = 0.07$, $t = 4.06$, $p < 0.0001$. However, none of our parent/family support factors buffer against this type of stress to internalizing relationship. Additionally, these were not moderated by risk status, suggesting that for both groups, this buffering association was non-existent.

Discussion

We tested whether parent and family supportive factors buffered the association between exposure to stressful life events and internalizing symptoms and whether this was the case for both youth at high and low risk for depression. We found a strong relationship between exposure

to stressful life events and higher levels of internalizing symptoms. However, none of the parent or family supportive factors were associated with a buffering of the life stress to internalizing relationships, and this did not vary between teens at high and low familial risk for depression. Post-hoc analyses tested several potential explanations of these non-effects by using multiple conceptually different measures of stress. While these alternative stress measures were linked to higher internalizing symptoms, none of the parent and family supportive factors moderated the link between stress and internalizing. Therefore, contrary to expectation, our findings suggest that parents may have a very limited role in buffering the impact of stress on adolescents internalizing symptoms.

Overall, our results consistently failed to suggest that parental and family factors have a buffering effect on the impact of life stress on internalizing symptoms in the adolescents in this sample. This lack of buffering effect was consistent across all of our parenting and family functioning measures, including perceived parental support after a laboratory stressor, overall (non-stress related) perceived parenting support, and adaptive family functioning. This lack of buffering effect was also consistent regardless of how we measured stress, including experimenter-rated severity of life events using contextual information, participant-rated subjective rating of stress, as well as measures of early life stress, and perceived recent stress. These findings are inconsistent with previous findings related to the stress-buffering effect of parent support on negative mental health outcomes (Czyz, Liu, & King, 2012; Kang et al., 2017; Mackin et al., 2017). For example, perceived parental support in emotional, academic, and financial areas all were related to a buffering effect of life stress on suicidal ideation in a non-depression sample (Kang et al., 2017). Additionally, adolescent perceived parent connection was associated with fewer depression symptoms across the year post-discharge for psychiatrically

hospitalized youth (Czyz et al., 2012). However, our study differs significantly from these past studies in our unique measurement of stress and population sample. To our knowledge, we are the first study to use an individual differences approach to explore whether these parent and family factors mitigate the impact of stressful life events in a population oversampled for higher familial risk for depression. Our findings may reflect that this type of parenting support is not associated with buffering of the association of stress to internalizing symptoms when the stress exposure is broader and over the course of a larger amount of time in this unique population.

Nonetheless, our findings are consistent with literature that suggests that as youth advance through adolescence, family and parent support are less important than other sources of support, such as that from peers (Furman & Buhrmester, 1992). Specifically, peers who are low on parent support, but higher on peer support were shown to be more well-adjusted than those who reported low peer support but high parent support (Laible, Carlo, & Raffaelli, 2000). Additionally, nuances in how these two support systems influence adolescent outcomes remain. For instance, some findings suggest that the benefits of peer support depend slightly on whether or not those teens also have supportive parents (Helsen, Vollebergh, & Meeus, 2000). In addition, other factors of the adolescent-parent relationship may matter more for facilitating coping, such as parent-teen relationship quality (Pool & Ford, 2019), involvement and communication (Brody et al., 2005), or parental warmth (Butterfield et al., 2021). It is possible that our measures of positive parenting failed to capture specific aspects of parenting that may be most supportive during the adolescent period.

Our findings from our exploratory Aim 3 also suggest that the potential buffering effect of parents was not moderated by parental history of depression, such that there were no differences in buffering (or lack thereof) for high-risk and low-risk teens in our sample. While

this is inconsistent with research that suggests parental depression negatively impacts positive parenting behaviors (Lovejoy et al., 2000; Wilson & Durbin, 2010), other research suggests some aspects of family functioning and parenting may not be impacted by parental history of depression (Bouma et al., 2008). The differences in these studies may be related to the current depressive state versus a history of depression. However, we did not examine parental current depression symptoms, and thus these differences may be only present when parents are depressed and are not based on a history of depression.

Another unexpected finding relates to our measure of family functioning. Contrary to our hypothesis, greater amounts of "positive" family functioning were associated with greater amounts of internalizing symptoms. In addition, teens at higher familial risk for depression reported higher levels of positive family functioning than their low-risk peers. Multiple explanations for these findings exist. First, it is possible that high levels of positive family functioning, as measured in our study, maybe harmful during this developmental stage. For example, enmeshment, which is a form of over-functioning in families, is associated with greater levels of adolescent and emerging adult distress (Prioste, Tavares, & Magalhães, 2019). Additionally, high levels of positive family functioning may reflect overprotection, such as parents getting in the way of their adolescents pursuing independence or other forms of support (e.g., peer support) that might be more beneficial to them. Another potential explanation is that given the stress generation hypothesis of depression, internalizing symptoms are associated with increased experiences of stress over time (Hammen, 2006), and the transactional nature of parent/adolescent relationships, perhaps those with greater internalizing symptoms are eliciting more support from their families. This may also explain why youth at high familial risk for depression reported greater positive family functioning than their low-risk peers. Likewise, it is

also possible that this effect is due to a sampling bias impacting our high-risk sample. Specifically, our sample of parents with a history of depression may include an overrepresentation of parents with greater positive family functioning given their interest in supporting science that aims to understand the risk experienced by their children. Research on depression in adults is known to be especially difficult because depression impacts research participation (Hughes-Morley, Young, Waheed, Small, & Bower, 2015). Given this, our high-risk parent participants may reflect a unique subpopulation characterized by positive family functioning.

The findings of the current study should be considered in light of several limitations. First, while our unique sample of low and high risk for depression adolescents provides the opportunity for exploring how protective factors may impact these two groups differently, it is not a representative sample of typically developing adolescents, and therefore these findings should not be applied to other groups of adolescents. Additionally, our sample was considered "High Risk" based on familial risk for depression only, but did not include risk for other commonly co-occurring psychiatric disorders, such as anxiety disorders, and are therefore a very specific definition of high risk of depression. Given that the majority of our participant parents were mothers, are larger sample may also allow for additional examination of differences in these associations that may arise when parents and adolescents are participating with the same gender parent versus participating with their opposite gender parent. Additionally, for models where we did not find a significant result, it is possible that these relationships have smaller effect sizes and therefore we may not have been powered to detect such a small effect. In these cases, a larger sample may provide power to detect relationships with a smaller effect. Additionally, because we only had behavioral data for half of our sample, this limited our ability

to compare observed behaviors and self-report measures across both parent present and absent groups. The behavioral data itself was also summarized into one variable which included positive minus negative parenting behaviors. Using this type of summary variable creates heterogeneity in the definition of "High" levels of parenting support behaviors as it may include people with different levels of negative parenting behaviors. Additionally, as this study was cross-sectional, we are not able to say anything definitive about the causation between parenting factors and internalizing symptoms. Longitudinal data are needed to further answer these questions. Finally, our study did not include measurements of teen-level factors that may influence these bidirectional relationships. For instance, teen irritability, their propensity to accept support, or their preferred style of support were not included and may have had an impact on how the parents were behaving in the interactions.

In conclusion, we found no evidence that parenting or family factors, as measured in this study, buffered the relationship between stress exposure and internalizing symptoms in a sample of adolescents at high and low familial risk for depression. This lack of buffering effect was found across multiple measures of parenting support and stress. These findings suggest that parenting support may have limited utility in minimizing the effect of stressful life events in adolescents. Future studies should examine other aspects of parenting that may be more relevant to this population with hopes to elucidate stronger parenting support interventions and additional non-parenting factors that do take a more prominent role in buffering stress in this population.

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Chapter 4 Parental Caregiving Stress and the Implementation of Adolescent Safety Recommendations for Parents of Adolescents at High Risk for Suicide

Abstract

Youth hospitalizations for suicide attempts and ideation are increasing and parents are tasked with implementing important safety recommendations post-discharge. Parents experience increased stress during this post-discharge time, but little is known about how this impacts their ability to implement safety recommendations. The current study examines caregivers and their offspring in the three months post-discharge for a sample of parents of teens who were psychiatrically hospitalized (n = 80). Parents and teens completed measures of psychopathology and levels of parental implementation of safety recommendations post-discharge. Parents also reported on their level of caregiving stress and their perceived parenting self-efficacy. We found that parents experience high levels of caregiving stress that decrease linearly across the three months post-discharge. We also found that baseline self-efficacy was associated with greater decreases in stress and that baseline teen severity of illness was associated with less steep decreases in stress over time. Increases in parental psychopathology from baseline to 3-months were associated with less steep declines in stress over time. Stress and self-efficacy were associated with increases in parental ability to implement safety recommendations post-discharge. This study adds to the growing literature focused on parental experiences of stress and self-efficacy in this high-risk population and suggests that parental self-efficacy might be an important target for intervention.

Keywords: adolescent, suicide, caregiving stress, stress, self-efficacy, parents, parenting.

Introduction

Suicide is one of the leading causes of death for adolescents in the United States and has been increasing in recent years (Centers for Disease Control and Prevention, 2018). While most research has focused on the pain and impairment endured by the teens themselves, suicide attempts often cause severe stress and strain on the parents and caregivers (Lindqvist, Johansson, & Karlsson, 2008). During this stressful time, caregivers are tasked with implementing important safety measures to help keep their teens safe post-discharge (e.g., means restriction and monitoring) and are often responsible for ensuring the teens' treatment compliance and adherence (King, Ewell-Foster, & Rogalski, 2013; Nock & Ferriter, 2005; O'Brien, Crickard, Lee, & Holmes, 2013). However, little is known about how the post-discharge stress experienced by parents might change over time nor what factors may contribute to and mitigate parental stress during this period. In addition, little is known about how parental stress and other parental characteristics impact their ability to implement safety recommendations. A deeper understanding of how parents are coping with stress during the post-discharge period may be beneficial in helping to develop novel parental support interventions to help reduce youth suicide.

Youth Suicide Epidemiology, Risk and Protective Factors

Rates of completed adolescent suicide have been increasing in recent years (Centers for Disease Control and Prevention, 2018). Likewise, the rates of hospital and emergency department visits due to suicide attempts have also increased (Plemmons et al., 2018). Each year, about 19% of high school students report suicidal ideation, and about 9% experience non-lethal suicide attempts (Ivey-Stephenson et al., 2020). In addition, suicidal behaviors impact some demographic groups more than others. For example, there are higher rates of suicide attempts

among females than males, black non-Hispanic youth than other racial/ethnic groups (Ivey-Stephenson et al., 2020), and teens identifying as LGBTQ than their non-LGBTQ peers (Berona, Horwitz, Czyz, & King, 2020; McNeil, Ellis, & Eccles, 2017; Miranda-Mendizábal et al., 2017).

In light of these recent trends, there has been an emphasis on identifying risk factors for suicide. Known risk factors include a history of previous suicidal behavior and ideation (Berman, 2018; Bostwick, Pabbati, Geske, & McKean, 2016; Large, Corderoy, & McHugh, 2020), and depression and hopelessness (Ribeiro, Huang, Fox, & Franklin, 2018). Further, the interpersonal theory of suicide (Van Orden et al., 2010) posits that the overlap of two interpersonal experiences (thwarted belongingness and perceived burdensomeness) are related to suicidal behavior, and as such, research has examined interpersonal risk factors such as bullying and peer victimization (van Geel, Vedder, & Tanilon, 2014).

Given that adolescents reside within a family social context, parent and family processes may play an important role in *reducing* the risk for suicide (Kang et al., 2017; Mackin, Perlman, Davila, Kotov, & Klein, 2017). Parents and families help to facilitate coping in their adolescents, which may be especially true for parents of adolescents who are at high risk for suicide (Mackin et al., 2017). For example, parental involvement is an important protective factor in reducing suicide risk (Brausch & Gutierrez, 2010; Flouri & Buchanan, 2002; Kang et al., 2017; Miller, Esposito-Smythers, & Leichtweis, 2015) and greater parental support may increase adolescent help-seeking behaviors as those with greater support are more likely to disclose a suicide attempt to their parents (Levi-Belz et al., 2019).

Caregiving Stress in Parents of Youth at High Risk for Suicide

Caring for a child with a severe mental health condition is a potent stressor for parents (Brannan, Heflinger, & Bickman, 1997; Ha, Hong, Seltzer, & Greenberg, 2008) and this may

impact parental ability to contribute to their child's care. This may be especially true when caring for a teen who displays suicidal behaviors. For example, parents of children who deliberately self-harm have been shown to have lower levels of social support and parenting satisfaction (Morgan et al., 2013) and face unique stigma and blame (Calhoun, Selby, & Faulstich, 1980). Additionally, qualitative interviews suggest that parents experience a range of emotions, including shock, fear, and shame, following their child's suicide attempt and that these parents have a strong desire for additional social support during that time (Ngwane & van der Wath, 2019). However, surprisingly little is known about how parents of psychiatrically hospitalized youth cope across the post-discharge time or how risk or protective factors might influence stress and their ability to implement safety recommendations. To our knowledge, no quantitative assessment of parent stress during the post-discharge period after hospitalization exists.

Impact of parental self-efficacy on caregiving stress

In addition, little is known about parental and family-level factors that can contribute to or mitigate parental stress during this time and therefore influence their effectiveness in their supportive role. Parental self-efficacy may play an important role in a parent's ability to implement safety recommendations and help teens regulate. According to self-efficacy theory, a personal sense that a challenge can be overcome is the mechanism through which people may initiate certain coping strategies during times of difficulty (Bandura, 1977; Bandura, 1982) and is related to the implementation of other public health behaviors (Sheeran et al., 2016). Parental self-efficacy, which is a specific type of self-efficacy related to parenting behaviors, has been shown to have important benefits to teen mental health outcomes (Albanese, Russo, & Geller, 2019). Parents of teens with fewer suicidal events have reported greater self-efficacy to keep their teen safe suggesting that parental self-efficacy may be a targetable factor for suicide risk

prevention interventions (Ewa K Czyz, Horwitz, Yeguez, Ewell Foster, & King, 2018). Additionally, parental self-efficacy may have the power to reduce parental stress as self-efficacy is a type of perceived control and therefore a potent anti-stress construct (Skinner & Zimmer-Gembeck, 2010). Therefore, parental self-efficacy might be a modifiable factor that could buffer some of the strain that parents experience after their teen is discharged from a psychiatric inpatient unit.

Impact of Parental and Adolescent Mental Health on Caregiving Stress and Teen Outcomes

Parental mental health can also affect post-discharge parental stress and teen outcomes. Specifically, parental psychopathology and parental suicidal ideation have been associated with increases in adolescent suicidal ideation (Lee, Lee, & Han, 2019), attempts (Burrell, Mehlum, & Qin, 2018; Goodday, Shuldiner, Bondy, & Rhodes, 2019; Hua, Bugeja, & Maple, 2019; Mok et al., 2016), as well as earlier time to first attempt (Itzhaky et al., 2020). Further, psychopathology itself increases stress and stress sensitivity (Liu & Alloy, 2010). Parent stress and teen suicidal behaviors also likely have transactional causal links. Specifically, having a teen that struggles with suicidal behaviors and/or attempts may increase parental stress (Ha et al., 2008), which in turn may increase suicide risk for the adolescents (Lee et al., 2019). Despite this cycle, levels of caregiver stress and subsequent negative impacts may vary as a function of the level of risk factors (e.g., the severity of teen difficulties, parent's mental health). However, to our knowledge, the impact of parental and adolescent severity of psychopathology on parental caregiving stress has not been examined in a recently psychiatrically hospitalized population.

Parental Role in Facilitating Safety Recommendations

Parents also play an important role in monitoring and responding to suicidal behavior and implementing safety recommendations within their homes. For example, parents who have teens at high risk for suicide are often asked by clinicians to monitor their teens for suicidal behaviors and warning signs, as this reduces the risk for suicide (Moon, Kim, & Parrish, 2020). In cases where a teen is hospitalized for suicidal behavior or attempts, parents are also given post-discharge safety recommendations that include means restriction: removing or securing firearms, medications, and/or sharp objects in the home. Additionally, parents play a role in supporting adolescent treatment compliance with pharmacotherapy and psychotherapeutic treatments (e.g., supporting and monitoring medication regimens, transportation to appointments, etc.). In sum, parental involvement and support during and after a suicidal crisis are paramount in keeping high-risk teens safe.

However, parental ability to adhere to these safety recommendations and support their teen may be negatively influenced by caregiver stress and this may likely be mitigated by modifiable protective factors. For example, self-efficacy has been shown to mitigate the negative effect of stress on the ability to cope with bullying (Benatov, 2019) and teachers' ability to avoid stress-induced burnout (Boujut, Popa-Roch, Palomares, Dean, & Cappe, 2017). Additionally, there is emerging evidence that parental self-efficacy may be linked to the ability to implement safety recommendations. For example, parents of emergency treatment-seeking youth who had fewer suicide behaviors post-emergency visit reported greater self-efficacy, (Czyz et al., 2018). Given the stress-reducing capabilities, it is likely that parental self-efficacy would buffer the negative impact of caregiving stress on parents' ability to implement safety recommendations for

their teens. However, to our knowledge, no previous work has examined this relationship in parents of high risk for suicide teens.

Conclusion and Proposed Aims and Hypotheses

In conclusion, attending to a distressed teen after suicide-related hospitalization is likely very stressful for parents but few studies have characterized post-discharge parental stress. It is also unknown whether the same factors that contribute to and mitigate parental stress generally also play a role in post-discharge stress in this population. Therefore, this study aims to characterize caregiver stress over three months post-discharge for a sample of parents whose teens have been psychiatrically hospitalized for suicidal ideation or attempt. Additionally, we will explore how parent and teen factors might covary with caregiver stress over time. Finally, we will examine whether caregiver stress predicts parental ability to implement follow-up recommendations (e.g., asking about suicide, encouraging coping) and we will test whether parental efficacy might buffer the negative effects of caregiver stress on recommendations. We examined the following aims and associated hypotheses:

Aim 1

In Aim 1, we characterized levels of parental caregiving stress over the three months after their adolescent was discharged from psychiatric hospitalization due to a suicide attempt and/or ideation. This exploratory aim examines how parents of this high-risk population experience stress related to caregiving responsibilities. Although this aim is exploratory, we speculate that stress levels will peak immediately after discharge when the trauma of the hospitalization is fresh and would decline over time.

Aim 2

In Aim 2, we examined how risk and protective factors (parent and adolescent level factors) might relate to caregiver stress trajectories over these three months. Specifically, we characterized how parental self-efficacy, parental psychopathology, parent-family support, and adolescent severity of illness might covary with caregiving stress over the three-month time. We hypothesize that risk factors (parental psychopathology, adolescent illness severity) will be associated with less decline in caregiving stress and that the protective factors (parental self-efficacy, parent-family support) will be associated with greater declines in caregiving stress.

Aim 3

In Aim 3, we characterized the association between caregiving stress and parental ability to implement safety recommendations. We also explored whether parental self-efficacy moderates the potential association between parental caregiving stress and the implementation of safety recommendations. We hypothesized that greater levels of caregiving stress at baseline would be related to decreased ability of parents to implement recommendations to keep their teens safe. We also hypothesized that greater levels of self-efficacy at baseline would be associated with greater ability for parental safety implementation. Further, we hypothesized that parental self-efficacy would moderate the association between stress and safety implementation, such that greater parental self-efficacy at baseline would buffer the stress to safety implementation relationship.

Methods

Participants

Participants are 80 adolescents and one of their parents participating in a pilot trial for an adaptive intervention for the prevention of adolescent suicidal behavior in a sample of

hospitalized youth (PI: Ewa Czyz). The development and feasibility/acceptability study (Czyz, King, & Biermann, 2019), as well as the main outcome study (Czyz et al., 2021), have been published elsewhere. Participants were recruited on a child and adolescent inpatient unit at a Midwest children's hospital. Participants were enrolled if they were psychiatrically hospitalized in the inpatient unit with a presenting concern of a recent suicide attempt (within last month) and/or recent suicidal ideation (within last week, with thoughts of method, intent, and/or plan). Participants were excluded if they did not have an available legal guardian, were going to be discharged to a long-term residential care facility, did not have access to a personal cell phone, or had severe cognitive impairment or altered mental status (e.g., mania or psychosis). The majority of the parents who participated in this study with their teen were mothers (74%). The average participant age was 15.16 (SD = 1.35, Range 13-17) and 67.5% (n = 54) of the adolescents were female. The participants' self-identified race was 83.8% (n = 67) White, 6.3% (n = 4) Black or African American, 5.0% (n = 4) Asian, 5.0% (n = 4) American Indian or Alaskan Native, and 1.3% (n = 1) Native Hawaiian or Other Pacific Islander. Two participants (2.5%) reported their race as "Other" and nine participants (11.3%) identified ethnically as Hispanic or Latino/a. Half of the sample (n = 40) reported at least one lifetime suicide attempt, with 35% (n = 28) reporting multiple lifetime suicide attempts. More than a third (37.5%, n =30) of the sample attempted suicide in the month before hospitalization. Retention across the three months of the study was high (95.0% adolescents and 93.8% parents at 1 month; 91.3% adolescents and 82.5% parents at 3 months).

This study was approved by the University Institutional Review Board and informed consent and assent were obtained from all adolescent participants and their parents before

participation. The adolescent participants were compensated up to $222 for their study participation, and the parents were paid up to $50 for their participation.

Procedures

Data came from a larger pilot sequential multiple assignment randomized trial (SMART) intervention aimed at reducing the risk of suicide in adolescents. In brief, though not of central importance to the current study, participants and their parents completed motivational interviewing-enhanced safety planning sessions (individual and teen/parent conjoint sessions). Following the intervention, participants were then randomized at two time points to various follow-up support conditions. All teens filled out daily mood questionnaires for four weeks (28 days) after discharge and these questionnaires were monitored for risk and a comprehensive risk management protocol was followed. Given the potential impact of these treatment randomizations on our variables of interest in the current study, randomization to the treatment group will be treated as a potential covariate that may impact our results and will be controlled for when this is the case. In these cases, randomization to "Booster/No Booster" was treated as one covariate, while randomization to "Text Support/No Text Support" was treated as another covariate.

The current study utilized the parent and adolescent self-report survey data from this trial. Parents and adolescents completed baseline surveys in the hospital before receiving the intervention. They then completed follow-up surveys at one month and three months post-discharge via phone calls administered by interviewers who were blind to participant condition.

Measures

Parental caregiving stress

Parents self-reported their level of caregiving stress at baseline, one month, and three months post-discharge by completing the short form Caregiver Strain Questionnaire (CSQ-Short Form 7; (Brannan, Athay, & de Andrade, 2012). This scale was developed with parents of children with autism spectrum disorders and is a valid measure of overall stress associated with caring for a child with a mental health condition (Brannan et al., 1997). This short version has been shown to have similar psychometric properties (Brannan et al., 2012). The measure has been used by parents of children and adolescents with depression and other mental health difficulties (Jaycox et al., 2009; Sales, Greeno, Shear, & Anderson, 2004). This measure asks parents to rate their agreement with various statements over the past month on a scale of 0 "Not at all" to 4 "Very Much". Items include questions about subjective strain (e.g., "How worried did you feel about your child's future?") and objective strain related to caregiving (e.g., "Financial strain for your family as a result of your child's emotional or behavioral problem? "). Cronbach's alpha for our sample was 0.87 at baseline, 0.89 at one month, and 0.91 at three months indicating good internal consistency.

Parental self-efficacy

Parents self-reported their feelings of self-efficacy to support their teens during a time of crisis via the Parent Self-Efficacy scale (Czyz et al., 2018), which was developed in consultation with experts in suicide prevention research. The scale was developed using a sample of parents of emergency department treatment-seeking youth, and modeled after self-efficacy scales for other behaviors. The 10-item scale asks parents to rate how confident they are (0 "Not at all Confident to 10 "Completely Confident") on various aspects of support for high-risk adolescents

including asking about adolescent mood and thoughts of suicide, supporting adolescents in using coping strategies, and helping to limit access to lethal means. Cronbach's alpha for our sample was 0.79 at baseline, 0.85 at one month, and 0.80 at three months indicating acceptable internal consistency.

Parental psychopathology

Parents self-reported their depression and anxiety symptoms at baseline, one month, and three months post-discharge by completing the Patient Health Questionnaire-4 (PHQ-4; Kroenke, Spitzer, Williams, & Löwe, 2009). The PHQ-4 is a brief screener which combines the PHQ-2 (Kroenke, Spitzer, & Williams, 2003) which assesses core symptoms of depression ("little interest or pleasure in doing things " and "feeling down, depressed, or hopeless") and the GAD-2 (Staples et al., 2019) which assesses core symptoms of anxiety ("feeling nervous, anxious or on edge" and "not being able to stop and control worrying"). This brief screener asks parents to rate how often they have been bothered by each of these four core symptoms over the past two weeks on a scale of 0 "Not at all" to 3 "Nearly every day". The PHQ-4 is a reliable and valid brief screener of the core anxiety and depression symptoms in the general population (Löwe et al., 2010). Cronbach's alpha for our sample was 0.92 at baseline, 0.84 at one month, and 0.89 at three months, indicating good internal consistency.

Parental Ability to Implement Safety Recommendations

Parent report. Parents reported the degree to which they were implementing important safety and coping recommendations at one month and three months post-discharge by completing the Parent Behavior Follow-Up Outcomes. This measure asks eight questions about the extent to which parents have implemented recommendations regarding asking their children about their moods and thoughts of suicide and talking with their children about using their safety

plan. The measure asks how much they have done certain activities on a scale of 0 “Not at all” to 4 “Very much”. Two of the questions ask parents to report on what they have done to lock away or remove firearms and medications. This collection of items was based on standard clinical care recommendations (King et al., 2013) and modeled after a measure used in a study of parents of youth who sought treatment from an emergency department (Jackson et al., 2018). We excluded the items related to means restriction because adolescents were not asked about these items, and summed the six other items, with higher scores indicating a greater report of implementing recommendations. Cronbach’s alpha for our sample was 0.77 at one month and 0.77 at three months, indicating acceptable internal consistency.

Adolescent report. Adolescents reported the degree to which they felt that their parents were implementing important safety and coping recommendations at one month and three months post-discharge by completing the Behavior Follow-Up Outcomes. This measure asks six questions about the extent to which adolescents perceive that their parents have implemented recommendations regarding asking about the adolescents’ mood and thoughts of suicide and talking with them about using their safety plan. The measure asks how much they perceive their parents to have done certain activities on a scale of 0 “Not at all” to 4 “Very much”. The adolescent version does not ask the two items about means restriction. This collection of items was based on standard clinical care recommendations (King et al., 2013) and modeled after a measure used in a study of parents of youth who sought treatment from an emergency department (Jackson et al., 2018). We summed the six items, with higher scores indicating a greater report of implementing recommendations. Cronbach’s alpha for our sample was 0.87 at one month and 0.86 at three months, indicating good internal consistency.

Adolescent severity of illness (suicidal ideation, attempts, depression, and anxiety)

Severity of Suicidal Ideation and history of attempts. Clinical interviewers ascertain adolescent severity of suicidal ideation at baseline, one month, and three months post-discharge by completing the Columbia-Suicide Severity Rating Scale (C-SSRS; Posner et al., 2011), which assesses the severity of suicidal ideation on a scale of 1 "Wish to be dead" to 6 "Suicidal ideation with specific plan and intent". The number of lifetime suicide attempts is also asked. The C-SSRS is a widely used measure of severity of suicidal ideation that has demonstrated strong psychometric properties and strong convergent validity with other measures of suicidal ideation (Posner et al., 2011).

Adolescent Depression Symptoms. Adolescents self-reported their depression symptoms at baseline, one month, and three months post-discharge by completing the Patient Health Questionnaire-9 adapted for adolescents (PHQ-9; Nandakumar et al., 2019). The PHQ-9 for adolescents is a widely used measure of depression symptoms and is a reliable and valid instrument in adolescent populations (Richardson et al., 2010). This measure asks participants to rate how much each symptom of depression has bothered them over the past two weeks on a scale of 0 "Not at all" to 3 "Nearly every day" and includes core symptoms of depression including low mood, lack of motivation, sleep, and appetite changes. Cronbach's alpha for our sample was 0.83 at baseline, 0.82 at one month, and 0.88 at three months, indicating good internal consistency.

Adolescent Anxiety Symptoms. Adolescents self-reported their anxiety symptoms at baseline only by completing the (GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006). The GAD-7 is a reliable and valid screener for anxiety symptoms in acute psychiatric care (Kertz, Bigda-Peyton, & Bjorgvinsson, 2013) and asks participants to rate how much they agree with

items on a scale of 0 “Not at all“ to 3 “Nearly every day“. Items include core symptoms of anxiety including feeling anxious or worried and the inability to control worry. Cronbach’s alpha for our sample was 0.88, indicating good internal consistency.

Adolescent Severity Index Score. Adolescent severity of illness was operationalized by creating an index score at each of the time points, which included adolescent depression symptoms, adolescent severity of suicidal ideation, number of lifetime suicide attempts, and adolescent anxiety symptoms. We created an optimally-weighted factor score for each time point, by running separate factor analyses for each time point to derive each item’s weight from their factor loading. We then multiplied that weight by the individual scores and summed them together. Cronbach’s alpha of the index scores ranged from 0.83 to 0.89 across the three time points, indicating good internal consistency.

Parent-Family Support

Adolescents reported how they perceive their families as supportive using the Parent-Family Connectedness scale, which was used in the National Longitudinal Study on Adolescent Health (Sieving et al., 2001). This 11-item scale asks three items related to how much the family is perceived as supportive and then four items that are specific to maternal support and four items specific to paternal support. Each question is rated on a 1 “Not at all” to 5 “Very Much” scale. For participants who participated in the study with their mother, their total family support variable was calculated by averaging the four items related to maternal support. For those who participated with their fathers, their family support total was calculated by averaging the four items related to paternal support. For those that participated with another type of family member, their family support total was calculated by averaging the three items related to general family support. Greater scores indicate higher levels of perceived family support. Cronbach’s alpha for

maternal support ranged from 0.86 to 0.90, paternal support from 0.90 to 0.93, and family support from 0.74 to 0.78, indicating acceptable to good internal consistency.

Planned Analyses

Sensitivity and interaction analyses

All analyses were conducted on SAS statistical software (SAS Studio v. 5.2). For each aim, we conducted covariate analyses to test the impact of covariates on dependent variables. These covariates include randomization to text condition, randomization to booster session condition, age, sex, gender, race, ethnicity, and parental education (For Aims 1-3). For aim 3, covariate analyses also included parental psychopathology and adolescent severity of illness. We subsequently controlled for all covariates that predicted our dependent variables in our main models. Then, to address the possibility that the overall findings were impacted by reduced power due to a greater number of covariates, we conducted additional single-variable sensitivity analyses, where the main effects were examined when controlling for each covariate at a time in individual models. We conducted post-hoc power analysis, based on the sample size and number of parameters in the most complicated model, to determine whether we were sufficiently powered to detect moderate effect sizes (0.35) at 80% power. This power analysis achieved a power of 0.99, which is above the minimum threshold value of 0.80. Thus, for these parameters and sample size, there is sufficient power to support the analyses results.

Aim 1

The first aim was to characterize parental caregiving stress throughout the three months post-discharge. We used a mixed-effects model to predict caregiving stress trajectory using growth curve modeling to determine whether there are linear or non-linear trajectories of caregiving stress over time. Due to sample size, we were unable to conduct models that allow for

examination of specific groups (e.g. growth mixture modeling). However, we examined general group membership by estimating how many participants had stable, increasing, or decreasing caregiving stress over time to verify that our models explained the trajectory of stress for a large proportion of our sample.

Aim 2

Aim 2 examined risk and protective factors that might covary with increasing or decreasing levels of parental caregiving stress over time. We predicted caregiving stress trajectory using mixed-effects growth curve modeling (the base model from Aim 1) and then predicted these caregiving trajectories from the following predictors: parental psychopathology, teen severity of illness, parent-family support, and parental self-efficacy. We conducted two different types of models: one set of models that predicted caregiving stress trajectories from baseline levels of the predictors and one set of models predicting caregiving stress trajectories from time-varying levels of the predictors (predictor slopes). Slopes of the predictors were individually derived for each participant and reflected the change from baseline to three months post-discharge. We used individual unadjusted models of each factor predicting caregiving stress trajectories individually as well as a fully adjusted model with all predictors.

Aim 3

For Aim 3, we used repeated measures general linear models to a) characterize the relationship between baseline parental *caregiving stress* and parents' ability to implement safety recommendations for their teen from both parent's perspective and adolescent perspective; b) characterize the relationship between baseline *parental self-efficacy* and parents' ability to implement safety recommendations for their teen from both parent's perspective and adolescent

perspective; and c) test whether parental self-efficacy might moderate the caregiving stress to safety implementation relationship from both parent and adolescent perspective.

Results

Sample Characteristics and Covariate Analyses

Means and standard deviations of key variables are reported in Table 4.1 and Table 4.2 for parent and adolescent participants. Covariate analyses for Aim 1 and Aim 2, which tested covariates that may impact caregiving stress over time in mixed-effects growth models revealed that randomization to text, randomization to boosters, age, sex, gender, race, ethnicity, maternal and paternal education, and parent participant's relationship to the teen were *not* associated with caregiving stress trajectories. Therefore, models predicting caregiver stress in Aims 1 and 2 will be reported *without* covariates, except where covariates impacted the findings in single variable sensitivity analyses. Covariate analyses for Aim 3 which tested covariates that may impact safety recommendations from the parent's report revealed that randomization to boosters, age, sex, race, ethnicity, parental relationship, and mother education did not impact safety implementation. However, the impact of randomization to text support, adolescent gender (girl vs. other), and parental psychopathology significantly predicted safety implementations. The only covariates that predicted parental safety implementation from the adolescent perspective were adolescent sex (female vs. other) and father education level (college education or higher vs. other). Aim 3 models predicting safety implementation will control for covariates that significantly predicted the outcome of interest, and all models reported are those that remain after single-variable sensitivity analyses are done with all covariates individually, except where noted (e.g. in the case where covariates impacted the results in the single-variable sensitivity analyses).

Table 4.1: Adolescent Sample Characteristics

	Baseline	1 month post-discharge	3 months post-discharge
Count	$n = 80$	$n = 74$	$n = 68$
Lifetime suicide attempts	$M = 0.49$, $SD = 0.50$		
Anxiety (GAD-7)	$M = 14.19$, $SD = 5.46$		
Depression (PHQ-9)	$M = 18.13$, $SD = 5.47$	$M = 10.01$, $SD = 5.65$	$M = 9.71$, $SD = 6.30$
Severity of SI (CSSRS)	$M = 3.91$, $SD = 0.90$	$M = 1.80$, $SD = 1.49$	$M = 1.27$, $SD = 1.47$
Family Support	$M = 19.24$, $SD = 5.64$	$M = 20.08$, $SD = 5.97$	$M = 20.27$, $SD = 5.62$
Safety Implementation		$M = 16.37$, $SD = 5.58$	$M = 14.99$, $SD = 5.59$

Table 4.2: Parent Sample Characteristics

	Baseline	1 month post-discharge	3 months post-discharge
Count	$n = 80$	$n = 74$	$n = 68$
Parental Psychopathology (PHQ4)	$M = 4.76$, $SD = 3.67$	$M = 4.52$, $SD = 3.22$	$M = 4.89$, $SD = 3.95$
Self-Efficacy	$M = 78.60$, $SD = 12.73$	$M = 86.20$, $SD = 11.27$	$M = 87.14$, $SD = 8.95$
Caregiving Stress (CSQ-SF7)	$M = 16.56$, $SD = 6.60$	$M = 14.71$, $SD = 7.45$	$M = 13.72$, $SD = 7.89$

Aim 1: Modeling Caregiving Stress Trajectory

We characterized changes in caregiving stress by first conducting a mixed-effects growth model to predict total parental caregiving stress trajectory over the three time points. We found that, overall, total parental caregiving stress declined over the three months post-discharge in a linear fashion, $b = -0.94$, $t = -3.66$, $p = 0.0004$ (Table 4.3). See Figure 4.1. A model with a non-linear term was not significant, $b = 0.44$, $t = 1.32$, $p = 0.19$, confirming that stress declined linearly overtime. We did a simple post-hoc verification to test whether this effect represented a significant number of parents by identifying people who displayed a decline in stress over time.

We found that 52.5% of the parents experienced a decline of 3.12 points (mean change in caregiving stress) or more. Additionally, 20% of the sample experienced either high stable (greater than mean at baseline plus less than average decline) or low stable (1 SD below the mean at baseline plus less than average decline). This suggests that our linear decline in caregiving stress is indeed driven by parents for whom stress was high and then declined over time (the majority of participants). See Figure 4.2.

Figure 4.1: Linear Association of Caregiving Stress Across Time

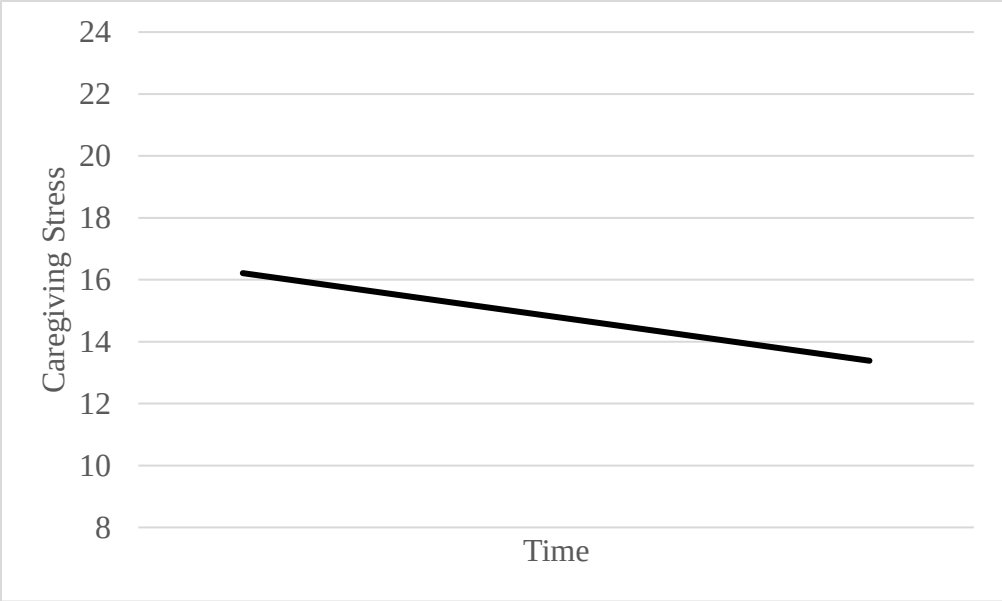

Figure 4.2: Change in Caregiving Stress Categories

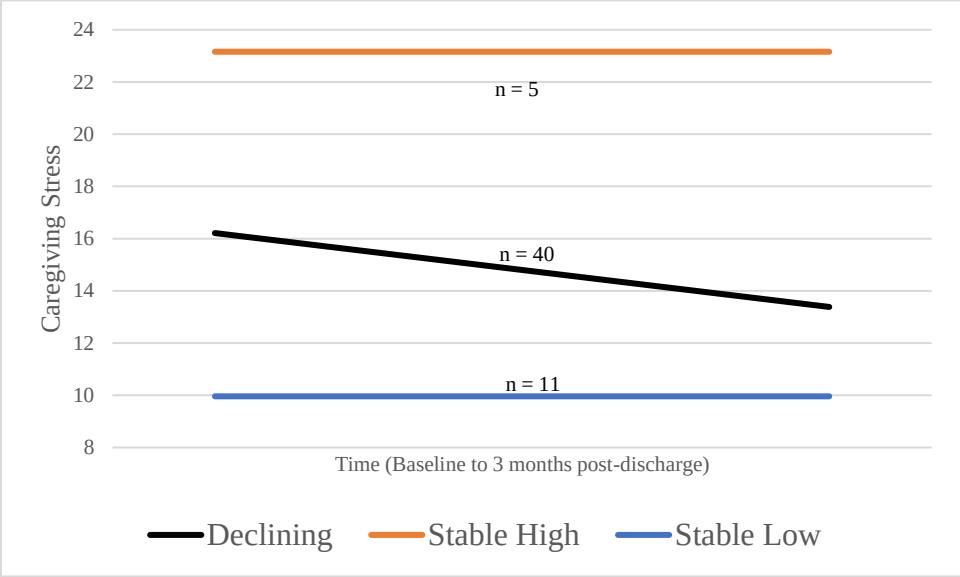

Table 4.3: The Impact of Time on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	16.214	0.768	79	21.1	<.0001
Time	-0.943	0.258	140	-3.66	0.0004

Aim 2: Factors that predict parental caregiving stress over time

Baseline Factors Predicting Caregiving Stress

We conducted four separate models that tested whether baseline total parental psychopathology, baseline parental self-efficacy, baseline parent-family support, and baseline adolescent severity of illness each predicted changes in caregiving stress. Baseline parental psychopathology did not significantly impact caregiving stress trajectory, ParentPHQ4*Time b = 0.02, t = 0.30, p = 0.76 (Table 4.4). We found Baseline parental self-efficacy significantly impacted trajectory, Self-efficacy*Time b = -0.04, t = -2.18, p = 0.03 (Table 4.5), such that greater self-efficacy at baseline was associated with greater declines in stress over time. See Figure 4.3. Baseline adolescent severity of illness also significantly impacted caregiving stress

trajectory, Severity*Time, b = 1.32, t = 3.11, p = 0.002 (Table 4.6), such that greater baseline severity causes a less steep decline of stress over time. See figure 4.4. Baseline parent-family support predicted caregiving stress trajectory at trend level b = -0.13, t = -1.66, p = 0.10 (Table 4.7), suggesting evidence that higher levels of baseline parent-family support were associated with more steep declines in caregiving stress over time.

Figure 4.3: Change in parental caregiving stress across time for parents with high and low self-efficacy at baseline

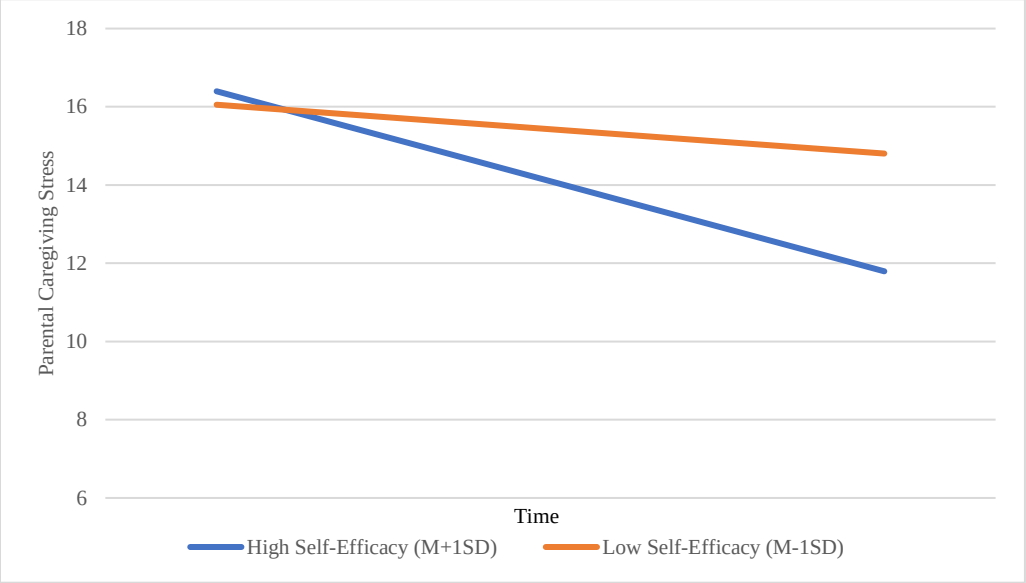

Figure 4.4: Change in parental caregiving stress across time for adolescents with high and low illness severity at baseline

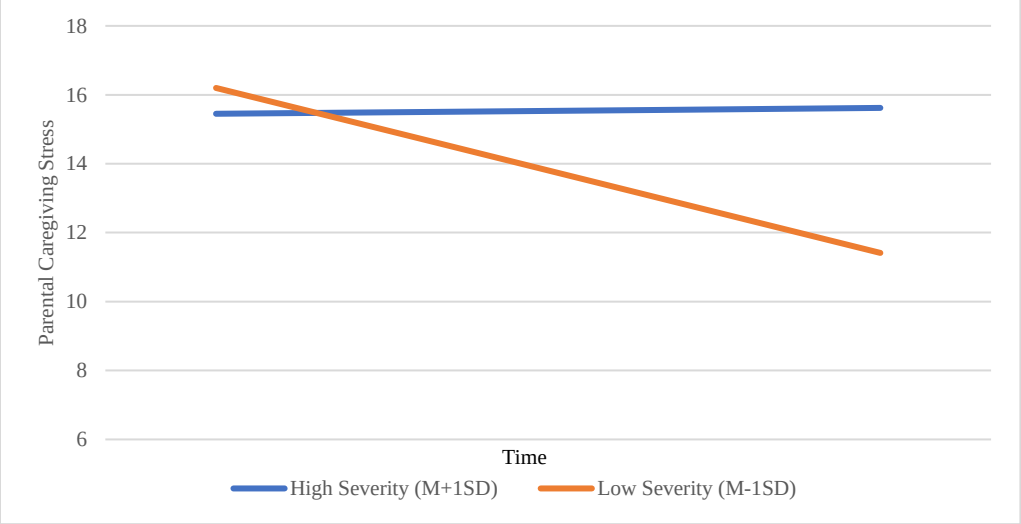

Table 4.4: Impact of Parental Psychopathology on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	12.054	1.116	78	10.8	<.0001
Parent PhQ4	0.872	0.186	78	4.7	<.0001
Time	-1.071	0.427	139	-2.51	0.013
ParentPHQ4*Time	0.021	0.068	139	0.3	0.761

Table 4.5: Impact of Parental Self-Efficacy on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	15.163	4.848	78	3.13	0.003
Parent Self-Efficacy	0.014	0.061	78	0.22	0.825
Time	2.476	1.588	139	1.56	0.121
ParentSelf-Efficacy*Time	-0.044	0.020	139	-2.18	0.031

Table 4.6: Impact of Teen Severity of Illness on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	15.811	0.791	73	19.99	<.0001
Teen Severity of Illness	-0.598	1.279	73	-0.47	0.641
Time	-0.741	0.260	128	-2.85	0.005
TeenSeverity*Time	1.320	0.424	128	3.11	0.002

Table 4.7: Impact of Parent-Family Support on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	16.644	2.983	78	5.58	<.0001
Parent-Family Support	-0.035	0.229	78	-0.15	0.881
Time	0.643	0.987	139	0.65	0.516
Parent-FamilySupport*Time	-0.125	0.075	139	-1.66	0.099

In a fully adjusted model that included all four of these baseline predictors, we found that baseline parental psychopathology remained a non-significant predictor of caregiving stress, $b = 0.04$, $t = 0.52$, $p = 0.61$ and family support was no longer a trend level predictor of caregiving

stress, b = -0.02, t = -0.31, p = 0.75. Self-efficacy became trend level significant in predicting greater decreases of parental caregiving stress, b = -0.04, t = -1.67, p = 0.10. In this model, baseline teen severity was the only significant predictor of caregiving stress trajectory, b = 1.13, t = 2.56, p = 0.01, when accounting for the conditional effects of parental psychopathology, self-efficacy, and parent-family support (Table 4.8).

Table 4.8: Fully Adjusted Model of Predictors on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	11.091	4.815	70	2.3	0.024
Parent PhQ4	0.892	0.197	70	4.53	<.0001
Parent Self-Efficacy	0.012	0.057	70	0.21	0.834
Teen Severity of Illness	0.463	1.183	70	0.39	0.697
Parent-Family Support	-0.026	0.215	70	-0.12	0.906
Time	2.227	1.732	125	1.29	0.201
ParentPHQ4*Time	0.037	0.071	125	0.52	0.606
ParentSelf-Efficacy*Time	-0.037	0.022	125	-1.67	0.098
TeenSeverity*Time	1.129	0.441	125	2.56	0.012
Parent-FamilySupport*Time	-0.025	0.079	125	-0.31	0.753

Time-varying factors predicting caregiving stress

We conducted four separate models to test whether change in these factors (individually derived slopes from baseline to three months for each participant) would impact changes in caregiving stress. We found that increasing parental psychopathology over time (positive slope) was associated with less of a decline in caregiving stress, b = 0.89, t = 4.48, p = <0.0001 (Table 4.9). See Figure 4.5. We found that changes in parental self-efficacy across time did not impact changes in caregiving stress, b = 0.01, t = 0.10, p = 0.92 (Table 4.10). Likewise, changes in adolescent severity of illness across time did not predict changes in caregiving stress, b = 0.79, t = 1.02, p = 0.31 (Table 4.11). Increases in parent-family support was associated with decreases in caregiving stress across time at trend level, b = -0.66, t = -1.76, p = 0.08 (Table 4.12). In a

fully adjusted model, which accounted for the conditional effect of each of these slopes, we found that the slope of parental psychopathology across time remained the only significant predictor of changes in caregiving stress, $b = 0.92$, $t = .56$, $p < 0.0001$ (Table 4.13).

Figure 4.5: Change in parental caregiving stress across time for parents with increasing, stable, and declining psychopathology across time

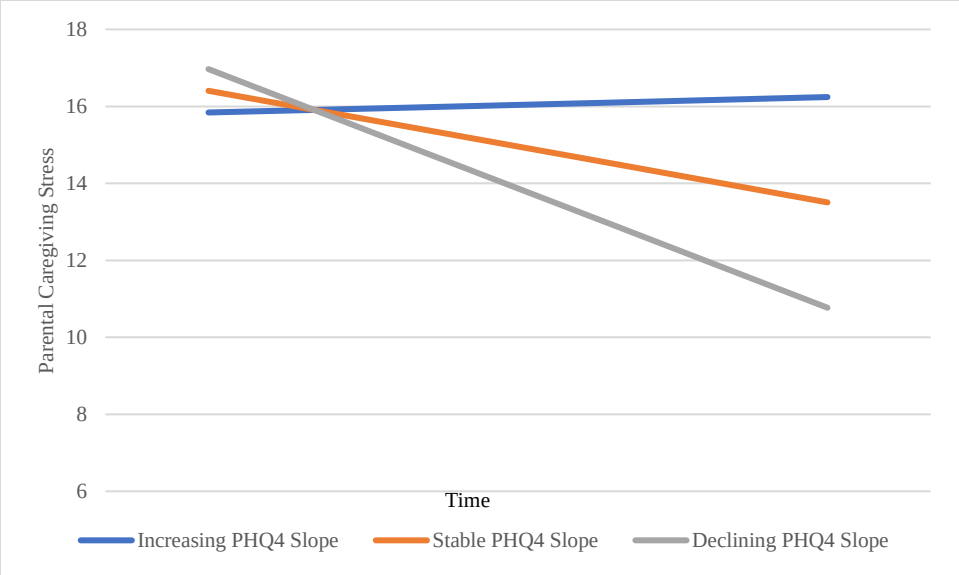

Table 4.9: Impact of Parental Psychopathology Slope on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	16.383	0.852	64	19.23	<.0001
Parent PHQ4 Slope	-0.458	0.697	64	-0.66	0.513
Time	-0.926	0.244	129	-3.8	0.0002
ParentPHQ4Slope*Time	0.894	0.199	129	4.48	<.0001

Table 4.10: Impact of Parental Self-Efficacy Slope on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	17.643	1.111	64	15.88	<.0001
Parent Self-Efficacy Slope	-0.398	0.230	64	-1.73	0.089
Time	-0.989	0.342	129	-2.89	0.005
ParentSelf-EfficacySlope*Time	0.007	0.071	129	0.1	0.920

Table 4.11: Impact of Teen Severity of Illness Slope on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	15.95	0.848	62	18.82	<.0001
Teen Severity of Illness Slope	6.437	3.092	62	2.08	0.042
Time	-0.878	0.273	120	-3.21	0.002
TeenSeveritySlope*Time	0.674	0.990	120	0.68	0.497

Table 4.12: Impact of Parent-Family Support Slope on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	16.116	0.765	70	21.05	<.0001
Parent-Family Support Slope	-2.170	1.147	70	-1.89	0.063
Time	-0.983	0.259	133	-3.79	0.0002
Parent-FamilySupportSlope*Time	-0.660	0.374	133	-1.76	0.080

Table 4.13: Fully Adjusted Model of Predictor Slopes on Caregiving Stress Trajectory

	b	*SE*	*df*	*t* value	*p* value
Intercept	17.034	1.101	56	15.47	<.0001
Time	-0.811	0.335	114	-2.42	0.017
Parent PHQ4 Slope	-0.999	0.750	56	-1.33	0.188
Parent Self-Efficacy Slope	-0.344	0.228	56	-1.51	0.138
Teen Severity of Illness	4.439	3.374	56	1.32	0.194
Parent-Family Support Slope	-2.123	1.442	56	-1.47	0.147
ParentPHQ4Slope*Time	1.031	0.228	114	4.52	<.0001
ParentSelf-EfficacySlope*Time	-0.010	0.071	114	-0.14	0.887
TeenSeveritySlope*Time	1.192	1.021	114	1.17	0.245
Parent-FamilySupportSlope*Time	0.300	0.437	114	0.69	0.494

Aim 3: Parental Caregiving Stress and Implementation of Safety Recommendations

Parents and adolescents reported on their perception of how much parents were implementing safety recommendations. These perceptions were not consistently strongly correlated (1-month $r(72) = 0.23$, $p = 0.055$; 3-months $r(72) = 0.04$, $p = 0.743$), so we conducted our Aim 3 analyses using parent report and adolescent report separately.

Parent Report of Safety Implementation – Impact of Caregiving Stress and Self-Efficacy

First, we explored how baseline levels of caregiving stress and parental self-efficacy impacted parental safety implementation (from the parent perspective) at one-month post-discharge and three months post-discharge using repeated measures general linear models. We found a significant positive effect of baseline caregiving stress on safety implementation, such that greater stress was associated with greater safety implementation from the parent perspective, $b = 0.19$, $t = 2.59$, $p = 0.01$. However, the interaction between stress and time was not significant, $b = -0.05$, $t = -0.73$, $p = 0.47$ (Table 4.14), suggesting that stress impacted safety implementation equally at both one month and three months post-discharge. Notably, this impact of stress on safety implementation was the opposite direction than we hypothesized. Greater baseline parental self-efficacy was also associated with higher parent-reported safety implementation $b = 0.07$, $t = 2.18$, $p = 0.03$, and the interaction with time was trend level, $b = 0.07$, $t = 1.98$, $p = 0.052$ (Table 4.15), suggesting that this association between self-efficacy and safety implementation may get stronger at three months post-discharge. We also explored whether baseline self-efficacy would moderate the association between baseline stress and safety implementation, and found that this was not the case, Stress*Self-efficacy $b = -0.007$, $t = -0.84$, $p = 0.40$ (Table 4.16). These findings suggest that stress and self-efficacy have a significant, albeit independent, impact on parental reports of safety implementation, both in the positive direction.

Table 4.14: Impact of Baseline Caregiving Stress on Parent-Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		13.278	1.714	71	7.75	<.0001
Baseline Parent PHQ4		0.020	0.118	71	0.17	0.865
Group – No Text		1.490	0.746	71	2	0.050
Group - Text		0	.	.	.	.
Gender - Female		-1.442	0.755	71	-1.91	0.060

Gender - Other		0	.	.	.	.
Time	0	1.828	1.601	63	1.14	0.258
	1	0	.	.	.	.
Baseline Stress		0.192	0.074	71	2.59	0.012
BaselineStress*Time	0	-0.047	0.065	63	-0.73	0.468
	1	0	.	.	.	.

Table 4.15: Impact of Baseline Parent Self-Efficacy on Parent-Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		11.3161	2.7224	71	4.16	<.0001
Baseline Parent PHQ4		0.1571	0.09573	71	1.64	0.105
Group – No Text		1.2931	0.7091	71	1.82	0.072
Group - Text		0	.	.	.	.
Gender - Female		-1.1214	0.7192	71	-1.56	0.123
Gender - Other		0	.	.	.	.
Time	0	-4.4717	2.6214	63	-1.71	0.093
	1	0	.	.	.	.
Baseline Parent Self-Efficacy		0.07303	0.03349	71	2.18	0.033
BaselineParentSelf-Efficacy*Time	0	0.06595	0.03322	63	1.98	0.052
	1	0	.	.	.	.

Table 4.16: Moderation of Baseline Parental Self-Efficacy on Relationship between Baseline Caregiving Stress and Parent-Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		-6.459	15.714	138	-0.41	0.682
Baseline Parent PHQ4		-0.216	0.145	138	-1.49	0.139
Group – No Text		-0.837	0.911	138	-0.92	0.360
Group - Text		0	.	.	.	.
Gender - Female		2.385	0.931	138	2.56	0.012
Gender - Other		0	.	.	.	.
Time	0	10.490	21.889	138	0.48	0.633
	1	0	.	.	.	.
Baseline Parent Self-Efficacy		0.242	0.194	138	1.25	0.215
Baseline Stress		0.648	0.663	138	0.98	0.330
BaselineParentSelf-Efficacy*Time	0	-0.131	0.272	138	-0.48	0.630
	1	0	.	.	.	.
BaselineStress*Time	0	-0.478	0.929	138	-0.51	0.607

	1	0	.	.	.	.
Stress*Self-Efficacy		-0.007	0.008	138	-0.84	0.402
Stress*Self-Efficacy*Time	0	0.007	0.011	138	0.59	0.560
	1	0	.	.	.	.

Adolescent Report of Safety Implementation – Impact of Caregiving Stress and Self-Efficacy

Next, we explored how baseline levels of parental caregiving stress and parental self-efficacy impacted adolescents' reports of their parents' safety implementation using repeated measures general linear models. We found that baseline caregiving stress was not associated with greater adolescent report of safety implementation, $b = 0.06$, $t = 0.65$, $p = 0.52$, and that the interaction with time was also non-significant, $b = 0.05$, $t = 0.55$, $p = 0.59$ (Table 4.17). Greater baseline parental self-efficacy was associated with greater adolescent reported safety implementation $b = 0.10$, $t = 2.13$, $p = 0.04$, and the association was the same across time, Self-efficacy*Time, $b = 0.02$, $t = 0.37$, $p = 0.71$ (Table 4.18). This effect became trend level (though in the same direction) in models controlling individually for sex and father education. In these models, the covariates themselves had a significant impact on the model, suggesting that the effect of self-efficacy on teen reported safety implementation may be different for different sex or paternal education groups. Similar to parent-reported safety recommendation, baseline self-efficacy did not moderate the association between baseline stress and adolescent report of parental safety implementation, Stress*Self-efficacy, $b = -0.008$, $t = -1.06$, $p = 0.29$ (Table 4.19).

Table 4.17: Impact of Baseline Caregiving Stress on Teen Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		10.251	2.597	73	3.95	0.0002
Sex - Female		2.496	1.157	73	2.16	0.034
Sex - Male		0	.	.	.	.
Father Education – College or higher		2.698	1.105	73	2.44	0.017
Father Education - Other		0	.	.	.	.

Time	0	0.175	2.275	70	0.08	0.939
	1	0	.	.	.	.
Baseline Stress		0.062	0.096	73	0.65	0.519
BaselineStress*Time	0	0.051	0.093	70	0.55	0.586
	1	0	.	.	.	.

Table 4.18: Impact of Baseline Parent Self-Efficacy on Teen Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		3.152	4.151	73	0.76	0.450
Sex - Female		2.760	1.120	73	2.47	0.016
Sex – Male		0	.	.	.	.
Father Education – College or higher		3.027	1.073	73	2.82	0.006
Father Education - Other		0	.	.	.	.
Time	0	-0.018	3.811	70	0	0.9962
	1	0	.	.	.	.
Baseline Parent Self-Efficacy		0.104	0.049	73	2.13	0.037
BaselineParentSelf-Efficacy*Time	0	0.018	0.048	70	0.37	0.714
	1	0	.	.	.	.

Table 4.19: Moderation of Baseline Parental Self-Efficacy on Relationship between Baseline Caregiving Stress and Teen Reported Safety Implementation Across Time (Controlling for Covariates)

	Time	*b*	*SE*	*df*	*t* value	*p* value
Intercept		-13.415	15.238	71	-0.88	0.382
Sex - Female		2.940	1.134	71	2.59	0.012
Sex - Male		0	.	.	.	.
Father Education – College or higher		3.096	1.077	71	2.87	0.005
Father Education - Other		0	.	.	.	.
Time	0	11.630	14.828	68	0.78	0.436
	1	0	.	.	.	.
Baseline Parent Self-Efficacy		0.293	0.187	71	1.57	0.121
Baseline Stress		0.720	0.636	71	1.13	0.262
BaselineParentSelf-Efficacy*Time	0	-0.142	0.184	68	-0.77	0.442
	1	0	.	.	.	.
BaselineStress*Time	0	-0.507	0.629	68	-0.81	0.423
	1	0	.	.	.	.
Stress*Self-Efficacy		-0.008	0.008	71	-1.06	0.295

Stress*Self-Efficacy*Time	0 1	0.007 0	0.008 .	68 .	0.89 .	0.376 .

Discussion

In this study, we characterized caregiver stress over three months post-discharge in a sample of parents whose teens had been psychiatrically hospitalized for suicidal ideation or attempt. We explored how parent and teen factors covary with stress over time and whether caregiver stress impacts the parental implementation of safety recommendations. We also examined whether parental efficacy might buffer the effects of caregiver stress on safety implementation. Overall, we found that caregiving stress declines linearly over the three-month post-discharge period. We found evidence that some factors led to greater declines (self-efficacy, parent-family support), and other factors were associated with less steep declines (parental psychopathology and adolescent severity of illness) in caregiving stress over time. While we found evidence that baseline stress and self-efficacy predicted parental implementation of safety recommendations, the impact of stress was in the opposite direction than we hypothesized. Further, we found no evidence that parental self-efficacy buffered the impact of caregiving stress on the ability to implement safety recommendations, suggesting that these factors are independently related to safety implementation. Because parents are crucial in following through with discharge safety recommendations, a better understanding of parental experiences during the post-discharge time may help build more effective interventions for this high-risk population of adolescents and their parents.

Notably, in Aim 1, we found that overall caregiver stress decreases from baseline to three months post-discharge in a linear fashion. In addition, further examination of individual trajectories suggests that most parents (52%) experienced a declining trajectory, confirming that

the average trajectory reflects the experience of most parents. To our knowledge, no other research has examined caregiving stress in parents of psychiatrically hospitalized youth longitudinally after discharge. This decline is likely explained by very high levels of stress experienced by parents at the time of the hospitalization, which represents the likely peak of a mental health crisis for the adolescent. This is consistent with recent work that found that parents of adolescents who visited the emergency department for suicidal crises also experienced high levels of parental stressors at baseline (Ewell Foster et al., 2021). Additionally, our sample displayed higher levels of caregiving stress at baseline than the population of mental health treatment-seeking adolescents in the study which developed the stress scale (Brannan et al., 2012). This gives quantitative evidence to what is known anecdotally and qualitatively (Ngwane & van der Wath, 2019) - that having a teen who attempts suicide is a very stressful experience for caregivers but this stress declines in the months post-discharge.

We also found that baseline parent and teen level factors were associated with caregiving stress trajectories. Specifically, we found that baseline levels of parental self-efficacy served as a protective factor for stress across time, such that greater levels of baseline self-efficacy were associated with greater decreases in caregiving stress. We also found that baseline levels of teen severity of illness served as a risk factor for greater stress. Further, the impact of teen severity remained the only significant predictor above and beyond the impact of the other factors, suggesting that of the baseline factors, the severity of illness has the greatest influence on caregiving stress trajectories. Those in our sample who reported higher baseline severity include teens who had more severe suicide attempts, including those that result in more medical complications. It is possible that over a longer follow-up period, efficacy may be more strongly

related to stress, but in the short period post-hospitalization, it is not surprising that teen severity at the time of hospitalization has the greatest impact on caregivers' stress post-discharge.

When looking at time-varying levels of these parent and teen factors, the only significant finding was that increases in parental psychopathology over time were associated with less decline in stress over time. While this is the first study of its kind to look at how parental caregiving stress changes over the post-discharge period for this population, it is not surprising that the severity of illness was related to greater stress over time. Parental psychopathology has been associated with greater suicidality in their teens (Lee et al., 2019) which in turn may be related to increased stress for parents. Additionally, psychopathology itself is associated with increases in stress and sensitivity to stress (Liu & Alloy, 2010). Given that *changes* in parental self-efficacy were not associated with steeper caregiving stress declines (though baseline levels were), but increasing parental psychopathology was associated with less steep declines in stress, this impact of psychopathology likely cancels out the protective impact of baseline self-efficacy in this sample. Increasing parental self-efficacy while also decreasing parental psychopathology (e.g. through connecting parents to their own mental health support), maybe a beneficial intervention to decrease stress for caregivers of psychiatrically hospitalized youth.

In examining whether caregiving stress and parental self-efficacy impacted parental ability to implement safety recommendations, we found that *both* caregiving stress and parental self-efficacy were associated with *greater* safety implementation from parent reports. The impact of stress was the opposite of what we hypothesized, where greater stress was associated with greater implementation of safety recommendations. It is possible that in the relatively short follow-up period of this study, greater acute stress mobilized parents to be more sensitive to asking their teens about coping strategies. In fact, parents of chronically ill teens report a greater

perception of their teen as vulnerable and greater uncertainty related to illness (Mullins et al., 2007). It is possible that in the short term, viewing the teen as vulnerable and increased uncertainty could both increase stress *and* cause parents to be more likely to implement safety recommendations in our sample. If examined over a longer follow-up period, we speculate that more chronic stress might produce negative consequences on the parental ability to support their teen.

From the adolescent report of how their parents were implementing safety recommendations, only baseline parental self-efficacy was associated with increases in safety implementation. Given that this was the same finding for parents' reports of safety implementation as well, these findings are in line with research that suggests the protective factor of self-efficacy in increasing the implementation of health behaviors (Sheeran et al., 2016). Self-efficacy may be a mechanism by which parents can help support their teens after a crisis. These findings strengthen the assertion that parental self-efficacy is a target for intervention in this population (Czyz et al., 2018).

Our findings should be interpreted in light of several limitations. First, given that this was a pilot study, our sample was relatively small and relatively heterogeneous. It will be important to replicate these findings in a larger, representative sample. Given that the majority of our participant parents were mothers, a larger sample may also allow for additional examination of differences in these associations that may arise when parents and adolescents are participating with the same gender parent versus participating with their opposite gender parent. Additionally, for models where we did not find a significant result, it is possible that these relationships have smaller effect sizes and therefore we may not have been powered to detect such a small effect. In these cases, a larger sample may provide power to detect relationships with a smaller effect.

Second, each of these measures was based on participants' *perceptions* of how often they implemented safety recommendations. Future studies should look into whether this can be examined objectively. Additionally, our study did not include measurements of teen level factors that may influence these bidirectional relationships. For instance, teen impulsivity or their propensity to accept support from parents, may have impacted their parents' perceptions of stress in this study. Finally, while a strength of our study was the longitudinal nature of our data across the three months post-discharge, there are likely dynamic shifts in perception of stress that can change more frequently. Approaches such as ecological momentary assessments of caregiving stress, as well as some of these other parents and teen level factors, might elucidate more complicated associations between these factors that are shifting more rapidly over time. Additionally, a longer follow-up period might allow the examination of acute versus chronic caregiving stress.

In conclusion, our study was the first of its kind to examine parental caregiving stress across the three-month post-discharge period following psychiatric hospitalization of their adolescent. Our findings suggest that this time is very stressful for parents and that teen severity of illness and parents' own psychopathology have the biggest impact on these changes in stress. This study highlights the importance of increasing parental support for parents who have a teen who is hospitalized, which may also improve teen functioning and safety during this stressful period.

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Chapter 5 Dissertation Integration and Discussion

This dissertation examined the role of parents as regulators of teen distress in two unique high-risk samples. Study 1 and Study 2 examined parent and family supportive factors in a sample of adolescents at high risk for depression both after a controlled acute stressor (regulating after a laboratory stress task) and less acute, naturalistic stressors (stressful life events over the past year). Study 3 examined how an acute high-stress situation (psychiatrically hospitalization of adolescents) affects parental stress and their ability to help their teen regulate post-discharge. Overall, we found that greater perceived parent and family support was associated with a greater ability to regulate following an acute stressor (Study 1), but that these factors did not buffer against the impact of naturalistic stressors on internalizing symptoms (Study 2). We also found that caregiving stress and parenting self-efficacy were associated with increases in parental support of their teen post-discharge, both in the positive direction. These three studies provide a unique exploration of parental factors that help adolescents regulate in stressful contexts and highlight the role that stress contexts may play in the effectiveness of parents to help teens cope with stressful situations.

Overall, we found significant inconsistencies in the extent to which parents can help teens cope, which are likely affected by specific characteristics of the stressors. For instance, in study 1 we found that more positive parental support and behaviors were associated with an increased ability of teens to regulate their neuroendocrine stress response after an acute laboratory stressor. In contrast, in study 2, which used the same sample, we looked at how these same perceived and observed parent and family level supportive factors might protect against the harmful effects of

recent life stressors on adolescent internalizing symptoms. We did not find any evidence that any of the parent-level factors examined protected against the harmful effects of stress on mental health, despite some of the factors being associated with internalizing symptoms more broadly. Study 3 was similar to study 1 in that it was examining whether parent level protective or risk factors might be the mechanism of parental ease or disease with implementing safety recommendations to help their teen regulate post-hospitalization. In this study, stress was associated with *more* positive outcomes (e.g. higher rates of parental safety implementation). Taken together, while it is clear that parenting support provides some protection, this may be context limited.

These inconsistencies may exist as a function of the timing of when parent support matters most across development. For example, we found differences in HPA axis reactivity based on levels of parenting support, but what we might be capturing is a longer developmental sequence for trait-like supportive behaviors on the development of HPA regulation (Gunnar & Donzella, 2002). In this conceptualization, trait-like parenting support may have a beneficial impact on the development of the HPA axis earlier on in development and is thus associated with trait-like HPA regulation. Evidence for this conceptualization comes from our findings related to parental presence. Specifically, the experimental manipulation of parental presence did not impact cortisol trajectories even after accounting for parental support. Therefore, the links between parent-level factors and HPA axis regulation may reflect the impact of long-term parental practices on HPA axis regulation. However, in study 2, we are capturing stress on a different time scale (e.g. report of stressors over the past year) along with more proximal internalizing symptoms (e.g. past two weeks). Positive parenting perception and in-the-moment behaviors may offer limited protection against the effect of life events in this age group, which

may be due to the acuteness of the life events or the highly stressful environments that teens are experiencing in contemporary society (Collins & Steinberg, 2007).

Another explanation for these inconsistencies could be that these parenting supportive factors help in the moment when it comes to HPA regulation but not necessarily when it comes to longer-term or more complex outcomes such as mental health. Although decades of research links dysregulation in the HPA axis to mental health (Lopez-Duran, Kovacs, & George, 2009), the associations are weak (Zorn et al., 2017) and there is still a lot of variability in these outcomes, which means that most of the variance in mental health outcomes is *not* related to HPA axis dysregulation. In this conceptualization, whatever support parents are providing for the HPA regulation in study 1 might not be enough to provide the protection that is needed in the context of real-life stressful events that teens experience. In this sense, parenting might be a critical component for the development of neurobiological stress regulatory systems because the factors that influence HPA axis development are very narrow (e.g. parenting, trauma exposure, and sex hormones) while parenting might only be a minor factor in protection against harmful mental health outcomes as those are more multidetermined.

Study 3 adds nuance to the exploration of the role of parental support by examining what factors might play a role in the parental ability to help their adolescent cope. While we assumed that stress would negatively impact these parents' ability to implement safety recommendations, we found the opposite – greater levels of stress in this population seemed to have mobilized parents to ask their teens more about coping strategies and suicidal thoughts post-discharge. This finding may also reflect a timing issue. Longer follow-up may show that acute stress is activating while chronic stress has deleterious outcomes. For example, acute stress may promote healthy adaptation in the short term, but when it becomes chronic, it adds to the allostatic load and has

harmful effects on mental and physical health (McEwen, 2004). Despite this, parental self-efficacy played a role in safety implementation from both the parent and teen reports of post-discharge safety implementation, suggesting the important role of self-efficacy in enhancing parental behaviors. This also strengthens the assertion that self-efficacy is a good target for intervention amongst these parents.

Overall, this dissertation found that parents played a role in helping their teens regulate in some contexts, but not in others. This highlights the importance of future research examining additional contexts under which parents may protect high-risk adolescents. This dissertation was unique in that it explored these parent and family supportive factors in two high-risk populations and used multi-informant and multi-method measurements of parent/family support. Additional research should continue to explore the contexts in which parent support is helpful for these populations, which might help to inform more supportive parenting interventions.

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Appendix A. Emotion Socialization Strategies Coding Manual

Emotion Socialization Strategies: Observational Coding Manual
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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