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SOCIAL STRESS REACTIVITY: LINKS WITH EARLY ENVIRONMENTAL INSTABILITY AND ADOLESCENT ANXIETY

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ABSTRACT

Although developmental increases in social sensitivity across adolescence are likely adaptive for aiding in the successful navigation of their changing social landscape, some teens might be placed at greater risk for internalizing symptom development (Pfeifer & Allen, 2021). Two studies within the present dissertation seek to better understand for whom, and in what social and developmental contexts, HPA responsivity to social stress might be associated with elevated anxiety in adolescence. Both studies drew from an epidemiological sample of 1292 diverse, lower-income families from rural communities, followed repeatedly from infancy through adolescence.

Study 1 focused on developmental unpredictability within the home environment, measuring cumulative household chaos across early life, as well as wave-to-wave fluctuations in caregiver support from infancy through adolescence. Adolescent HPA functioning was additionally examined as, 1) a potential mechanism by which the early environment influenced later anxiety symptoms, and 2) a child-level marker of biological sensitivity that modulated the effects of the environment on anxiety. Results indicated that lability in caregiver support from infancy through adolescence was associated with lesser cortisol reactivity to social stress, though cortisol did not significantly mediate the effects of caregiver support or household chaos on anxiety symptoms. However, for those who displayed greater cortisol reactivity to social stress or lower cortisol at baseline, greater early household disorganization was associated with elevated anxiety. Effects were buffered for those who displayed lesser cortisol reactivity or higher cortisol levels at rest. These results are partly consistent with the biological sensitivity to context framework, highlighting certain patterns of HPA functioning that might increase

children's susceptibility to ongoing or future stressors – or that might impact regulatory abilities – within their surrounding environment.

Study 2 focused on salient contexts within adolescence, examining the association between concurrent cortisol functioning and anxiety, and the moderating role of peer problems and perceived pubertal stage. Results indicated no direct association between cortisol and anxiety, emphasizing the importance of incorporating developmental and contextual factors when considering the effects of physiological functioning on behavior. Lesser cortisol reactivity was associated with higher trait anxiety for adolescents who reported an earlier perceived pubertal stage. Conversely, greater reactivity was associated with higher risk for a social anxiety diagnosis for those who reported a mid-perceived pubertal stage. Trait anxiety symptoms might be more enduring, such that cortisol hyporeactivity could reflect physiological burnout within the context of developmentally less mature coping skills. However, for those who view themselves as undergoing physical pubertal changes, high physiological sensitivity to social stress might be a more potent risk factor for elevated social anxiety. Overall, these findings point to the complex role of pubertal maturation in anxiety symptom development.

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Chapter 1

General Introduction

Humans are posited to have a fundamental need to belong. From an evolutionary perspective, the desire to create and maintain positive social bonds offered essential reproductive and survival benefits to our ancestors (Baumeister & Leary, 1995). Social groups allowed for the sharing of food and offspring care, the provision of mates, and provided enhanced protection against threat. This evolutionary selection likely resulted in an innate motivation toward social belonging in humans. Basic reinforcement principles suggest that the positive affect and pleasure derived from social affiliation, alongside the distress and pain resulting from social bond rupture, maintains this underlying desire for long-lasting bonds. Thus, alongside an innate motive to protect one's physical self from harm, humans are also motivated to preserve a positive social self (Dickerson et al., 2004; Kemeny et al., 2004).

Given the value of social group affiliation, a threat to one's social status – including potential or explicit rejection, isolation, or interpersonal conflict – elicits a set of evolved psychobiological processes (Dickerson et al., 2004; Gruenewald et al., 2004). This integrated psychobiological response is characterized by a decrease in feelings of social worth and self-esteem, as well as an increase in cortisol production. Beginning with the emotional component, threats to one's social self are often accompanied by self-conscious emotions such shame, guilt, and submissiveness, which in turn are associated with behavioral disengagement and withdrawal. The display of these emotions following a violation of social norms might help to regain favor and acceptance within the group,

while de-escalating the situation to maintain cohesion (Dickerson, Gruenewald, et al., 2009; Kemeny et al., 2004). Empirical work supports that individuals with low perceived social status self-reported higher levels of shame (Kemeny et al., 2004), while observers who viewed nonverbal displays of shame in others reported higher levels of sympathy relative to other emotion displays (e.g., amusement; Keltner et al., 1997). Thus, the emotional aspect of social threat responding likely enhances survival from an evolutionary standpoint by eliciting affiliative behaviors in others.

Moving to the physiological component, social stressors are consistent and potent activators of the hypothalamic-pituitary-adrenal (HPA) axis (Dickerson & Kemeny, 2004). The HPA axis elicits a cascade of physiological processes that culminate in elevated cortisol production, while inhibiting the acute stress response to help restore the body to homeostasis (Bauer et al., 2002). Gruenewald et al. (2004) found that participants who were asked to complete speech and mental arithmetic tasks in front of an evaluative panel of strangers displayed a larger increase in cortisol relative to those who completed the tasks alone. Elevated cortisol in the socio-evaluative group was also associated with an increase in self-reported feelings of shame and a decrease in self-esteem, suggesting integration between psychological and physiological components of the stress response.

Although increased cortisol production is often framed from the perspective of risk for physical and mental health problems, acute increases are adaptive for coping with social stress. At the cognitive level, cortisol helps to consolidate emotional learning and memories for salient events (Buchanan & Lovallo, 2001; Erickson et al., 2003). This process helps individuals to learn from and later avoid actions that contributed to negative social evaluation. The HPA axis also plays an important role in flexibly up- and

down-regulating other physiological processes, to include metabolic, immune, and sympathetic systems (Bauer et al., 2002). Regulation of metabolic processes helps to mobilize energy resources for the body to use. Within the context of social threat, energy is necessary to initiate coping responses that help to re-establish social bonds, or to respond to more antagonistic social threats (Dickerson, Gruenewald, et al., 2009). Further, cortisol release is thought to have acute mood-protective effects. Cortisol release has been associated with lower negative affect following experimental stress induction (Het et al., 2012), while momentary increases in cortisol have been linked with a subsequent increase in activeness and alertness in daily life (Hoyt et al., 2016). Within the context of social stress, these mood-buffering effects might facilitate the downregulation of negative emotional arousal while giving a "boost" needed for mental coping and problem-solving. Finally, as part of a negative feedback loop, the HPA axis suppresses sympathetic fight-or-flight and related immune activation, as well as its own activation, to return the body to homeostasis (Adam et al., 2007). Following the offset of an acute social threat, this down-regulation protects the body from excess "wear-and-tear" due to highly-taxing sympathetic-adrenal activity, while further supporting calm social engagement and repair. Overall, each of these physiological processes are widely adaptive for coping with and learning from acute social stress while returning the body to homeostasis, ultimately aiding in social group re-acceptance.

Developmental Perspective of Social Stress

Social relationships remain a core component of emotional well-being throughout the lifespan. However, the structure and significance of these relationships changes substantially across development, characterized by a progressive shift in engagement and

attention from one social target to another (Nelson et al., 2016). Briefly, infancy through early childhood is characterized by the formation and reliance on caregiver attachment to meet physical and social needs. Social motivation then shifts towards peer play in middle childhood. School entry at this stage brings increased independence and more time spent outside of the home, though the caregiver relationship is retained as a safe home base (McHale et al., 2003). This increase in peer experience prepares children for their next shift into the adolescent phase, when social goals are re-oriented more heavily towards peer group integration and move further away from familial attachment. Social re-orientation across adolescence is driven, in part, by a range of integrated biological, socio-emotional, and cognitive changes – these include development of brain regions involved in social cognition and affective processing, the onset of puberty, as well as contextual changes characterized by school transitions and the formation of new peer groups (Blakemore & Mills, 2014).

Given this substantial shift in social motivation toward peer group integration, adolescents display an enhanced attunement to both positive and negative social evaluation, as evidenced by functioning of the evolved psychobiological processes described above (Dickerson et al., 2004; Gruenewald et al., 2004; Somerville, 2013). Beginning with self-conscious emotion – adolescence is characterized by an improvement in mentalizing, defined as the ability to think about the mental state of others (e.g., their thoughts, feelings, beliefs; Andrews et al., 2021). This ability comes alongside increased self-consciousness and the tendency to view oneself from the perspective of others (Pfeifer et al., 2009; Guyer et al., 2016), as well as enhanced mentalizing during the processing of social emotion (e.g., embarrassment, guilt; Klapwijk

et al., 2013). At the physiological level, adolescents display heightened cortisol reactivity to social evaluation and stress relative to children, suggesting that negative social evaluation and threat become particularly salient at this age (Tottenham & Galván, 2016). Altogether, these developmental changes support the social sensitivity and skills necessary for successful peer group affiliation, emphasizing the adaptive nature of enhanced psychobiological responding to instances of social threat at this age.

Despite the widely normative and adaptive nature of increased responsivity to social stress in adolescence, this sensitization might also increase risk for psychopathology in some teens. When individuals feel that they are consistently or frequently negatively evaluated by others, chronic activation of the integrated psychobiological response system could contribute to adverse physical and mental health outcomes (Dickerson, Gruenewald, et al., 2009). Similarly, some teens might be more sensitive to social threat, leading to more frequent or exaggerated activation. The overarching goal of the present dissertation is to consider for whom, and in what contexts, might specific patterns of cortisol responsivity to social stress become maladaptive. Specifically, I will situate functioning of the HPA axis within intra- and inter-personal contexts spanning infancy through adolescence, ultimately considering links between environmental instability, social stress reactivity, and anxiety symptoms in teens.

Study 1 will leverage a more holistic, developmental perspective, examining how unpredictability within both the home and the caregiver-child relationship predict HPA functioning – both resting cortisol levels and reactivity to social stress – and anxiety into adolescence. Additionally, I will consider whether cortisol in adolescence acts as a

mediator or moderator of the association between environmental instability across early development and anxiety symptoms in adolescence. Household and caregiver factors will be examined repeatedly from infancy through middle childhood and adolescence, allowing us to capture chronicity and instability over time. Study 2 will focus specifically on the adolescent period, examining the association between anxiety outcomes and cortisol at baseline and in response to social stress. Homing in on salient aspects of this developmental stage, I will test the moderating effect of peer functioning and pubertal status. Altogether, this dissertation should point to several key aspects of an individual's internal state and external environment that might be leveraged for prevention and intervention during two potentially impactful windows of opportunity – early life and the teenage years.

Chapter 2

Environmental Instability Across Development: Interplay with the HPA Axis and Anxiety Development

Introduction

Building upon foundational evolutionary and developmental theory, the Adaptive Calibration Model points to two roles that the stress-response system might play in psychopathology development (Del Giudice et al., 2011). First, neural and physiological systems involved in the stress response are thought to remain plastic across early life, such that the proximal environment tunes children's physiological systems to aid in optimal responsivity (Del Giudice et al., 2011). In line with this perspective, stressful early life environments might lead to later mental health problems by adjusting responsivity of the stress response system (Smith & Pollak, 2020). Indeed, stressful family and household environments have been associated with more dysregulated patterns of stress reactivity in youth (Koss & Gunnar, 2018; Marsh et al., 2020). Second, building upon the biological sensitivity to context framework, the stress response system is also thought to play a role in modulating openness and sensitivity to the surrounding environment (Belsky & Pluess, 2009; Belsky et al., 2007; Boyce & Ellis, 2005). All children exposed to early environmental risk do not go on to display dysregulated physiological and behavioral phenotypes, such that certain children are more or less resilient in the face of early life stress (Belsky & Pluess, 2009; Belsky et al., 2007; Boyce & Ellis, 2005). Greater physiological reactivity is thought to confer heightened sensitivity to ongoing or future environmental exposures, thereby increasing risk for psychopathology within more stressful contexts. Overall, it is important to consider both

how the environment shapes physiological systems, as well as the active interplay between person and context across developmental time.

The present study will incorporate these two theoretical perspectives by examining the stress-response system as a mediator and a moderator of the association between environmental instability and later anxiety in adolescence. Specifically, this paper will begin with a discussion of cortisol within the context of anxiety, considering evidence for the HPA axis as a mechanism by which unpredictability in the household and parent-child contexts influence later anxiety symptoms. I will then expand into a consideration of biological sensitivity to context, examining if cortisol confers openness to the larger environment, thereby moderating the association between instability and anxiety. Overall, the present study leveraged measures of household chaos and caregiver behavior spanning from infancy through adolescence to address how these environmental experiences vary and accumulate across developmental time (see Figure 2-1 for our longitudinal data collection overview).

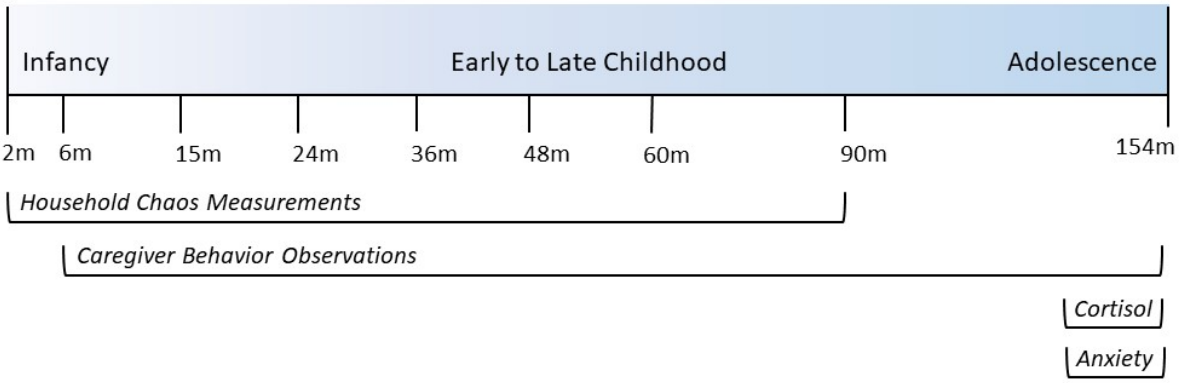

Figure **2-1**: Longitudinal Data Collection Timeline

Note. FLP data were collected from when the target child was approximately 2 months to 154 months of age. Each tick in the above timeline denotes a wave of data collection, resulting in a total of 8 waves. The lower brackets represent how long each primary measure in the present study was collected – specifically, measures of household chaos were collected across 8 assessments (at 2, 6, 15, 24, 36, 48, 60, and 90 months), observations of caregiver behavior were collected across 7 assessments (at 6, 15, 24, 36, 60, 90, and 154 months), and cortisol and anxiety were measured once at the adolescent assessment.

HPA Axis and Social Stress Regulation: Implications for Anxiety

Stress regulation involves multiple physiological systems, including the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenal (HPA) axis (Bauer et al., 2002). Activation of these systems follows a coordinated, temporal sequence. The sympathetic system is activated most quickly, stimulating the "fight-or-flight" response to mobilize bodily resources for engagement with environmental demand. This response includes an increase in heart rate, blood flow, and respiratory rate. Sympathetic nerve fibers innervate visceral organs and the adrenal medulla, which is responsible for releasing the hormones epinephrine and norepinephrine for distribution throughout the body. This direct innervation by the sympathetic system allows for rapid, though shorter-lived, physiological changes (Bauer et al., 2002).

Following acute sympathetic activation, the HPA axis mounts a slower and longer-acting response (Adam et al., 2007). This system is activated most strongly in response to events that are perceived as uncontrollable and unpredictable – particularly

those that threaten one's social self (Dickerson & Kemeney, 2004). Experimental work supports that social-evaluative stressors (e.g., giving a speech in front of an evaluative panel) evoke larger increases in HPA activity relative to other tasks, particularly if the stressor is coupled with uncontrollability (e.g., false negative feedback about performance; Dickerson & Kemeney, 2004). Increased activation of the HPA axis sets off a cascade of events that culminate in elevated cortisol levels, the release of which forms an inverted U-shaped curve across time (Adam et al., 2007). This curve comprises a cortisol peak approximately 20-30 minutes post-stressor onset, reflecting increased mobilization of metabolic resources. These resources have a range of functional implications, including enhanced attention, learning and memory consolidation, and the facilitation of necessary behaviors and coping mechanisms (e.g., social avoidance behaviors; Erickson et al., 2003; Kaldewaij et al., 2016). Peak levels are followed by a rapid decline back to baseline within 40-60 minutes, reflecting function of the negative feedback loop (Bauer et al., 2002). The HPA axis terminates its own activation, as well as acute activity of the sympathetic system, helping to restore the body to homeostasis.

Patterns of moderate cortisol reactivity and subsequent recovery are considered necessary for effective self-regulation and coping. For example, Blair et al. (2005) found that a moderate increase in cortisol across a child's assessment session (e.g., meeting the investigator, completing cognitive tasks) was associated with better executive function skills, higher teacher-reported self-regulation, and lower aggressive behavior relative to children who did not demonstrate this increase. Additional work supports the adaptive role of cortisol within the context of stress, such that greater cortisol output during an experimental social stressor – measured from the start of the stressor to 40-minutes post-

task – has been associated with less socially anxious behaviors (e.g., shaky voice, fidgeting, wincing) observed during the task (Perry et al., 2022). Engagement of the HPA axis likely enables resources needed for cognitive effort and emotional and behavioral regulation necessary for coping with environmental demand in-the-moment, while also inhibiting acute sympathetic activation to facilitate physiological recovery over time.

Variation from the normative, U-shaped curve of moderate cortisol reactivity is thought to reflect a dysregulated stress response, implicated in physical and mental health problems across the lifespan (Adam et al., 2007). Two main profiles of HPA dysregulation have been identified in both youth and adult samples. First, *hyperactivity* refers to excessive elevations in cortisol at rest or in response to stress. Elevated baseline levels of cortisol have been linked with concurrent internalizing problems (Lopez-Duran et al., 2009), while greater increases in resting levels across one year have been found to predict later anxiety symptoms (Schiefelbein & Susman, 2006). Cortisol hyper-reactivity in response to social stress and conflict has also been associated with greater anxiety in childhood and adolescence (Hartman et al., 2013). Overall, elevations in cortisol have been associated with greater behavioral inhibition, withdrawal, and increased sensitivity to punishment, which together might serve as the underlying psychobiological mechanism by which social stress influences and reinforces anxiety symptoms (Buss & Qu, 2018).

The second profile of dysregulation – *hypoactivity* – reflects lower cortisol levels at rest and/or a smaller change or decrease in cortisol from baseline in response to external challenge. Blunted profiles of activity characterized by both lower resting levels

of cortisol and reduced cortisol output during a laboratory stressor – as well as profiles characterized by moderate resting levels and lowered reactivity – have been associated with deficits in regulating emotion and behavior (Ayer et al., 2013; Joos et al., 2019). For example, children and adolescents with higher anxiety symptoms have been shown to display lower cortisol output across a social stress task (Stadelmann et al., 2018). These patterns of cortisol hypo-reactivity might reflect an inability to effectively mobilize resources in response to stress, suggesting a lack of flexibility and disengagement in response to social challenge that reduces one's ability to cope effectively (Seiss et al., 2014). Given that anxiety has been empirically linked to both hyper- and hypo-cortisol activity, it is important to consider the early environmental factors that might contribute to individual differences in stress response system functioning.

Beyond cross-sectional associations with psychopathology, downregulation of cortisol responsivity across developmental time has been predominantly linked with more chronic or extreme stressors (Miller et al., 2007; Trickett et al., 2010). For example, children raised in harsh deprived conditions were found to display blunted cortisol reactivity to psychosocial stress, while more typical response patterns were observed in those adopted into a high-quality foster care (McLaughlin et al., 2015). Similarly, greater cumulative psychosocial (e.g., family chaos) and physical (e.g., housing quality) risk exposure has been associated with lowered baseline arousal from middle childhood through adolescence within the context of low maternal responsiveness (Evans et al., 2007). These findings highlight the need to model environmental stressors across longer periods of time to account for stress accumulation and chronicity in a more accurate manner. Considering sensitivity of the HPA axis to indicators of unpredictability within

the environment, the present study first examined whether cortisol mediated the effects of cumulative household chaos and longitudinal instability in caregiver behavior across early youth development on anxiety symptoms in adolescence.

Household Chaos

Household chaos denotes a lack of structure and predictability in the home environment, encapsulating dimensions of both instability and disorganization (Bronfenbrenner & Evans, 2000; Evans & Wachs, 2010; Vernon-Feagans et al., 2021). Briefly, household instability captures changes in residence and relationships in the home, reflecting a range of periodic, destabilizing events. Household disorganization indexes crowding, clutter, noise, and a lack of structure. This dimension denotes the degree of routine within the home, where high levels of background stimulation and activity likely contribute to greater unpredictability in proximal family processes that might otherwise be sustained and consistent in a more organized environment – for example, consistency in mealtimes, as well as homework and bedtime routines (Evans, 2006; Zvara et al., 2014).

Both dimensions of household chaos might increase risk for anxiety in childhood and adolescence. Specifically, chaos contributes to a chronically unpredictable environment, producing frequent, mild stressors that necessitate a constant state of vigilance and preparation for those in the home (Doom et al., 2018). High unpredictability might also instill a sense of helplessness in children as they are unable to consistently manage and regulate their surrounding context. These early experiences with diminished control – defined as "the ability to personally influence events and outcomes in one's environment" – are thought to foster an increased tendency towards processing

future events as outside of one's control, which is a hallmark vulnerability for anxiety (Chorpita & Barlow, 1998, pg. 5). In support of these negative implications, there is considerable evidence that greater levels of household chaos are linked to adverse socio-emotional and behavioral outcomes in both younger children and adolescents (Marsh et al., 2020; Raver et al., 2015; Tucker et al., 2018), though there is a need for more systematic examinations of anxiety.

Given the high degree of unpredictability and uncontrollability observed in chaotic households, this context is also a salient contributor to the development and function of the HPA axis (Marsh et al., 2020). According to the adaptive calibration model, early environments that signal high levels of unpredictability and harshness calibrate biological response systems towards heightened responsivity (Boyce & Ellis, 2005; Del Giudice et al., 2011). This developmental fine-tuning serves to facilitate vigilance, enhanced attention, and rapid engagement toward threat-related cues. Indeed, cross-sectional research supports that greater household chaos and more frequent disruptions to attachment relationships (e.g., instability in the child's primary or secondary caregiver) are associated with higher daily cortisol output in infancy and early childhood, reflecting HPA sensitization (Brown et al., 2021; Tarullo et al., 2020).

The longer-term effects of household chaos are also highlighted in longitudinal work. For example, previous work in the present sample found that markers of household instability in infancy – including changes in both residence and adults within the home – predicted stable, elevated baseline cortisol levels from infancy through toddlerhood (Blair, Raver, et al., 2011). Similarly, in a sample of children and adolescents aged 9-18, greater household chaos was found to mediate the association between higher poverty

and longitudinal increases in daily cortisol output across 2 years (Chen et al., 2010). Though relatively less research has focused on measures of cortisol *reactivity*, greater household chaos in preschool has been shown to predict heightened cortisol reactivity during social stress into middle childhood (Doom et al., 2018). These patterns of increased vigilance might reflect preparation for engagement with future chaotic social environments across development, such that the early home context creates an internal working model of environmental unpredictability. Aligning with the perspective that vigilance for threat or stress is a core element of anxiety (Eysenck, 1997), previous work further supports elevated emotional reactivity as a mechanism linking early childhood stress with anxiety disorders later in life, though more work is needed on physiological mechanisms (McLaughlin et al., 2010).

Despite cross-sectional and longitudinal evidence for sensitization of the HPA axis within chaotic home environments, the literature is not entirely consistent. For example, Suor et al. (2015) found that greater family instability (e.g., caregiver and residential changes, caregiver relationship changes) in early life predicted stable membership in either elevated-basal-cortisol or low-basal-cortisol groups across toddlerhood. This additional pattern of lowered cortisol might serve as a protective factor against repeated HPA activation in response to unpredictable and recurrent stressors within the household. However, as evidenced by the work discussed thus far, chronicity of contextual stressors is important to capture when considering effects on stress responding and self-regulatory processes (Evans & Kim, 2013; Miller et al., 2007). Although there is a budding literature that examines the influence of early chaos on developmental changes in cortisol and related internalizing symptoms, there is a need to

expand beyond single-time-point measures of chaos itself to understand how the influence of disorganization and instability accumulates across development. Thus, the present study considered cumulative household chaos measured repeatedly across infancy and childhood.

As a final note, although less research has simultaneously investigated the two dimensions of household disorganization and instability, previous research within the current sample has found more robust effects of disorganization (relative to instability) on cognitive, emotional, and behavioral outcomes in early childhood (Garrett-Peters et al., 2019; Mills-Koonce et al., 2016; Raver et al., 2015; Vernon-Feagans et al., 2012). However, the present focus on cumulative chaos across a longer developmental time window might be especially important when considering the instability dimension. Fluctuations in residence and household-based relationships likely become more salient as children age. Progression into middle childhood comes with increased integration into social networks outside of the home (e.g., peer and student-teacher relationships at school entry), as well as increased active engagement and mutuality in relationships within the home, making changes to these networks particularly disruptive (McHale et al., 2003). The present study builds upon previous research by extending markers of chaos longitudinally across the middle childhood years, and further testing whether these earlier measures of household chaos continue to influence physiological and behavioral outcomes into adolescence.

Parent-Child Context

While chaos captures stability and organization within the larger household context, Bronfenbrenner's bioecological systems theory posits that development

primarily occurs through more “proximal processes” (Bronfenbrenner & Evans, 2000; Bronfenbrenner & Morris, 2006). Proximal processes are complex, reciprocal interactions between the child and their immediate environment, and are thought to be most effective when they occur regularly over an extended period of time. Examples of these processes include frequent parent-child or peer-child interactions, reading books, or playing with toys, all of which drive cognitive, socio-emotional, and underlying biological development. The current study tied in bioecological theory to examine the stability of proximal caregiver processes (e.g., supportive interactions with their child) across development on adolescent outcomes.

Though the negative effects of more extreme caregiver behaviors and contexts (e.g., maltreatment, institutionalization) have long been a focus of child development research, normative variation in caregiver behavior from infancy through the adolescent years has also been shown to have a strong influence on emotional and behavioral outcomes (Hackman et al., 2010). Research has expanded over the years to encapsulate a range of parenting practices, to include dimensions ranging from harsh to sensitive caregiver behaviors (e.g., Lovejoy et al., 1999). Harsh and intrusive parenting includes diminished responsivity that undermines children’s developing autonomy and self-regulation. More supportive forms of parenting capture developmentally sensitive responsivity and warmth directed from caregiver to child, allowing for reciprocal exchanges that provide stimulation and reward (Blair, Granger, et al., 2011; Morris et al., 2007). Developmental theory suggests that, beginning in early life, young children learn how to regulate their emotions through warm, supportive, and responsive caregiver engagement and communication (Eisenberg et al., 1998), such that higher caregiver

sensitivity and lower control has been associated with lowered child anxiety and internalizing symptoms (Kok et al., 2013; McLeod et al., 2007). Despite a shift towards peer influence into adolescence, these positive caregiver behaviors continue to be impactful throughout development. Evidence suggests that higher caregiver warmth in adolescence predicts lower anxiety symptoms by influencing how teens process stressful social stimuli (e.g., criticism; Butterfield et al., 2021). Thus, caregiver behavior is important for scaffolding and promoting adaptive emotion processing and regulation.

In addition to effects on anxiety, caregiver-child relationships have a strong regulatory effect on the HPA axis, with experimental studies in rodent and primate models supporting the role of maternal behavior in the development of HPA stress responding (Gunnar & Donzella, 2002; Hostinar & Gunnar, 2013; Hostinar et al., 2014). For example, rat pups raised by dams higher in licking and grooming behaviors displayed lower corticosterone release to acute stress in adulthood, while pups with limited maternal licking and grooming behaviors displayed elevated corticosterone and more anxious, fearful behaviors (Caldji et al., 1998; Meaney, 2010; Weaver et al., 2004). Thus, normative variation in sensitive maternal behavior directly programs HPA function in experimental animal models. In humans, caregivers serve as children's primary social environment for a more protracted period, meaning that caregiver engagement lays the foundation for adaptive neurobiological development.

There is robust evidence for the effects of different forms of caregiver behavior on physiological responsivity. For example, Suor et al. (2015) found that greater maternal unresponsiveness – operationalized as parental insensitivity and disengagement during a parent-child play task – was associated with stable-elevated cortisol across early

childhood. Expanding across longer periods of development, cumulative measures of parental harshness and hostility from infancy through adolescence have been associated with higher chronic cortisol output in adolescence (Doom et al., 2022; Ouellet-Morin et al., 2021). A harsh and more threatening parent-child context might lead to elevated stress perception and exposure over time. However, multiple studies in the present sample suggest that positive forms of caregiver behavior are more robust indicators of HPA function in childhood, such that associations observed for negative parenting might simply capture a lack of sensitive caregiving within a harsher family context. For example, positive (but not negative) aspects of parenting were found to be inversely related to child baseline cortisol across the first two years of life (Blair, Granger, et al., 2011). Maternal sensitivity has similarly been associated with more normative reactivity patterns to emotional challenge in infancy and toddlerhood (Blair et al., 2006; Hibel et al., 2011). These positive forms of parent-child engagement likely foster the development of adaptive stress responsivity by providing a supportive and secure space for children to learn how to cope with and regulate their emotions, which might in turn influence internalizing outcomes (Calkins et al., 2013). Overall, there is a gap in the literature on the longitudinal effects of caregiver support on acute *adolescent* HPA stress reactivity and anxiety, with a particular need to explicitly model instability and unpredictability within the parent-child context across time.

Caregiver Lability

Children's needs, autonomy, and social relationships undergo drastic changes from infancy through adolescence. For example, we see shifts from caregiver-directed emotion regulation in infancy to more self-directed regulation into childhood, as well as

shifts in attachment focus from caregivers toward peer and romantic relationships across adolescence (Nelson et al., 2016). As children enter school, join after-school extracurriculars, and spend increasing amounts of time outside of the home, the nature of the parent-child relationship must adjust to meet these changes (McHale et al., 2003). Indeed, family systems theory suggests that developmental transitions can induce instability within the family system, initiating changes in interaction patterns within and between subsystems (e.g., caregiver-child, partner-partner, etc.; Cox & Paley, 1997, 2003). A key concept within this theoretical framework is adaptive self-organization, meaning that family systems have the capacity to re-organize and re-stabilize following perturbation. Eventually, new patterns unfold as roles and boundaries are re-negotiated within the context of the new family structure and situation.

Caregiver behaviors and patterns of engagement within the caregiver-child subsystem fluctuate across development. For example, beginning in early life, positive emotional expressiveness directed from parent to child was found to peak in infancy and decline across the "terrible twos" (Barry & Kochanska, 2010), with maternal-child conflict subsequently declining from 2 to 4 years of age (Weaver et al., 2015). Longitudinal work suggests that parent-child warmth declines from childhood though the transition into adolescence, while conflict frequency peaks around early adolescence and declines into late adolescence (Shanahan et al., 2007a, 2007b; Smetana et al., 2006). Variation in these developmental trajectories has also been linked to child outcomes. Trentacosta et al. (2011) found that a trajectory of high-stable conflict across childhood was associated with greater behavioral problems in adolescence. Similarly, steeper

decreases in parental warmth and increases in hostility have been associated with increases in internalizing problems across early adolescence (Lippold et al., 2021).

Though the work cited above has advanced our understanding of both normative and maladaptive changes in caregiver-child relationship quality, there are two key gaps in the literature that need to be addressed. Firstly, as discussed in previous sections, limited work encompasses measurements of caregiver-child engagement from infancy through adolescence. This more extended longitudinal design is important for capturing the wide range of developmental transitions that require repeated adjustments to the family system. Secondly, there has been an overwhelming emphasis on sample-level trajectories of change in caregiver behavior – however, change can be characterized both by averaged developmental trends *and* by variability around these trends (Lippold et al., 2018; Marceau et al., 2015). Variability captures more dynamic fluctuations in behavior within persons and families. This distinction is visualized in Figure 2-2, where family A displays much higher wave-to-wave lability in caregiver behavior relative to family B, despite similar developmental trends. Multiple studies suggest that the majority of variance in parenting from year-to-year is captured by within-person lability, over and above that attributed to between-person, systematic change (e.g., Lippold et al., 2018; Marceau et al., 2020; Zheng & McMahon, 2022). Thus, addressing caregiver lability in intervention and prevention programs might carry more weight in changing parenting phenotypes beyond a more global focus on averaged change across development, opening a new avenue of future research.

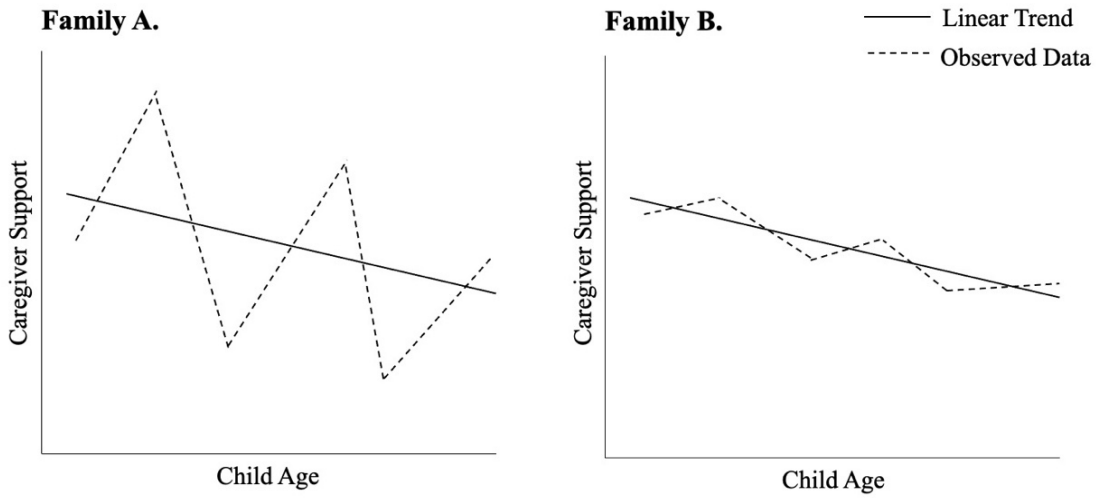

Figure **2-2**: Distinguishing Between Developmental Trends and Lability in Caregiver Behavior

Note. Mock longitudinal data from two families. Solid lines represent averaged linear trends, and dashed lines represent fluctuations of each data point around the linear trend. Figure adapted from Lippold et al. (2018).

Fluctuations in the quality of caregiver-child engagement across development has important implications for children's anxiety outcomes. For example, Zheng and McMahon (2022) found that greater year-to-year lability in caregiver warmth from kindergarten through grade 5 was associated with higher internalizing problems and lower social competence at the transition into middle school. Extending across early adolescence, greater lability in paternal hostility and warmth from grade 6 to grade 8 was associated with higher adolescent internalizing problems, and greater lability in maternal warmth was associated with higher internalizing problems in girls (Lippold et al., 2021). While some degree of lability in caregiver behavior likely reflects responsive and

adaptive adjustment of familial roles and boundaries, high levels of variability can indicate difficulty re-organizing and stabilizing the family system around children's rapidly changing developmental needs. Lower levels of lability foster a stable, safe environment where children can expect clear and consistent interactions within the parent-child subsystem, likely lowering vulnerability for later anxiety symptoms.

Although no research to our knowledge has directly examined the effects of caregiver lability on youth biological stress responsivity, the high degree of unpredictability and uncontrollability that comes alongside inconsistency in caregiver behavior likely has a strong influence on the developing HPA axis. Physiological dysregulation might serve as the underlying mechanism by which lability in supportive parenting influences the internalizing outcomes discussed above.

Biological Sensitivity to Context

Despite established effects of instability in the household and parent-child contexts on psychopathology, not all children are equally susceptible to environmental risk. Traditional models of the "diathesis-stress" hypothesis suggest that children with high levels of biological or behavioral reactivity are more sensitive to stress within their environment, showing higher levels of emotional and behavioral problems relative to their low-reactive counterparts (Belsky et al., 2007). In line with this view, high stress reactivity is thought to be uniformly maladaptive, serving only as a vulnerability factor for psychopathology and environmental stress. However, more recent theoretical perspectives contend that heightened biological sensitivity to context makes children more susceptible to both negative *and* positive environmental influences (Belsky & Pluess, 2009; Boyce & Ellis, 2005). Thus, heightened stress reactivity might be

maladaptive in the context of a harsh and risky environment, but adaptive in the context of a nurturing and supportive environment. This perspective highlights the need to better understand how biological processes interact with the quality and stability of the surrounding environment to predict psychological well-being.

Empirical work supports the role of stress reactivity in moderating sensitivity to the environment. Beginning with the larger familial and household contexts, Obradovic et al. (2010) found that biological reactivity to stress moderated the association between family adversity and socioemotional behavior in childhood. Relative to their low-reactive peers, more highly reactive children displayed higher levels of problem behaviors and lower prosocial competence within the context of high adversity, but the lowest levels of problem behaviors and the highest prosociality within the context of low adversity. Similarly, Steeger et al. (2016) found that cortisol reactivity moderated the association between stressful family life events and psychopathology in adolescence. Family stress was associated with more internalizing symptoms for those who displayed greater cortisol reactivity. These effects were buffered by patterns of lower cortisol reactivity. Finally, household chaos has been associated with greater adolescent problem behavior for those with higher maternal-reported emotional reactivity, though more work is needed on the intersections between chaos and biological sensitivity (Shapero & Steinberg, 2013). Overall, these findings support the role of heightened physiological reactivity in increasing sensitivity to the surrounding environment – for better or for worse. The present study built upon these findings by focusing on specific dimensions of household chaos across important sensitive periods from infancy through middle childhood.

Moving to the parent-child context, cortisol reactivity has been found to moderate the link between observed parental behaviors and child psychopathology. Relative to their low-reactive peers, greater caregiver hostility has been associated with an increase in internalizing symptoms from early to middle childhood for children who displayed high cortisol reactivity (Barrios et al., 2017). Considering both positive and negative contexts, for toddlers who displayed greater cortisol reactivity to social stress, higher maternal overprotection was found to predict higher child anxiety, while higher maternal support predicted lower anxiety (Kalomiris et al., 2019). These effects were buffered for children who displayed patterns of lower reactivity. The present study built upon these findings by delving into the stability of caregiver support over time, above and beyond a strict focus on quality of parent-child engagement.

Sensitive Periods

As a final component, the present study considered sensitive periods for the effects of household chaos across both mediation and moderation models. Sensitive periods refer to the developmental windows during which children are particularly susceptible to influence from their surrounding environment (Woodard & Pollak, 2020). Work on post-institutionalized youth offers key evidence for sensitive periods of HPA and psychopathology development, allowing for a better understanding of how changes in the home environment might differentially impact biobehavioral systems depending upon the developmental period in which those changes occur (Hostinar et al., 2015a). Focusing in early life, initial environmental inputs that denote instability and adversity might become embedded into children's developing neurobiological systems. For example, children adopted later in childhood were found to display more severe problem

behaviors relative to post-institutionalized children adopted earlier in life, to include elevations in internalizing, externalizing, and attentional problems (Hawk & McCall, 2010). McLaughlin et al. (2015) found that institutionalized children placed into high-quality family care before 24 months of age displayed more typical patterns of HPA functioning (comparable to never-institutionalized children) relative to those placed in later childhood. Thus, stable and supportive home environments in early life actively shape functioning of the stress response system and related behavioral outcomes. Building upon this work, the present study compared the effects of household chaos across the first two years of life (2-24 months) relative to more chronic forms of chaos across infancy and middle childhood (2-90 months). Given the present modeling approach, caregiver lability was not split into separate developmental periods.

Current Study

The present study moved beyond the cross-sectional space to consider whether instability within the home environment across development influenced anxiety in adolescence. Additionally, I examined whether exposure to instability influenced anxiety risk by altering HPA function for optimal responsivity within an unpredictable environment (mediation model), or if cortisol denoted sensitivity to the environment by modulating the effects of instability on later anxiety development (moderation model). Measures of the home environment included both proximal family processes (e.g., caregiver-child engagement) and larger contextual factors (e.g., household chaos). Given that the current study incorporated repeated measures spanning key developmental periods (e.g., infancy, childhood, adolescence; see Figure 2-1), I also leveraged the unique opportunity to delve into *lability* around general developmental trends in observed

caregiver-child interactions. Longitudinal data were further leveraged to test potential developmental sensitive periods for the effects of household chaos on adolescent outcomes.

Overall, the present study tested two primary sets of research questions: 1) does environmental instability directly predict later cortisol functioning and anxiety symptoms, such that cortisol acts as a *mediator* between instability and anxiety, and 2) does cortisol act as a *moderator* of the association between instability and anxiety? Theoretically, treating cortisol as a developmental mediator assumes that the functioning of the HPA axis is actively shaped by the stability of the early home environment, such that cortisol is the output of environment experiences. Conversely, because cortisol was measured in adolescence within the present study, the biological sensitivity to context approach (moderation model) assumes some rank-order stability in cortisol functioning across developmental time, such that the association between early environmental instability and later anxiety differs depending upon certain patterns of more time-invariant HPA functioning.

Beginning with the first research question, the present study tested both direct and indirect effects of instability on anxiety. First, for direct effects, I hypothesized that greater lability in supportive caregiver behavior and greater cumulative household chaos across development would each be associated with higher adolescent anxiety and elevated cortisol at baseline and in response to a social stressor. These predictions align with the adaptive calibration model, which proposes a link between unpredictable environments and vigilant physiological profiles that facilitate high attention and engagement (Del Giudice et al., 2011). Secondly, for indirect effects, I hypothesized that

the association between greater lability in caregiver support and higher anxiety would be explained by elevated cortisol measures, with similar effects for household chaos.

Moving to the second research question, the present study tested the moderating effects of cortisol on the association between instability and anxiety. Aligning with the sensitivity to context framework (Boyce & Ellis, 2005), I hypothesized that the association between greater lability in caregiver behavior and higher anxiety would only be significant for those with elevated cortisol at baseline and in response to social stress, with similar effects for household chaos. Given limited work that distinguishes between household instability and disorganization, particularly beyond early childhood, the current study explored the effects of both dimensions across all models.

Methods

Participants

The Family Life Project (FLP) is an epidemiological study designed to study the effects of rural poverty on child development. A representative sample of 1292 families were recruited in three counties in Central Pennsylvania (n = 519) and three counties in Eastern North Carolina (n = 773) selected to represent Appalachia and the Black South, respectively. Recruitment was conducted in local hospitals from September 2003-2004, covering over 90% of all births in the target counties. Low-income families in both states and Black families in North Carolina were oversampled to ensure adequate power for dynamic and longitudinal modeling of psychosocial risk. Home visits were conducted by trained research assistants when the child was approximately 2, 6, 15, 24, 36, 48, 60, 90 months, and 13 years of age. Visits included interviews and questionnaires with the primary caregiver, child assessments, and videotaped interactions between the child and

their caregiver(s). The present study leveraged the full expanse of data, incorporating cumulative household measures averaged from age 2 months to 90 months, and parent-child observation measures collected from age 6 months to 13 years. Cortisol and anxiety data were collected at the adolescent visit (see Figure 2-1).

Patterns of missing data were assessed across all waves of data collection, to include the number of families who provided complete and various levels of partial data. Out of the original 1292 families enrolled in the Family Life project, n = 711 children provided cortisol and questionnaire data at the adolescent visit, reflecting attrition of n = 581 families over time. This subsample comprised 49.8% females and the racial/ethnic breakdown was as follows: 54.3% White, 44.4% Black or African American, and < 1% each of Pacific Islander, American Indian or Alaska Native, and Asian participants. This subsample did not differ from the full sample in sex ($\chi^2(1) = .04$, $p = .83$) or racial/ethnic group (tested as White vs. Black/African American combined with the three smaller groups; $\chi^2(1) = .80$, $p = .37$). Incorporating in the longitudinal data, caregiver-related analyses were limited to families who provided at least 3 waves of data to ensure precision of our lability measurement (as suggested in Lippold et al., 2016). Out of the 711 families with adolescent data, n = 707 had at least 3 waves of caregiver-child interaction data, and n = 392 had complete interaction data across the full 7 waves. Additionally, out of those with complete adolescent data, n = 679 families had cumulative household chaos data collected across 2 to 90 months of age.

Procedure

Each home visit lasted for approximately 2 to 3 hours in total. Research assistants collected information from the primary caregiver on household and family demographics.

An array of survey measures and interviews were collected from caregivers, children completed several assessments, and the child and their primary caregiver completed an observed interaction task. At the adolescent assessment, teens were asked to complete the Trier Social Stress Test (TSST) while physiological data were collected. All questionnaires and tasks are described in detail below.

Measures

Cumulative Household Chaos

Household chaos was operationalized using two dimensions – instability and disorganization (Vernon-Feagans et al., 2012). *Household instability* was measured via five indicators: the total number of people residing in the household (defined as anyone who sleeps in the household for 3 or more nights per week) at the time of assessment, the total number of household members who have moved into or out of the household since the previous assessment, the number of physical residential moves the child experienced since the previous assessment, and the number of changes in primary and secondary caregivers since the previous assessment. These measures were collected from primary caregiver report at each visit (see Figure 2-1) and items were averaged across all 8 assessments.

Household disorganization was measured using five indicators collected at each home visit: average number of hours the TV is on in the home, average household density, preparation for home visits, cleanliness, and neighborhood noise level. TV time was reported by the primary caregiver. Household density was calculated for each visit by dividing the number of rooms in the home by the total number of people residing in the home. The final three indicators represent consensus ratings by the two research

assistants who conducted each home visit, and were collected at 7 of 8 assessments (missing at age 15 months). These measures were drawn from the post-visit Inventory in the Fast Track intervention study (Dodge et al., 1994) – preparation was rated from 1 (*surprise/difficulty*) to 4 (*good hosts*), cleanliness from 1 (*very dirty*) to 4 (*clean*), and neighborhood noise from 1 (*very quiet*) to 4 (*very noisy*). Household preparation and cleanliness were reverse scored, such that higher values across measures represent more disorganization.

Altogether, cumulative measures of instability and disorganization were created for the present study, reflecting an average of all non-missing items across the 8 repeated data collection waves from when the target child was 2 months of age through adolescence. All individual indicators were standardized ($M = 0$, $SD = 1$) prior to averaging. See Table 2-1 for raw cross-sectional and standardized cumulative descriptives, as well as correlations across assessments. Available cross-sectional data suggested moderate rank-order stability in household disorganization ($rs = .47$ to $.61$) from 24 to 90 months, and weaker rank-order stability in household instability over time ($rs = .20$ to $.41$). Early disorganization measured from 2-24 months ($M = .02$, $SD = .62$) was significantly correlated with more chronic disorganization measured from 2-90 months ($M = .01$, $SD = .67$; $r = .87$). Similar effects were observed for instability measured across the 2-24-month ($M = -.01$, $SD = .69$) and 2-90-month ($M = -.04$, $SD = .70$; $r = .75$) time windows.

Table **2-1:** Household Chaos Correlations and Descriptives Across Waves

	1	2	3	4	5	6	7
Disorganization							
1. 24 months	–						
2. 36 months	.57	–					
3. 48 months	.53	.59	–				
4. 60 months	.51	.56	.61	–			
5. 90 months	.47	.51	.57	.56	–		
6. 2-24 cumulative	.85	.62	.60	.59	.53	–	
7. 2-90 cumulative	.76	.78	.82	.79	.76	.87	–
Mean	2.14	2.06	2.66	2.56	2.73	.02	.01
SD	.58	.57	1.30	1.27	1.28	.62	.67
Range	.98-3.75	.73-4.08	.92-8.92	.89-12.57	1.02-9.46	-1.48-1.99	-1.44-2.10
Instability							
1. 24 months	–						
2. 36 months	.32	–					
3. 48 months	.26	.38	–				
4. 60 months	.20	.28	.41	–			
5. 90 months	.23	.23	.36	.34	–		
6. 2-24 cumulative	.72	.33	.24	.18	.26	–	
7. 2-90 cumulative	.56	.58	.58	.52	.59	.75	–
Mean	1.12	1.17	1.16	1.16	1.28	-.01	-.04
SD	.52	.52	.53	.52	.59	.69	.70
Range	.40-4.80	.40-4.20	.40-3.60	.40-3.80	.40-4.60	-1.07-3.27	-1.15-3.11

Note. N = 640. Cross-sectional data were available beginning at 24 months of age. Mean, standard deviation, and range for cross-sectional data represent raw disorganization and instability item averages. Individual items were standardized prior to averaging for cumulative measures and correlation analyses. Significance of all correlation coefficients $p < .001$.

Caregiver-Child Interactions

Children and their primary caregiver completed a videotaped interaction task during home visits from 6 months to adolescence – note that interaction tasks were not conducted at 2 or 48 months of age. See Figure 2-3 for a visual depiction of tasks given at each assessment. At the 6- and 15-month assessment, dyads completed a 10-minute, semi-structured free-play task. Caregivers were provided with a standardized set of toys and asked to play with their child as they normally would. At the 24- and 35- month assessments, the child and caregiver completed a 10-minute puzzle task. They were given three jig-saw puzzles of increasing difficulty. Caregivers were instructed that the child was to complete the task, though they could provide help. At the 60-month assessment, dyads completed two 15-minute tasks. The first task involved building a tower together with instructions to replicate a tower built by the research assistant. In the second task, they played a competitive "slap-jack" card game where the caregiver and child competed to collect the most cards. At the 90-month assessment, three 5-minute tasks were completed. First, the child was instructed to re-create 2-D and 3-D shapes presented in a flipbook using a set of magnets. The caregiver was instructed to assist in any way they chose. Second, the caregiver and child were instructed to replicate a line drawing of a house and tree using an Etch-A-Sketch, with each member of the dyad controlling one of the knobs. For the third task, they played a "slap-jack" card game. Finally, at the adolescent assessment, the caregiver and child completed two tasks. The first involved a 10-minute "hot topic" discussion, where dyads chose and discussed their top three topics of conflict or disagreement. Participants were provided with a list of 23 topics to choose from (e.g., bedtime, privacy, taking responsibility, friends), with the additional option to

come up with their own topic(s) beyond those provided. If the three topics had been thoroughly covered before time was up, they were instructed to pick an additional topic(s) to discuss. The second was a 5-minute interactive teamwork task, where the caregiver was provided instructions for folding an origami fox, and the teen was given lined, colored paper to create the fox. The caregiver was not allowed to look at what the teen was doing, and the teen could not see the instructions while folding (but could ask their caregiver for direction).

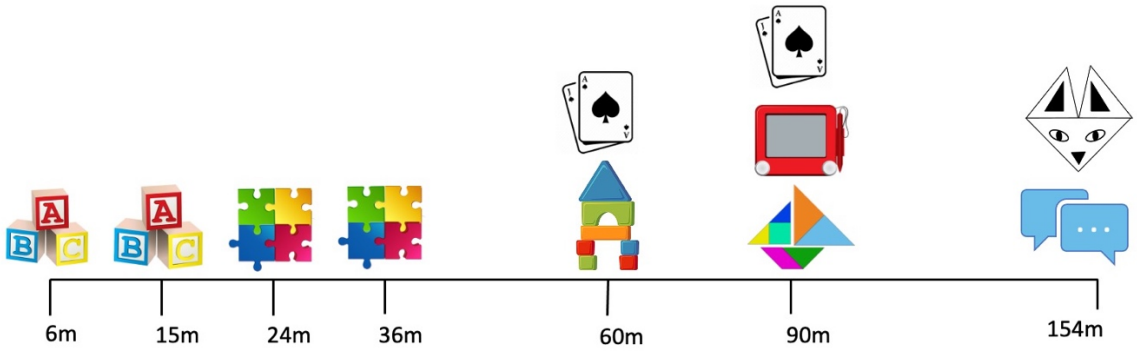

Figure **2-3**: Visualization of Caregiver-Child Interaction Tasks at Each Home Visit

Note. The horizontal axis denotes child age at each assessment, and each image represents the type of interaction task completed. Multiple stacked images indicate multiple tasks completed during one visit.

All videos were later coded for several aspects of parenting quality (adapted from NICHD Early Child Care Research Network, 1999). The present study used observations of caregiver support, defined broadly as expressions of positive regard and emotional support directed from caregiver to child. Caregivers rated high in support consistently

provided high quality verbal and non-verbal support, and positively interacted with their child by offering validation, agreement, and engaged interaction. Conversely, caregivers low in support were more aloof and unavailable or hostile when the child displayed a need for assistance. Support was rated on a 5-point scale from 1 (*not at all characteristic*) to 5 (*highly characteristic*) based on the quality and quantity of observed behaviors. Caregiver interactions at the adolescent assessment were originally rated on a 7-point scale but were subsequently re-coded to match earlier assessments. For assessments during which multiple tasks were completed by the caregiver and child, the tasks were coded globally to create a single score. All videos were coded by teams of 4-5 coders, which included 1-2 "masters" with whom coders were trained to be reliable. Each coder completed approximately 30% of the videos with the master(s). Descriptives for each time point and correlations across time are reported in Table 2-2. The full possible range of caregiver support ratings were observed at each assessment. Support ratings were significantly and positively correlated with one another across all time points (*rs* = .17 to .55), with the weakest correlations observed between earlier caregiver-child interactions and caregiver support at the adolescent assessment.

Table **2-2:** Caregiver Support Correlations and Descriptives Across Waves

	1	2	3	4	5	6	7
1. 6 months	–						
2. 15 months	.41	–					
3. 24 months	.40	.43	–				
4. 35 months	.35	.42	.50	–			
5. 60 months	.43	.44	.47	.48	–		
6. 90 months	.38	.47	.43	.44	.55	–	
7. 154 months	.20	.17	.20	.21	.20	.32	–
Mean	2.71	2.71	2.77	2.95	2.46	2.39	2.17

SD	.83	.81	1.00	.99	.89	.87	.79
Range	1-5	1-5	1-5	1-5	1-5	1-5	1-5

Note. $N = 672$. Significance of all correlation coefficients $p < .001$.

Trier Social Stress Test

Procedure. At the beginning of the adolescent home visit, teens completed a modified version of the TSST (DeJoseph et al., 2019; Kirschbaum et al., 1993; Yim et al., 2010). This socio-evaluative stressor has been shown to elicit a strong stress response in children and teens (Gunnar, Talge, et al., 2009). See Figure 2-4 for an illustration of the TSST protocol and salivary sampling timeline. Trained research assistants evaluated each home environment to find an open but quiet space to conduct assessments with the teen. This space was typically the kitchen/eating area, situated in a separate room and out of direct sight of the living room where the primary caregiver completed their portion of the experiment. Following arrival, the experimenters unpacked study materials and chatted with the teen for the first 10-15 minutes. Participants were then provided with a *National Geographic* book and instructed to relax during the first baseline phase. After being outfitted with heart rate and blood pressure monitors, a 5-minute heart rate baseline was collected, immediately followed by the first saliva sample and blood pressure measure (time = 0). Participants then responded to a series of questionnaires about general health and medications. After receiving initial task instructions (described below), participants completed the TSST.

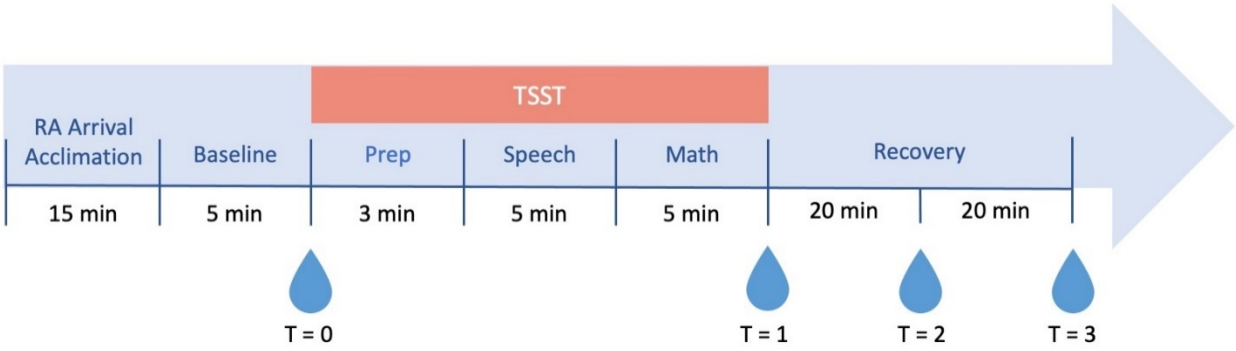

Figure **2-4**: Trier Social Stress Test and Saliva Sampling Protocol

A 5-minute heart rate measurement, followed by saliva and blood pressure samples, were taken immediately after the TSST (time = 1). Participants were then debriefed on task deception. Additional saliva and blood pressure samples were collected 20-minutes (time = 2) and 40-minutes (time = 3) post-task as participants completed additional home visit assessments. After quietly resting and completing questionnaires, the final 40-minute sample should reflect physiological recovery of the HPA axis. At the time of collection, all four saliva samples were time-stamped by experimenters within the data entry software *Blaise*. These time stamps were used to estimate intervals between saliva samples and session time of day.

Task Paradigm. The TSST comprised a three-minute preparation period, followed by a five-minute free speech and a five-minute math task (Kirschbaum et al., 1993; Yim et al., 2010). During their speech, participants were shown a video of two older teenage panelists (aged 14-16) seated at a table in white laboratory coats. The panelists displayed expressions of disinterest and boredom (e.g., yawning, playing on their mobile phone, and occasionally taking notes) during the task. Panelists were

matched to the participant's own gender, with one Black and one White member so all participants had one racially matched panelist. Though the video was pre-recorded, participants were told that they were conducting a live video conference with teenage researchers from New York University using NYU's "Blue Jeans" system. The recording was presented on a laptop with a built-in webcam, placed approximately three feet away from the participant.

Research assistants read task instructions verbatim to each participant to ensure standardization across sessions. For the first phase of the TSST, teens were told that they were going to introduce themselves as they would to a new class in school, including at least one good thing and one bad thing about themselves. Their goal was to convince the class that they were very likeable and would be a good student. Participants were told that the teen researchers at NYU were going to listen and take notes, and that their speech would be videotaped for later analysis by experts. Prior to beginning the speech, participants were given a three-minute preparation period during which they could take notes and write down things to talk about on paper. After the three minutes were up, participants were asked to stand and put away their notes, while the research assistant started the video camera and began the "conference call." Facing the panel shown on the laptop, participants introduced themselves and began their speech. If they paused and remained silent for 20 seconds, the research assistant began asking a prepared list of questions (e.g., "Tell us the three best things about you," "Do you like being part of a team?"). If participants spoke freely for four minutes, they were interrupted at an appropriate juncture and asked the same list of questions.

Next, teens completed a five-minute serial subtraction task. The sham panel was not shown during this segment of the TSST due to extreme levels of distress observed during the pilot, but their performance was still recorded. Participants were instructed to start at 1027 and subtract by 5 until they reached zero, moving as fast as possible while maintaining accuracy. If they made a mistake, they were corrected and told to start over from the beginning. After one minute, if participants could not get past three to four correct responses, they were moved down to an easier level (e.g., starting at 200 and subtracting by 3's). If participants were flying through the answers with ease, they were moved up to a harder level (e.g., starting at 1000 and subtracting by 6's). At specified points throughout the task, research assistants cued the participants to "answer a little faster." When the five minutes were up, the camera was turned off, participants were debriefed, and the additional saliva samples were collected.

Salivary Cortisol

Four saliva samples were collected using the Salimetrics passive drool collection method, and later assayed using ELISA kits (Salimetrics, Carlsbad, CA). Each sample was assayed in duplicate using a highly sensitive enzyme immunoassay (Salimetrics, Carlsbad, CA). The tests used a sample volume of 25 µL per determination, a sensitivity lower limit of 0.007 µg/dL, and a standard curve ranging from 0.012 to 3.0 µg/dL. The intra-assay coefficient of variation was less than 10%, and the inter-assay coefficient was less than 15%. Preliminary descriptives were assessed to identify outliers in cortisol values above or below 3 SD from the sample-level mean. Initial distributions were highly kurtotic and positively skewed, so all values were square root transformed after outlier removal to normalize distributions.

Initial growth models suggested that cortisol values displayed a significant linear decline from baseline to 40-minutes post-TSST (γ = -.02, p < .001; left plot in Figure 2-5), with no significant quadratic effect (γ = -.00, p = .22). However, as displayed through a subsample of participants in Figure 2-5 (right plot), there was substantial variability in patterns of cortisol reactivity across the TSST. To help capture this variability, cortisol was simplified into two individual measures: baseline (t = 0) and a reactivity change score from baseline to 20-minutes post-TSST ($t_2 - t_0$). The 20-minute saliva sample was chosen based on previous work denoting peak cortisol values approximately 20-30 minutes following stressor onset and a subsequent return to pre-test levels within 40-60 minutes (Adam et al., 2007). A positive reactivity score indicates an increase in cortisol from baseline in response to the stressor task, while negative scores indicate a decrease. Reactivity scores ranged from -0.5 to 0.26 (M = -.04, SD = .09), and approximately 26% of participants displayed an increase in cortisol from baseline to task.

Finally, it should be noted that, despite the present protocol, there was inevitable variability in the time intervals between saliva samples collected. Although active task time between baseline and 20-minutes post-TSST – capturing the period following rest through the theoretical peak of cortisol values after the social stressor – should have been approximately 33 minutes (see Figure 2-4), this does not directly account for variation in the length of time it took each RA to explain instructions, answer questions, and navigate any technical issues. According to time stamps collected at the start of each saliva sample, timing intervals between samples collected at baseline and 20-minutes post-TSST varied from 31 to 89 minutes (M = 51.85, SD = 7.32). Therefore, timing intervals were included as a covariate in all cortisol-related analyses.

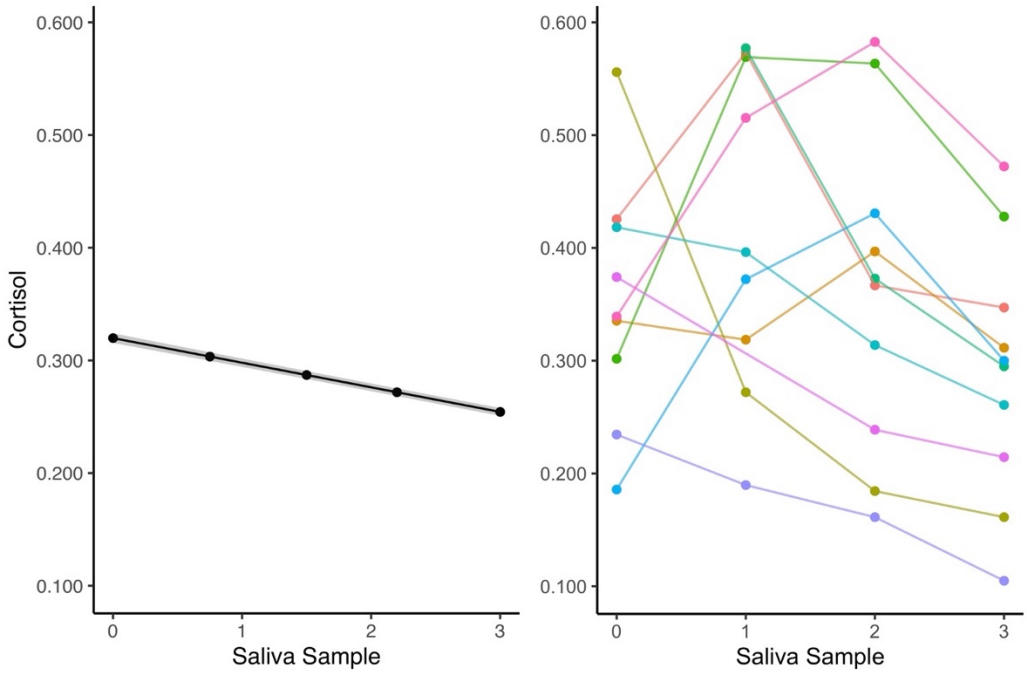

Figure **2-5**: Cortisol Trend and Inter-Individual Variability Across the TSST

Note. Change in cortisol from baseline (t = 0) to 40-minutes post-TSST (t = 3). The left plot depicts the linear sample-level trend. The right plot depicts raw cortisol trajectories for a subsample of 10 participants, highlighting inter-individual variability in patterns of cortisol change over time.

Health and Medications

As part of the saliva sampling protocol, participants were asked a series of self-report questions about their general health, behaviors, and medications. Recalling from the previous day(s), teens were asked to report how many hours they had slept the night prior to the home visit, whether or not they had taken any medications in the past 24 hours, and how they would rate their health over the last two days compared to other

same-age children (from 1 = *excellent* to 5 = *poor*). Additionally, teens were asked to report the number of hours and minutes that had passed since they last had something to eat/drink other than water.

Trait Anxiety

At the adolescent assessment, participants completed the self-report State Trait Anxiety Inventory for Children (STAIC; Spielberger et al., 1973). This measure is divided into two 20-item subscales: state anxiety that captures transient emotional states characterized by increased tension and arousal, and trait anxiety that captures disposition or proneness towards anxious responding. The present study used the trait anxiety subscale. Participants rated how each item described how they usually feel, from 1 (*hardly ever*) to 3 (*often*). Example items included, "I worry about making mistakes" and "I notice my heart beats fast." Item scores were summed together, with higher total scores reflecting greater trait anxiety (Cronbach's $\alpha = 0.89$). Trait anxiety was normally distributed within the present adolescent sample ($M = 35.26$, $SD = 7.68$; skew = .47, kurtosis = -.10), with scores extending across the full possible range of severity from 20 to 60.

Covariates

Associations between primary study variables and demographic and session-specific variables were examined to determine the inclusion of covariates. Race/ethnicity was re-coded to combine Black participants with those in the three smaller racial groups. Cortisol change scores ($t = 3.58$, $p < .001$), household chaos dimensions ($ps < .001$), and trait anxiety ($t = -3.09$, $p = .002$) varied by race, such that White participants displayed greater reactivity, lower chaos, and lower anxiety relative to those in the minoritized

racial groups. Females displayed greater reactivity (t = 2.23, p = .03) and higher anxiety (t = -6.72, p < .001) relative to males. Age at the adolescent assessment was positively correlated with baseline cortisol (r = .12, p = 001) and trait anxiety (r = .09, p = .01).

Moving to confounds related to health and session timing: cortisol was not associated with hours slept the previous night (ps > .86), medications taken in the previous 24 hours (ps > .10), or general self-reported health (ps > .58). Baseline cortisol values were positively correlated with the number of hours prior to the session that the participant had food or drink (r = .10, p = .02), and significantly declined across the time of day that sessions were held (r = -.37, p < .001). Overall, in addition to the timing interval between saliva samples, primary models covaried for age, sex, race/ethnicity, hours since eating or drinking, and session time of day. For models that did not include cortisol, the timing interval, eat/drinking, and session time covariates were removed.

Data Analysis

Preliminary Analyses

To prepare for the primary analyses, lability scores were first calculated for supportive caregiver behavior. Given empirical support for typical developmental change in caregiver warmth and support across childhood and adolescence, person-level trends were estimated from a larger sample-level model rather than specifying fully personalized trajectories for each participant. Consistent with previous literature (Lippold et al., 2016, 2018, 2021; Marceau et al., 2015), an initial two-level growth model was estimated in the following form

$$\text{Level 1: } Support_{it} = \beta_{0i} + \beta_{1i}(Age_{it}) + \beta_{2i}(Age_{it})^2 + e_{it}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + u_{0i} \quad (1)$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

$$\beta_{2i} = \gamma_{20} + u_{2i}$$

Where $Support_{it}$ represents repeated measures of observed caregiver support for individual *i* at assessment *t* modeled as a function of the person-specific intercept (β_{0i}), linear (β_{1i}) and quadratic (β_{2i}) slopes of developmental change, and residual error (e_{it}). At level two, γ parameters represent the sample-level intercept and slopes, and *u* parameters represent individual deviations from those terms. "Age" in our model refers to continuous age in years, allowing for greater variability within assessments (SD_{age} = .10 to .54 across waves). Both linear and quadratic growth curves were tested at the sample-level. The quadratic model was not significant, so linear effects are used in all subsequent steps.

Next, the estimated growth model above was used to estimate the person-specific *intercept* (e.g., initial level of sensitive caregiver behavior), *developmental trend* (e.g., linear change in behavior over time), and *lability* (e.g., within-person standard deviation [σ_i] of the growth model residuals [e_{it}]) for each caregiver *i*. Lability scores were calculated as

$$Lability_i = \sqrt{\sigma_i^2} = \sqrt{\frac{1}{T-1}\sum_{t=1}^{T}(e_{it} - \bar{e}_i)^2} \quad (2)$$

where higher scores indicate greater wave-to-wave, within-person fluctuations in caregiver behavior, independent from systematic, between-person change over time. Lower scores reflect a more stable pattern of caregiver support, but do not indicate the degree of the quality (e.g., high vs. low support) of that behavior.

We additionally decomposed the variance in repeated measures of caregiver support attributed to developmental change vs. that attributed to lability, following

procedures outlined by Snijders and Bosker (1999) and Lippold et al. (2016). An estimate of residual variance (σ^2_e) (from equation 1) that incorporates our time metrics was compared to residual variance obtained from an unconditional baseline model without time as a predictor ($\sigma^2_{e(baseline)}$), calculated as

$$\% \text{ Developmental Variance} = \frac{(\sigma^2_{e(baseline)} - \sigma^2_e)}{\sigma^2_{e(baseline)}} \quad (3)$$

This estimate quantifies the percentage of the total variance in longitudinal caregiver support captured by developmental trends. The remaining residual variance can be conceptualized as lability (meaningful fluctuations around the developmental trend), as well as measurement error and time-specific error (non-meaningful fluctuations).

Primary Analyses

Research Question 1: Mediation

Our first research question addressed whether adolescent cortisol measures mediated the effects of environmental instability on later anxiety outcomes. Mediation models were conducted in three steps, beginning with the direct effects of 1) instability on adolescent anxiety, and 2) instability on adolescent cortisol. If these initial criteria were met, the indirect effect of instability on adolescent anxiety through cortisol was tested in the final step (Baron & Kenny, 1986). Adolescent cortisol measures at baseline and in response to the TSST were analyzed separately, as were the two measures of instability (caregiver support lability and household chaos dimensions).

Lability in caregiver support was analyzed first. In addition to person-specific lability scores, models also included each caregiver's linear slope as a covariate (calculated using equations 1 and 2). This approach allowed us to understand the effects

of lability above and beyond the effects of developmental change in caregiver behavior from infancy through adolescence. The person-specific intercept was not included due to high collinearity with the linear slope (r = -.90).

Household instability and disorganization were analyzed second. The cumulative effects of instability and disorganization were modeled separately across two phases of development to explore potential sensitive periods. The first period extended across early life (2 to 24 months) and the second period extended into middle childhood (2 to 90 months).

Research Question 2: Moderation

Our second research question addressed the moderating effect of cortisol functioning on the association between environmental instability and later anxiety. The first set of regression models tested the interaction between each cortisol measure (baseline and reactivity) and lability in caregiver support on adolescent anxiety symptoms. The person-specific liner slope in caregiver support was included as a covariate. The second set of models separately tested the interaction between cortisol and each dimension of cumulative household chaos – disorganization and instability – across the two developmental periods of interest (2-24 months and 2-90 months).

Preliminary Results

As part of the preliminary analyses, I estimated developmental change in caregiver support from infancy through adolescence, and further decomposed the variance in behavior captured by developmental change relative to lability. Results indicated that caregiver support displayed a significant negative linear (γ = -.05, p < .001) pattern of change over time (see Table 2-3 and Figure 2-6). Variance decomposition

calculations indicated that 12.0% of the variance in caregiver support was attributed to linear developmental trends from infancy through adolescence, with the remaining 88% of variance attributed to meaningful wave-to-wave fluctuations in behavior (lability) and additional error. Thus, variability in observations of supportive caregiver behavior is likely driven by both averaged developmental trends and lability around those trends.

Table **2-3:** Caregiver Support Developmental Trend: Infancy to Adolescence

Fixed Effects	*b (SE)*	*t*
Intercept	2.83 (.03)	98.84***
Age	-.05 (.00)	-17.76***
Random Effects	*SD*	*95% CI*
Intercept	.64	[.59, .69]
Residual	.70	[.68, .71]
Linear Slope	.03	[.02, .04]
Intercept/Linear Cov.	-.71	[-.82, -.53]

Note. Age represents continuous age in years. Cov = covariance. *** $p < .001$

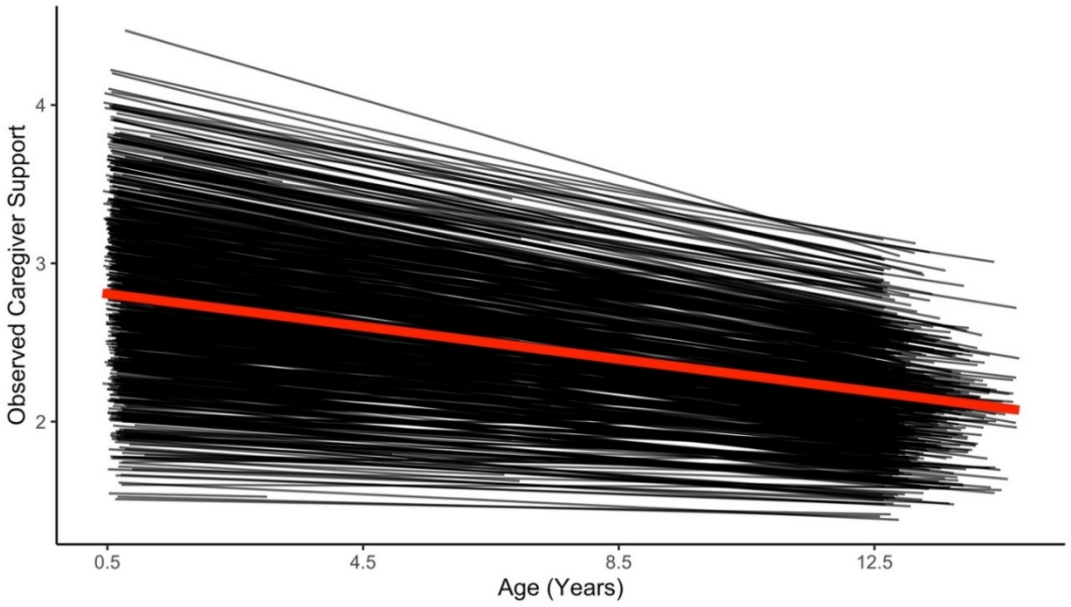

Figure **2-6**: Linear Developmental Trend in Caregiver Support

Note. Black lines display person-level predicted trajectories. Red line displays the sample-level averaged trend. Note the wide spread of variability across participants.

Table **2-4:** Bivariate Correlations, Means, and Standard Deviations of Main Study Variables

	1	2	3	4	5	6	7	8	9	10
1. Baseline	–									
2. Reactivity	**-.59**	–								
3. Intercept	-.03	**.12**	–							
4. Trend	.03	**-.12**	**-.90**	–						
5. Lability	.02	-.05	**.16**	**-.09**	–					
6. Disorg. 2-24	.04	**-.08**	**-.45**	**.41**	**-.09**	–				
7. Instab. 2-24	.03	-.01	**-.22**	**.19**	-.02	**.35**	–			
8. Disorg. 2-90	.04	-.05	**-.47**	**.43**	**-.11**	**.87**	**.35**	–		
9. Instab. 2-90	.01	-.01	**-.34**	**.29**	-.05	**.45**	**.75**	**.47**	–	
10. Anxiety	-.01	-.03	**-.15**	**.13**	.02	**.18**	**.10**	**.18**	**.15**	–
Mean	.32	-.04	2.84	-.05	.66	.02	.00	.01	-.03	35.25
SD	.12	.09	.57	.02	.21	.61	.69	.67	.70	7.61
Range	.08-.93	-.50-.26	1.51-4.56	-.12-.00	.05-1.32	-1.48-1.99	-1.07-3.27	-1.44-2.10	-1.15-3.11	20-60

Note. N = 678. Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Person-specific caregiver support *intercept* = initial level of behavior at 6 months, *trend* = linear change in behavior from infancy to adolescence, *lability* = wave-to-wave variation in behavior. Disorganization and instability reflect standardized early (cumulative 2 to 24 months) and chronic (cumulative 2 to 90 months) chaos. Bolded values indicate $p < .05$.

Primary Results

Descriptive Statistics

Initial bivariate correlations and primary variable descriptives are reported in Table 2-4. Briefly, cumulative household disorganization and instability across both developmental periods were positively correlated with adolescent anxiety symptoms (rs > .10, ps < .008). Chaos measures were not correlated with adolescent baseline cortisol (ps > .28), though disorganization across 2-24 months of age was significantly correlated with cortisol reactivity (r = -.08, p = .04), such that greater early disorganization was associated with lesser reactivity to social stress. Lability in caregiver support was not significantly correlated with adolescent anxiety or cortisol measures (ps > .16).

Research Question 1: Mediation

Caregiver Support Lability

Beginning with the first research question, I first tested the direct effects of lability in caregiver support from infancy through adolescence on adolescent anxiety and cortisol. Results indicated that lability in caregiver support was not associated with anxiety (b = 1.30, p = .32), though a lesser decline in caregiver support over time was significantly associated with higher anxiety symptoms in adolescence (b = 43.98, p = .007). Caregiver lability was not significantly associated with baseline cortisol levels (b = .02, p = .36; Table 2-5). However, the effect of lability on cortisol reactivity across the social stress task was significant (b = -.03, p = .04; see Figure 2-7), such that children who experienced a greater level of lability in caregiver support across development displayed a greater decrease in cortisol from baseline to 20-minutes post-TSST (e.g.,

lesser reactivity to social stress). The linear slope of caregiver support was significantly associated with cortisol reactivity (b = -.44, p = .02; see Figure 2-7), such that children who experienced a lesser decline in caregiver support over time also displayed lesser cortisol reactivity. Given the lack of significant main effects of caregiver lability on adolescent anxiety, indirect effects were not pursued further.

Table **2-5:** Caregiver Lability Main Effects on Adolescent Anxiety and Cortisol

	Anxiety	Cortisol Baseline	Cortisol Reactivity
	b (SE)	*b (SE)*	*b (SE)*
Intercept	28.29 (7.00)***	0.30 (.12)**	-0.28 (.09)**
Sex	-3.82 (.56)***	-.02 (.01)	.02 (.01)*
Race	-.77 (.63)	.01 (.01)	.01 (.00)
Age	.82 (.52)	.02 (.01)*	.01 (.01)*
Session Time	–	-.02 (.00)***	.01 (.00)***
Eat/Drink Time	–	.00 (.00)	-.00 (.00)
Saliva Interval	–	.00 (.00)	.00 (.00)*
Trend	43.98 (16.40)**	.36 (.26)	-.44 (.19)*
Lability	1.31 (1.31)	.02 (.02)	-.03 (.01)*

Note. Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples collected at baseline and 20-minutes post-TSST. Person-specific caregiver support *trend* = linear change in behavior from infancy to adolescence, *lability* = wave-to-wave variation in behavior. Sex coded as 0 = female and 1 = male. * $p < .05$, ** $p < .01$, *** $p < .001$

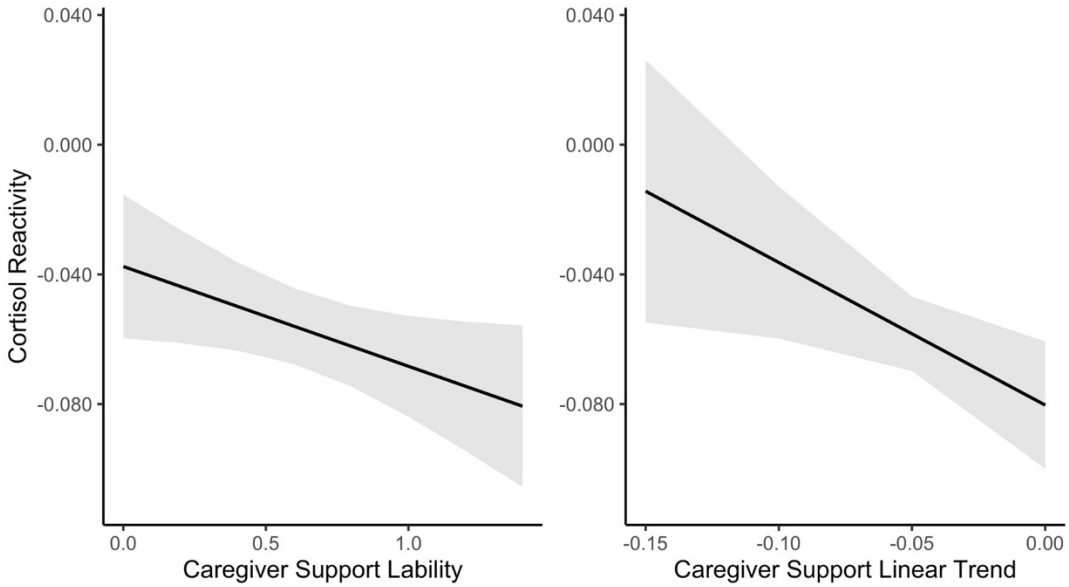

Figure **2-7**: Caregiver Support Lability and Linear Trend Predict Cortisol Reactivity

Note. Higher lability (left plot) and a lesser decline (right plot) in caregiver support from infancy through adolescence are each associated with a greater decline (e.g., lesser reactivity) in cortisol from baseline to social stress task.

Household Chaos

Next, I tested the effects of cumulative household chaos from 2 to 24 months on adolescent anxiety and cortisol functioning. All results for household disorganization are presented in Table 2-6, and results for household instability are presented in Table 2-7. Findings indicated that greater early household disorganization ($b = 1.80$, $p < .001$) and instability ($b = .84$, $p = .04$) significantly predicted higher anxiety in adolescence. However, there was no significant effect of either household dimension on adolescent cortisol at baseline or in response to the social stressor ($ps > .13$).

Widening our developmental window to span from infancy through middle childhood (2 to 90 months), similar significant effects were observed for household disorganization ($b = 1.71$, $p < .001$) and instability ($b = 1.26$, $p = .002$) on adolescent anxiety. However, these more chronic chaos dimensions did not predict adolescent cortisol at baseline or in response to the social stressor (ps > .27). Given the lack of significant direct effects of household chaos on cortisol across both developmental periods, indirect effects were not pursued further.

Table **2-6:** Household Disorganization Main Effects on Adolescent Anxiety and Cortisol

	Anxiety *b (SE)*	Cortisol Baseline *b (SE)*	Cortisol Reactivity *b (SE)*
2-24 Months:			
Intercept	28.48 (6.89)***	.30 (.12)*	-.30 (.09)***
Sex	-3.52 (.56)***	-.02 (.01)	.01 (.01)*
Race	-1.15 (.59)	.00 (.01)	.02 (.01)**
Age	.70 (.52)	.02 (.01)*	.01 (.01)*
Session Time	–	-.02 (.00)***	.01 (.00)***
Eat/Drink Time	–	.00 (.00)	-.00 (.00)
Saliva Interval	–	.00 (.00)	-.00 (.0)*
Disorganization	1.80 (.48)***	.01 (.01)	-.01 (.01)
2-90 Months:			
Intercept	29.05 (6.89)	.30 (.12)*	-.30 (.09)***
Sex	-3.52 (.56)***	-.02 (.01)	.01 (.01)*
Race	-1.24 (.58)*	-.00 (.01)	.02 (.01)**
Age	.66 (.52)	.02 (.01)*	.01 (.01)*
Session Time	–	-.02 (.00)***	.01 (.00)***
Eat/Drink Time	–	.00 (.00)	-.00 (.00)
Saliva Interval	–	.00 (.00)	-.00 (.00)*
Disorganization	1.71 (.43)***	.01 (.01)	-.00 (.01)

Note. Cumulative household disorganization separately estimated into infancy (2-24 months) and middle childhood (2-90 months). Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples

collected at baseline and 20-minutes post-TSST. Sex coded as 0 = female and 1 = male.

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

Table **2-7:** Household Instability Main Effects on Adolescent Anxiety and Cortisol

	Anxiety *b (SE)*	Cortisol Baseline *b (SE)*	Cortisol Reactivity *b (SE)*
2-24 Months:			
Intercept	29.41 (6.98)***	.30 (.12)*	-.29 (.09)**
Sex	-3.56 (.56)***	-.02 (.01)	.01 (.01)*
Race	-1.62 (.57)**	-.00 (.01)	.02 (.01)**
Age	.65 (.52)	.02 (.01)*	.01 (.01)*
Session Time	–	-.02 (.00)***	.01 (.00)***
Eat/Drink Time	–	.00 (.00)	-.00 (.00)
Saliva Interval	–	.00 (.00)	-.00 (.00)
Instability	.84 (.41)*	.00 (.01)	-.00 (.00)
2-90 Months:			
Intercept	30.83 (6.98)***	.30 (.12)*	-.29 (.09)**
Sex	-3.53 (.56)***	-.02 (.01)	.01 (.01)*
Race	-1.43 (.58)*	-.00 (.01)	.03 (.01)**
Age	.53 (.53)	.02 (.01)*	.01 (.01)*
Session Time	–	-.02 (.00)***	.01 (.00)***
Eat/Drink Time	–	.00 (.00)	-.00 (.00)
Saliva Interval	–	.00 (.00)	-.00 (.00)
Instability	1.26 (.41)**	.00 (.00)	.00 (.00)

Note. Cumulative household instability separately estimated into infancy (2-24 months) and middle childhood (2-90 months). Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples collected at baseline and 20-minutes post-TSST. Sex coded as 0 = female and 1 = male.

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

Research Question 2: Moderation

Caregiver Support Lability

Moving to the second research question, I tested the moderating effects of cortisol at baseline and in response to the social stressor on the association between caregiver support lability and adolescent anxiety (Table 2-8). Results indicated no significant interaction between lability and baseline (b = 3.10, p = .78) or reactivity (b = -16.57, p = .28) values.

Table **2-8:** Cortisol Moderation: Caregiver Lability and Anxiety

	Baseline Moderation *b (SE)*	Reactivity Moderation *b (SE)*
Intercept	23.28 (8.15)**	22.96 (7.98)**
Sex	-3.82 (.57)***	-3.81 (.58)***
Race	-.78 (.65)	-.85 (.66)
Age	.77 (.53)	.77 (.54)
Session Time	.15 (.12)	.18 (.11)
Eat/Drink Time	-.06 (.09)	-.07 (.09)
Saliva Interval	.07 (.04)	.06 (.04)
Trend	37.80 (16.68)*	36.74 (16.81)*
Lability	.90 (3.78)	1.13 (1.52)
Cortisol	-2.82 (7.54)	10.13 (10.67)
Lability x Cortisol	3.10 (10.92)	-16.57 (15.57)

Note. Left-hand column displays the interaction between cortisol at baseline and caregiver lability, and right-hand column displays the interaction between cortisol reactivity and lability predicting anxiety. Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples collected at baseline and 20-minutes post-TSST. Person-specific caregiver support *trend* = linear change in behavior from infancy to adolescence, *lability* = wave-to-wave

variation in behavior. Sex coded as 0 = female and 1 = male. * $p < .05$, ** $p < .01$, *** $p < .001$

Household Chaos

Next, I tested the moderating effects of cortisol on the association between early cumulative household chaos from 2 to 24 months and adolescent anxiety. All results for household disorganization are displayed in Table 2-9, and household instability in Table 2-10. The interaction between early household disorganization and baseline cortisol was significant ($b = -8.87$, $p = .04$; Figure 2-8). Simple slopes indicated that, for those with lower baseline cortisol, greater early disorganization was significantly associated with higher anxiety symptoms in adolescence ($b = 2.67$, $p < .001$). This slope was not significant for those with higher baseline cortisol ($b = .51$, $p = .49$). The interaction between early household disorganization and cortisol reactivity was also significant ($b = 16.02$, $p = .006$; Figure 2-9). The slope of disorganization on anxiety was significant for those who displayed greater cortisol reactivity to social stress ($b = 3.11$, $p < .001$), such that higher disorganization was associated with higher anxiety symptoms. This slope was not significant for those who displayed lesser cortisol reactivity ($b = .38$, $p = .58$). Cortisol measures did not significantly moderate the association between early household instability and later anxiety ($ps > .44$).

Moving to more chronic household chaos measured from 2 to 90 months of age, results indicated that the interactions between household disorganization and both baseline cortisol ($b = -7.39$, $p = .07$) and cortisol reactivity ($b = 9.50$, $p = .07$) were not significant. Cortisol measures did not moderate the association between chronic

household instability and later anxiety (*p*s > .65). Overall, relative to the earlier developmental period estimated above, the effects of disorganization on adolescent anxiety were muted when chaos was estimated over a longer period of childhood, offering preliminary evidence for a sensitive period.

Table **2-9:** Cortisol Moderation: Household Disorganization and Anxiety

	Baseline Moderation *b (SE)*	Reactivity Moderation *b (SE)*
2-24 months:		
Intercept	23.33 (7.67)**	24.29 (7.82)**
Sex	-3.56 (.57)***	-3.40 (.58)***
Race	-1.06 (.62)	-1.17 (.63)
Age	.55 (.53)	.63 (.54)
Session Time	.19 (.12)	.17 (.11)
Eat/Drink Time	-.10 (.09)	-.10 (.09)
Saliva Interval	.08 (.04)*	.05 (.04)
Disorganization	4.40 (1.39)**	2.43 (.57)***
Cortisol	.33 (2.57)	-1.78 (3.49)
Disorganization x Cortisol	-8.87 (4.20)*	16.02 (5.79)**
2-90 months:		
Intercept	23.89 (7.68)**	24.46 (7.84)**
Sex	-3.56 (.57)***	-3.42 (.58)***
Race	-1.15 (.61)	-1.27 (.63)*
Age	.50 (.53)	.60 (.54)
Session Time	.20 (.12)	.19 (.11)
Eat/Drink Time	-.10 (.09)	-.11 (.09)
Saliva Interval	.08 (.04)*	.05 (.04)
Disorganization	3.89 (1.32)**	2.02 (.50)***
Cortisol	.41 (2.59)	-2.07 (3.51)
Disorganization x Cortisol	-7.39 (4.01)	9.50 (5.22)

Note. Left-hand column displays the interaction between cortisol at baseline and disorganization, and right-hand column displays the interaction between cortisol reactivity and disorganization predicting anxiety. Cumulative household disorganization separately estimated into infancy (2-24 months) and middle childhood (2-90 months).

Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples collected at baseline and 20-minutes post-TSST. Sex coded as 0 = female and 1 = male. * $p < .05$, ** $p < .01$, *** $p < .001$

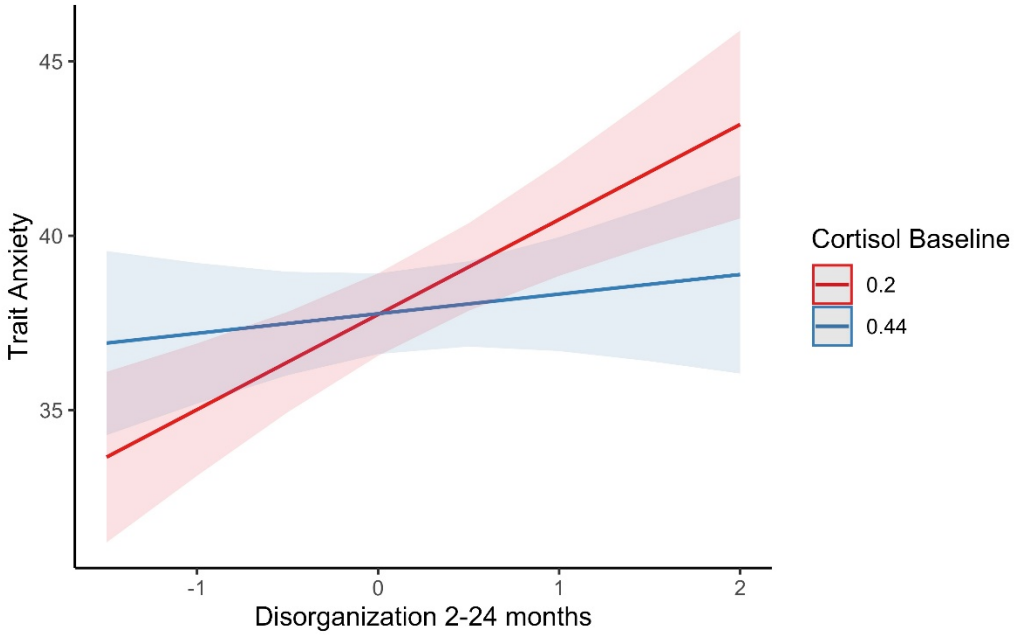

Figure **2-8**: Baseline Cortisol Moderates the Association Between Early Disorganization and Later Anxiety

Note. Cortisol at baseline is plotted at + 1 SD (.44; blue) and -1 SD (.20; red). The association between greater household disorganization across 2-24 months and higher adolescent anxiety symptoms was significant only for those who displayed higher baseline cortisol levels.

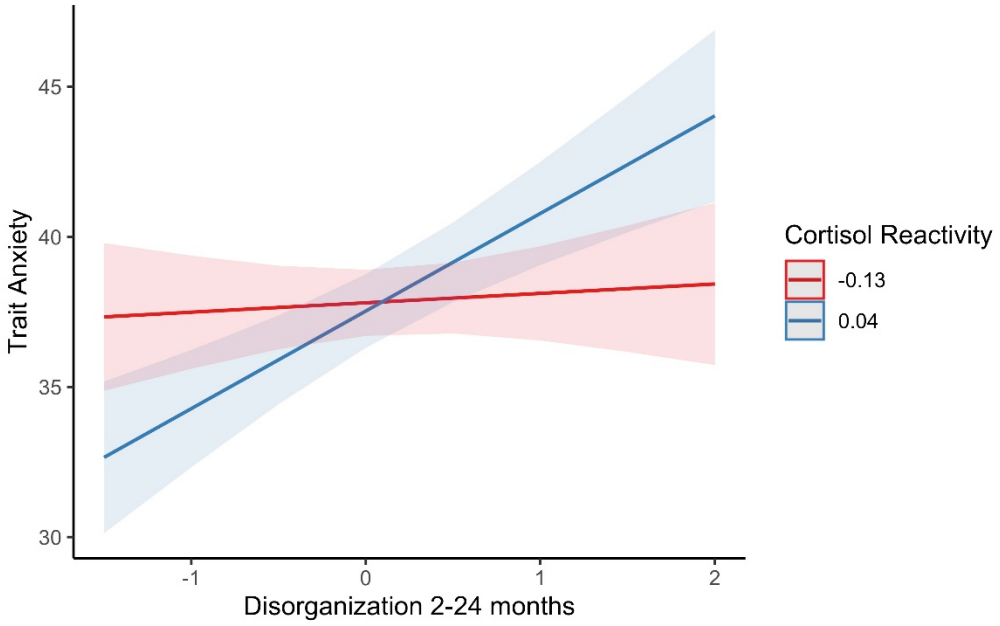

Figure **2-9**: Cortisol Reactivity Moderates the Association Between Early Disorganization and Later Anxiety

Note. Cortisol reactivity is plotted at + 1 SD (.04; blue) and -1 SD (-.13; red). The association between greater household disorganization across 2-24 months and higher adolescent anxiety symptoms was significant only for those who displayed greater reactivity to the social stress task.

Table **2-10:** Cortisol Moderation: Household Instability and Anxiety

	Baseline Moderation *b (SE)*	Reactivity Moderation *b (SE)*
2-24 months:		
Intercept	27.98 (7.48)***	26.01 (7.65)***
Sex	-3.67 (.57)***	-3.58 (.58)***
Race	-1.74 (.58)**	-1.78 (.60)**
Age	.60 (.53)	.71 (.54)
Session Time	.16 (.12)	.18 (.11)
Eat/Drink Time	-.10 (.09)	-.10 (.09)

Saliva Interval		
Instability	.61 (1.05)	.75 (.49)
Cortisol	-.59 (2.55)	-1.13 (3.48)
Instability x Cortisol	.51 (2.94)	-2.65 (4.93)
2-90 months:		
Intercept	24.90 (7.80)**	25.05 (7.95)**
Sex	-3.61 (.58)***	-3.52 (.58)***
Race	-1.37 (.61)*	-1.51 (.62)*
Age	.48 (.54)	.57 (.55)
Session Time	.20 (.12)	.22 (.11)
Eat/Drink Time	-.08 (.09)	-.08 (.09)
Saliva Interval	.07 (.04)	.04 (.04)
Instability	1.38 (1.02)	1.06 (.46)*
Cortisol	-.16 (2.55)	-1.80 (3.50)
Instability x Cortisol	-1.00 (2.70)	-2.03 (4.50)

Note. Left-hand column displays the interaction between cortisol at baseline and instability, right-hand column displays the interaction between cortisol reactivity and instability. Cumulative household instability separately estimated into infancy (2-24 months) and middle childhood (2-90 months). Cortisol values are square root transformed (reactivity = $t_2 - t_0$). Saliva interval refers to the amount of time between samples collected at baseline and 20-minutes post-TSST. Sex coded as 0 = female and 1 = male.

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

Discussion

As the prevalence of anxiety increases into adolescence, understanding the earlier developmental contexts that contribute to this heightened risk is important for establishing early prevention and intervention efforts (Beesdo et al., 2009; Kessler et al., 2012). The present study examined the effects of environmental instability across development on adolescent anxiety symptoms. Cortisol functioning was also tested as a mediator and moderator of this association. Results indicated a significant direct effect of

greater household disorganization on increased adolescent anxiety, as well as an effect of greater caregiver support lability on lowered cortisol reactivity to social stress. These findings offer initial evidence for the effects of environmental instability on later adolescent functioning, but did not support cortisol as a mediator. However, moderation effects were significant, such that greater household disorganization was associated with higher anxiety for those who displayed greater cortisol reactivity or lower baseline cortisol, aligning with the biological sensitivity to context framework (Belsky & Pluess, 2009; Ellis & Boyce, 2011). These effects were more robust for disorganization measured from 2 to 24 months of age relative to measurements across middle childhood, emphasizing the first two years of life as a potential sensitive period for the effects of environmental risk on later behavior. Findings are discussed in detail below.

Household Chaos

Beginning with direct effects, hypotheses were partially supported, such that greater cumulative household disorganization across infancy and middle childhood was associated with higher adolescent anxiety. These findings build upon previous work supporting the deleterious effects of household chaos on child emotional and behavioral problems (Marsh et al., 2020). Disorganization captures higher levels of stimulation (e.g., noise) and instability in routine and structure within the home, which likely undermines children's developing ability to self-regulate and manage emotion (Evans et al., 2005). However, results did not support the effects of household disorganization on later cortisol functioning. This finding contrasts with previous work within the present sample, which reported a significant association between cumulative household disorganization from 2 to 48 months of age and elevated cortisol across a mildly stressful data collection session

in early childhood (Blair et al., 2013). Additional previous work reported significant effects of household chaos in preschool on social stress reactivity in middle childhood (Doom et al., 2018). Given that chaos in the present sample was measured substantially earlier in development relative to cortisol, this direct association may have been obscured by protective factors in the intervening time period – for example, the presence of stable and supportive environments outside of the home (e.g., relationships with teachers and peers at school). Additionally, Suor et al. (2015) found that unpredictability within the home was associated with both elevated *and* lowered trajectories of basal cortisol from age 2 to 4 within one sample of children. These patterns of multifinality might have been obscured by the averaged, between-person approach used in the present study. However, it is also possible that household chaos predicts stress-response system development only within the context of additional risk factors that function as more proximal processes (e.g., variability in time spent with caregivers, quality of parenting behaviors; Bronfenbrenner & Evans, 2000). Future work is needed to better understand multiple aspects of the home environment that continue to influence HPA functioning across both childhood and adolescence, and whether household chaos predicts multiple profiles of change in cortisol across development, with an emphasis on person-centered modeling approaches.

Despite the lack of significant mediation effects, our findings did support moderation by cortisol on the association between household disorganization and adolescent anxiety. Aligning with differential susceptibility models, greater disorganization from infancy through 24 months of age was associated with higher anxiety for those who displayed greater cortisol reactivity to social stress (Belsky &

Pluess, 2009; Ellis & Boyce, 2011). This pattern of elevated reactivity might render children more susceptible to a stressful home environment. To use the classic analogy, highly reactive adolescents in the present sample could be conceptualized as "orchids," whose risk for maladjustment was closely tied to the quality of their early environment, while those who displayed lower reactivity could be thought of as "dandelions," whose outcomes did not vary according to their surroundings (Ellis & Boyce, 2008). However, this interpretation is predicated on the assumption that patterns of cortisol functioning reflect a more stable characteristic of the child from infancy through adolescence. Given that the stress-response system is thought to sample from and adjust to the surrounding environment, a continually disorganized household context might facilitate this rank-order stability over time for some children (Del Giudice et al., 2011). Future work should directly test this assumption by examining the effects of both stability and change in household disorganization on trajectories of HPA functioning across development.

Next, although diverging from our initial hypothesis, results indicated that greater early disorganization was associated with higher anxiety for those who displayed lower baseline cortisol. These findings align with previous work supporting elevated emotional problems for children with lower resting cortisol within home environments characterized by greater stress and a lack of routine (Badanes et al., 2011; Miller et al., 2017). However, it should be highlighted that baseline cortisol values in the present study were likely confounded by anticipatory arousal, as initial salivary samples were collected approximately 20 minutes after research assistants arrived at the home (e.g., around when cortisol typically peaks following stressor onset). Blunted anticipatory cortisol has been associated with greater emotional lability and dysregulation in teens (Kliewer et al.,

2016), while higher anticipatory cortisol within the present context might reflect early engagement of behavioral and cognitive processes needed to effectively prepare for the unpredictability of the home visit (Blair et al., 2005). Overall, regularity, structure, and routine within the home might be particularly necessary for youth displaying difficulty with flexible stress responsivity and regulation (Lo et al., 2021; Miller et al., 2017), such that a history of household disorganization might impede the development of effective emotion regulation strategies by increasing feelings of uncertainty and anxiety. More work is needed to explicitly test all three phases of cortisol responsivity – anticipation, stress reactivity, and recovery – in relation to household chaos and anxiety. Given that higher cortisol at baseline was significantly correlated with a greater decline in cortisol from baseline to task in the present sample, lower resting or anticipatory arousal might allow for a stronger subsequent stress response. Future work should consider temporal links between each phase of responsivity across an acute stressor.

Importantly, the moderation effects discussed above were examined across multiple, early sensitive periods of development. The present study compared the effects of early household chaos measured across the first two years of life (2-24 months) to more chronic measures from infancy through middle childhood (2-90 months). Results indicated that the interactions between household disorganization and cortisol in predicting adolescent anxiety were more robust for the earlier measure of disorganization, such that the inclusion of household experiences across middle childhood muted the effects. These findings align with previous work emphasizing the importance of a high-quality family and home environment across the first two years of life for adaptive stress responsivity and behavioral outcomes (Hostinar et al., 2015a; McLaughlin et al., 2015).

Given that the present study only examined later measures of physiological and psychological functioning in adolescence, future intervention work should consider if active changes to the surrounding household environment in the first few years of life reciprocally contribute to declines in anxiety for those who display profiles of high biological sensitivity (e.g., elevated cortisol reactivity). Additionally, though chaos was not measured into adolescence for the present study, pubertal maturation has been considered a recalibration period for reshaping biological systems and related behaviors to adjust to the current environment (Doom et al., 2015; Gunnar et al., 2019). Future work should longitudinally extend measures of environmental risk to test if adolescence might serve as another sensitive period for the effects of instability on psychopathology for biologically sensitive youth, particularly if the quality of the surrounding environment has changed since the first few years of life.

Finally, it should be noted that, although greater cumulative household instability was associated with higher adolescent anxiety, all other effects were not significant. This clear divide in our findings might be due to differences between the two chaos dimensions. Household disorganization captures more proximal, day-to-day emotion regulation challenges within an unstructured home environment, while household instability captures less frequent destabilizing events (e.g., people moving in and out of the home). Therefore, despite variation in the degree of biological sensitivity across the present sample, our measure of instability might not be powerful enough to pull out individual differences in anxiety across levels of cortisol functioning.

Caregiver Support

Moving to instability within the caregiver-child context, direct effects indicated that greater lability in caregiver support was associated with lesser cortisol reactivity to social stress in adolescence. Given the importance of caregiver support for teaching children how to regulate emotion, lower behavioral lability likely provides the consistent scaffolding needed for the development of adaptive coping abilities. Indeed, moderate physiological reactivity to stress has been shown to promote effective self-regulation, cognitive functioning, and engagement with the environment (Blair et al., 2005). Conversely, high lability in caregiver support creates an unpredictable environment across development, such that repeated caregiver-related stressors might lead to lesser cortisol reactivity in adolescence to protect against repeated activation of the stress response system. This pattern of cortisol hypoactivity likely reflects a stress response system that is unable to effectively mobilize and engage with social challenge, mitigating coping resources (Seiss et al., 2014). However, lability in caregiver support was not associated with anxiety outcomes, and therefore the mediation effects of cortisol were not supported. Perhaps lability in more explicit measures of caregiver autonomy-granting and control would explain greater variance in adolescent anxiety symptoms, as control and self-efficacy are considered important determinants of anxiety (Chorpita & Barlow, 1998; McLeod et al., 2007). Future work should also expand beyond internalizing symptoms, as the patterns of cortisol hyporeactivity and lability in positive caregiver behaviors observed in the present study have been found to increase risk for adolescent externalizing and delinquent behaviors (Alink et al., 2008; Lippold et al., 2018). Further, the biological sensitivity to context framework includes hypotheses about the beneficial

effects of positive, nurturing environments on positive youth development. Although present findings did not support the effects of stable caregiver support on psychopathology, perhaps physiologically sensitive children would be more likely to display positive developmental outcomes within this context – for example, higher prosocial and academic competencies (Obradovic et al., 2010). In addition to capturing the full breadth of the quality of a child's environment, future work should also explicitly measure both positive and negative aspects of child functioning.

Next, contrary to hypotheses, results indicated that cortisol functioning did not moderate the association between caregiver support lability and later anxiety. Similar to the household disorganization findings discussed above, there might be a sensitive period for the effects of caregiver lability on developmental outcomes. Due to the need for more frequent waves of observed caregiver behavior for an accurate measure of lability, caregiver-child interactions were not collected with sufficient frequency in the present study to compare effects in early life (e.g., up to 24 months of age) relative to later in childhood and adolescence. Building from previous work, perhaps stability in caregiver support specifically across the first few years of life provides the foundation for adaptive emotional and regulatory development, such that more physiologically sensitive children benefit the most from a positive environment (Hostinar et al., 2015a; McLaughlin et al., 2015). These benefits might subsequently decline into adolescence as other contexts (e.g., school, peers) increase in salience (Hostinar et al., 2015b). Future work should seek to sample more intensively across different phases of development and across different contexts to examine whether biological sensitivity is uniform or if it varies according to developmental salience of the surrounding environment.

Finally, although not an explicit component of our hypotheses, the person-specific linear trend in caregiver support across development was associated with both anxiety and cortisol reactivity, above and beyond the effects of lability in caregiver behavior. A lesser decline in caregiver support from infancy through adolescence was associated with lower cortisol reactivity to social stress and higher anxiety symptoms. Although initially counterintuitive, observed declines in caregiver support across development might represent normative and successful increases in children's independence and autonomy away from the parent-child context, as adolescents require less active parental support and guidance (Kobak et al., 2017). Further, given the strong negative correlation between the person-specific linear trend and intercept (initial levels of behavior at 6 months) of caregiver support, a lesser decline might also capture those caregivers who are low and stable in support over time. Without progressive scaffolding offered through developmentally-sensitive changes in parenting behavior – to include higher support in early life and higher autonomy later in adolescence – a child might not ultimately develop adaptive emotion regulation skills and perceptions of self-efficacy (Brenning et al., 2015). Future work should consider exploring different longitudinal profiles of caregiver behavior across development to better differentiate between those who are low-stable, high-stable, or changing in both positive and negative engagement.

Conclusion

Extending beyond singular measures of household chaos or caregiver support, the present study highlights the importance of a developmental perspective for capturing how environmental risk accumulates and varies across time. Addressing developmental variability *in addition to* longitudinal changes in caregiver behavior might serve to

optimally promote adaptive physiological regulation in adolescence. Further, individual differences in biological sensitivity to the effects of early household disorganization were found to be potent predictors of adolescent anxiety, with the first few years of life serving as a period of elevated sensitivity to the environment. Particularly in samples characterized by higher levels of poverty, solutions to creating a less crowded and less stimulating household environment are needed to promote positive mental health outcomes across the board. Further, prevention and intervention work might consider incorporating physiological measures into child assessment, as children who are most physiologically sensitive to the negative effects of a harsh or unstable environment might also be especially responsive to intervention measures.

Chapter 3

Pubertal and Peer Influence on Social Stress Sensitivity and Anxiety Outcomes in Adolescence

Introduction

Progression into and through adolescence comes with a confluence of transitions across social, biological, emotional, and cognitive domains (Blakemore & Mills, 2014). These transitions are thought to drive an increase in attention and salience given to social evaluation and standing within the peer context, referred to as *social sensitivity* (Somerville, 2013). A growing body of work seeks to understand the role of social sensitivity in adjustment, emphasizing how the larger developmental context – both external and internal to the child – contributes to changes in sensitivity from childhood into adolescence.

Beginning with the social context, teens are met with the developmental challenge of shifting attachment away from the family system and towards peer relationships outside of the home (Blakemore & Mills, 2014). This social re-orientation is facilitated by transitions from elementary to secondary educational settings, which come with an increase in the number of peers at school and more time spent with friends relative to family members across a typical day (Anderson, 2013). Peer relationships at this age are characterized by increased intimacy and disclosure, but also tend to exist in a state of flux (Brown & Larson, 2009; Somerville, 2013). School transitions – alongside entry into new classroom groups across the middle school years – continually place teens into new social environments without all their same peers. This process injects substantial discontinuity

and variability into social networks during a developmental phase characterized by an increased need for belonging (Felmlee et al., 2018; Temkin et al., 2018). Thus, age-related changes in social relationships both at home and at school might contribute to increases in social sensitivity following childhood (Nelson et al., 2016).

Biological transitions complement these age-related changes in social affiliation, such that neural and pubertal maturation contribute to the underlying mechanisms of developmental change in social sensitivity. Adolescence is characterized by significant brain development, particularly frontal and limbic regions implicated in the activation and regulation of stress and emotion (Guyer et al., 2016). Compared to children and adults, adolescents show a unique attunement both to socially appetitive stimuli (e.g., images of social acceptance; Perino et al., 2016) and to negative social evaluation (e.g., exclusion; Somerville, 2013), suggesting enhanced affective processing within social contexts. Further, puberty encompasses a cascade of hormonal changes that have an organizing effect on neural regions implicated in socio-affective processing (Guyer et al., 2016; Pfeifer & Allen, 2021). Sex hormones have been shown to increase amygdala activation to affective social stimuli, and to alter connectivity between fronto-limbic regions, supporting the role of biological development in social-related stress and emotion regulation (Scherf et al., 2013). While many social transitions are common to a chronological stage of adolescence (e.g., the transition into middle school), there is substantial between-person variability in the timing of biological changes (Mendle et al., 2019). This variability likely contributes to differential sensitivity to stressors within the larger social environment, highlighting the importance of biological development for understanding links between social sensitivity and adolescent adjustment.

Heightened social attunement and salience across adolescence is likely adaptive for the successful navigation and integration into new peer networks (Nelson et al., 2016), but might also increase vulnerability to anxiety in teens who have difficulty coping with these developmental transitions. Given that an increase in sensitivity does not lead to elevated anxiety in *all* teens, there is a need to better understand how variation in pertinent developmental contexts might elevate risk (Pfeifer & Allen, 2021). The present study built upon the salience of social and biological transitions in adolescence to examine how peer problems and perceived pubertal stage each moderated the association between physiological sensitivity to social stress and concurrent anxiety outcomes.

Anxiety in Adolescence

Anxiety can be conceptualized and measured in multiple ways, spanning from normative variation in anxious tendencies to more severe disorders that impact only a smaller segment of the population. Some degree of anxiety in response to stress is considered an adaptive response, alerting the body to danger to prepare for action. Variability in this tendency to become anxious can be conceptualized as trait anxiety. Relative to those lower in trait anxiety, those with higher levels are more likely to experience frequent and more intense bouts of state-level (e.g., momentary) anxiety and worry in the presence of stress, as well as heightened attentional bias toward threatening stimuli (Bar-Haim et al., 2007; Eysenck, 2000; Eysenck & Berkum, 1992). Within the context of adolescence, high trait anxiety might enhance developmental changes in sensitivity and increase vulnerability to processing a wide range of situations as threatening. Further, individuals high in trait anxiety are thought to have enhanced long-term memory and recall of threat-related information (Mitte, 2008; Reidy & Richards,

1997). These biases facilitate and are further strengthened by increased worry even in the absence of present threat (Mitte, 2008; Reidy & Richards, 1997). Overall, trait anxiety is considered an enduring personality characteristic that exists along a continuum, such that those with a higher predisposition might display worse anxiety outcomes in adolescence.

Rates of anxiety disorders increase into adolescence, with symptoms that often cluster into specific subtypes (Beesdo et al., 2009; Kessler et al., 2012). One subtype that is particularly pertinent to this developmental stage is social anxiety, which describes feelings of apprehension, fear, and distress around social situations (Beesdo et al., 2009). While trait anxiety is thought to capture typical worry and responsivity across a broader range of situations, social anxiety manifests specifically within interpersonal contexts. For example, adolescents with social anxiety disorder report high levels of distress and avoidance when working with a group, answering questions in class, and initiating conversations (Rao et al., 2007). Social anxiety disorder and its associated symptoms often worsen or increase in early adolescence, as some teens might begin to interpret typical social-contextual changes and challenges as threatening or ambiguous (Tillfors et al., 2012). Overall, elevated anxiety in adolescence has been associated with higher life stress, poorer social relationships, and lower life satisfaction into adulthood (Essau et al., 2014). These long-term implications highlight the teenage years as a potentially impactful window for prevention and intervention.

Anxiety phenotypes are rooted in a dysregulated response to stress or threat, pointing to the importance of how *biological* stress response systems contribute to symptom profiles. Although the contextual and behavioral manifestations of anxiety vary between persons, the underlying physiological stress response might serve as a

transdiagnostic risk factor. Yet, given the wide range of developmental changes that take place across adolescence, a sole focus on physiological risk offers only a small portion of the larger picture. Instead, it is the interactions between various risk factors, assessed at multiple levels of analysis, that allow for rich descriptive models of psychopathology vulnerability (Beauchaine & Gatzke-Kopp, 2012). The present study considered interactions across multiple domains of biological and social-contextual functioning that are in flux across the transition into adolescence. Specifically, I examined how interactions between cortisol functioning and both puberty and peer functioning were associated with concurrent trait anxiety levels and social anxiety disorder.

The Stress Response System

The stress response comprises intersecting influence from the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenal (HPA) axis (Bauer et al., 2002). These neuroendocrine systems are distinguished not only by anatomical features, but by the timing and duration of response to psychosocial challenge (Del Giudice et al., 2011). The sympathetic system is activated most quickly, driving a "fight-or-flight" response that increases heart rate, respiratory rate, and blood flow to prepare the body for immediate action. Through direct innervation of multiple tissues and organ systems throughout the body, sympathetic nerve fibers facilitate rapid – although brief – physiological changes in response to stress (Bauer et al., 2002).

Following rapid engagement of the sympathetic system, the HPA axis mounts a slower-acting and longer-term response to the same stressor (Adam et al., 2007). Increased HPA activation culminates in the release of cortisol, which occurs alongside a cascade of additional hormonal and behavioral responses that aid in physiological

mobilization, as well as negative feedback that facilitates recovery and a return to homeostasis. The HPA axis is activated most strongly to unpredictable and uncontrollable stressors (Dickerson & Kemeney, 2004). These can include threats to the physical self (e.g., safety, survival) that generate a fear response, but also threats to one's *social* self. Social self-preservation theory suggests that situations with the potential to harm one's social standing or acceptance elicit decrements in self-esteem, self-worth, and – importantly – increases in cortisol (Gruenewald et al., 2004; Kemeny et al., 2004). Experimental work supports that cortisol elevations are highest in response to tasks with a social-evaluative stressor (e.g., presence of an evaluative audience during a public speaking task; Dickerson & Kemeney, 2004). This response is increased even further when social evaluation is paired with aspects of uncontrollability (e.g., receiving false feedback of poor performance). Thus, the HPA axis is responsive to one's social surroundings and experiences, to which adolescents are particularly attuned and sensitive (Somerville, 2013).

Cortisol and Anxiety Outcomes

The HPA axis can be measured at multiple time points, each capturing a unique aspect of function. Firstly, basal activity captures levels of arousal when unstimulated, and is often measured at a single time point during rest within a laboratory or home setting (Adam et al., 2007). By accounting for the underlying circadian rhythm present in cortisol secretion (e.g., controlling for time of day), baseline can be considered a measure of typical HPA function. While moderate levels of cortisol are widely adaptive for cognition and behavior (e.g., Suor et al., 2015), chronic overproduction of cortisol – resulting in higher levels at baseline – has been associated with affective disorders in

youth (Lopez-Duran et al., 2009). Theoretical and empirical work suggests that higher baseline cortisol reflects a tendency towards behavioral withdrawal and inhibition, as well as increased sensitivity to punishment (Blair et al., 2004; Buss & Qu, 2018; Kagan et al., 1997). Behavioral inhibition maintains vigilance for unexpected stimuli or events, and directs attention towards social threat (Gray & McNaughton, 2000; Roelofs et al., 2007). These behavioral mechanisms might underlie the link between baseline cortisol and anxiety outcomes. Indeed, elevated basal cortisol measured within a laboratory setting has been found to predict greater concurrent general and social anxiety symptoms in adolescent girls (Schiefelbein & Susman, 2006).

Considering change from baseline levels within a laboratory setting, a normative cortisol response to acute stress forms a U-shaped curve across time. Cortisol levels increase from baseline and peak approximately 20-30 minutes following the onset of a stressor, reflecting mobilization of resources that are important for regulating a range of physiological functions (Adam et al., 2007). This peak is followed by a decline to re-establish homeostasis within 40-60 minutes. Patterns of moderate reactivity are considered necessary for effective self-regulation, cognitive functioning, and engagement with the environment (Blair et al., 2005). For example, acute increases in cortisol from baseline have been associated with less socially anxious behaviors (e.g., facial fear expressions, shaking voice) observed in-the-moment during a social stress task, potentially reflecting mobilization of effortful stress regulation (Perry et al., 2022). However, previous work supports various forms of “dysregulated” cortisol responding, to include larger or smaller initial peaks and slower post-stressor recovery in those higher in

psychopathology symptoms relative to those lower in symptoms (Alink et al., 2008; Fiksdal et al., 2019; Lopez-Duran et al., 2009).

Beginning with a pattern of exaggerated responding, higher levels of internalizing symptoms and related ruminative tendencies have been associated with elevated cortisol reactivity to social stress in early adolescence (Poon et al., 2016). van West et al. (2018) found that trait-level anxiety was associated with greater cortisol output during a social stress task across a sample of children aged 6-12 years, with socially anxious children displaying higher cortisol relative to those low in social anxiety. This profile of HPA hyperresponsivity has been linked to increased avoidance behaviors toward social threat (e.g., in response to angry facial stimuli; Roelofs et al., 2009), likely increasing acute affective responding to, and withdrawal from, stressful stimuli. Thus, cortisol might play a role in facilitating threat avoidance that underlies and reinforces anxiety symptoms.

Next, patterns of hyporeactivity have also been associated with difficulty regulating behavior, attention, and emotion (Ayer et al., 2013). In a sample of pre- and young adolescents aged 8-14 years, those with an anxiety disorder displayed lower total cortisol output across a social stress task relative to healthy controls (Stadelmann et al., 2018). Similarly, despite higher subjective reports of anxiety and fear during a social stress task, young adult participants who met diagnostic criteria for clinical or subclinical social anxiety were found to display lower levels of cortisol at baseline and during social stress relative to those who reported low social anxiety (Beaton et al., 2006; Crisan et al., 2016). This pattern of hyporesponsivity might be specific to those with more chronic or severe symptoms, reflecting a less flexible physiological system that is unable to effectively respond to repeated stressors, or an inability to curb sympathetic arousal via

the HPA-mediated negative feedback loop (Booij et al., 2013; Siess et al., 2014). To begin testing this theory, the present study will examine trait anxiety symptoms – which are often normally distributed and capture the full continuum of anxiety – and a social anxiety diagnosis, which captures only severe and context-specific symptoms. Perhaps those with anxiety that manifests most strongly to social stress will display lower cortisol baseline and reactivity, serving as a protective adaptation to the more persistent and frequent anxious arousal they experience within social contexts – similar to the stressful social environment constructed in laboratory settings. Hyperresponsivity might serve to reinforce and maintain the more stable, trait-level anxiety symptoms, facilitating the regulation of negative affect and stress across various contexts through more maladaptive coping strategies (e.g., behavioral avoidance).

Despite evidence that physiological function is associated with anxiety outcomes, it is only one component of risk within the larger developmental context. Stress response systems are thought to mediate openness of the individual to their environment, with contextual information feeding back in to calibrate the stress response system itself (Boyce & Ellis, 2005; Del Giudice et al., 2011). This conditional adaptation suggests that a child's surrounding social environment plays an important role in determining whether certain physiological profiles contribute to maladjustment – for example, a pattern of high social sensitivity might contribute to poorer emotional and behavioral outcomes within a negative social environment but facilitate better developmental outcomes within a positive environment (e.g., Rudolph et al., 2011). Thus, individual differences in patterns of social stress sensitivity should be considered alongside the developmental stages and contexts in which these systems are situated and engaged. Given the salience

of peer relationships in adolescence, the present study will consider how the association between HPA functioning and anxiety might vary according to the degree of peer problems experienced within their surrounding social context.

Peer Context

The HPA axis is strongly regulated by social relationships, with substantial evidence supporting the role of warm, positive social interactions in fostering adaptive HPA development across early life (Adam et al., 2007). Shifting into adolescence, social goals and motivations begin to orient away from caregiver attachment and towards a focus on peer relationships (Nelson et al., 2016). This re-orientation aligns with increases in social sensitivity during pubertal development, when difficulties with peer relationships become particularly salient. Poor social evaluation can lead to general social rejection and exclusion (e.g., lower social status), as well as more targeted threats to well-being (e.g., victimization) – all of which run counter to the updated social goals of the adolescent developmental stage (Prinstein & Giletta, 2016). Considering this amplified salience, researchers should consider the quality of the social context in which adolescents are engaging regulatory processes.

Social relationships come with innate challenges and stressors that activate the HPA axis, with dysregulated responsivity likely contributing to anxiety symptoms. For example, in a college-aged sample, individuals high in social anxiety displayed flatter declines in cortisol (e.g., overall elevated levels) while completing a self-disclosure task with an unknown partner, as well as decreased levels of self-reported closeness with their partner (Ketay et al., 2019). Elevated cortisol during stressful social interactions might contribute to the maintenance of social anxiety, and reciprocally contribute to problems

with peer relationships. Further, Rudolph et al. (2011) found that, specifically within the context of high peer victimization, heightened anticipatory HPA activity prior to interacting with an unfamiliar peer was associated with increased depression and ruminative responding to social challenge in children. This hypervigilance likely increases negative affect to perceived or actual social threat in everyday life. Interestingly, within the context of lower victimization, high levels of cortisol were protective, potentially mobilizing effective self-regulation and positive rather than negative engagement with the surrounding social environment. It should be noted that mental and physical health problems have also been associated with cortisol *hypo*reactivitiy within the context of high peer victimization, though these findings might be specific to samples with more chronic and severe forms of bullying (Knack et al., 2011; Ouellet-Morin et al., 2011) – more severe than the peer experiences captured in the present study. Overall, the emotional and behavioral implications of physiological processes are likely contingent upon the social context in which these systems are often engaged.

The Role of Puberty

Given developmental trends in mental health problems, increased efforts have been made to understand specific characteristics of adolescence that might drive the onset and intensification of psychopathology. Pubertal increases in hormones – initiated by the hypothalamic-pituitary-gonadal (HPG) axis – are a central component of adolescent development that likely interplay with the HPA axis, emphasizing the need for a "dual-axis" approach to research (Shiftcliff et al., 2015). Puberty is a protracted process that reflects the maturation of internal reproductive (e.g., ovaries, testes), neuroendocrine, and

brain function that influence social and emotional behaviors (Dorn & Biro, 2011). However, puberty is most frequently measured through external, observable characteristics, including breast, genital, and pubic hair growth. These characteristics have traditionally been used to categorize teens into discrete stages from pre-pubertal (e.g., no visible signs of puberty) to post-pubertal (e.g., full physical maturation). Interactions between pubertal stages and cortisol responsivity to social stress have been studied minimally in relation to anxiety (Marceau et al., 2015), leaving a gap in the literature to be explored.

Evidence from both rodent and human studies suggest that functioning of the HPA axis changes from childhood into adolescence (Shirtcliff et al., 2015). Beginning briefly with general age-related change – adolescents display greater cortisol reactivity to social stress relative to younger children (Tottenham & Galván, 2016), while older adolescents display greater reactivity and slower recovery in comparison to younger adolescents, potentially signifying the mediating role puberty (Ji et al., 2015). Indeed, emerging work points to the effects of pubertal stage over and above the effects of age. Cross-sectional work suggests that cortisol baseline and reactivity to psychosocial stress increases with pubertal maturation (Gunnar, Wewerka, et al., 2009; Sumter et al., 2010). Longitudinally, van den Bos et al. (2014) found that cortisol reactivity to social stress was associated with between- and within-person differences in self-reported pubertal stage after controlling for age. Cortisol reactivity increased with puberty progression, reflecting heightened physiological sensitivity to social evaluation. These findings emphasize the adaptive and normative process of increasing social sensitivity across adolescence, which likely facilitates shifts from caregiver-focused attachment to peer-orientation integration.

Yet, these typical maturational processes might exacerbate or contribute to anxiety in a smaller subset of teens who struggle to navigate the range of developmental transitions that take place at this age.

Incorporating the role of psychopathology, Hankin et al. (2010) found that pubertal stage was associated with developmental shifts in cortisol responsivity in dysphoric youth, dichotomizing their sample into pre-pubertal (e.g., no development) and post-pubertal (e.g., completed development) groups based on the self-reported Pubertal Development Scale. Alongside slightly elevated cortisol levels at baseline, pre-pubertal children displayed hyporeactivity (e.g., a decrease from baseline) to psychosocial challenge relative to healthy age-matched controls. Post-pubertal youth displayed hyperreactivity relative to controls, with an exaggerated increase from baseline. Similarly, Colich et al. (2015) examined the interaction between cortisol reactivity and continuous pubertal development, using the self-reported Tanner Staging to group girls from stage 1 (pre-pubertal) to 5 (post-pubertal). Results indicated that cortisol hyporeactivity to social stress prospectively predicted onset of depression in early pubertal girls (below Tanner Stage 2.40) relative to those without a depressive episode, while hyperreactivity predicted onset for girls who were later in pubertal maturation (above Tanner Stage 4.28). Baseline cortisol was similar between groups. These findings suggest divergent psychobiological profiles at different salient stages of development. For post-pubertal adolescents who display hyperreactivity – high social sensitivity is likely closely associated with internalizing symptoms at this stage. Psychosocial stressors become more frequent and salient into adolescence, and negative self-evaluation increases with advancements in pubertal maturation (Pfeifer et al., 2021). Conversely, for

pre- and early-pubertal adolescents – the onset of depressive symptoms at this earlier stage in life might reflect atypically high, chronic stress that contributes to a switch towards protective HPA attenuation over time (e.g., Trickett et al., 2010).

Finally, pulling from the limited work that has investigated anxiety-specific outcomes, results are inconsistent with those reported by Hankin et al. (2010) and Colich et al. (2015). van den Bos et al. (2017) estimated puberty with the Pubertal Development Scale, finding that higher social anxiety was associated with elevated cortisol reactivity to social stress at earlier pubertal stages (estimated at stage 1.5 out of 4). This effect inverted at later pubertal stages (estimated at stage 3.6 out of 4), such that lower cortisol reactivity was associated with higher social anxiety. Altogether, though normative puberty-related increases in cortisol responsivity might be exacerbated in some teens at-risk for depression, this developmental effect could be inverted towards a blunted response for those with elevated and/or more chronic anxiety symptoms, reflecting a less-flexible stress response system (Siess et al., 2014). Given the social nature of the stressor tasks used across research studies, a lowered cortisol response in more mature socially anxious youth could also reflect an allostatic response to stressful social situations over time – situations that might be less salient for those with more recent depressive symptoms. The present study will consider multiple measures of anxiety to potentially disentangle correlates of variation in more general trait-level anxiety symptoms from more context-specific social anxiety diagnoses.

Current Study

The present study examined the associations between cortisol baseline and reactivity to social stress and concurrent anxiety outcomes in a diverse sample of

adolescents, as well as the moderating role of peer functioning and perceived pubertal status. Incorporating these contextual factors might help to explain previous mixed findings or uncover more robust links between cortisol and anxiety. Given the increase in anxiety symptoms and disorders at this age, the present study examined both trait-level anxiety and social anxiety disorder.

First, I hypothesized that elevated cortisol at baseline or in response to a social stressor would be associated with higher concurrent anxiety levels. It should be noted that participants with high cortisol values at baseline are less likely to also display a greater cortisol increase to a stressor task (e.g., law of initial values), highlighting the importance of including both resting and reactivity measures to fully parse what component of the stress response might be driving effects. Additionally, the present study explored whether findings would vary depending upon the type of anxiety examined. I hypothesized that trait anxiety would be associated with cortisol hyperactivity, and youth with elevated social anxiety would display hypoactivity. While trait anxiety captures more global symptoms measured along the full continuum of severity, social anxiety diagnoses capture only those with more severe and context-specific symptoms, where protective lowering of the stress response to a social-specific stressor might be more likely (Siess et al., 2014). Finally, I hypothesized that the association between cortisol baseline or reactivity and each anxiety type would be more robust for those who are, 1) higher in peer problems and 2) at a more advanced perceived pubertal stage. These intra- and inter-personal factors have been linked to heightened social sensitivity and anxiety in adolescence.

Methods

Participants

Data were collected as part of the Family Life Project (FLP), a longitudinal, epidemiological study examining the effects of rural poverty on child development. Briefly, 1,292 families in regions of Pennsylvania (n = 519) and North Carolina (n = 773) were recruited through local hospitals at the time of childbirth. Low-income families in both states and African American families in North Carolina were oversampled to ensure adequate power for longitudinal analyses of psychosocial risk. Research assistants conducted home visits when the children were approximately 2, 6, 15, 24, 36, 48, 60, 90 months, and 13 years of age. Visits included interview and questionnaires completed by the caregiver, child assessments, and videotaped interactions between the child and caregiver(s).

The present study utilized physiological and survey measures collected at the adolescent visit. Full data were available for n = 686 adolescents (M_{age} = 13.15, SD = .54, range = 12.52 to 15.09; 49.7% female). This subsample was comprised of 55.5% White, 43.4% Black or African American, and < 1% each of Pacific Islander, American Indian or Alaska Native, and Asian participants. The original full sample did not significantly differ from the present subsample in sex ($\chi^2(1) = .03$, $p = .86$) or racial/ethnic group (tested as White vs. Black/African American combined with the three smaller groups; $\chi^2(1) = .12$, $p = .73$). Income-to-needs ratio did not significantly differ between the full sample at 6 months and our present adolescent subsample (t = -.04, p = .97).

Procedure

Families were visited for at-home data collection procedures over a 2.5-year window when the target child was between approximately 12 and 15 years of age. Home visits lasted around 2 to 3 hours in total. Primary caregivers were asked to complete a range of questionnaire measures about family demographics and child behavior. Adolescents completed questionnaires alongside multiple computerized tasks that measured aspects of executive function and self-regulation. These tasks included a modified, home-based version of the Trier Social Stress Task (TSST; DeJoseph et al., 2019; Kirschbaum et al., 1993; Yim et al., 2010), which is the focus of the present study. The TSST has been shown to elicit increased stress and arousal in both adult and youth samples (Gunnar, Talge, et al., 2009; Yim et al., 2015). See Figure 2-4 for an illustration of the TSST protocol and salivary sampling timeline.

Research assistants were trained to evaluate each home environment to find an open but quiet room in which to conduct the adolescent assessments. This space was often the kitchen/eating area, typically in a separate room and out of direct sight of the living room where the primary caregiver completed their portion of the experiment. At the beginning of the home visit, research assistants unpacked study materials and chatted with the teen for the first 10-15 minutes. Participants were then outfitted with heart rate and blood pressure monitors (not examined in the present study), provided with a *National Geographic* book, and instructed to relax during the baseline phase. An initial 5-minute heart rate baseline recording was conducted, followed by collection of the first saliva sample and blood pressure measure (time = 0). Teens then responded to a series of

questionnaires about general health and medications. After receiving initial task instructions (described below), participants completed the TSST.

Immediately after the TSST, a 5-minute post-heart rate measurement was collected, followed by saliva and blood pressure samples (time = 1). Participants were then debriefed on deception within the task. Additional saliva and blood pressure samples were collected 20-minutes (time = 2) and 40-minutes (time = 3) post-task as participants quietly completed additional home visit questionnaires. At the time of collection, all four saliva samples were time-stamped by experimenters within the data entry software *Blaise*. Time stamps were used to estimate intervals between saliva samples and session time of day.

Trier Social Stress Test

The TSST included a three-minute preparation period and five-minute free speech task, followed by a five-minute math task. Participants completed the task in front of a sham panel of older teenagers (aged 14-16). Though the panel was a pre-recorded video, participants were told they were conducting a live video conference with teen researchers using NYU's "Blue Jeans" system. Each panel consisted of two adolescents matched to the participant's own gender, with one Black and one White member so all participants had one racially matched panelist. Panelists were seated at a table in white laboratory coats, displaying expressions of disinterest and boredom (e.g., yawning, playing on their mobile phone) while occasionally taking notes. The recording was presented on a laptop with a built-in webcam, placed approximately three feet away from the participant.

Research assistants read instructions verbatim to each participant to ensure standardization across sessions. Before beginning the preparation period and speech task,

teens were told that they were to introduce themselves as they would to a new class in school. Specifically, they were asked to say at least one good thing and one bad thing about themselves, with the goal of convincing the class that they are likeable and a good student. Participants were told that teen researchers at NYU were going to listen and take notes, and that their speech would be videotaped and recorded for later analysis by experts. During the three-minute preparation period, participants were allowed to take notes and write down things to talk about on paper, but could not use their notes during the talk. After three minutes were up, the participant was asked to stand and face a laptop displaying the sham panel, and to begin their speech. During the speech phase, if the participant paused for 20 seconds, the research assistant began asking a list of outlined questions (e.g., "Tell us the three best things about you," "Do you like being part of a team?"). If participants spoke freely for four minutes, they were interrupted at an appropriate juncture and asked the same list of questions.

Following their speech, participants completed a five-minute math task involving serial subtraction. The sham panel was not shown during the math task given extreme levels of participant distress during the pilot, though their performance was recorded. Participants were instructed to go as quickly as possible while maintaining accuracy, and were corrected and told to start over from the beginning following a mistake. At specified points throughout the task, research assistants asked the participant to "answer a little faster." All participants were instructed to start at 1027 and subtract by 5 until they reached zero. If they could not get past three to four correct responses, participants were moved down to an easier level after one minute (e.g., subtracting 3's from 200). If they

were answering the problems quickly and easily, they were moved up to a more difficult level after one minute (e.g., subtracting 6's from 1000).

Measures

Salivary Cortisol

Four saliva samples were obtained from each participant using the Salimetrics passive drool collection method. Samples were assayed in duplicate using a highly sensitive enzyme immunoassay through ELISA kits (Salimetrics, Carlsbad, CA). The tests used a sample volume of 25 μL per determination, a sensitivity lower limit of 0.007 μg/dL, and a standard curve ranging from 0.012 to 3.0 μg/dL. The intra- and inter-assay coefficients of variation were less than 10% and 15%, respectively. Preliminary descriptives were assessed to identify outliers in cortisol values above 3 SD from the sample-level mean (n = 9 samples were removed). Initial distributions were highly kurtotic and positively skewed, so all values were square root transformed after outlier removal to normalize distributions.

Cortisol values significantly declined from baseline to 40-minutes post-TSST (γ = -.02, $p < .001$), with no significant quadratic effect (γ = -.00, p = .20). This averaged linear trend is depicted in Figure 2-5 alongside a subsample of person-level trajectories to highlight substantial variability in cortisol reactivity patterns. To help capture this variability, cortisol was examined using two measures: baseline (t = 0) and a reactivity change score from baseline to 20-minutes post-TSST ($t_2 - t_0$). The 20-minute saliva sample was chosen based on previous work denoting peak cortisol values approximately 20-30 minutes following stressor onset, followed by a return to pre-test levels within 40-60 minutes (Adam et al., 2007). A positive reactivity score indicates an increase in

cortisol from baseline in response to the stressor task, while negative scores indicate a decrease. Reactivity scores ranged from -.50 to .26 (M = -.04, SD = .09), and approximately 26% of the present sample displayed an increased in cortisol from baseline to 20-minutes post-task.

Finally, despite the task protocol, there was inevitable variability in the time intervals between saliva samples collected. Although active task time between baseline and 20-minutes post-TSST – capturing the speech preparation period through the theoretical peak of cortisol values following the social stressor – should have been approximately 33 minutes (see Figure 2-4), this does not directly account for variation in how long it took each RA to explain instructions, answer questions, and navigate any technical issues. According to time stamps collected at the start of each saliva sample, timing intervals between samples collected at baseline and 20-minutes post-TSST varied from 31 to 89 minutes (M = 51.54, SD = 7.20). Longer time intervals were significantly correlated with more negative cortisol reactivity scores (r = -.12, p = .002). Therefore, timing intervals were included as a covariate in all models.

Trait Anxiety

Adolescents completed the State Trait Anxiety Inventory for Children (STAIC; Spielberger et al., 1973). This measure is divided into two subscales: state anxiety that captures transient emotional states characterized by increased tension and arousal, and trait anxiety that captures disposition or proneness towards anxious responding. The present study uses the 20-item trait anxiety subscale. Participants are asked how each item describes how they usually feel, rated from 1 (*hardly ever*) to 3 (*often*). Example items include “I worry about making mistakes” and “I notice my heart beats fast.” Item

scores are summed together, with higher total scores reflecting greater trait anxiety (Cronbach's $\alpha = 0.89$). Trait anxiety was normally distributed within the present sample (M = 35.31, SD = 7.71; skew = .48, kurtosis = -.09), with scores extending across the full possible range of severity from 20 to 60.

Social Anxiety

Research assistants conducted the structured Diagnostic Interview Schedule for Children (DISC-IV; Shaffer et al., 2000) with each child's primary caregiver. This measure has demonstrated acceptable reliability and validity for both parent- and child-report versions (Shaffer et al., 2000). The DISC uses diagnostic criteria specified by the DSM-IV to identify 30 different childhood psychiatric disorders. The present sample was only measured on a subset of 5 disorders, including social anxiety. The DISC generates categorical social anxiety diagnoses, as well as symptom and criterion counts. Consistent with prevalence rates in adolescent populations (Beesdo et al., 2009), *n* = 26 adolescents (3.8%) in the present sample were diagnosed with a clinical social anxiety disorder, and *n* = 100 (14.6%) were classified as having a sub-threshold disorder. Those with clinical and subthreshold disorders were combined into one group for present analyses, referred to as having an "elevated" social anxiety diagnosis from here on.

Perceived Pubertal Stage

Pubertal stage was assessed using the self-reported Tanner Staging questionnaire (Marshall & Tanner, 1969, 1970). Teen self-report of pubertal stage has been shown to correlate with physician examinations (Shirtcliff et al., 2009). This measure uses schematic line drawings of secondary sex characteristics to categorize individuals across an ordinal scale from pubertal stage 1 (pre-pubertal) to stage 5 (post-pubertal). Boys were

shown five images that displayed pubic hair maturation in combination with genital maturation, with depictions of the size and proportion of penis, testes, and scrotum as observed in early childhood up to adulthood. Girls were shown five images of progressive breast maturation and five images of progressive public hair development (e.g., from no hair to hair that is adult-like in quantity and location). Given that boys in the current sample were not directly asked to classify their genital and pubic hair stages separately, present analyses only used breast development for girls to maintain consistency in single-item gonadal measurement between sexes. Participants were asked to circle the drawing that most closely resembled their own stage of development. The present sample spanned all five pubertal stages (M = 3.75, SD = 1.02). Most adolescents (60.8%) reported stage 4 or 5, while 11.6% reported stage 1 or 2. It is important to highlight that each pubertal stage encompassed a wide range of ages – for example, stages 1 and 2 ranged from age 12.6 to 14.5 years, while stages 4 and 5 ranged from age 12.5 to 15.1 years (see Figure 3-1). These distributions emphasize the importance of incorporating puberty as a developmental mechanism that is distinct from age-related change. Additionally, given limitations in self-report, the present Tanner stages are more likely to represent *perceived* pubertal stage, capturing important subjective psychosocial aspects of self-perception in addition to more objective markers of pubertal maturation (Dorn & Biro, 2011; Mendle, 2014). For the present analyses, perceived pubertal stage was trichotomized into pre/early (Tanner Stages 1-2; n = 79), mid (Tanner Stage 3; n = 189), and late/post-pubertal (Tanner Stages 4-5; n = 415).

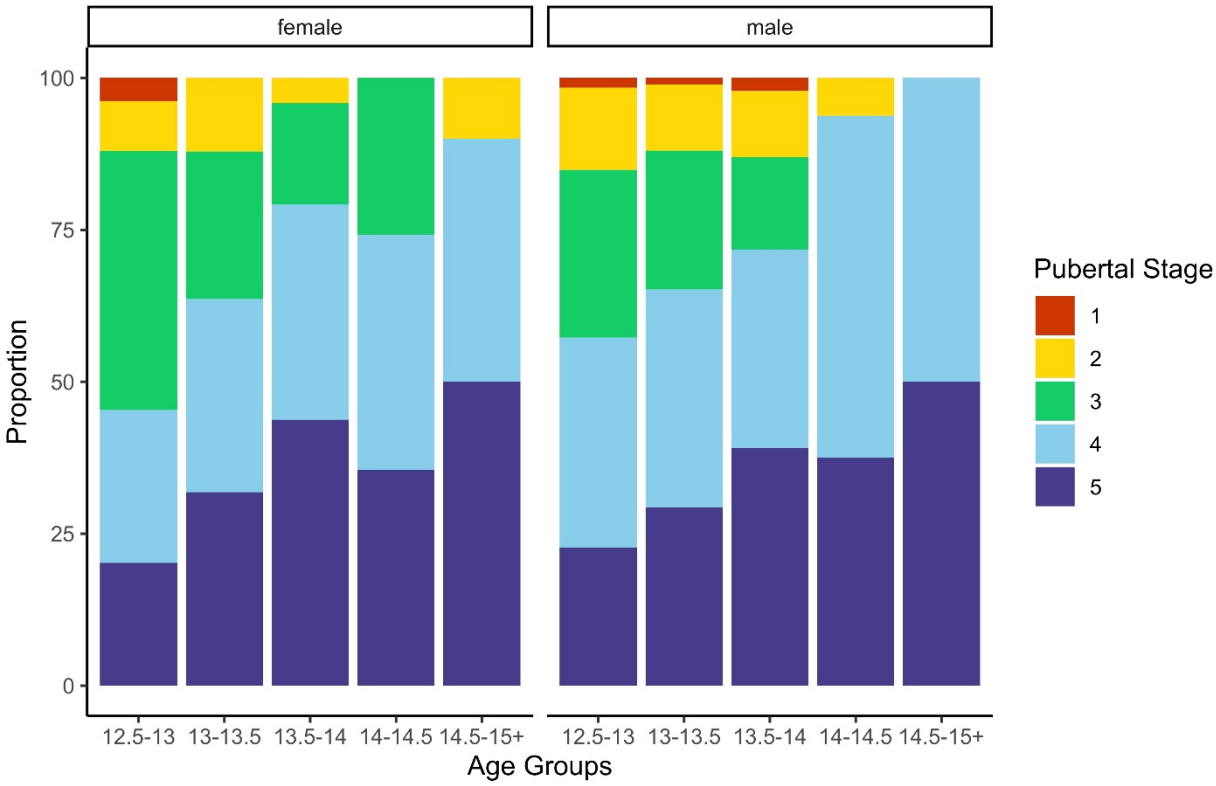

Figure **3-1**: Variability in Perceived Pubertal Stage Across Age

Note. Proportion of each pubertal stage represented in incremental age groupings, broken down by sex. Age is grouped from 12.5 to 15+ years in increments of .5 years. Note the spread and variability in pubertal stage across age.

Peer Problems

Primary caregivers completed the social problems subscale of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). This scale comprises 5 items rated on a scale from 0 (*not true*) to 2 (*certainly true*). Items ask about the child's behavior over the past six months. Example items include: "rather solitary, prefers to play alone," "has at least one good friend," and "picked on or bullied by other children." Values are

averaged for each child, with higher scores reflecting more peer problems (Cronbach's α = 0.60). Peer problems were relatively low in the present sample (M = .34, SD = .35), though the more upper end of severity was represented (range = 0 to 1.6).

Health and BMI

As part of the saliva sampling protocol, participants were asked a series of self-report questions about their general health, behaviors, and medications. Teens were asked to report how many hours they had slept the night prior to the home visit (M = 7.88, SD = 2.68), if they had taken any medications in the past 24 hours (yes = 185, no = 501), and how they would rate their health over the last two days compared to other same-age children (from 1 = *excellent* to 5 = *poor*; M = 2.52, SD = .99). Additionally, teens were asked to report the number of hours that had passed since they last had something to eat or drink other than water (M = 2.60, SD = 3.13).

Finally, participants' height and weight were measured once by trained research assistants. Heavy clothing and shoes were removed prior to measurement. Weight (kg) and standing height (cm) were used to calculate age- and sex-specific body mass index based on Centers for Disease Control and Prevention guidelines (Kuczmarski et al., 2002). BMI values were z-scored in the present study (M = .86, SD = 1.19).

Covariates

Associations between primary study measures and demographic and session-specific variables were examined to determine the inclusion of covariates. Race/ethnicity was re-coded to combine Black participants with those in the three smaller racial groups. Cortisol reactivity and anxiety varied by race, such that White participants displayed higher reactivity (t = 3.90, p < .001) and lower trait anxiety (t = -3.27, p = .001) relative

to those in minoritized racial groups. In terms of sex, males displayed significantly lower anxiety (*p*s < .03) and lesser cortisol reactivity ($t = 2.06$, $p = .04$) relative to females. Age was positively correlated with baseline cortisol ($r = .12$, $p = .001$) and trait anxiety ($r = .10$, $p = .01$). Finally, pubertal stage was positively correlated with physical assessment of body mass index (BMI; $r = .22$, $p < .001$).

Considering confounds related to session timing: cortisol measures were not associated with hours slept the previous night (*p*s > .88), medications taken in the previous 24 hours (*p*s > .09), or self-reported health (*ps* > .44). Cortisol values at baseline were positively correlated with the number of hours prior to the session that the participant had food or drink ($r = .11$, $p = .005$). Cortisol baseline significantly decreased ($r = -.38$, $p < .001$) and reactivity increased ($r = .19$, $p < .001$) across the time of day that sessions were held. In summary, significant covariates included in analyses were racial/ethnic group, sex, age, hours since last food/drink, session time of day, and timing interval between saliva samples. Puberty-related analyses were adjusted for BMI.

Data Analysis

Regression models were conducted to examine the associations between adolescent cortisol and anxiety outcomes, to include moderation by peer problems and perceived pubertal stage. Two primary sets of analyses were conducted: (1) trait anxiety symptoms were estimated within a hierarchical regression framework, and (2) elevated social anxiety diagnoses were estimated using bivariate logistic regression. Step one of each model set examined the main effects of cortisol on anxiety, while step two incorporated the interactions between cortisol measures and each moderator. Cortisol

baseline and reactivity change scores were modeled separately, as were interactions with peer problems and pubertal stage.

Results

Descriptive Analyses

See Table 3-1 for initial bivariate correlations and variable descriptives. Briefly, cortisol at baseline and reactivity scores were not significantly correlated with either form of anxiety. Cortisol values at baseline were higher among older children ($r = .12$, $p < .001$), though additional analyses suggested that neither cortisol at baseline ($F(2) = .17$, $p = .85$) nor reactivity ($F(2) = .67$, $p = .51$) varied across the trichotomized perceived pubertal groups after adjusting for age. Both trait and social anxiety were positively correlated with peer problems (rs $> .12$, ps $< .001$), but were not associated with pubertal stage. Trait anxiety was modestly positively correlated with social anxiety ($r = .11$, $p = .003$), with higher symptoms in those with a social anxiety diagnosis ($M = 37.43$, $SD = 7.95$, range = 23-58) relative to those without a diagnosis ($M = 34.95$, $SD = 7.61$, range = 20-60).

Table **3-1:** Bivariate Correlations, Means, and Standard Deviations of Main Study Variables

	1	2	3	4	5	6
1. Cortisol Baseline	–					
2. Cortisol Reactivity	**-.59**	–				
3. Peer problems	.01	-.02	–			
4. Pubertal stage	.02	.04	.05	–		
5. Trait anxiety	-.01	-.03	**.21**	.04	–	
6. Social anxiety	-.08	.05	**.12**	.01	**.11**	–
Mean	.32	-.04	.34	3.75	35.31	.15
SD	.12	.09	.35	1.02	7.71	.35
Range	.08-.93	-.50-.26	0-1.60	1-5	20-60	0-1

Note. N = 686. Cortisol values are square root transformed. Cortisol Reactivity = baseline subtracted by 20-minutes post-TSST. Elevated social anxiety coded as 0 = no diagnosis and 1 = diagnosis. Sex coded as 0 = female and 1 = male. Bolded values indicate $p < .05$.

Primary Analyses

Trait Anxiety

The first set of regression models tested the association between adolescent cortisol and trait anxiety symptoms, to include the moderating effects of peer problems and perceived pubertal stage. In step one of analyses, main effects indicated no significant association between cortisol baseline or reactivity and trait anxiety symptoms (*p*s > .68; Table 3-2). For step two in the first set of moderation models, interactions between peer problems and cortisol baseline and reactivity were not significant (*p*s > .95; Table 3-2).

Table **3-2:** Trait Anxiety and Cortisol: Main Effects and Peer Moderation

	Step One *b (SE)*	Step Two *b (SE)*
Cortisol Baseline:		
Intercept	18.83 (7.79)*	16.59 (7.66)*
Sex	-3.79 (.58)***	-3.88 (.56)***
Race	-1.51 (.59)*	-1.46 (.58)*
Age	.94 (.54)	1.02 (.53)
Session Time	.19 (.12)	.18 (.12)
Eat/Drink Time	-.07 (.09)	-.11 (.09)
Saliva Interval	.08 (.04)	.08 (.04)
Baseline	-.78 (2.56)	-.64 (3.36)
Peer		4.88 (2.24)*
Peer x Baseline		-.41 (6.55)
Cortisol Reactivity:		
Intercept	18.51 (8.01)*	16.29 (7.85)*
Sex	-3.74 (.58)***	-3.86 (.57)***
Race	-1.55 (.61)*	-1.51 (.60)*
Age	.95 (.55)	1.04 (.54)
Session Time	.22 (.12)	.22 (.11)
Eat/Drink Time	-.08 (.10)	-.12 (.09)
Saliva Interval	.07 (.04)	.06 (.04)

Reactivity	-1.48 (3.52)	-1.16 (4.68)
Peer		4.74 (.95)***
Peer x Reactivity		-.53 (9.13)

Note. Sex coded as 0 = female, 1 = male; Race group coded as 0 = Black participants and those in smaller racial groups, 1 = White participants. *** $p < .001$, ** $p < .01$, * $p < .05$

Moving to the second set of moderation models, interactions between perceived pubertal stages and cortisol baseline were not significant (*p*s > .23; Table 3-3). However, the association between cortisol reactivity and trait anxiety was significantly different between those in early relative to mid perceived pubertal stages ($b = 23.36$, $p = .03$; see Figure 3-2). Simple slopes suggested a significant, negative association between cortisol reactivity and anxiety for those in earlier puberty ($b = -17.15$, $p = .04$), such that higher anxiety was observed in those who displayed a greater decline in cortisol from baseline to task (e.g., lesser reactivity). The slope of cortisol on anxiety was not significant for those in the mid pubertal stage ($b = 6.21$, $p = .37$). Similarly, those later in the later pubertal stage displayed no significant association between cortisol and anxiety ($b = -.89$, $p = .85$), though the interaction effect suggested only a marginal difference between the late and early pubertal groups ($b = 16.26$, $p = .09$).

Table **3-3:** Trait Anxiety and Cortisol: Perceived Pubertal Moderation

	b (SE)
Cortisol Baseline:	
Intercept	16.03 (8.35)
BMI	.12 (.25)
Sex	-3.70 (.59)***
Race	-1.44 (.60)*

Age	.98 (.56)
Session Time	.20 (.12)
Eat/Drink Time	-.07 (.09)
Saliva Interval	.08 (.04)
Baseline	6.55 (7.53)
Puberty Mid	3.39 (2.92)
Puberty Late	2.13 (2.69)
Puberty Mid x Baseline	-10.52 (8.74)
Puberty Late x Baseline	-6.90 (8.07)
Cortisol Reactivity:	
Intercept	17.56 (8.18)*
BMI	.10 (.25)
Sex	-3.65 (.59)***
Race	-1.48 (.62)*
Age	.96 (.56)
Session Time	.23 (.12)*
Eat/Drink Time	-.06 (.10)
Saliva Interval	.06 (.04)
Reactivity	-17.15 (8.48)*
Puberty Mid	1.29 (1.16)
Puberty Late	.89 (1.07)
Puberty Mid x Reactivity	23.36 (10.95)*
Puberty Late x Reactivity	16.26 (9.55) †

Note. Sex coded as 0 = female, 1 = male; Race group coded as 0 = Black participants and those in smaller racial groups, 1 = White participants. Pubertal stage trichotomized as pre/early (Tanner stages 1-2), mid (Tanner stage 3), and late/post-pubertal (Tanner stages 4-5). The early pubertal group served as the reference group. *** $p < .001$, ** $p < .01$, * $p < .05$, † $p = .09$

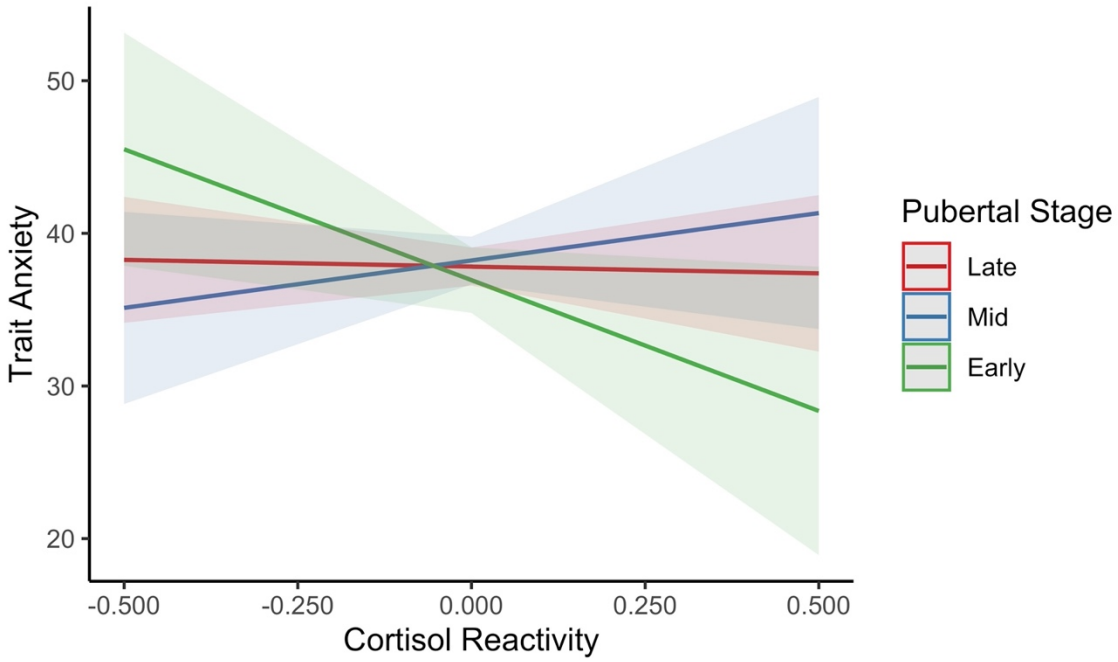

Figure **3-2:** Interaction Between Cortisol Reactivity and Perceived Pubertal Stage Predicting Trait Anxiety

Note. Significant difference in slopes between early (green) and mid (blue) perceived pubertal groups. Marginal difference between early and late (red) groups. For only the early pubertal group, higher trait anxiety is observed in those who display a greater decline in cortisol from baseline to social stressor task.

Elevated Social Anxiety

The second set of primary models examined the association between cortisol measures and an elevated social anxiety diagnosis, to include the moderating effects of peer and pubertal contexts. In step one, the main effects of cortisol baseline and reactivity on social anxiety were not significant (*p*s > .13; Table 3-4). For step two in the first set of

moderation models, interactions between cortisol measures and peer problems were not significant (*p*s > .68; Table 3-5).

Table **3-4:** Social Anxiety and Cortisol: Main Effects and Peer Moderation

	Step One *b (SE)*	Step Two *b (SE)*
Cortisol Baseline:		
Intercept	-3.30 (3.13)	-3.45 (3.19)
Sex	-.50 (.23)*	-.54 (.23)*
Race	-.27 (.23)	-.30 (.23)
Age	.01 (.21)	.00 (.22)
Session Time	.10 (.05)	.10 (.05)
Eat/Drink Time	-.05 (.05)	-.06 (.05)
Saliva Interval	.01 (.02)	.02 (.02)
Baseline	-1.56 (1.13)	-1.76 (1.65)
Peer		0.69 (.86)
Peer x Baseline		0.61 (2.68)
Cortisol Reactivity:		
Intercept	-3.54 (3.19)	-3.71 (3.25)
Sex	-.55 (.23)*	-.61 (.24)**
Race	-.25 (.24)	-.29 (.24)
Age	-.03 (.22)	-.03 (.22)
Session Time	.11 (.05)*	.10 (.05)*
Eat/Drink Time	-.06 (.05)	-.07 (.05)
Saliva Interval	.02 (.02)	.02 (.02)
Reactivity	2.23 (1.48)	3.14 (2.21)
Peer		.87 (.35)*
Peer x Reactivity		-1.48 (3.60)

Note. Sex coded as 0 = female, 1 = male; Race group coded as 0 = Black participants and those in smaller racial groups, 1 = White participants. *** $p < .001$, ** $p < .01$, * $p < .05$

Finally, for the second set of moderation models, the effects of baseline cortisol on social anxiety did not vary between perceived pubertal stages (*p*s > .35; Table 3-5).

However, there was a significant association between cortisol reactivity and the odds of having a social anxiety diagnosis for those in the mid-pubertal group (b = 8.44, p = .008). Interaction effects further indicated that the association between cortisol reactivity and social anxiety significantly varied between mid and both early (b = -10.24, p = .02) and late (b = -7.33, p = .048) pubertal stages. Estimated odds ratios suggested that, for a .10-unit increase in cortisol reactivity, the odds of having an elevated social anxiety diagnosis increased by a factor of 2.33 (95% confidence interval = [1.27, 4.50]) for those in the mid-puberty group. The odds of having a diagnosis decreased by a factor of .84 (95% CI = [0.47, 1.55]) for those in the early pubertal group and increased by a factor of 1.12 for those in the late pubertal group (95% CI = [0.77, 1.64]), though these values were not statistically significant. To help visualize effects, the predicted probabilities were plotted, representing the probability of having a social anxiety diagnosis across cortisol reactivity values for each pubertal stage (see Figure 3-3). However, these findings should be interpreted caution because there were a limited number of participants with an elevated social anxiety diagnosis in each perceived pubertal grouping (early pubertal n = 12, mid pubertal n = 26, late pubertal n = 61), as evidenced by the large standard error represented in Figure 3-3.

Table **3-5:** Social Anxiety and Cortisol: Perceived Pubertal Moderation

	b (SE)
Cortisol Baseline:	
Intercept	-3.25 (3.22)
BMI	.10 (.10)
Sex	-.49 (.23)*
Race	-.21 (.24)

Age	.02 (.22)
Session Time	.11 (.05)*
Eat/Drink Time	-.05 (.05)
Saliva Interval	.01 (.02)
Baseline	-2.91 (2.16)
Puberty Early	.17 (1.16)
Puberty Late	-.70 (.77)
Puberty Early x Baseline	.37 (3.79)
Puberty Late x Baseline	2.35 (2.49)
Cortisol Reactivity:	
Intercept	-4.14 (3.26)
BMI	.10 (.11)
Sex	-.52 (.24)*
Race	-.22 (.25)
Age	.03 (.22)
Session Time	.12 (.05)*
Eat/Drink Time	-.06 (.05)
Saliva Interval	.01 (.02)
Reactivity	8.44 (3.19)**
Puberty Early	-.08 (.44)
Puberty Late	-.26 (.29)
Puberty Early x Reactivity	-10.24 (4.30)*
Puberty Late x Reactivity	-7.33 (3.71)*

Note. Sex coded as 0 = female, 1 = male; Race group coded as 0 = Black participants and those in smaller racial groups, 1 = White participants. Pubertal stage trichotomized as pre/early (Tanner stages 1-2), mid (Tanner stage 3), and late/post-pubertal (Tanner stages 4-5). The mid-pubertal group served as the reference group. *** $p < .001$, ** $p < .01$, * $p < .05$

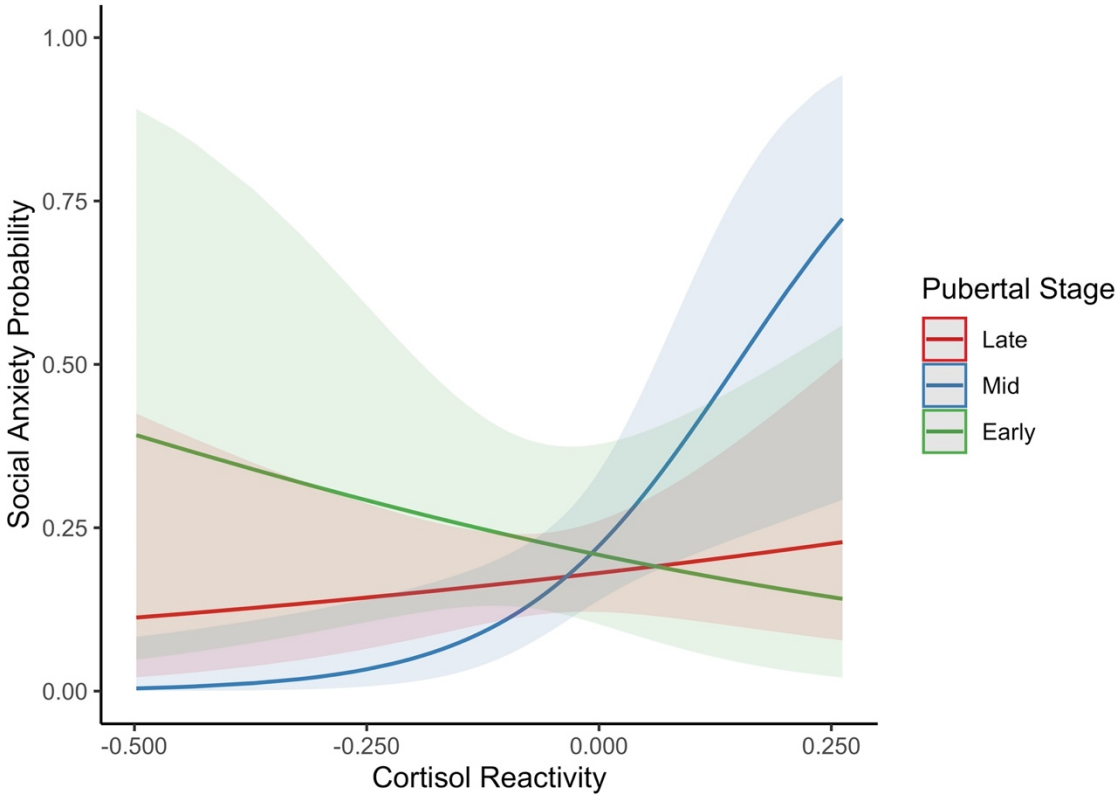

Figure **3-3:** Predicted Probability of a Social Anxiety Diagnosis by Cortisol Reactivity and Perceived Pubertal Stage

Note. There is a significant increase in the probability of a social anxiety diagnosis as cortisol reactivity increases for those who perceive themselves to be in the mid-pubertal stage (blue).

Discussion

Adolescence is characterized by an uptick in time, intimacy, and salience dedicated to peer relationships (Somerville, 2013). Although this shift represents a normative developmental transition, the changing peer landscape often comes alongside

an increase in social stressors, highlighting the importance of adaptive stress responding and coping at this age (Pfeifer & Allen, 2021). Given the importance of the HPA axis for responding to unpredictable social stressors, the present study sought to examine the effects of cortisol baseline and reactivity to social stress on adolescent anxiety, further testing whether these links were moderated by peer problems or perceived pubertal stage. Both trait-level anxiety symptoms and an elevated social anxiety diagnosis were examined to explore the potential role of anxiety specificity and severity. Results indicated no direct associations between cortisol baseline or reactivity and anxiety symptoms. However, lesser cortisol reactivity to the social stressor task was associated with increased trait anxiety for those who reported an earlier perceived pubertal status, while greater reactivity was associated with increased odds of having a social anxiety diagnosis for those who reported a mid-pubertal status. These findings highlight the importance of a developmental and contextual perspective when considering the role of physiological functioning on behavior.

To begin, the present study found no direct link between cortisol baseline or reactivity and concurrent forms of anxiety. Although diverging from our initial hypothesis, these findings do emphasize apparent inter-individual variability in links between psychophysiology and psychopathology. The effects of individual differences in cortisol functioning on behavior may not be inherently beneficial or risky, but are rather dependent upon additional aspects of a person and their environment. For example, despite a similar lack of significant main effects of cortisol on adjustment, multiple previous studies support the association between elevated cortisol reactivity and internalizing symptoms within environments characterized by high stress or adversity,

but lower symptoms within less stressful environments (Obradovic et al., 2010; Steeger et al., 2017). The present study built upon this previous work by emphasizing the developmental aspects of context in adolescence, examining both peer functioning and perceived pubertal stage as contextual moderators.

Peer Moderation

Initial results indicated no significant interaction between peer problems and cortisol with respect to anxiety, which was contrary to previous research on peer victimization (e.g., Rudolph et al., 2011). This null finding might be due, in part, to limitations regarding the present measurement approach. First, our peer problems scale relied upon parent report, though a multi-informant (self, peer, teacher, parent) approach to measuring peer rejection has been found to be both more reliable and a better predictor of children's relational adjustment over time (Ladd & Kochenderfer-Ladd, 2002). Second, the present study used a general measure of peer "problems," which predominantly includes more mild markers of social maladjustment (e.g., "rather solitary, prefers to play alone," "gets along better with adults than with other children"). To offer greater specificity, future work should incorporate and differentiate between additional components of the peer experience (e.g., victimization, rejection, neglect), to include examinations of variation in the chronicity and severity of different forms of mistreatment. Relatedly, physiological sensitivity to social stress might be a more potent predictor of anxiety symptoms for individuals with negative peer experiences who *also* lack the presence of supportive others, emphasizing the importance of considering one's larger social network both within and outside of the peer context (e.g., friends, siblings, caregivers; Hostinar & Gunnar, 2013; Peters et al., 2011). Overall, future research should

pursue a more comprehensive approach to understanding the role of social functioning on cortisol responsivity and risk for anxiety symptoms.

Puberty Moderation

Beginning with trait anxiety, the present study emphasized the importance of perceived pubertal stage in moderating the association between cortisol and behavior, over and above the effects of age. However, the directionality of results was unexpected, as *lesser* cortisol reactivity to social stress was associated with greater trait anxiety for those in the *earlier* pubertal stages. This pattern of hyporeactivity to the TSST might reflect a stress-response system that is unable to engage sufficient resources for adaptive coping, or that lacks the HPA-mediated feedback loop to suppress sympathetic arousal (Siess et al., 2014). In terms of perceived maturation, if an individual views themselves as not yet undergoing puberty, their internal self-evaluations, emotions, and cognitions will be based upon that view (Mendle, 2014). For example, teens might be more likely to interpret negative social cues and stressors as relating to their perceived appearance or degree of maturation. Thus, perceptions of oneself as less physically mature within the context of lowered cortisol reactivity might increase vulnerability for anxiety, particularly as it relates to body image, concern about peer perceptions, and self-esteem.

However, these interpretations should be considered alongside a few study limitations. First, teens were not directly asked about their level of negative affect following the TSST, so it is unclear if our task was truly successful in inducing stress across participants. If a subset of participants did not experience the task as stressful, associations between elevated cortisol reactivity and anxiety might have been obscured across pubertal groups. Second, the present baseline saliva sample was collected

approximately 20 minutes following initial RA arrival at the home. Given the typical 20- to 30-minute peak in cortisol following a stressor, our "baseline" measure of cortisol was likely confounded by anticipatory stress at the start of the visit, which might further obscure effects. Additionally, given that multiple items in the trait anxiety questionnaire captured future-focused worry (e.g., "I worry too much," "I worry about things that may happen"), a sharp decline in cortisol across the visit might alternatively capture highly anxious teens who displayed an anticipatory cortisol peak even prior to RA arrival at their home. Future work should incorporate a longer acclimation period prior to baseline assessment, as well as more frequent saliva sampling across the task to allow for more dynamic modeling of cortisol across anticipatory, reactivity, and recovery phases.

Despite limitations, these findings are partially consistent with Colich et al. (2015) and Hankin et al. (2010), who found that cortisol hyporeactivity in early pubertal adolescents was associated with concurrent or later depressive symptoms. However, in contrast to their findings, our results did not support the additional association between cortisol hyperreactivity and trait anxiety at later pubertal stages. Perhaps the previously observed inversion of effects across puberty was driven by the biological influence of sex hormones on HPA axis activity, as the developmental increase in physiological sensitivity to social stress has been shown to be amplified during pubertal maturation (Gunnar, Wewerka, et al., 2009; Sumter et al., 2010; van den Bos et al., 2014). The present measure of self-reported *perceived* puberty may not have accurately captured true status, making it difficult to dissociate between the psychological and biological aspects of puberty. While psychological aspects include self-perceptions and feelings about maturation (e.g., embarrassment), biological aspects capture the underlying

neuroendocrine changes that drive internal and external maturation (Mendle, 2014). Cortisol baseline and reactivity did not vary as expected by pubertal stage in the present sample, potentially highlighting the more psychological aspects captured. Future work should seek to incorporate self- and clinician-reported Tanner staging to allow for the simultaneous investigation of both perceived and objective stages of maturation.

Moving to social anxiety, present findings partially aligned with hypotheses. Results indicated that greater cortisol reactivity to social stress was associated with increased odds of having a social anxiety diagnosis for those in the mid-pubertal stage. Classic models of social anxiety disorder emphasize the role of heightened self-focused attention and perceptions of an "audience" in the maintenance of symptoms (Clark & Wells, 1995; Rapee & Heimberg, 1997). These cognitive biases might be particularly salient for adolescents who perceive themselves as actively experiencing puberty, which comprises a range of physical changes (body composition, acne, etc.) associated with an increase in self-conscious emotion and appearance-related concerns (La Greca & Ranta, 2015; Pfeifer & Allen, 2021). Thus, greater cortisol reactivity might amplify social anxiety risk for those in the mid-pubertal stage by further increasing sensitivity to peer perceptions and the surrounding social context. However, it should be emphasized that these findings are not robust and need to be replicated in future work with a more equal distribution of participants across both Tanner stages and anxiety diagnoses.

Interestingly, the intersecting profile of risk for trait anxiety (*hypo*reactivity and *early* perceived pubertal stage) almost directly conflicts with risk for social anxiety (*hyper*reactivity and *mid*-perceived pubertal stage). It should again be noted that the present study did not directly assess stress induction by the TSST. The task might have

been stressful predominantly for those with elevated social anxiety given the social nature of the task, such that elevated cortisol reactivity was less likely to be observed for those higher in trait anxiety. However, I also speculate that these divergent findings could be attributed to the chronicity of anxiety symptoms. On one hand, trait anxiety is considered a more stable, person-level characteristic, such that higher symptoms are likely enduring over time (Spielberger et al., 1970; Spielberger & Rickman, 1990). Within this context of chronic exposure, cortisol hyporeactivity may be evidence of physiological burnout, particularly for less mature adolescents who have been shown to display lower coping self-efficacy relative to those who report a more advanced pubertal status (Hankin et al., 2010; ten Brink et al., 2021). On the other hand, a diagnosis of social anxiety in the present study was based upon retrospective report of behaviors over the past year. This approach might capture some adolescents with relatively newer symptoms, as social anxiety has been shown to emerge with the progression of puberty in some teens (La Greca & Ranta, 2015). Building upon these speculations, future longitudinal work should test if higher anxiety early in life is associated with initial elevations in cortisol reactivity that downregulate over time as symptoms become more chronic, as well as whether risk for social anxiety emerges later in pubertal maturation relative to trait anxiety. Additionally, despite significant group-level differences, there was considerable overlap in the range of trait anxiety levels between those with and without a social anxiety diagnosis, to include a weak correlation between the two variables in the present study. These measures might capture different subsets of teens with different life histories, emphasizing the importance of future work to 1) distinguish between different forms of anxiety in adolescence, and 2) better understand precursors to and chronicity of

symptoms across development. Further, trait and social anxiety were captured using different informants – self- and caregiver-report, respectively – which might have further contributed to lower concordance between the two measures. Integrating information across multiple informants (e.g., teen, caregiver, teacher) might better characterize anxiety symptoms, to include variation in the severity of subjective and observed symptoms across contexts (e.g., home, school; De Los Reyes & Kazdin, 2005).

Present analyses were additionally limited to an examination of perceived pubertal stage. However, perception of one's own maturation is influenced by comparisons with fellow peers, emphasizing the importance of also examining pubertal *timing.* As a distinct construct from pubertal stage, timing captures inter-individual differences in the timing of one's puberty onset relative to their same-age, same-sex peers (Dorn & Biro, 2011). Early pubertal timing – defined as beginning puberty at an earlier age than one's peers – has been associated with a range of negative psychosocial outcomes, to include depression, anxiety, substance use, and risky sexual behaviors (Graber et al., 2010, 2013). Though early pubertal onset was initially considered advantageous for boys, more recent evidence points to a similar link with psychopathology symptoms as shown in girls (Hamlat et al., 2019; Ullsperger & Nikolas, 2017). Pubertal timing itself might play a role in generating increased interpersonal stress that younger adolescents are unprepared to cope with – for example, elevated social anxiety and depressive symptoms have been found in early maturing teens who show an increased tendency to select older and more deviant peer groups (Rudolph et al., 2014), to start dating early (Benoit et al., 2013), and in those with more problematic peer

relationships (Blumenthal et al., 2009). Thus, physiological responding to social stress might mitigate or exacerbate pubertal timing-related vulnerability to anxiety.

Conclusion

Overall, given the population-level increase in anxiety symptoms and disorders in early adolescence (Beesdo et al., 2009; Kessler et al., 2012), it is important to better understand which teens are at greatest risk and how this risk can be mitigated. The present study examined how the association between physiological sensitivity to social stress and anxiety varied across peer and pubertal contexts. The effects of cortisol reactivity on both trait anxiety and risk for elevated social anxiety was found to vary according to perceived pubertal stage. Interventions aimed at improving anxiety symptoms should consider the larger developmental context – both internal and external to the child – in which an individual typically experiences and regulates social stress to address psychopathology in a more holistic manner.

Chapter 4

General Discussion

This dissertation broadly aimed to examine developmentally salient risk factors for elevated anxiety in adolescence. Findings emphasized the context-dependent nature of physiological responsivity. Patterns of both lesser and greater cortisol activity were associated with elevated anxiety in adolescence, depending upon the surrounding social and developmental context.

Study 1 explored associations between developmental instability within the household and caregiver-child contexts and adolescent anxiety. Building upon theoretical models of adaptive calibration and biological sensitivity, I also examined if cortisol functioning, 1) acted as a mechanism by which early environmental instability influenced later anxiety symptoms, and 2) modulated the association between instability and anxiety across development. Results indicated that greater developmental lability in caregiver support was associated with lesser cortisol reactivity to social stress, though cortisol did not significantly mediate the link between measures of instability and anxiety. However, biological sensitivity models were largely supported, such that greater cortisol reactivity and lower cortisol at baseline were associated with higher anxiety for children raised in more disorganized households. These effects were only significant for household disorganization measured across the first two years of life, highlighting infancy as a potentially impactful sensitive period.

Study 2 focused specifically within adolescence, examining the moderating role of perceived pubertal stage and peer problems on the association between HPA

functioning and anxiety. Findings indicated no direct association between cortisol and anxiety, although pubertal stage served as a significant moderator. For those who reported an earlier pubertal stage, lower cortisol reactivity to social stress was associated with higher levels of trait anxiety. Conversely, for those who reported a mid-perceived pubertal stage, greater cortisol reactivity was associated with increased risk for an elevated social anxiety diagnosis.

Overall, both hypo- and hyper- patterns of cortisol activity were important for predicting anxiety symptoms in adolescence. Beginning with lowered cortisol activity, proximal parenting processes were found to have a more direct and potent effect on children's cortisol functioning, aligning with both human and animal research (Hostinar et al., 2014). Unpredictability within the caregiver-child context from infancy through adolescence might contribute to downregulation of HPA sensitivity across development, protecting against repeated physiological activation within the child's primary social context. Further, lowered cortisol at rest was associated with greater anxiety within a more disorganized household in early life, while lowered reactivity increased anxiety risk within the context of earlier pubertal stages in adolescence. This blunting of HPA activity at rest and in response to a social stressor might again reflect physiological downregulation that makes it difficult for children to successfully regulate and cope with ongoing household-related stressors (e.g., over-stimulation, lack of routine) and development-related stressors prior to puberty. Future work is needed to explicitly model changes in cortisol across development to understand if hypoactivity represents a stable, trait-level risk factor or if cortisol activity downregulates over time.

Despite risk associated with cortisol hypoactivity, elevated cortisol reactivity to social stress was found to be less adaptive within a few developmental contexts. Greater household disorganization in early life, as well as a mid-perceived pubertal stage in adolescence, were associated with higher anxiety for those with greater cortisol reactivity to social stress. Although lowered cortisol might reflect difficulty engaging resources for cognitive and regulatory abilities (Suor et al., 2015), elevated reactivity might confer similar risk in a subgroup of children by increasing sensitivity to the acute, frequent stressors within chaotic home environments (Boyce & Ellis, 2005; Evans et al., 2005). Extending the developmental context into adolescence, increases in cortisol reactivity might also confer risk for social anxiety in teens who perceive themselves to be actively undergoing puberty, which comes alongside an uptick in self-consciousness and appearance-related concerns within social contexts (Pfeifer & Allen, 2021). Thus, elevated physiological reactivity might increase children's sensitivity to negative aspects of their surrounding physical context, as well as sensitivity to negative perceptions of their surrounding social context.

Moving to implications for prevention and intervention in early life, poverty has been associated with greater household chaos (Chen et al., 2010; Garrett-Peters et al., 2016) and higher lability in caregiver behaviors (Zheng & McMahon, 2022) across childhood. These early experiences are thought to have lasting effects on physiological and behavioral health, with present findings emphasizing the first two years of life as a particularly impactful time of household-based intervention. Building upon the lower-SES nature of the present sample, future work should target both child-level skills (e.g., regulatory abilities) and environmental risk (e.g., stability within the home) to mitigate

later elevations in internalizing symptoms. Within adolescence, attention should be paid to the role of puberty in social sensitivity and psychopathology, with the understanding that risk for different forms of anxiety might change across pubertal development. These research-based approaches offer a more holistic view of anxiety risk, pointing to multiple malleable contextual factors that might be addressed across youth development.

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Rogers, C. R., **Fry, C. M.**, Lee, T. H., Galvan, M., Gates, K. M., & Telzer, E. H. (2022). Neural connectivity underlying adolescent social learning in sibling dyads. *Social Cognitive and Affective Neuroscience*, *17*(11), 1007-1020.

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