

THE MULTIGENERATIONAL PROCESS OF ADVERSITY AND RESILIENCE

by

RACHEL A BROWN

(Under the Direction of Kalsea J. Koss)

ABSTRACT

Research to date has examined the negative developmental effects of adverse childhood experiences (ACEs), but fails to examine adversity experienced across the life course including prenatal and intergenerational experiences of adversity. Utilizing a life course theory and developmental psychopathology approach, the current dissertation utilizes two separate datasets to elucidate the multigenerational process of adversity and resilience. Study one examines effects of cumulative prenatal adversity on adolescent offspring depressive and anxiety symptoms, and investigates the buffering role of attachment against this association. Study two examines effects of parental ACEs on adolescent offspring depressive and anxiety symptoms. The mediating role of nurturant parenting, and the moderating role of parental benevolent childhood experiences (BCEs) and family cohesion were investigated as well. Findings suggest that prenatal adversity, as well as adversity experienced before pregnancy, is predictive of offspring depressive symptoms. Additionally, family level processes (e.g., secure attachment) operated as a buffer against these associations. This dissertation highlights family processes as especially protective after exposure to adversity, and informs intervention programs that target diverse samples.

INDEX WORDS: Adversity; Prenatal; Resilience; Intergenerational; Parenting; Family cohesion; Attachment

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B.S., Arizona State University, 2019

M.S., University of Georgia, 2023

A Dissertation to be Submitted to the Graduate Faculty of The University of Georgia in Partial
Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2025

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DEDICATION

I would like to dedicate this dissertation to all of the children experiencing adversity. I hope that, above all else, the science that I conduct will reach and inform families and children who are experiencing adversity.

ACKNOWLEDGEMENTS

To my major professor, Dr. Kalsea Koss, words cannot adequately capture the gratitude I have for you as my advisor in a professional and personal sense. You have supported me through 5 years of research and have dedicated your unwavering and constant support through many life events that I've experienced. I began my doctorate during a global pandemic, endured 2 moves, 2 pregnancies, and 2 postpartum experiences. You have extended grace, kindness, and motivation to promote my progress in this program all the while. Thank you for your support. It has been a wonderful privilege to learn from you throughout the last five years.

Thank you to my dissertation committee members, Drs. Steven Kogan, Geoffrey Brown, and Katie Ehrlich, for your support and commitment to helping me grow as a developmental scientist. I appreciate your willingness to allow me to utilize your data, time, and energy toward my professional growth.

Evin, thank you for being such an incredible sounding board and mentor throughout the years. I'm so thankful for your guidance!

To my boys, Brody and Bryce, Mommy loves you so much. I hope my kids learn that anything is possible, and that hard work is always rewarded.

To Patrick, I'm so appreciative of your encouragement that has pushed me forward for the last 5 years. You're an incredible partner, and an incredible dad to our children. I'm looking forward to this next chapter of life that we get to share together.

To my family, thank you for making this possible. To Sarah, thank you for always being a listening ear and for being my rock. Mom and Dad, I love you both and can't thank you enough for your continued support during the past 5 years.

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CHAPTER 1

INTRODUCTION AND OVERVIEW OF THE STUDIES

Introduction

Adverse childhood experiences (ACEs) reflect the experience of significant adversity exposure prior to age 18 (e.g., death of a loved one, abuse/neglect, intimate partner violence) that impact psychological and physical well-being across the lifespan (McLaughlin, 2018; Ridout et al., 2018). Original development of an ACEs index reflects a cumulative metric of early life adversity that encompasses a set of 10 specific adverse experiences that include physical abuse, sexual abuse, emotional abuse, neglect, living with someone who abuses substances or is mentally ill, losing a parent through divorce, having a relative who has been sent to jail or prison, losing a parent through death or abandonment, and being exposed to domestic violence against a mother (Felitti et al., 1998). Minoritized populations are at higher risk of being exposed to adversity through systemic disadvantage (Crenshaw, 1991). In particular, Black youth may be exposed to more chronic adversity due to inequality (Pais, 2014). Exposure to ACEs may lead to greater physical and mental health disparities. ACEs are associated with accelerated aging, psychiatric diagnoses, and risk for disease later in life (Hogan et al., 2024; McCrory et al., 2015; Pietrek et al., 2013). Research demonstrates a consistent association between exposure to ACEs and psychopathology with childhood adversity exposure predicting higher rates of psychopathology at later times in development, which highlights the long-term psychological implications of ACEs across the lifespan (Schilling et al., 2008; Smith & Pollak, 2021). Most research to date examines adversity that begins after a child is born. Overlooked in adversity

research are experiences of adversity prior to a child's birth including both the prenatal period and intergenerational experiences. Existing research demonstrates that the prenatal period may have profound effects on development. For example, adversity experienced by a mother that occurs during pregnancy, preceding the child's birth, is significantly associated with both child well-being as well as the quality of the parent-child relationship (Seckl, 2004). Moreover, intergenerational effects of early life adversity are understudied and the mechanisms through which early life adversity exposure for a caregiver affects their offspring in the next generation remain unanswered. As such, there is a crucial need for researchers to examine adversity among diverse populations through a holistic lens that extends developmental investigations prior to a child's birth and throughout the life course.

Adolescence may be a particularly important developmental period to assess psychopathology outcomes following adversity in the form of depressive and anxiety symptoms due to mental health outcome divergence that typically occurs in adolescence (Jones, 2013). Mental health symptom onset typically occurs during adolescence, or shortly thereafter (Paus et al., 2008). For example, research shows that prevalence for elevated depressive and anxiety symptoms is roughly 25% and 20%, respectively, during adolescence (Racine et al., 2021). Diagnoses of anxiety and depression also occur at higher rates in adolescence compared to earlier in childhood (Kessler et al., 2007). Prevalence of depressive and anxiety symptoms in Black populations have also increased in recent years (Weinberger et al., 2018). Higher rates of diagnoses and elevated depressive and anxiety symptoms highlight adolescence as a sensitive period for internalizing psychopathology especially for minoritized youth (Gabard-Durnam & McLaughlin, 2020). Environmental experiences that occur during sensitive periods may have lasting effects that extend throughout the lifespan above and beyond those that occur during

other developmental periods (Gabard-Durnam & McLaughlin, 2020). As such, examining adolescent psychopathology outcomes as a result of early prenatal adversity may be most informative during this developmental period. The transition to adolescence is marked as a sensitive period due to many physical (e.g., puberty) and behavioral (e.g., increasing independence, identity formation) developmental changes that occur (Branje et al., 2021; Wray-Lake et al., 2016). However, not all adolescents exposed to adversity develop physical and mental health disorders; understanding the protective factors that contribute to resilience following adversity is imperative for promoting better health and well-being. A central goal of my dissertation research is to examine the protective factors that promote resilience among diverse populations and identify the protective factors that may offset the effects of ACEs on adolescent psychological well-being.

Resilience

Despite exposure to adversity, not all youth will develop deleterious outcomes following the experiencing of adversity and instead demonstrate resilience. Resilience captures the *protective* effects of various individual and contextual factors for an individual and reflects the tendency to maintain positive development despite exposure to adversity (Luther, 2015; Wright et al., 2013). In contrast, a promotive effect is beneficial for all youth, despite level of risk and this positive development inherently differs from resilience in the context of adversity (Wright & Masten, 2005). A salient goal of resilience research is to delineate the protective factors within individuals and their environments that help to maintain positive developmental outcomes despite experiencing adversity. Consequently, it is necessary to examine the factors that account for this heterogeneity and identify those factors that contribute to resilience (Luthar, 2015).

Indeed, research has examined resilience in individuals who have experienced childhood adversity. Resilience research focus on protective factors that span across ecological contexts including investigations of individual traits, healthy family dynamics, a positive child-caregiver relationship, and positive meaningful engagement with the community or one's school as potential sources of protective factors (Masten & Barnes, 2018). A large body of research has supported this argument with evidence demonstrating a wide range of protective factors for individuals who have experienced adversity (Darling Rasmussen et al., 2019; Hu et al., 2015; Yule et al., 2019). Research has also supported these findings across minoritized populations with varying intersectional ties (Brown & Koss, 2025). Additionally, a wide range of outcomes have been examined in resilience research among individuals who have experienced adversity including mental health outcomes such as anxiety and depressive symptoms. Indeed, Watters and colleagues (2023) conducted a meta-analysis that found that, despite individuals experiencing ACEs, some individuals demonstrated resilience as measured by fewer depressive symptoms in adulthood in the presence of protective factors. Furthermore, a systematic review conducted by Abate and colleagues (2024) found that resilience-promoting interventions also significantly protect individuals against maladaptive mental health outcomes during adulthood from ACEs exposure. Findings that suggest psychological resilience for individuals despite being exposed to ACEs expands across the lifespan, and have been found in studies of diverse samples with adolescents, young adults, and older adults (Haczkeicz et al., 2024; Han et al., 2023; Leung et al., 2022). To conclude, individuals who experience ACEs during their childhood may be at risk for maladaptive psychological outcomes, but the presence of protective contextual factors reduces this risk.

THEORETICAL PERSPECTIVES

Developmental Psychopathology

Developmental psychopathology is a scientific discipline that pertains to adversity and resilience which aims to better elucidate the nature of psychopathology and how psychopathology develops over time (Rutter & Sroufe, 2000). Developmental psychopathology highlights the heterogeneity in developmental pathways, primarily through the conceptualization of multifinality and equifinality (Sroufe, 1997). Multifinality reflects the notion that multiple outcomes can occur despite two individuals undergoing the same experience (Sroufe, 1997). Research demonstrates that despite individuals experiencing ACEs, only some individuals develop maladaptive psychological outcomes, which highlights the need to examine the mechanisms and developmental pathways that contribute to the process of resilience (McLaughlin, 2018). In contrast, equifinality depicts the theoretical tenet that two individuals may experience unique forms or different levels of exposure to adversity, but both reach the same symptom level or type of psychopathology as an outcome. Cumulative models that measure adversity, like ACEs, with the inclusion of multiple forms of adversity exposure take into account that different types of adversity may lead to the same outcome. For example, children who are exposed to higher levels of trauma are more prone to negative consequences than children who have only been exposed to a singular form or type of trauma (Ogle et al., 2013) which highlights that exposure to multiple adverse life experiences may have more compounding effects than adverse experiences that occur in singularity. Both prenatal and parental ACEs may also lead to similar outcomes of depressive and anxiety symptoms despite unique developmental cascades of adversity.

Developmental Cascades

Extending beyond acute, short-term outcomes, Masten and Cicchetti (2010) also implore resilience researchers to identify developmental cascades in resilience research that extend across time and levels of functioning. Developmental cascades are the accumulation of consequences for development of myriad interactions that occur in an individual's environment across contextual levels. Developmental cascades highlight the impact of early life experiences on an individual and recognize that there may be a "snowball effect" to experiencing adversity which can interfere with resilience (Repetti et al., 2009). These developmental cascades may also occur across generations. For instance, research shows that when parents are exposed to adversity as children, this may negatively impact their parenting which in turn, may impact their offspring's development creating a "snowball effect" of negative consequences across generations (Zhang et al., 2023). However, developmental cascades also highlight the potential for this "snowball effect" to occur in a positive direction, such that resilience is more likely when an individual is exposed to more and continues to experience protective factors. Despite accumulation of exposure to chronic adversity being associated with negative outcomes, developmental psychopathology highlights that a child is an active organism, and actively participates in shaping their own environment and development. As such, development is viewed as being bidirectional, and understanding the context in which a child demonstrates resilience is critical to ascertaining how risk factors and protective factors together contribute to an individual's developmental pathway throughout the lifespan.

Life Course Theory

Life course theory suggests that an individual is continually influenced by processes and experiences that unfold throughout the entire life span (Elder, 2013). Furthermore, life course

theory suggests that in order to adequately conduct developmental science, more extended periods of time within development need to be considered. Indeed, developmental scientists have found processes are better understood when considering the entire life course as opposed to considering more narrow portions of an individual's life (Clark et al., 2010; Karatsoreos & McEwen, 2013; Kuh, 2007). For example, mental health and developmental psychopathology is better understood when considering the entire lifespan (Westerhof & Keyes, 2010). Mental health outcome divergence typically occurs during adolescence and young adulthood (Jones, 2013) especially for those youth who experienced adversity earlier in life consistent with a long-term developmental cascade. Additionally, psychopathology may not remain stable throughout the lifespan (Southwick et al., 2011; Sulis, 2021). As such, considering mental health throughout an individual's lifespan, as well as comparing mental health outcomes throughout different points of development, is imperative to better understand how these processes occur.

Prenatal Influences on Development

Developmental science adopting a life course theory perspective should incorporate the influence of the prenatal period alongside consideration of postnatal experiences. The examination of both prenatal and postnatal experiences is understudied in regard to their associations with internalizing symptoms later in life, and necessitates further examination, as both reflect sensitive developmental periods throughout the life course. Research demonstrates that prenatal experiences do exert effects immediately after birth (Gillespie et al., 2019), but also across the lifespan (Tobi et al., 2018). Developmental Origins of Health and Disease (DoHaD) is a theoretical perspective that highlights the influence of the prenatal environment on postnatal health and disease (Barker, 2004; Silveira et al., 2007). This perspective posits that if a pregnant mother experiences adversity, the fetal offspring may develop in ways that anticipate a more

adverse postnatal environment, which may negatively impact their physical and mental health later in the postnatal period. Mothers' own experiences of adversity during her childhood may also impact how she perceives stress during pregnancy (Bowers et al., 2018), which could lead to variations in development during the prenatal period for the developing fetus. Minoritized pregnant women may experience more chronic stressors due to racism and inequality and this increased stress may transmit to their offspring (Conradt, 2017). Research consistently demonstrates an association between the prenatal experience and postnatal physical health outcomes (Slopen et al., 2015), but mental health outcomes as a result of prenatal adversity exposure are not as well understood, especially the examination of mediating pathways between prenatal adversity and postnatal mental health outcomes (Liu et al., 2022). Fetal programming research may also inform the examination of mediating pathways between prenatal adversity and postnatal outcomes. Fetal programming research indicates an ability to map the origins of mental and physical health variations prior to birth (Conradt et al., 2018). The match-mismatch model, a dominant theoretical framework informing fetal programming, suggests that a developing fetus may anticipate their postnatal environment by gathering cues from their prenatal environment (Gluckman et al., 2005). Fetal programming theories support the measurement of adversity during the prenatal period in order to better understand how and why individuals maintain between-individual variations in behavioral and physical health after birth.

Utilizing a life course perspective when examining prenatal adversity extends beyond examining health outcomes directly following birth. Rather, examining mental health outcomes as a result of prenatal adversity with a life course perspective would include the examination of outcomes during adolescence or later in life. Existing research suggests prenatal adversity exposure in the form of maternal depressive symptoms predicts later psychopathology in the

form of offspring depressive symptoms during adolescence (Braithwaite et al., 2014) and in later adulthood (Liu et al., 2022). Utilizing a life course perspective promotes the incorporation of prenatal exposure to adversity in conjunction with postnatal experiences and should be utilized to better capture an individual's risk for psychopathology due to adversity exposure.

Intergenerational Cascades of Risk and Resilience

Intergenerational effects of adversity and its effect on later well-being in offspring are also understudied, but are important to consider in developmental science as the life course also acknowledges the effects of the previous generation and adversity at all life points needs to be considered. When parents are exposed to their own ACEs, those experiences can exert negative effects onto their offspring's development across their lifespan (Barnert et al., 2023; Racine et al., 2018). Studies have shown these negative effects of parental ACEs may influence offspring development through behavioral mechanisms including maternal mental health and parenting (Swords et al., 2024). Individuals often reside with their caregiver for a large portion of their early life (Láftman, 2010), which further highlights the need to acknowledge parental exposure to adversity and understand how it impacts their offspring. Researchers who are proponents of life course theory have also encouraged developmental researchers to consider intergenerational effects when examining development through a life course perspective, as they believe this is the “missing element” of life course research (VanderVen, 2013). This “missing element” of life course research, intergenerational influences, acknowledges that parents cannot raise children without their own experiences affecting the way they interact with their child (Wang et al., 2023). However, not all children will exhibit negative outcomes despite having parents who were exposed to adversity. Studies have found that despite parents experiencing adversity, positive family characteristics such as parental nurturance and family social support may protect

offspring from the effects of parental adversity exposure (Hatch et al., 2020; Woods-Jaeger, 2018).

OVERVIEW OF STUDIES

This dissertation utilized two separate datasets to examine the effects of ACEs on adolescent mental health in diverse populations. Study 1 examined the longitudinal effects of prenatal exposure to adversity on offspring's mental health in adolescence and examined the protective effects of attachment during toddlerhood. Study 2 examined the longitudinal effects of parental exposure to adverse childhood experiences on their adolescent children's mental health and tested whether low levels of nurturant parenting served as a mechanism in this intergenerational cascade. Both of these studies intend to delineate the pathways between adversity and offspring well-being in minoritized populations, specifically through understudied forms of transmission (prenatal adversity and intergenerational effects of adversity).

Manuscript 1 overview

Despite the experience of prenatal adversity exerting negative effects on child well-being, not all individuals who experience prenatal adversity will experience later negative outcomes. However, research has not yet examined more long-term effects of multiple forms of prenatal adversity that contribute to later adolescent well-being. Research has demonstrated that positive parenting during childhood may offset the risk associated with prenatal adversity (Corcoran & McNulty, 2018). A large body of research demonstrates the many positive and protective effects of having a secure parent-child attachment relationship for all youth, despite level of risk (Peterson & Park, 2007). Furthermore, attachment as a protective factor to offset the risk of prenatal adversity on adolescent well-being has not yet been examined. Using a large nationally representative, multi-ethnic birth cohort study, the first manuscript of my dissertation

had two specific aims. The first aim was to determine whether prenatal adversity impacts adolescent offspring mental health (at age 15 years) including depressive symptoms and anxiety symptoms. Next, I examined whether a secure parent-child attachment during toddlerhood (at age 3 years reflecting early postnatal caregiving) diminished the association between prenatal adversity and adolescent mental health. Mirroring investigations of postnatal ACEs, I created a cumulative index for prenatal adversity that included prenatal experiences including maternal substance use, poverty, material hardship, inadequate prenatal maternal nutrition, paternal incarceration, and intimate partner violence.

Manuscript 2 overview

Research in adults finds that ACEs are common experiences. In fact, 64% of adults report that they have experienced at least one ACE during their childhood (Swedo et al., 2023) and individuals who have experienced ACEs are more likely to experience physical and mental health problems (Lang et al., 2020). Further, the effects of ACEs may extend beyond individual well-being; for example, ACEs may perpetuate across generations to their offspring (Narayan et al., 2021). Indeed, when individuals become parents, their own childhood ACEs may impact their parenting, family functioning, and their child's mental health (Zhang et al., 2023). Parenting may also be impacted by how an individual was parented themselves. As such, these associations may extend beyond a single generation, and may affect developmental outcomes in subsequent generations. It is necessary to better elucidate the mechanisms and protective factors that may offset this multigenerational cascade. Protective factors, however, may operate at different time points in this intergenerational cascade. Thus, a central goal of my dissertation research is to elucidate the protective factors that offset disruptions in parenting as well as protective factors that offset the development of psychopathology for adolescents. Research on resilience shows

that it is the balance of risk and protective factors that predict maladaptive outcomes. Thus, when examining parental ACEs, it is imperative to also examine positive childhood experiences that may co-occur during this developmental period to offset the negative effects of ACEs. A frequently utilized scale to measure these experiences is the benevolent childhood experiences scale (BCEs) (Narayan et al., 2023). Indeed, BCEs have been shown to offset the risk against poorer family functioning and parent-child relationships when a parent has also experienced ACEs as a child (Johnson et al., 2022). For youth, the broader family climate may offset the effects of parenting on adolescents' psychopathology. In particular, family cohesion, reflecting the emotional and physical bonding that family members have with one another, has been found to buffer against maladaptive adolescent psychopathology outcomes (Moreira & Telzer, 2015).

Using data from long-term, multigenerational longitudinal study of Black families, I examined the intergenerational cascade between parent-experienced childhood ACEs (experiences before age 18), their parenting of their own children (e.g., reduced nurturant parenting), and youth's anxiety and depressive symptoms in a sample of youth ages 8-16 years old. In my dissertation, I tested a) whether parent's own positive life experiences BCEs moderated the pathway between ACEs and reduced nurturant parenting and b) whether a current cohesive family environment protected against the negative effects of low nurturant parenting on child well-being.

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CHAPTER 2

**CUMULATIVE PRENATAL ADVERSITY AND ITS ASSOCIATION WITH
ADOLESCENT INTERNALIZING SYMPTOMS: THE PROTECTIVE ROLE OF
PARENT-CHILD ATTACHMENT**

Brown, R., et al. To be submitted to a peer-reviewed journal.

Abstract

Research to date has examined the negative effects of prenatal adversity on offspring well-being, however much of the existing literature fails to examine outcomes that extend beyond infancy and early childhood. Additionally, existing research fails to examine potential postnatal protective effects of family processes against the effects of prenatal adversity on offspring well-being. Utilizing data from the Future Families and Child Well-being Study, the current study examined the protective role of a secure attachment against the association of cumulative prenatal adversity on adolescent offspring depressive and anxiety symptoms. Results showed that cumulative prenatal adversity predicted higher levels of depressive symptoms at age 15 for offspring. Additionally, secure attachment served as a buffer to disrupt the association between cumulative prenatal adversity and adolescent depressive symptoms. Implications of this research highlight the protective effects of a secure parent-child attachment in the context of prenatal adversity.

INDEX WORDS: Prenatal; Adversity; Depression; Anxiety; Attachment; Resilience

Introduction

Adverse childhood experiences (ACEs) refer to adversity that occurs prior to the age of 18 (Felitti et al., 1998). ACEs include, but are not limited to, significant negative events such as the death of an immediate family member, exposure to violence, neglect, or abuse (Ports et al., 2020). ACEs are associated with worse emotional, physical, and psychological developmental outcomes both directly after exposure (Varese et al., 2012) as well as across the lifespan (Champagne, 2010). ACEs are a strong indicator of psychological well-being during childhood (Liming & Grube, 2018) and adulthood (Von Cheong et al., 2017), which highlights the long-term implications of exposure to adversity. Adolescent anxiety symptoms, depressive symptoms, and externalizing symptoms have all been examined as outcomes of ACEs exposure (Busso et al., 2017; Hazel et al., 2008; Phillips et al., 2005; Stroud et al., 2019). Additionally, minoritized youth and White youth may experience early life adversity at different rates, with minoritized youth experiencing ACEs at a higher rates (Fagan & Novak, 2017). While a large body of research demonstrates the importance of exposure to ACEs across childhood, these effects may begin during the prenatal period. Existing research also lacks life course investigations of cumulative prenatal adversity on psychosocial functioning later in life. Life course theory suggests that contexts throughout an individual's lifespan continue to influence later points of development, and in order to adequately conduct developmental science, multiple developmental periods in the lifespan should be considered (Elder, 2013).

Experiences during the prenatal period, an especially sensitive period in development, may predict the trajectory of future development (Camerota & Willoughby, 2021). Indeed,

exposure to adversity in the prenatal period has been found to have immediate effects in the postnatal period (Triplett et al., 2022) as well as across the lifespan (King et al., 2012). Fetal programming theories posit that prenatal experience may contribute to variations in postnatal functioning (Conradt et al., 2018). Further, these theories posit that these variations occur through adaptations that the fetus makes based off the prenatal experience (Hochoer et al., 2001). Indeed, existing research demonstrates offspring ontogenic vulnerability to poorer outcomes in the experience of prenatal maternal malnourishment, maternal depression, and maternal stress (Del Giudice, 2012; Langley-Evans, 2001; Lewis et al., 2015), and this vulnerability may be exacerbated for individuals who identify as a racial/ethnic minority (Silva et al., 2022). Prenatal exposure to adversity is thought to have unique additive effects with adversity exposure in the postnatal period and during childhood, suggesting that these experiences may have unique influences on later development (Camerota & Willoughby, 2021). As such, the postnatal period may be a salient developmental period for examining protective effects of prenatal adversity onto postnatal outcomes. Examining these associations among diverse samples is important due to variations in vulnerability and responses to adversity amongst racial groups.

Cumulative prenatal risk

There is a need for researchers to include prenatal adversity exposure in their assessments when examining an individual's exposure to adversity across the lifespan. The prenatal period is overlooked in adversity research and unique types of prenatal risk are often studied in isolation from one another (e.g., examining maternal drug use alone or maternal poverty during pregnancy alone). Furthermore, examining the combination of multiple forms of adversity through cumulative metrics of adversity during the prenatal developmental period is an important consideration of prenatal risk that is often not considered. Cumulative models to conceptualize

adversity account for the fact that different types of adversity co-occur and the overall accumulation of adversity may have additive effects that contribute to increasingly more maladaptation (McLaughlin et al., 2021). Cumulative models are frequently seen in research with ACEs, which totals the exposure to adversity by sum-scoring each adverse experience (e.g., sexual abuse, physical abuse, parental separation or divorce, living with an alcoholic parent). Cumulative stress models, which combine multiple forms of risk, are still being developed into research on fetal programming (Conradt et al., 2018; Nederhof & Schmidt, 2012). However, the limited amount of research that has examined this association finds that mothers who experienced higher cumulative prenatal adversity were more likely to have children with heightened depressive symptoms in adolescence (Maxwell et al., 2018). Research also reflects higher prevalence rates of myriad forms of prenatal adversity amongst minoritized populations than White populations (Campbell et al., 2020; Johnson et al., 2023). The present study seeks to develop a cumulative adversity measure during the prenatal period that parallels investigations of cumulative ACEs to examine the long-term impacts of prenatal adversity on adolescent psychopathology in a diverse sample. Included in this index, are individual forms of prenatal adversity that have been found to be associated with offspring well-being as well as those that parallel forms of adversity studied during childhood. Next, I summarize research on existing types of prenatal adversity.

Nutritional inadequacy

Nutritional inadequacy refers to circumstances in which a pregnant mother does not consume enough essential nutrients such as calcium, folate, and other vital nutrients during pregnancy (Rosso, 1981) which may serve as a prenatal risk factor for the developing fetus. Nutritional inadequacy may occur due to lack of resources or may occur when a pregnant mother

suffers from an eating disorder. The prevalence of maternal eating disorders, often resulting in nutritional inadequacy, is higher than ever before (Triunfo & Lanzone, 2015). Prenatal nutritional inadequacy is frequently associated with postnatal outcomes of offspring well-being such as physical health and growth over the life course (Gonzalez-Bulnes et al., 2020). For example, mothers who experience nutritional inadequacy throughout their pregnancy were more likely to have children who struggled with adiposity and BMI in adolescence (Shapira, 2008; Cao-Lei et al., 2015). Studies have found that minoritized pregnant women are at the highest risk for nutritional inadequacy during pregnancy when considering variations of risk across racial and ethnic groups (Sauder et al., 2021). Prenatal nutritional inadequacy is also associated with deficits in mental health and cognitive health outcomes (Brown & Susser, 2008; Li et al., 2019). However, much of the research examining mental health as an outcome of prenatal nutritional inadequacy examines maternal mental health as opposed to offspring mental health (Rechenberg & Humphries, 2013; Sparling et al., 2017). As such, nutritional inadequacy during pregnancy is essential to investigate in diverse samples with postnatal psychological outcomes for the offspring of nutrient deficient mothers.

Intimate partner violence

Prenatal exposure to intimate partner violence (IPV) occurs when the pregnant mother is a victim of physical, sexual, or emotional abuse by their intimate partner (Cha & Masho, 2014). Meta-analysis findings (Kim et al., 2024) suggest that prenatal exposure to IPV may be associated with child developmental outcomes such as higher psychological problems, dysregulated physiological functioning, poorer physical health outcomes, and worse behavioral health outcomes. For example, IPV during the prenatal period has been found to be predictive of child psychosocial and emotional well-being after birth during childhood and adolescence

(Murray et al., 2020). IPV is also seen at significantly higher rates for individuals positioned in lower socioeconomic statuses and for those who face more social inequalities (Rennison & Planty, 2003). Prenatal IPV has been associated with heightened internalizing symptoms throughout childhood (Bianchi et al., 2016; Martinez-Torteya et al., 2016; Martinez-Torteya et al., 2017; McFarlane et al., 2014). Research on exposure to prenatal IPV and adolescent well-being shows more overall mental health problems (Serpeloni et al., 2019) including higher depressive and anxiety symptoms (Chen et al., 2022) reported among adolescents who were exposed to prenatal IPV.

Maternal substance use

Maternal substance use during the prenatal period occurs when the pregnant mother uses illicit drugs, nicotine, or alcohol during pregnancy (Sithisarn et al., 2012). With the legalization of marijuana in a portion of the United States (Gnofam et al., 2020) and the rise of the opioid epidemic (Haight, 2018), rates of prenatal substance use have risen in recent years (Guille & Aujla, 2019). Prenatal illicit substance exposure has been associated with poorer offspring physical health outcomes such as growth restriction and memory impairments (Gorman et al., 2014; Richardson et al., 2015) as well as poorer psychological outcomes such as externalizing symptoms (Eze et al., 2016). Prenatal substance exposure is also associated with adolescent mental health including both depressive symptoms (Duko et al., 2021) and anxiety symptoms (Easey et al., 2019). Existing research also reflects variation among rates of prenatal substance use when comparing racial and ethnic groups (Loggins Clay et al., 2018). As such, maternal prenatal substance use is an important prenatal risk factor when examining the effects of prenatal risk on adolescent mental health outcomes.

Poverty

Poverty is a measure of inadequate income relative to needs based on household size (Chambers, 2006). According to the US census, roughly 11% of Americans are living in poverty (US Census, 2024) and roughly 1 in 5 pregnant women are at or below the poverty line (Bravenman et al., 2010). Minoritized individuals, specifically Black and non-White Hispanics individuals, experience poverty at rates twice as high as White individuals (Gradin, 2011). Although governmental assistance programs have been designed to aid pregnant women living in poverty, many women do not take advantage of these resources, as they may not be aware of these programs nor have the necessary resources to access them (Gago et al., 2022). Prenatal poverty is associated with negative outcomes for offspring such as low birth weight (Strully et al., 2010) and postnatal cognitive development (Vore, 1973). Prenatal experiences of poverty have also been found to be associated with child well-being after birth (Lefmann & Combs-Orme, 2014). Negative associations with prenatal poverty are also seen across the lifespan in offspring. Bleil and colleagues (2018) found that when females were exposed to prenatal poverty, they were more likely to have fewer ovarian reserves during adulthood. Furthermore, depressive and anxiety symptoms may be higher for those individuals exposed to prenatal poverty (Lefmann & Combs-Orme, 2014). Bruce (2002) finds that adolescent rates of depressive and anxiety symptoms are higher when they were exposed to prenatal poverty.

Material hardship

Material hardship is a consumption-based measure reflecting households who are unable to meet basic needs that includes housing, food, and healthcare (Beverly, 2001). Examples of material hardship indicators include the inability to pay rent or utilities or buy sufficient food. Material hardship is only moderately correlated with poverty (Mayer & Jencks, 1989) as material

hardship and the inability to financially make ends meet may be experienced across a range of household incomes. Similarly, families living in poverty may be able to meet basic needs despite financial challenges. Material hardship is also reported at higher rates among minoritized groups and may be associated with poorer prenatal care due to insufficient financial resources (Liu et al., 2024). Material hardship during pregnancy is associated with postnatal outcomes for offspring such as emotion regulation (Fuller et al., 2018) and cognitive development (Rauh et al., 2004). Prenatal material hardship is also significantly associated with offspring diagnoses of ADHD during preadolescence (Perera et al., 2018). Adolescent offspring mental health may also be impacted by early material hardship; however, studies to date have only examined these associations with postnatal exposure to material hardship at age 1 and older (Miller & Johnston, 2024).

Parental incarceration

Parental incarceration reflects either the pregnant mother or biological father residing in jail or prison during the prenatal period. When fathers are involved in the criminal justice system during the prenatal period, they may be unable to attend prenatal appointments, maintain involvement in the pregnancy, or provide emotional support to the pregnant mother (Hairston, 2002; Hessami et al., 2022; Testa et al., 2022). Paternal incarceration may also increase stress for expecting mothers. Additionally, when fathers are in prison during the prenatal period, offspring are more likely to be born to unmarried parents, and have lower rates of acknowledgement of paternity (Testa et al., 2022). There is a large gap for incarceration rates among racial groups, with Black men being imprisoned at significantly higher rates than their White counterparts (Pettit & Western, 2004). Adolescent indicators of well-being such as depressive symptoms and delinquency have also been associated as outcomes of paternal involvement in the criminal

justice system (Poehlmann, 2009). Shlafer and colleagues (2012) found that when adolescent offspring experienced parental incarceration, they were more likely to exhibit antisocial behaviors. Much of the research examining prenatal parental incarceration examines the effects on either the pregnant mother's well-being or the immediate postnatal well-being of the child. As such, longitudinal examinations should determine whether parental incarceration during the mother's pregnancy among diverse samples has long-term effects on offspring psychological well-being.

Fetal Programming of Adversity

The cumulative stress model, a dominant theoretical framework in prenatal adversity research, suggests prenatal exposures to stress cumulatively impact the fetus and cause disruptions in ongoing development (Glover, 2011). Mechanisms of impact from prenatal adversity exposure to psychopathology may be explained through increased maternal distress and activation of stress biology that operates through increased maternal cortisol as well as through maternal nutrition mechanisms, both of which can cross the placental barrier (Briffa et al., 2017; Conradt et al., 2018). The placenta is the primary fetal organ that develops to filter toxins and transport nutrients between the mother and the fetus (O'Connor et al., 2019). An additional purpose of the placenta is to protect the fetus against negative forms of exposure that the mother experiences (e.g., viruses, toxins) (O'Connor et al., 2019). Indeed, higher levels of placental cortisol exposure have been associated with poorer offspring outcomes such as preterm birth, poor gestational health, and low birth weight (Lester et al., 2013). Additionally, much of the research on fetal programming also considers nutritional conditions as a context that provides cues for the fetus to organize development in preparation for the postnatal environment (Gluckman et al., 2005). The current study examines forms of prenatal adversity that may

operate through these nutritional and stress biology signals, which may explain how exposure to adversity affects offspring psychopathology in adolescence (Conradt et al., 2018).

Adversity and adolescent well-being

Examining the longitudinal psychological impact of prenatal adversity exposure should expand upon and extend into particularly vulnerable developmental periods. Adolescence marks an especially important developmental period that consists of vast biological (e.g., puberty) and social transitions. Adolescence is an important developmental period to examine due to the development of mental health problems that typically emerge and rise during the transition from childhood to adolescence (Saugstad, 1989). Developmental psychopathology researchers also highlight adolescence as a developmental period in which divergence of mental health outcomes occurs (e.g., multifinality)(Jones, 2013). The developmental turning point from childhood to adolescence is a particularly vulnerable period for American families, and even more so for those who have experienced adversity (Burt & Masten, 2010). Additionally, adolescence is a particularly vulnerable developmental period for minoritized youth, who may begin internalizing systemic inequalities and experience racism more frequently during this developmental period (Tyler et al., 2020). Consequently, adolescence is a developmental period that should be examined in minoritized youth when researching early life adversity and psychosocial functioning. Despite research existing that highlights the link between prenatal adversity and child developmental outcomes in adolescence, not all individuals will experience the deleterious effects of prenatal adversity. As such, moderators should be included to examine the potential protective effects of postnatal contextual influences.

Resilience

Not all individuals who experience adversity develop psychopathology (Luthar et al., 2000). Indeed, research demonstrates that individuals display resilience despite exposure to ACEs (Masten & Barnes, 2018). When an individual experiences adversity, even in the context of familial adversity, the child may often lean on their most essential relationship to promote resilience or offer solace (Hostinar & Miller, 2019). As such, the parent-child relationship may serve as a buffer between the effects of prenatal adversity and adolescent well-being (Tung et al., 2024). The parent-child relationship is a key player in the process of resilience (Masten, 2007). Developmental theorists posit that infancy and toddlerhood may be a particularly salient developmental period that could protect against the deleterious effects of prenatal adversity (Schuetze et al., 2020). However, there is minimal research that assesses the long-term impacts of early postnatal protective factors to promote adolescent psychological well-being despite experiencing prenatal adversity. Research demonstrates that parenting may serve as a protective factor rather than a mechanism following early life adversity (Whittaker et al., 2011; Schuetze et al., 2020). As such, it is necessary to examine the early postnatal parent-child relationship as a protective factor in the association of prenatal adversity and adolescent psychopathology. The prenatal and postnatal periods should be integrated in lifespan developmental studies on adversity and resilience.

Mother-child attachment as a postnatal protective factor

Mother-child attachment refers to the level of security in the relationship between a mother and child (Ainsworth, 1978). Attachment can be measured throughout development, but is often first formed in parent-child relationships during infancy (Bowlby, 1979). Attachment is most frequently categorized into secure and insecure classifications (Ainsworth, 1978). A secure

attachment is predictive of better parent-child relationship functioning and emotional functioning for the child (Sroufe, 2001; Walsh & Zadorian, 2023). Additionally, the effects of attachment may exert themselves beyond the early childhood developmental period. Adolescents may also evince positive effects in the form of emotional development and interpersonal development from having a secure attachment with their caregiver during infancy and toddlerhood (Brumariu & Kerns, 2010). Additionally, adolescents with an insecure attachment with their caregivers have more mental health problems than those individuals with a secure attachment (Rosenstein & Horowitz, 1996). A secure attachment bond between a caregiver and child may also be protective for those who have experienced adversity. Research shows that secure parent-child attachment can protect against the effects of poor emotion regulation on mental health disorder diagnoses (Kim et al., 2013). A secure attachment style to caregivers may also protect against maladaptive child outcomes by weakening the association between prenatal adversity and child psychosocial functioning (Bergman et al., 2008). The long-term protective effects of attachment should be examined longitudinally and across developmental stages to elucidate the protective effects of having a secure attachment between a caregiver and child despite experiencing adversity.

The current study

Although research demonstrates the effects of singular forms of prenatal adversity on adolescent well-being, investigations of cumulative prenatal adversity are lacking. Excluding cumulative metrics of prenatal adversity fails to account for the tendency for multiple types of co-occurring adversity. Additionally, minoritized individuals may be at higher risk of experiencing compounding forms of prenatal adversity due to social position and systemic inequalities. Despite experiencing risk, a secure mother-child bond may repair the deleterious effects of prenatal adversity on adolescent anxiety and depressive symptoms, but has not been

utilized in research to examine later offspring mental health during adolescence. Thus, I utilized a diverse multiethnic sample to investigate attachment during toddlerhood as a moderator of the association between cumulative prenatal adversity and adolescent anxiety and depressive symptoms (See Figure 1). The present study includes two aims. First, I investigated the extent to which cumulative prenatal adversity is associated with adolescent anxiety and depressive symptoms at age 15. Second, I examined whether secure attachment during toddlerhood served as a protective factor to attenuate the association between cumulative prenatal adversity and adolescent anxiety and depressive symptoms. I hypothesize that higher levels of cumulative prenatal adversity will result in higher levels of adolescent anxiety and depressive symptoms. I also hypothesize that attachment during toddlerhood will significantly moderate the association of cumulative prenatal adversity and adolescent anxiety and depressive symptoms such that secure attachment will weaken the influence of prenatal adversity on adolescent anxiety and depressive symptoms at age 15.

Method

Participants

Participants were from the Future Families and Child Well-being Study (FFCWS). FFCWS is a stratified birth cohort study of 4898 children who were born in large U.S cities between 1998 and 2000 (populations over 200,000) (See Table 1). Participants were oversampled for unmarried mothers by a 3 to 1 ratio, which resulted in a larger number of Black, Hispanic, and low-income families. Baseline measurement was collected shortly after birth, and follow-up interviews of mothers and fathers were conducted when children were ages 1, 3, 5, and 9 years old. Primary caregivers were interviewed at ages 15 and 22 years old. Interviews with children were conducted at ages 9, 15, and 22 years old. Medical records were obtained for mothers and

children from the birth hospitalization record at baseline. Birth hospitalization records were collected by hospital liaisons at hospitals where mothers delivered their babies. FFCWS research staff coded the medical records for prenatal care, labor, and postnatal health information. Medical records were obtained for 3,684 participants. Missing records occurred for one of three reasons: hospitals were unwilling to provide records or unable to provide documentation due to financial feasibility (38%), the mother did not provide consent (33%), or hospital staff were unable to locate medical records (29%). Attachment was measured during the in-home assessment at age three; 2,268 families had available attachment data. The current study maintained two separate analytic samples, which was a result of missing data due to availability of medical records and completion of parent-child attachment classification at age three. Analyses pertaining to the long-term impacts of prenatal adversity utilize a larger sample of mothers with available medical records ($N = 3119$) and analyses pertaining to moderation by attachment classification utilize a smaller subset of families with both medical record and attachment data ($N = 1482$). As such, demographics are reported for both the larger analytic sample for main effects of prenatal adversity analyses, and the reduced subsample for moderation analyses that examined protective effects of attachment. At the time of the child's birth, the majority of parents were unmarried (87.60%). The average family income at baseline was reported at \$45,450.81 ($SD = \$45,007.08$). The majority of the sample were minoritized families (54.60% Black, 29.30% Hispanic 16.10% White). Due to analyses examining findings stratified by race/ethnicity, families reporting other racial/ethnic backgrounds were omitted (due to insufficient sample size).

Measures

Prenatal Adversity

To create an index of prenatal exposure to adversity, baseline surveys at birth from mothers and medical records from birth were examined. To achieve an index of prenatal adversity that is akin to postnatal ACEs measures, a broad range of adversity types were included from various ecological contexts (e.g., experiencing violence or abuse, parental mental health problems, and instability due to parental separation). As such, all variables were scaled such that higher scores reflected higher adversity exposure during pregnancy. Items that were scored on a yes/no scale were scored as 0=No and 1=Yes. The absence of prenatal adversity reflected as a 0 within the construct, and 1 reflects the presence of prenatal adversity within the construct. Scores were summed for all participants ($0 = \text{minimum score}$; $6 = \text{maximum score}$) to indicate the total amount of prenatal adversity a mother had experienced.

Inadequate Prenatal Nutrition. Nutritional inadequacy during pregnancy was extracted from the medical records. A single code for ‘nutritional inadequacy’ was coded by FFCWS research staff for any indication of nutritional inadequacy during pregnancy and scored a 1 if yes and 0 if no. If there were no indications of maternal nutritional inadequacy throughout her pregnancy in the medical records, nutritional inadequacy was scored as 0. Although nutritional adequacy was measured with a single variable, it has been included in prior research to examine prenatal conditions in the FFCWS sample (Burdette et al., 2012).

Interpartner Violence. Interpartner violence reflects the presence of physical abuse in the couple relationship perpetrated by the child’s father toward the mother. Mothers were asked one item regarding how often their partner engaged in violent behavior with them (e.g., how often would you say that he hits or slaps you when he is angry) ($1 = \text{Often}$; $2 = \text{Sometimes}$; $3 =$

Never). This item has been utilized in prior research that examines interpartner violence in FFCWS (Juan et al., 2020; Chen and Yookyong, 2021). This item was recoded to reflect a dichotomous score for the presence of any IPV such that both *often* and *sometimes* were scored as 1 and *never* was scored as 0.

Maternal Substance Use. Maternal substance use was extracted from medical records to reflect whether mothers used illicit drugs, cigarettes, or alcohol while pregnant. FFCWS research staff coded for maternal substance use during pregnancy from the medical records reflecting medical notes written by medical personnel. Medical records were coded for the use of specific substances (e.g., alcohol, tobacco, amphetamines, cocaine, heroin, marijuana) during pregnancy. Medical records were also coded for laboratory tests for urine toxin screens. Medical records were also coded for newborn conditions reflecting prenatal substance use (e.g., fetal alcohol syndrome, drug withdrawal). Substance use was coded as 1 if the mother had any indication of illicit drug, cigarette, or alcohol use during pregnancy in the medical records. Substance use was coded as 0 if the mother had no indications of substance use during her pregnancy in the medical records. The method measuring prenatal substance use is consistent with previous research in FFCWS (Huang & Reid, 2006).

Poverty. Mothers reported family income and household size at baseline surveys, and FFCWS research staff computed their income to needs ratio relative to the appropriate federal poverty thresholds in accordance with family composition and year. If families were below the federal poverty line, poverty was coded as 1. If families were at or above the federal poverty line, poverty was coded as 0. Poverty level has been utilized in previous FFCWS studies (Assari & Zare, 2024; Kravitz-Wirtz et al., 2022).

Material Hardship. Material hardship data were collected from baseline surveys to reflect the presence of an inability to financially make ends meet, regardless of an indication of poverty. Mothers responded to four items that probed the extent to which they utilized federal assistance programs. Of these four items, two items assessed whether the participant lived in a public housing project at any point during pregnancy and if the federal/state/local government was helping to pay for their rent at the time of data collection. The remaining two items measured whether the participant had any income from public assistance, welfare, food stamps, unemployment, social security, or disability in the past year. Mothers responded to all four items on a yes/no scale. If mothers responded *yes* to any of the four items, material hardship was coded as 1. If mothers responded *no* to all 4 items, material hardship was coded as 0. Prior studies utilizing the FFCWS sample have utilized housing assistance items as indicators of material hardship (Leifheit et al., 2020).

Paternal Incarceration. Paternal incarceration reflects whether or not the baby's father resided in prison during the mother's pregnancy or at the time of birth. Mothers responded to 2 items: one that asked whether the father was in jail or prison at any time during the pregnancy (*1 = Yes; 0 = No*) and one that asked whether the father was currently in jail or prison at birth (*1 = Yes; 0 = No*). If participants responded *yes* to either of these items, paternal incarceration was coded as 1. If mothers responded *no* to both of these items, paternal incarceration was coded as 0. Prior research from FFCWS has utilized this variable to examine paternal incarceration rates (Turney & Haskins, 2019).

Caregiver-Child Attachment

Mothers completed the 39-item Toddler Attachment Q-Sort (Andreassen & Fletcher, 2007) at age 3 to assess attachment. Mothers were asked to sort 39 cards that contained behaviors and

characteristics about their child based on the frequency of these characteristics and behaviors. Scoring of classifications was conducted as described by Kirkland and colleagues (2004). Briefly, data-driven models including multidimensional scaling, factor analysis, and hierarchical clustering were conducted to obtain three categorical attachment classifications: secure, insecure-avoidant, and insecure-resistant. Attachment Q-Sort has good convergent validity with the Strange Situation Procedure, a validated measure that examines attachment (Van Ijzendoorn et al., 2004). Due to small sample size for the insecure-avoidant classification (Secure = 75.8%; Insecure-resistant = 22.1%; Insecure-avoidant = 2.1%), attachment was scored insecure vs secure ($1 = \text{Secure attachment}$; $0 = \text{Insecure attachment}$). The practice of categorizing attachment by secure and insecure attachment is a common practice for researchers who utilize the FFCWS attachment data (Pudasainee-Kapri & Razza, 2015).

Adolescent Internalizing Symptoms

Adolescent internalizing symptoms was measured by self-reports of both anxiety and depressive symptoms.

Anxiety symptoms. Anxiety symptoms were measured utilizing an abbreviated version of the Brief Symptom Inventory (BSI) scale (Derogatis, 2001). Teens responded to 6-items from the original 18-item scale and reported how strongly they agreed with statements about a range of behaviors or feelings in the past four weeks (e.g., I have spells of terror or panic, I feel nervous or shaky inside). Higher scores reflected higher levels of anxiety symptoms ($1 = \text{Strongly disagree}$; $2 = \text{Disagree}$; $3 = \text{Agree}$; $4 = \text{Strongly agree}$). Reliability for anxiety symptoms in the current sample was adequate ($\alpha = .76$). A mean score was created, such that higher scores indicate higher levels of anxiety symptoms.

Depressive Symptoms. Depressive symptoms were measured utilizing an abbreviated set of 5 items from the Center for Epidemiologic Studies Depression Scale (CES-D)(Radloff, 1977). Youth reported how strongly they agreed with 5 statements during the last 4 weeks (e.g., In the last 4 weeks I felt sad, I felt life was not worth living). Higher scores reflected higher levels of depressive symptoms (*1 = Strongly disagree; 2 = Disagree; 3 = Agree; 4 = Strongly agree*). Reliability for depressive symptoms in the current sample was adequate ($\alpha = .76$). A mean score was created, such that higher scores indicate higher levels of depressive symptoms.

Covariates

Covariates include child sex, race and ethnicity, parental marital status at baseline, birth weight in grams, adolescent age at the time mental health symptoms were assessed, maternal depression at year 1, and child internalizing symptoms at age three. Child sex, race and ethnicity, and parental marital status at baseline were reported by mothers at baseline. Birth weight was extracted from medical records. Maternal depression at year 1 was assessed utilizing the Composite International Diagnostic Interview Short Form (CIDI-SF)(Kessler et al., 1998). The CIDI consists of 11 questions that mothers responded to regarding their mood in the last year (e.g., losing interest, feeling tired and worthless) reflecting the first year postpartum. Mothers were considered to be depressed if they met depression criteria for a diagnosis. Lastly, child internalizing symptoms at age three assessed the child's anxious/depressed and withdrawn symptoms utilizing the Child Behavior Check List (CBCL) (Achenbach, 1992). At age three, primary caregivers responded to 16 items and reported whether a given statement was true regarding their child's behaviors and characteristics (*0 = Not True; 2 = Very True*) (e.g., is nervous or high-strung) ($\alpha = .66$). Mean scores were computed such that a higher score indicated higher internalizing symptoms.

Data Analytic Plan

First, descriptive statistics were examined and reported. Descriptive statistics in the current study include prenatal risk indices and attachment frequencies as well as means and standard deviations among study variables. Bivariate correlations were calculated among all study variables. Additionally, analysis of variance (ANOVAs) were conducted to examine differences among in study variables by child sex, race/ethnicity, attachment classification, and maternal postnatal depression.

Regression analyses were conducted in Mplus 8.0 (Muthén and Muthén 1998-2017) to elucidate the effect of maternal prenatal adversity exposure on adolescent off-spring depressive and anxiety symptoms. As a follow-up to main analyses, a series of sensitivity analyses were conducted. Analyses were stratified by child sex and stratified by race/ethnicity to test whether associations among prenatal adversity and adolescent symptoms varied across these groups. Next, sensitivity analyses were conducted to examine whether associations between prenatal adversity and adolescent mental health persisted when controlling for postnatal maternal depression throughout the first year postpartum. This sensitivity analysis was conducted to examine whether the effects of prenatal adversity may operate through maternal postnatal depression. A sensitivity analysis was also conducted to examine whether associations with adolescent mental health persist when controlling for internalizing symptoms at age three. This sensitivity analysis examined whether associations with adolescent mental health hold over and above internalizing symptoms earlier in development.

Lastly, analyses were stratified by attachment classification (e.g., secure, insecure) to examine whether associations among prenatal adversity and adolescent mental health differ by attachment classification in toddlerhood. Across regression analyses, child sex, race and

ethnicity, parental marital status at baseline, birth weight in grams, and adolescent age at wave 6 were used as covariates when analyzing both the larger sample and the reduced sample.

Results

Descriptive Statistics

Means, standard deviations, and ranges for the prenatal adversity index and adolescent depressive and anxiety symptoms can be found in Table 2. Frequencies are reported for both the full analytic sample (e.g., prenatal risk sample), as well as the subsample of individuals who had attachment data during toddlerhood (e.g., attachment subsample). Experiences of prenatal adversity in this sample ranged from 1 to 5 prenatal risk experiences. Women, on average, experienced 1.76 ($SD = .82$) forms of prenatal adversity. Individual prenatal risk frequencies and means can be found in Table 3. Material hardship was the most frequently experienced type of prenatal risk for both samples. Bivariate correlations for all study variables are displayed in Table 4. Prenatal adversity was significantly correlated with insecure attachment, adolescent depressive symptoms, and adolescent anxiety symptoms such that higher prenatal adversity was correlated with more frequent classifications of insecure attachment and increased anxiety and depressive symptoms. Adolescent anxiety symptoms and adolescent depressive symptoms were also positively correlated with one another.

Race/Ethnicity Differences

A one-way between-subjects analysis of variance (ANOVA) was conducted to examine whether study variables differed by race and ethnicity (See Table 5). There were no significant differences among racial and ethnic groups in adolescent depressive and anxiety symptoms at age 15. However, there were significant differences among racial and ethnic groups in prenatal

risk exposure. Black mothers ($M = 1.90$; $SD = .84$) experienced more prenatal adversity than both White mothers ($M = 1.60$; $SD = .80$) and Hispanic mothers ($M = 1.60$; $SD = .75$).

Child Sex Differences

A one-way between-subjects ANOVA was conducted to examine whether mental health symptoms differed significantly by child sex (See Table 6). Indeed, consistent with research in adolescence, girls reported having significantly higher levels than boys in both depressive symptoms (Girls $M = .70$; $SD = .63$; Boys $M = .55$; $SD = .56$) and anxiety symptoms (Girls $M = 1.88$; $SD = .69$; Boys $M = 1.79$; $SD = .62$).

Attachment Classification Differences

A one-way between-subjects ANOVA was conducted to examine whether attachment classifications differed significantly for specific prenatal risk indicators (See Table 7). The majority of prenatal risk indicators were not related to postnatal attachment. The only prenatal risk indicator that significantly differed in attachment classification was domestic violence, such that mothers who were categorized as having insecure attachments with their children during toddlerhood reported higher rates of prenatal domestic violence exposure ($M = .08$; $SD = .27$) than those who were classified as having a secure attachment with their child ($M = .05$; $SD = .21$).

Postnatal Maternal Depression Differences

A one-way between-subjects ANOVA was conducted to examine whether prenatal risk indicators and attachment classifications differed by probable postnatal depression diagnoses (See Table 8). Indeed, mothers who met diagnostic criteria for depression 1 year after birth had higher percentage of children with insecure attachment classifications ($M = .31$; $SD = .47$) than for those mothers who did not meet depressive diagnostic criteria 1 year after birth ($M = .25$; $SD = .43$). Cumulative prenatal risk scores were also higher for women who later met depressive

diagnostic criteria 1 year after birth ($M=1.84$; $SD = .86$) than for women who did not meet depressive diagnostic criteria ($M=1.74$; $SD = .80$). Among the individual indicators of prenatal risk, there were no differences among mothers who experienced postnatal depression among most individual forms of prenatal risk with the exception of rates of maternal substance use. Mothers who met depressive diagnostic criteria during the first postnatal year had higher rates of prenatal substance use ($M=.37$; $SD = .48$) compared to those who did not experience postnatal depression ($M=.30$; $SD = .46$).

Prenatal Risk and Adolescent Depressive and Anxiety Symptoms

Regression analyses were conducted to examine the main effects of cumulative prenatal risk on adolescent depressive and anxiety symptoms at age 15. Covariates included child sex, race/ethnicity, parental marital status, youth age at the adolescent assessment, and birth weight in grams. Results are shown in Table 9. More cumulative prenatal risk was associated with higher adolescent depressive symptoms ($\beta = .06$, $p = .01$). The effects of cumulative prenatal risk on adolescent anxiety symptoms did not reach statistical significant but approached significance ($\beta = .04$, $p = .06$).

Sensitivity analyses

To examine the robustness of the effects of prenatal adversity on adolescent depressive and anxiety symptoms, a series of additional sensitivity analyses were conducted. First, results were stratified by child sex and separately stratified by race/ethnicity. Second, analyses controlled for postnatal maternal depression to account for potential confounding effects. Lastly, analyses controlled for children's earlier internalizing symptoms to examine whether associations with adolescent hold over and above earlier emerging internalizing problems.

Analyses Stratified by Sex. Analyses examined the effect of prenatal adversity on adolescent depressive and anxiety symptoms stratified by child sex. Results are shown in Table 10. More cumulative prenatal risk was associated with higher depressive symptoms for girls ($\beta = .07, p = .04$), but not for boys ($\beta = .05, p = .09$). Similar to the full sample analysis, cumulative prenatal risk were not associated with anxiety symptoms at age 15 for either boys ($\beta = .04, p = .20$) nor for girls ($\beta = .05, p = .17$).

Analyses Stratified by Race. Analyses examined the effect of prenatal adversity on adolescent depressive and anxiety symptoms stratified by race. Results are shown in Table 11. More cumulative prenatal risk was associated with both higher depressive symptoms ($\beta = .07, p = .02$) and higher anxiety symptoms ($\beta = .06, p = .04$) in Black adolescents. Cumulative effects of more cumulative prenatal risk for White youth were not associated with depressive symptoms ($\beta = .05, p = .34$), nor were they associated with anxiety symptoms ($\beta = .05, p = .44$). Lastly, cumulative effects of more cumulative prenatal risk for Hispanic youth were not associated with depressive symptoms ($\beta = .05, p = .34$), nor were they associated with anxiety symptoms ($\beta = -.00, p = .92$).

Sensitivity analyses controlling for maternal depression. Next, analyses examined the effect of prenatal adversity with postpartum maternal depression at one year included as a covariate to determine if associations between prenatal adversity hold over and above experiences of postnatal maternal depression. Results are shown in Table 12. Postnatal maternal depression was associated with higher levels of adolescent depressive symptoms ($\beta = .06, p = .01$). However, when controlling for all previously included covariates as well as postpartum maternal depression, cumulative prenatal risk remained a significant predictor of adolescent depressive symptoms ($\beta = .06, p = .01$). Results also remained non-significant at the cutoff value

of $p < .05$ for adolescent anxiety symptoms ($\beta = .04, p = .07$); however, effects approached significance.

Sensitivity analyses controlling for earlier internalizing problems in main analyses.

Next, children's internalizing symptoms at age three were included as a covariate to examine whether associations with prenatal adversity remain when accounting for earlier child symptoms. Results are shown in Table 13. When controlling for all previously included covariates as well as internalizing symptoms at age three, prenatal adversity remained associated with adolescent depressive symptoms ($\beta = .05, p = .03$). Results remained non-significant with adolescent anxiety symptoms ($\beta = .03, p = .21$).

Analyses examining Attachment Security as a Moderator

Regression analyses stratified by attachment classification were conducted to examine whether secure attachment served as a buffer between the effects of prenatal adversity on adolescent depressive and anxiety symptoms. Results are shown in Table 14. More prenatal risk was associated with increased adolescent depressive symptoms for offspring with an insecure attachment with their caregiver ($\beta = .13, p = .04$), but not for offspring with a secure attachment with their caregiver ($\beta = .03, p = .35$) consistent with hypotheses that secure attachment may buffer the effects of prenatal adversity. Prenatal cumulative risk was not associated with anxiety symptoms either attachment classification (secure: $\beta = -.00, p = .96$; insecure $\beta = .07, p = .27$).

Discussion

The current study aimed to investigate the effects of cumulative prenatal adversity exposure on adolescent offspring internalizing symptoms as well as test the protective role of a secure attachment during early childhood in these associations. Findings partially supported my first hypothesis; prenatal adversity was associated with depressive symptoms in adolescents and

anxiety symptoms approached significance, but did not meet cutoff scores for significance. These findings are consistent with prior research that suggests maternal prenatal adversity is associated with worse mental health outcomes for their offspring (Braithwaite et al., 2014; Clayborne et al., 2024; Kim et al., 2015; Silveira et al., 2017). Furthermore, research utilizing the Future Families and Child Well-being Study have shown prenatal adversity is associated with earlier behavioral outcomes during childhood in this study (Mack & Chavez, 2014). Sensitivity analyses stratified by child sex and race/ethnicity show these prenatal adversity effects are found among female youth as well as Black youth.

Secure attachment buffered against the negative effects of prenatal adversity on adolescent depressive symptoms. These findings suggest that a secure attachment with a caregiver is protective against adversity and these protective effects may extend across developmental periods throughout the lifespan. This highlights the notion that postnatal environments can have protective effects after the occurrence of prenatal adversity and are imperative to considering lifelong health following prenatal risk. This is consistent with a robust literature that suggests a secure attachment between a caregiver and child is protective against the negative effects of adversity more generally (Bergman et al., 2008; Dagan et al., 2018; Kim et al., 2015). These findings are also consistent with research that examines the protective role of attachment against the effects of prenatal adversity on early childhood development (Bergman et al., 2008; Roth et al., 2013; Perry et al., 2017). However, the current study demonstrates the long-term effect of attachment on later outcomes that emerge during adolescence. As such, the findings in this study highlight the need to examine the longitudinal protective role of a secure parent-child attachment, following the experiences of prenatal adversity, across the lifespan.

On the other hand, prenatal adversity was associated with increased depressive symptoms for children with an insecure attachment with their caregiver during toddlerhood. These findings are consistent with cumulative models of risk which reflect an accumulation of risk across multiple developmental periods being associated with maladaptive mental health outcomes for children (Chen, 2022; Evans & Whipple, 2013). Furthermore, these findings also reflect the importance of considering risk in both the prenatal and postnatal periods. Findings are consistent with prenatal cumulative risk models rather than prenatal-postnatal match-mismatch models (Conradt et al., 2018) highlighting the importance in reducing risk in multiple developmental periods.

Several sensitivity analyses were conducted to test the robustness of the main effects of prenatal adversity. Notably, results hold when controlling for postnatal maternal depression. This highlights that, even when accounting for having mothers who met diagnostic criteria for probable depression after birth, effects of prenatal adversity extended above and beyond maternal depression that may be an additional source of postnatal adversity. Findings also hold when controlling for internalizing symptoms measured during childhood. This suggests that prenatal adversity contributes to offspring mental health outcomes independently of the effects of both maternal postnatal depressive symptoms as well as earlier internalizing symptoms in childhood. Both maternal depression and earlier internalizing behaviors for offspring may have profound effects on adolescent depressive outcomes; however, controlling for these variables reflects a unique and separate influence of prenatal adversity on adolescent depressive and anxiety symptoms. This highlights that the effects of prenatal adversity continue to significantly predict anxiety and depressive symptoms in adolescence above and beyond earlier emerging internalizing problems.

Findings stratified by race/ethnicity and child sex suggest prenatal adversity is a risk factor for later adolescent mental health for females and Black youth. Findings for female youth may be explained by higher prevalence rates of depressive symptoms among females compared to males (Albert, 2015; Piccinelli & Wilkinson, 2000). These discrepancies among rates of depressive symptoms are also consistently found in adolescents and begin to emerge during early adolescence (Bennett et al., 2005; Nolen-Hoeksema & Girgus, 1994). Additionally, there were effects of prenatal adversity on adolescent depressive and anxiety symptoms at age 15 for Black youth when stratifying analyses by race/ethnicity. These results were specific to Black youth, and not found when analyzing the full sample. This finding is consistent with prior research that has found prenatal adversity to be associated with mental health outcomes for Black youth (Lefmann & Combs-Orme, 2014). Black mothers in this sample experienced significantly higher rates of prenatal adversity than White and Hispanic mothers (See Table 6), which may also contribute to these findings. Indeed, prior literature has also consistently found discrepancies among racial groups in the experience of adversity (Keyes, 2009), such that Black individuals are more likely to experience higher levels of adversity (Liu et al., 2018). Existing research also suggests that Black individuals may be more vulnerable to adversity than White individuals (Wickrama et al., 2016). It is important to point out that the sample of Black participants was larger than the samples of White and Hispanic mothers and these findings may in part also reflect differences in power due to sample size. Although the effects of prenatal adversity on White adolescent depressive and anxiety symptoms did not reach statistical significance, regression coefficients were similar in magnitude to those effects for Black youth. Similarly, the effects of prenatal adversity on adolescent depressive symptoms for Hispanic youth were similar in magnitude to those effects for Black youth even though these findings did not reach statistical significance.

This suggests that there may be similar patterns of anxiety and depressive symptoms as outcomes of prenatal adversity, but the significance of these findings may be explained by differences in sample size amongst racial and ethnic groups.

In this study, prenatal adversity exposure had direct effects on adolescent offspring depressive symptoms, and anxiety symptoms approached significance, but did not reach the cutoff value of $p < .05$. Although both depressive and anxiety symptoms contribute to the conceptualization of internalizing symptoms, they are unique in regard to their symptomology in their measurement which may result in findings similar in magnitude but leading to differences in their statistical significance. While both are related to mood dysfunction, are fundamentally similar, and often concordant, there are clinical distinctions between the two (Hamilton, 1983). Depressive symptoms reflect persistent feelings of sadness or low mood, a sense of hopelessness, and experiences of anhedonia (Stingaris, 2017). On the contrary, anxiety symptoms reflect intense unease or nervousness and a preoccupation of concern (Craske et al., 2011). Although results are consistent with past research for depressive symptoms (Clayborne et al., 2024), findings in the current study for associations between prenatal adversity and anxiety symptoms only trended toward significance. This suggests that there may be similar patterns between anxiety and depressive symptoms, but there may be variation in specific symptoms of the scales utilized in the current study. However, these studies differ in their measures of anxiety and may be limited by the use of an abbreviated measure in the current study. Future research is needed to disentangle the two outcomes separately as well as elucidate the pathways which predict their onset.

The present study is not without limitations which includes the attachment measure at age three being reported by primary caregivers. Caregivers may be inclined to report characteristics

of secure attachment due to social desirability. Furthermore, attachment may be impacted by the experience maternal adversity with mothers who experience higher levels of adversity more likely to be classified as having an insecure attachment with their child. The attachment measure is also limited as it may not capture insecure avoidant classifications. Additionally, not all mothers had available medical records which limited the investigation of adversity in the full FFCWS sample. Roughly one-third of medical records were unavailable due to either financial feasibility, the mother not providing consent, or hospital staff not being able to locate medical records. Consequently, participants without medical records were not included in this investigation due to a lack of data for prenatal adversity measures. Demographics of excluded mothers in main analyses differed such that mothers with missing medical record data were likely to be older, married to the child's father, report higher income, and have a higher level of education than mothers who had extracted medical record data. As such, mothers experiencing more social disadvantage were more likely to be in this analysis. Similar to this point, there were also missing data for mother-child attachment for families that did not participate in the in-home assessment at age 3. As such, lack of attachment data required the utilization of two separate analytic samples for main analyses and multigroup analyses based on attachment classification. It is important to note, the subsample with attachment data in early childhood were similar to those in the broader sample with regards to experiences of prenatal adversity.

The current study has notable strengths. Previous research that examines prenatal adversity to predict offspring well-being often fails to account for multiple forms of prenatal adversity and neglect the co-occurrence of these experiences. The current study extended past conceptualizations of prenatal adversity by developing a cumulative metric of prenatal adversity that incorporates six unique forms of adversity across different ecological levels. Consistent with

prenatal programming mechanisms, the prenatal index created in this study broadly capture adversity that may operate through stress biology or nutritional signals that cross the placental barrier. During the prenatal period, mothers serve as a filter through which adversity reaches the fetus; it may be the cumulative exposure to adversity through these stress and natural signals rather than individual types that reach the fetus. Lastly, a notable strength of the current study is that the utilized a diverse, multiethnic sample of families that included multiple racial and ethnic groups and geographical locations across the United States allows for greater generalizability.

Conclusion

The findings from the current study expand upon existing literature the examines the effects of prenatal adversity on adolescent mental health outcomes and adds to the broad attachment research to highlight the protective effects of a secure attachment in developmental pathways of prenatal adversity and adolescent mental health. The current study highlights the long-term importance of cumulative prenatal adversity consistent with lifespan perspectives. Higher exposure to prenatal adversity was associated with adolescent depressive symptoms 15 years later. Additionally, implications of this research provide evidence for the need for preventative interventions that help caregivers to form a secure attachment with their child despite their exposure to adversity as well as the need for programs to reduce exposure to prenatal adversity. Material hardship and poverty were the most prevalent form of prenatal adversity in this study; financial resources for pregnant mothers may substantially reduce the lifelong effects of prenatal adversity. The current study elucidates the protective effects of attachment against the negative effects of prenatal adversity that exert onto offspring, but specifically highlights the importance of measuring prenatal adversity with a cumulative lens given exposure to the fetus operates through maternal experiences. Researchers should continue

to examine the effects of adversity across the lifespan to ascertain processes that contribute to adolescent mental health outcomes.

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Table 1. Sample Demographics of Study 1

Construct	<i>M (SD) or N (%)</i>	
	Prenatal Risk Sample	Attachment Sample
<i>N</i>	3119	1482
Marital Status <i>N (%)</i>		
Married	388 (12.40)	156 (10.50)
Non-Married	2731 (87.60)	1326 (89.50)
Sex <i>N (%)</i>		
Female	1469 (47.10)	703 (47.40)
Male	1650 (52.90)	779 (52.60)
Youth Age at Wave 6 (Year) <i>M (SD)</i>	15.66 (.71)	15.46 (.51)
Family Income at Birth <i>M (SD)</i>	\$45,450.81 (\$45,007.08)	\$43,421.54 (\$43,679.14)
Race/Ethnicity <i>N (%)</i>		
White	501 (16.10)	218 (14.70)
Black	1704 (54.60)	930 (62.80)
Hispanic	914 (29.30)	334 (22.50)

Table 2. Descriptive Statistics for Study Variables

Construct	Prenatal Risk Sample			Attachment Sample		
	<i>M (SD)</i>	Min-Max	<i>N</i>	<i>M (SD)</i>	Min-Max	<i>N</i>
Prenatal Adversity	1.76 (.82)	1-5	3231	1.82 (.82)	1-5	1482
Anxiety Symptoms	1.83 (.66)	1-4	2256	1.86 (.66)	1-4	1227
Depressive Symptoms	.62 (.60)	0-3	2256	.63 (.59)	0-3	1227

Table 3. Prenatal Risk Frequencies and Descriptive Statistics

	Prenatal Risk Sample	Attachment Sample
Prenatal Risk Indicator	<i>N (%)</i>	<i>N (%)</i>
Interpartner Violence	180 (5.80%)	82 (5.50%)
Poverty	1782 (57.10%)	839 (56.60%)
Material Hardship	2204 (70.70%)	1110 (74.90%)
Maternal Substance Use	989 (31.70%)	478 (32.30%)
Parental Incarceration	180 (5.80%)	94 (6.30%)
Inadequate Nutrition	164 (5.30%)	89 (6.00%)

Table 4. Correlation Table

	1	2	3	4	5	6	7	8	9
1. Prenatal Adversity	1.00								
2. Insecure Attachment	.06*	1.00							
3. Adolescent Anxiety Symptoms	.06**	.04	1.00						
4. Adolescent Depressive Symptoms	.06**	.04	.65**	1.00					
5. Marital Status	-.20**	-.05	-.01	-.03	1.00				
6. Child Sex	-.00	.05	-.08**	-.12**	.00	1.00			
7. Youth Age at 15	.04*	.08**	-.03	.01	-.01	.02	1.00		
8. Birth Weight in Grams	.05**	.01	.06**	.02	-.04*	-.03	.02	1.00	
9. Maternal Depressive Symptoms	.05**	.07*	.04	.07**	-.03	.00	-.03	.04	1.00

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. Child Sex 0 = Girl, 1 = Boy; Marital Status 0=Unmarried, 1=Married; Attachment 0 = Secure, 1 = Insecure.

Table 5. Comparisons of Prenatal Risk and Adolescent Mental Health By Race and Ethnicity

Prenatal Risk Sample							
Construct	White <i>M (SD)</i>	Black <i>M (SD)</i>	Hispanic <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
Prenatal Risk	1.60 (.80)	1.90 (.84)	1.60 (.75)	36.33	2	<.001***	B > W; B > H
Anxiety Symptoms	1.87 (.66)	1.81 (.66)	1.86 (.64)	1.02	2	.38	Non-significant
Depressive Symptoms	.65 (.65)	.61 (.59)	.62 (.58)	.44	2	.73	Non-significant

Note. Significant pairwise comparisons are reported utilizing the Bonferonni posthoc comparison; *** $p < .001$; ** $p < .01$; * $p < .05$.

Table 6. Comparisons of Adolescent Mental Health Symptoms by Child Sex

Prenatal Risk Sample						
Construct	Girls <i>M (SD)</i>	Boys <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
Anxiety Symptoms	1.88 (.69)	1.79 (.62)	12.04	1	<.001	Girls > Boys
Depressive Symptoms	.70 (.63)	.55 (.56)	33.60	1	<.001	Girls > Boys

Table 7. Comparisons of Prenatal Risk Types By Attachment Classification

Construct	Attachment Sample		<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
	Secure <i>M (SD)</i>	Insecure <i>M (SD)</i>				
Maternal Substance Use	.32 (.47)	.32 (.47)	.01	1	.93	Non-significant
Parental Incarceration	.06 (.23)	.08 (.28)	3.07	1	.08	Non-significant
Domestic Violence	.05 (.21)	.08 (.27)	4.59	1	.03	Insecure > Secure
Prenatal Poverty	.56 (.50)	.57 (.50)	.14	1	.71	Non-significant
Material Hardship	.74 (.44)	.77 (.42)	1.67	1	.20	Non-significant
Nutritional Inadequacy	.06 (.24)	.05 (.23)	.42	1	.52	Non-significant
Cumulative Prenatal Risk	1.79 (.81)	1.89 (.83)	3.62	1	.06	Non-significant

Table 8. Comparisons of Prenatal Risk and Attachment By Postnatal Maternal Depression

Construct	Prenatal Risk Sample					<i>Pairwise Comparisons</i>
	Non-Depressed <i>M (SD)</i>	Depressed <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>p</i>	
Maternal Substance Use	.30 (.46)	.37 (.48)	7.64	1	.01	Depressed > Non-Depressed
Parental Incarceration	.05 (.23)	.07 (.25)	1.44	1	.23	Non-significant
Domestic Violence	.05 (.22)	.06 (.24)	.56	1	.45	Non-significant
Prenatal Poverty	.57 (.50)	.55 (.50)	.47	1	.49	Non-significant
Material Hardship	.71 (.45)	.72 (.45)	.19	1	.66	Non-significant
Nutritional Inadequacy	.05 (.22)	.07 (.25)	3.40	1	.07	Non-significant
Cumulative Prenatal Risk	1.74 (.80)	1.84 (.86)	6.00	1	.01	Depressed > Non-Depressed
Insecure Attachment	.25 (.43)	.31 (.47)	5.13	1	.02	Depressed > Non-Depressed

Table 9. Summary of Regression Analyses for the Main Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15

Construct	Depressive Symptoms				Anxiety Symptoms			
	β	B	SE	p	β	B	SE	p
Prenatal Risk	.06	.05	.02	.01	.04	.04	.02	.06
<i>Covariates</i>								
Marital Status	-.03	-.05	.04	.17	-.02	-.03	.04	.43
Child Sex	-.12	-.15	.03	<.001	-.07	-.01	.03	<.001
Black	-.06	-.07	.04	.08	-.05	-.07	.04	.08
Hispanic	-.03	-.04	.04	.33	-.01	-.02	.04	.72
Youth Age at 15	.01	.01	.02	.69	-.02	-.02	.02	.27
Birth Weight	.03	.03	.02	.24	.04	.04	.03	.15
R^2	.02				.01			
N	3119							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), race/ethnicity (Reference category=White), child sex (0=Girl; 1 = Boy), and birth weight in grams. Numbers in bold were significant at $p < .05$.

Table 10. Summary of Regression Analyses for the Main Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15 Stratified by Child Sex

Construct	Boys								Girls							
	Depressive Symptoms				Anxiety Symptoms				Depressive Symptoms				Anxiety Symptoms			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
Prenatal Risk	.05	.04	.02	.09	.04	.03	.02	.20	.07	.05	.03	.04	.05	.04	.03	.17
<i>Covariates</i>																
Marital Status	-.06	-.11	.05	.02	-.04	-.07	.05	.18	.00	.01	.07	.92	.00	..01	.07	.94
Black	.01	.01	.05	.79	-.03	-.04	.05	.44	-.13	-.17	.06	.01	-.08	-.11	.06	.08
Hispanic	.03	.04	.05	.48	.02	.02	.06	.69	-.10	-.13	.07	.05	-.04	-.06	.07	.36
Youth Age at 15	.01	.00	.03	.88	-.04	-.03	.03	.19	.01	.01	.03	.77	-.01	-.01	.03	.71
Birth Weight	.05	.05	.03	.10	.06	.06	.04	.09	.00	.00	.04	.91	.02	.02	.04	.64
R^2	.03				.02				.04				.04			
<i>N</i>	1650								1469							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), race/ethnicity (Reference category=White), and birth weight in grams. Numbers in bold were significant at $p < .05$.

Table 11. Summary of Regression Analyses for the Main Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15 Stratified by Race

Construct	White								Black								Hispanic							
	Depressive Symptoms				Anxiety Symptoms				Depressive Symptoms				Anxiety Symptoms				Depressive Symptoms				Anxiety Symptoms			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
Prenatal Risk	.05	.04	.04	.34	.05	.04	.05	.44	.07	.05	.02	.02	.06	.05	.02	.04	.05	.03	.03	.34	-.00	-.00	.04	.92
<i>Covariates</i>																								
Marital Status	-.04	-.06	.08	.45	.02	.03	.09	.69	-.03	-.06	.06	.32	-.03	-.07	.07	.31	-.01	-.02	.07	.73	-.03	-.05	.07	.54
Child Sex	-.24	-.32	.07	<.001	-.13	-.18	.07	.01	-.10	-.11	.03	<.001	-.07	-.09	.04	.02	-.11	-.13	.05	.01	-.06	-.08	.05	.14
Youth Age at 15	.15	.13	.05	<.001	.05	.04	.05	.39	-.01	-.01	.03	.82	-.03	-.03	.03	.29	-.05	-.04	.03	.24	-.04	-.04	.03	.22
Birth Weight	.04	.04	.05	.48	.03	.04	.06	.55	.01	.01	.03	.77	.02	.02	.04	.58	.06	.05	.05	.30	.08	.08	.06	.19
R^2	.09				.02				.01				.01				.02				.01			
<i>N</i>	501								1704								914							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), child sex (0=Girl; 1 = Boy), and birth weight in grams. Numbers in bold were significant at $p < .05$.

Table 12. Summary of Regression Analyses for the Main Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15 – Controlling for Maternal Depression at Age 1

	Depressive Symptoms				Anxiety Symptoms			
Construct	β	B	SE	p	β	B	SE	p
Prenatal Risk	.06	.04	.02	.01	.04	.03	.02	.07
<i>Covariates</i>								
Marital Status	-.03	-.05	.04	.19	-.02	-.03	.04	.45
Child Sex	-.12	-.15	.03	<.001	-.07	-.10	.03	<.001
Black	-.06	-.07	.04	.08	-.05	-.07	.04	.09
Hispanic	-.03	-.04	.04	.37	-.01	-.01	.04	.75
Youth Age at 15	.01	.01	.02	.62	-.02	-.02	.02	.30
Birth Weight	.03	.03	.02	.01	.04	.04	.03	.16
Postnatal Maternal Depression	.06	.10	.04	.01	.04	.06	.04	.10
R^2	.03				.01			
N	3119							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), race/ethnicity (Reference category=White), child sex (0=Girl; 1 = Boy), birth weight in grams, and maternal depression at age 1. Numbers in bold were significant at $p < .05$.

Table 13. Summary of Regression Analyses for the Main Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15 – Controlling for Internalizing Symptoms at Age 3

Construct	Depressive Symptoms				Anxiety Symptoms			
	β	B	SE	p	β	B	SE	p
Prenatal Risk	.05	.04	.02	.03	.03	.02	.02	.21
<i>Covariates</i>								
Marital Status	-.03	-.05	.04	.26	-.01	-.02	.04	.61
Child Sex	-.13	-.15	.03	<.001	-.08	-.10	.03	<.001
Black	-.06	-.07	.04	.07	-.06	-.07	.04	.07
Hispanic	-.03	-.05	.04	.28	-.02	-.02	.04	.61
Youth Age at 15	.00	.00	.02	.89	-.03	-.03	.02	.16
Birth Weight	.03	.03	.02	.29	.03	.04	.03	.19
Internalizing Symptoms at Age 3	.08	.19	.06	<.001	.10	.26	.07	<.001
R^2	.03				.02			
N	3119							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), race/ethnicity (Reference category=White), child sex (0=Girl; 1 = Boy), birth weight in grams, and child internalizing problems at age 3. Numbers in bold were significant at $p < .05$.

Table 14. Summary of Multigroup Analyses for the Effect of Cumulative Prenatal Risk on Adolescent Depressive and Anxiety Symptoms at Age 15 by Attachment Security

Construct	Secure Attachment								Insecure Attachment							
	Depressive Symptoms				Anxiety Symptoms				Depressive Symptoms				Anxiety Symptoms			
	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>	β	B	SE	<i>p</i>
Prenatal Risk	.03	.02	.03	.35	-.00	-.00	.03	.96	.13	.09	.04	.04	.07	.05	.05	.27
<i>Covariates</i>																
Marital Status	-.03	-.06	.06	.37	.00	.01	.07	.93	-.03	-.06	.09	.50	-.04	-.08	.13	.53
Child Sex	-.12	-.14	.04	<.001	-.06	-.08	.04	.07	-.06	-.08	.07	.25	-.07	-.10	.07	.20
Black	.01	.02	.06	.77	-.03	-.05	.06	.47	-.04	-.05	.09	.57	-.04	-.05	.09	.57
Hispanic	-.03	-.04	.06	.50	-.02	-.03	.07	.65	.07	.10	.11	.36	.09	.14	.11	.22
Youth Age at 15	-.01	-.01	.04	.83	-.03	-.04	.04	.30	.08	.09	.07	.19	.12	.15	.07	.04
Birth Weight	.08	.08	.03	.01	.07	.08	.04	.06	-.03	-.03	.07	.68	.02	.02	.07	.80
R^2	.03				.01				.04				.04			
N	1106								376							

Note. Control variables were youth age at 15, marital status (0=Unmarried; 1 = Married), race/ethnicity (Reference category=White), child sex (0=Girl; 1 = Boy), and birth weight in grams. Numbers in bold were significant at $p < .05$.

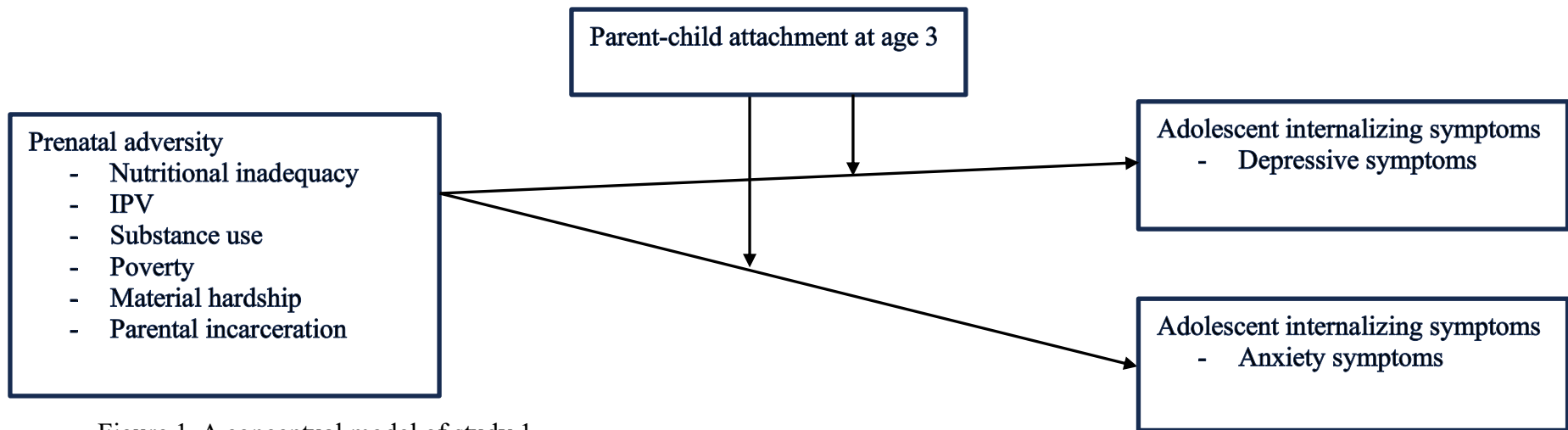


Figure 1. A conceptual model of study 1

CHAPTER 3

THE DEVELOPMENTAL CASCADE OF RISK ON MENTAL HEALTH: THE PROTECTIVE ROLE OF FAMILY COHESION AND PARENT BCES

Brown, R., et al. To be submitted to a peer-reviewed journal.

Abstract

Research to date has examined the negative effects of ACEs across generations; however, little is known about mechanisms through which these associations operate. Additionally, existing research fails to include protective factors at the familial level that may provide more insight toward resilience cascades. Utilizing data from a multigenerational study on 108 African American families, the current study examined the developmental cascade of adverse childhood experiences (ACEs) within and between families to elucidate how adversity may affect parenting, which in turn may affect offspring internalizing symptoms. Transmission of parenting was also examined between G1 and G2. Results showed that G2 ACEs were associated with lower G2 nurturant parenting, which in turn lead to higher G3 depressive symptoms. While moderation analyses were preliminary, there was support for protective effects of benevolent childhood experiences and protective and promotive effects of family cohesion. Black youth in America face unique stressors related to their social position, and these findings inform developmental research that examines multigenerational adversity and factors that offset maladaptive outcomes of adversity, particularly in vulnerable communities.

INDEX WORDS: Multigeneration; Adversity; Depression; Anxiety; Parenting; Family

Introduction

Adverse childhood experiences (ACEs) are negative and potentially traumatic early experiences that occur prior to age 18 (Felitti et al., 1998). ACEs include a broad range of childhood adversity experiences such as experiencing parental divorce, witnessing familial substance abuse, experiencing neglect and abuse, witnessing violence and other negative events (Ports et al., 2020). ACEs have been associated with maladaptive outcomes for individuals across the lifespan such as negative behavioral outcomes including internalizing symptoms, externalizing symptoms, and impulsivity (Bevilacqua et al., 2021; Muniz et al., 2019; Shin et al., 2018). Additionally, exposure to ACEs has been associated with negative physical health outcomes such as higher BMI, heart disease, and accelerated biological aging (Godoy et al., 2021; Mian et al., 2022; Wiss & Brewerton, 2020). Existing research suggests that nearly half (45%) of individuals in the United States have experienced at least one ACE (Sacks & Murphey, 2018), with nearly 10% of children experiencing three or more ACEs. Further, there are disparities in ACEs exposure among racial and ethnic groups. Minority youth experience ACEs at higher rates than their White peers, with 61% of Black youth experiencing at least one ACE (Sacks & Murphey, 2018). As such, the experience of ACEs and its impact on development among Black youth and families are necessary to better inform prevention and intervention research.

The experience of ACEs not only impacts an individual's later health and well-being but may also transfer to their offspring as well. Research shows negative intergenerational effects of adversity exposure (Zhang et al., 2023). However, the mechanisms through which ACEs impact

their offspring's well-being are unclear. Some research suggest that parenting behaviors and family environment may be potential mechanisms between the effects of parental ACEs and offspring well-being (Woods-Jaeger et al., 2018). Nevertheless, despite exposure to ACEs as children, some families may not experience disruptions in the family context as a result of parental ACEs exposure which, in turn, may lessen the negative impact of parental ACEs on offspring development. As such, it is imperative to investigate the parental experiences and family environments that operate as protective factors to buffer against the negative cascade of parental exposure to ACEs for their offspring.

Parental ACEs and child outcomes

The effects of experiencing ACEs during childhood may extend beyond an individual's own well-being to their offspring (McDonnell & Valentino, 2016). Further, these effects of parental ACEs on their offspring's well-being may operate through child-rearing processes. Parental exposure to ACEs has been associated with child well-being in myriad forms (Rowell & Neal-Barnett, 2022). For example, Le-Scherban and colleagues (2018) found that when parents reported higher experiences of ACEs, their offspring had worse physical health and engaged in poorer health behaviors such as increased TV and screen time. Additionally, parental ACEs has been associated with hyperactivity, behavioral problems, and psychopathology in their children (Rowell & Neal-Barnett, 2022; Schickedanz et al., 2018). Arnold and colleagues (2023) conducted a systematic review that examined the relation between parental ACEs and the health and well-being of their offspring. This review found that offspring physical growth (Noll et al., 2007), risk-taking behaviors (Pear et al., 2017), and educational attainment (Zvara & Burchinal, 2021) have all been associated with parental exposure to ACEs (Arnold et al., 2023).

Research has also found that parents with higher reports of ACEs tend to have children who report higher levels of psychopathology including higher depressive symptoms and anxiety symptoms (Fenerci & Allen, 2018; McDonald et al., 2019; Rowell & Neal-Barnett, 2022). Rowell & Neal-Barnett conducted a meta-analysis to investigate the association of parental ACEs and offspring psychopathology. Findings from this meta-analysis suggest that there is a significant direct association between parental exposure to ACEs and later offspring psychopathology (Rowell & Neal-Barnett, 2022) which has implications for lifelong health and well-being as earlier psychopathology significantly predicts poorer cognitive, social, and emotional functioning throughout the lifespan (Finsaas et al., 2020; McLaughlin et al., 2011; Romer & Pizzagalli, 2021). However, the majority of the studies that examine the effects of parental ACEs on offspring psychological and emotional development focus on developmental outcomes during early childhood (Hetherington et al., 2020; McDonald et al., 2019). Adolescence may be a particularly important stage of development to examine emotional and psychopathology outcomes in children of individuals who have experienced ACEs due to the divergence and rise of mental health outcomes during this stage (Kessler et al., 2007; Remschmidt, 2013). Ethnic and racial minority youth are also an especially important demographic to consider in this association due to increased prevalence rates of psychopathology including among Black youth (Lopez et al., 2017). Researchers also continue to hypothesize *how* the effects of parental ACEs may exert influence onto adolescent offspring. Rowell & Neal-Barnett (2022) posit parenting behaviors to be a potential mediator between parental ACEs and offspring psychopathology; however, there are limited studies examining this potential mechanism. As such, future research should examine the specific parenting behaviors and family

environment contexts to elucidate the mechanisms through which parental exposure to adversity impacts parenting which, in turn, impacts offspring psychopathology.

Parental ACEs and parenting

Parents who have reported experiencing higher levels of ACEs are more likely to report higher levels of parenting stress and aggravation in parenting (Lange et al., 2019; Schickedanz et al., 2018). Indeed, extant research suggests exposure to adversity negatively affects multiple aspects of parenting including higher levels of harshness, lower nurturance, and lower sensitivity (Bouvette-Turcot et al., 2020; Racine et al., 2018). The parent-child relationship and attachment bond may also be affected by parental ACE exposure (Racine et al., 2018). These characteristics of the dyadic relationship have consistently been associated with poorer outcomes for children across multiple domains including increased psychopathology such as depressive and anxiety symptoms, delinquency, and social aggression (Hinnant et al., 2015; Tang et al., 2018). Research has also found that higher ACE exposure for parents was associated with use of more harsh parenting behaviors (Shen & Wu, 2024). Despite clear associations between parental adversity exposure, parenting, and maladaptive outcomes for children, the mediating effect of parenting between parental adversity and child outcomes is not well understood. To my knowledge, there are only a handful of studies that examine parenting as a mediator between parental ACEs and offspring psychopathology. These studies that include parenting as a mediator between parental ACEs and child outcomes measure parenting in the form of discipline, harshness, hostility, and sensitivity (Banyard et al., 2003; Zvara et al., 2017). Specificity is important to utilize in future research to elucidate what type of parenting behaviors and what characteristics of family environment mediate the association between parental ACEs and offspring development (Rowell & Neal-Barnett, 2022; Zhang et al., 2023).

Determinants of parental nurturance

Nurturant parenting refers to a parent's extent to provide an emotionally warm environment for their child (Locke & Prinz, 2002). Parents who have been exposed to ACEs may also be less likely to engage in positive parenting practices due to deficits in emotional availability as a result of their trauma (Moran & Diamond, 2008). Babacan (2021) found that when mothers experienced higher rates of ACEs, they were significantly lower in parental nurturance. As such, nurturant parenting as a reflection of parent's emotional availability may mediate the association between parental ACEs and offspring development. Determinants of parenting are multi-causal; parent's nurturant parenting may be influenced by factors in addition to childhood adversity. Existing research also suggests that parenting behaviors may be transmissible across generations, and parenting behaviors in one generation are associated with parenting behaviors in future generations (Vermulst et al., 2017). Indeed, research has found multigenerational effects of nurturant parenting such that when first generation parents are higher in nurturance, second generation parents also exhibit higher levels of nurturance (Garcia et al., 2020). As such examining nurturant parenting across generations may inform research pertaining to multigenerational pathways of risk and resilience.

The effects of nurturant parenting on child development

The caregiver-child relationship is not only the first social relationship an individual will experience, but the most salient point of context in terms of proximity and influence (Acock, 1984). Nurturant parenting has been associated with many child outcomes such as behavioral functioning (Miller et al., 2022), emotional development (Pollak, 2012), and characteristics of the dyadic parent-child relationship such as attachment to caregiver (DePasquale & Gunnar, 2020). Additionally, in a study conducted on mother-child dyads, findings suggest that parental

nurturance and adolescent depressive symptoms are related to one another such that higher levels of parental nurturance predict lower adolescent depressive symptoms (Hou et al., 2020). Existing research also suggests that when parents are less nurturant, adolescent offspring may have higher levels of anxiety and depressive symptoms than their peers who experience more nurturant parenting (Urban, 2020). This may be explained by a tendency for nurturant parenting to lead to better self-esteem for offspring (Tafarodi et al., 2010), an individual characteristic highly correlated with anxiety and depressive symptoms (Sowislo & Orth, 2013). The role of nurturant parenting for Black youth, including among the parents in the sample utilized in this study, has been found to be protective against the effects of adversity (Brody & Flor, 1998; Brody & Ge, 2001; Brody et al., 2019). Consequently, nurturant parenting may be an important mechanism among Black families to promote positive psychological development in their children through their parenting.

Resilience

Despite parental exposure to adversity during childhood, disruptions to the parenting experience may not occur. Additionally, the effects of early life adversity may not exert themselves onto the next generation of offspring due to the presence of protective factors. Protective factors serve to buffer the negative association of adversity onto maladaptive outcomes. Minimal research has been conducted to examine what factors may offset the risk of parental ACEs onto adolescent psychopathology. Therefore, it is imperative for researchers to examine the association between parental ACEs and offspring psychopathology during adolescence with the inclusion of both mechanisms that facilitate this association and the protective factors that disrupt this association. Both risk and protective factors should be included to better understand heterogeneity in intergenerational effects of parental exposure to

adversity. In intergenerational research throughout the lifespan of both generations, there are important places for intervention following exposure to adverse childhood experiences.

Benevolent childhood experiences

Benevolent childhood experiences (BCEs), the counterpart to adverse childhood experiences, refers to a cumulative subset of positive experiences that an individual may experience prior to the age of 18 (e.g., having a safe caregiver, having a predictable home routine) (Narayan et al., 2023). Individuals with higher levels of BCEs are more likely to adjust positively across the lifespan for both behavioral and physical health outcomes (Doom et al., 2021; Raghunathan et al., 2024). Research also demonstrates that individuals who experience higher number of BCEs during childhood are less likely to develop mental health problems such as depression, anxiety, and post-traumatic stress disorder later in adulthood (Johnson et al., 2022; Lucke, 2024). Moreover, BCEs have also been shown to counteract the experience of ACEs (Crandall et al., 2019; Fabio et al., 2024). In a study conducted on adult individuals who ranged from 19-57 years old, researchers found that the presence of BCEs neutralized the effects of ACEs on adult physical and mental health outcomes (Crandall et al., 2019). BCEs may also be particularly promotive for parents, and their parenting behaviors. Extant research has been conducted on parents' childhood experiences of BCEs and their effects on positive behaviors in parenting (Lucke, 2024; Narayan et al., 2019; Tadjine & Swords, 2024). Indeed, research reflects that BCEs promote positive parenting behaviors such as parenting effectiveness (Raghunathan et al., 2024). Parents who have been exposed to more positive experiences during childhood are more likely to engage in positive parenting (Crandall et al., 2019). Taking this into consideration, positive parental childhood experiences should be investigated as a potential moderator that may offset risk of the negative effects of parental ACEs on nurturant parenting. However, much of the

research on the effects of BCEs on parenting examines parenting characteristics such as levels of parenting stress and parenting efficacy. Consequently, less research has investigated the effects of BCEs and their relation to positive parenting behaviors such as warmth or nurturance.

Research suggests that experiencing BCEs may be predictive of lower levels of stress later in life (Sever et al., 2024), which is associated with parenting warmth (Xu & Zheng, 2023). Positive experiences during childhood have been associated with better self-regulation skills (Feiler et al., 2023), which may explain this association, as self-regulation skills are correlated with better parent-child relationship characteristics (Lunkenheimer et al., 2023). Moreover, little research has examined whether BCEs may offset the influence of parental ACEs on their own nurturant parenting. Existing research also suggests that BCEs may interact uniquely with discrimination for Black individuals (McClinton, 2023).

Family cohesion

Along with parental experiences such as BCEs potentially offsetting parental adversity, positive family characteristics may also have the ability to offset parental exposure to adversity. The family environment as a whole should be considered in families that have parents who have been exposed to ACEs to understand the role of the family in protecting against this negative intergenerational cascade. Healthy family dynamics have been found to promote positive psychological development for children (Ani, 2024). A strong family bond may also function as a buffer against the negative effects of ACEs on child mental health outcomes (Lu & Xiao, 2019). Family cohesion refers to the level at which a family unit is emotionally close, supportive of one another, and accepting of one another (Cooper et al., 1983). Family cohesion has been found to positively influence parental well-being (Boyras & Sayger, 2011) as well as child well-being and behavioral health (Fosco & Lydon-Staley, 2020; Xiang et al., 2022). Additionally,

higher levels of family cohesion have been associated with less anxiety and depressive symptoms in adolescent children (Zahra & Saleem, 2021). Family cohesion has also served as a buffer against the negative effects of familial adversity (Daniels & Bryan, 2021). This highlights the notion that, despite parents engaging in reduced positive parenting behaviors toward their children, the perception of a close and accepting broader family unit as a whole may exert protective effects over and above parenting by one specific parent that is characterized by low warmth or nurturance. Family Systems theory posits that families are a system comprised of many subsystems that are larger than the whole (Cox & Paley, 1997). A parent may lack nurturant parenting; however, the impact of these behaviors on adolescent psychopathology may vary as a function of perceptions of the family as a whole. Perceptions of the family as a whole as cohesive and close may offset the low nurturant parenting from a single member of the larger family unit. As such, family cohesion, positive whole-system functioning, may offset a parent's lack of nurturant parenting. Protective components of the family system as a whole may influence the parent's well-being, offspring well-being, and the dyadic parent-child relationship. Schofield and colleagues (2016) found family cohesion as being protective against the effects of harsh parenting on child well-being. However, these associations should be expanded upon to examine the protective role of family cohesion against the effects of low nurturant parenting on adolescent depression and anxiety. Family cohesion may also be particularly promotive for Black individuals, as research suggests that family cohesion forecasts decreased anxiety symptoms in Black young adults (Augustine et al., 2022).

The current study

ACEs have been associated with a host of negative outcomes across the lifespan and across generations. However, the mechanisms through which these associations occur are

unclear. Furthermore, little is known about which factors at the familial level could offset the intergenerational risk of parents' exposure to adversity and how that impacts their children's mental health. Thus, the present study has four aims. First, the present study will examine the extent to which second generation (G2) ACEs are associated with third generation (G3) adolescent anxiety and depressive symptoms and determine whether low G2 nurturant parenting serves as a mechanism in this association. Second, consistent with intergenerational transmission of parenting, I also investigate whether first (G1) generation nurturant parenting influences G2 nurturant parenting. Third, I will also investigate whether G2 BCEs serve as a moderator to offset the association between G2 ACEs and G2 nurturant parenting. Lastly, I will examine whether family cohesion functions as a moderator to dampen the association between low G2 nurturant parenting and G3 adolescent anxiety and depressive symptoms. Consistent with research on the intergenerational effects of ACEs, I hypothesize that higher levels of G2 ACEs will result in higher levels of G3 depressive and anxiety symptoms. Additionally, I hypothesize that lower levels of G1 parenting will be associated with lower levels of G2 parenting. I also hypothesize that low G2 nurturant parenting will serve as a mediator linking G2 ACEs with G3 anxiety and depressive symptoms during adolescence. Next, I hypothesize that G2 BCEs will significantly moderate the association of G2 report of childhood ACEs and nurturant parenting such that higher reports of BCEs will attenuate the association between G2 ACEs and low G2 nurturant parenting. Last, I hypothesize that family cohesion will significantly moderate the association of low G2 nurturant parenting and G3 anxiety and depressive symptoms such that higher levels of family cohesion will mitigate the association between low nurturant parenting and G3 anxiety and depressive symptoms.

Method

Participants

The participants and measures used in this study are derived from a multi-generational study that is comprised of two projects: the Strong African American Families Healthy Adults Project (SAAF from 2001-2009, SHAPE from 2009-2024) and the Health and Resilience Project – Generations Study (HARP-G). The SAAF/SHAPE study was designed to examine the efficacy of an intervention program that aimed to mitigate the likelihood of adolescents engaging in externalizing behaviors such as substance use by promoting positive familial processes such as positive parenting. Participants in this study were a sample of Black families that resided in rural Southern counties in lower-income areas (See Table 15 for demographics; for more information regarding the sampling of the SAAF/SHAPE study, see Brody, 2016). During the first wave of data collection in SAAF, 495 families participated in data collection with 495 caregivers (first generation, G1) and 495 target youth (second generation, G2). There were 288 families in the intervention group, and 207 families were in the control group. At wave 1, target youth (G2) were roughly 11 years old. Data were collected through survey completion via private sessions with trained research personnel. After wave 1 was completed, data were collected again across a 20-year period with multiple rounds of data collection.

At the start of the HARP-G study, all participants who were the target children in the SHAPE study (G2) were contacted if they had at least one child ages 8-16 who spent at least 50% of their time with them. Of the G2s who were contacted to participate, 167 agreed to participate in the HARP-G follow-up study. Participants (G2) and their children (third generation, G3) completed questionnaires during an in-home session with trained research staff. For demographics of the HARP-G sample, see Table 15 G2 had, on average, 1.55 ($SD = .73$;

Min = 1; *Max* = 4) children participating in the study. Of these individuals, roughly 50.90% of G2 participants were in a relationship. The remaining G2 participants reported either being in a committed relationship or single. The majority of G2 participants (90.40% female, 9.60% male) were biological or birth parents of G3 participants (98.20%) with 1.80% being stepparents. Median annual income was reported at \$30,001-\$40,000 by G2 participants. For G3s, 49.40% were boys and 50.60% were girls. The average age for G3 participants was 11.09 years (*SD* = 2.21), ranging from 8 to 16 years old.

Measures

Adverse childhood experiences

ACEs were measured utilizing a 10-item list of adverse experiences during their first 18 years of life (Felitti et al., 1998). In the earlier waves of SHAPE, G2 (*Mean Age* = 26.34) participants reported (*0* = *No*; *1* = *Yes*) whether they experienced each of the 10 adverse experiences including physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect, violence against a mother, parental divorce, household members experiencing substance use/abuse, household members experiencing mental illness, and having an incarcerated household member. The ACEs questionnaire is a highly validated measure (Dobson et al., 2021). Higher scores on this scale indicate more adverse childhood experiences. In the current study, a sum score was utilized, totaling how many items individuals responded “yes”.

Nurturant Parenting

G1 nurturant parenting was measured utilizing the FACHS Nurturant-involved parenting scale. This scale has been utilized in previous studies (Brody et al., 2001; Ge et al., 1994). G1 responded to 9 items relating to their communication skills, and their tendency to reason with their child (*1* = *Never*; *4* = *Always*). A mean score was computed across waves 1, 2, 5, and 6

(Ages 11-16) of SAAF such that higher scores indicate higher levels of nurturant parenting. Reliability for this sample was good for G1 nurturant parenting and ranged from $\alpha = .74$ to $\alpha = .88$. G2 nurturant parenting was measured utilizing the same nurturant parenting scale as G1 (Brody et al., 2008). In wave 1 of HARP-G, G2 responded to 9 items by responding how often ($1 = \text{Never}$; $4 = \text{Always}$) they engaged in certain parenting behaviors with their child (e.g., how often do you discipline your child by reasoning, explaining, or talking to them?) A mean score was computed such that higher scores indicate higher levels of nurturant parenting. Reliability for this scale in G2 was good in the current sample ($\alpha = .86$).

Adolescent Internalizing Symptoms

The current study included depressive symptoms and anxiety symptoms to measure G3 adolescent internalizing symptoms.

Depressive symptoms. Depressive symptoms were examined utilizing the Center for Epidemiological Studies Depression Scale (CES-D)(Radloff, 1977). To measure depressive symptoms, G3 responded to a 20-item scale at wave 1 of HARP-G that assessed how frequently in the past week ($0 = \text{Rarely, or none of the time, less than 1 day}$; $3 = \text{Most or all of the time, 5-7 days}$) they were experiencing intense negative feelings they could not control (e.g., I felt that I could not shake off the blues even with help from my family and friends). A mean score was computed such that higher scores indicated higher levels of depressive symptoms. The CES-D has been utilized in many diverse samples and is a highly validated measure (Orme et al., 1986). Depressive symptoms maintained excellent reliability in the current sample ($\alpha = .92$).

Anxiety symptoms. Levels of anxiety symptoms were measured utilizing the Spence Children's Anxiety Scale (Ahlen et al., 2018; Spence, 1998). The SCAS is a highly validated measure that captures children's anxiety symptoms (Orgiles et al., 2016). To measure anxiety

symptoms, G3 responded to a 12-item scale at wave 1 of HARP-G that assessed how frequently ($1 = \text{Never}$; $4 = \text{Always}$) they were experiencing certain worries or concerns out of their control (e.g., I worry that something bad will happen to me). Both scales maintained good reliability in the current sample ($\alpha = .82$). A mean score was computed such that higher scores indicate higher levels of anxiety symptoms.

Benevolent Childhood Experiences (BCEs)

The Benevolent Childhood Experiences (BCEs) scale was used to measure positive experiences during G2's childhood (Narayan et al., 2018). In this study, G2 retrospectively responded to a 10-item scale during wave 1 of HARP-G that asked whether they experienced a specific event ($0 = \text{No}$; $1 = \text{Yes}$) in the first 18 years of their life (e.g., did you have at least one caregiver with whom you felt safe?) Sum scores were computed such that higher scores indicate more benevolent childhood experiences. BCEs have been validated to accurately measure positive childhood experiences (Narayan et al., 2023).

Family Cohesion

Family cohesion was measured using the family cohesion subscale of the self-report Family Functioning Scale (FFS)(Bloom, 1985). In wave 1 of HARP-G, G2 participants responded to 5 items and reported how true ($1 = \text{Very untrue for my family}$; $4 = \text{Very true for my family}$) a given statement was for their family environment (e.g., Family members really help and support each other). A mean score was created such that higher scores reflect higher levels of family cohesion. Reliability for this scale in the current sample was adequate ($\alpha = .81$), and this scale has been validated in other samples (Bloom, 1985).

Covariates

Covariates in the current study include G3 child sex and age, intervention status, family income at wave 1 of HARP-G, and G2 depressive symptoms across SHAPE waves. G2 depressive symptoms were measured with the CES-D at each wave in SHAPE except for wave 6 (W1-W5 and W7-W8; *Min Age = 19 ; Max Age = 34*), and were mean scored to reflect average depressive symptom scores across adolescence and adulthood.

Data Analytic Plan

Descriptive statistics and bivariate associations were conducted in SPSS. Due to clustering of data (e.g., G3 children within G2 caregivers), descriptive statistics are provided at the caregiver/family-level for G1 nurturant parenting, G2 ACEs, G2 BCEs, G2 depressive symptoms, and family cohesion and at the child-level for G2 nurturant parenting (pertaining to the specific child) and G3 depressive and anxiety symptoms.

Due to the clustered structure of G3 children clustered within G2 families, multilevel structural equation modeling (MSEM) was utilized for both tests of mediation (Preacher et al., 2010) and moderation (Preacher et al., 2016). Mental health symptoms for individual G3 children and G2 nurturant parenting about each child were nested within families at Level 1 and G2 ACEs, G2 BCEs, G1 nurturant parenting, and family cohesion were at Level 2. Analyses were conducted in Mplus 8.0 (Muthén and Muthén 1998-2017) to test hypotheses consistent with the overall conceptual model (See Figure 2). Covariates at the within-level were G3 age and G3 sex. Covariates at the between-level included G2 depressive symptoms, family income, G3 sex, G3 age, and intervention status.

MSEM Mediation Analyses

General multilevel models (MLM) may lead to biased or conflated effects for within- and between-level indirect effects (Preacher et al., 2010). MSEM models overcome these limitations by allowing any variable in the study to be specified as a latent variable by freeing relevant loadings. Additionally, MSEM overcomes these limitations by partitioning the Level 1 variance into orthogonal within and between components. In the current study this is necessary because G2 nurturant parenting contains variance due to both the within (child specific) as well as between- (parent-general) components. Thus, the current study utilizes a 2-1-1 mediation model, consistent with MSEM mediation models outlined in Preacher et al. (2010) for modeling hierarchically clustered data in mediation tests. A 2-1-1 mediation model utilizes a Level 2 predictor, Level 1 mediator, and Level 1 outcome. Indirect effects of G2 ACES on G3 mental health outcomes via G2 nurturant parenting were assessed using model constraints indirect in Mplus. The test of mediation is at the between level due to the nature of the measurement of ACEs which was assessed by the G2 parent. Due to the ability for MSEM models to partition Level 1 variance into both within- and between-level components, the current model can confidently calculate unbiased tests of between-level mediation.

MSEM Moderation Analyses

Next, moderation in both pathways of the model were tested using moderation models in MSEM consistent with Preacher et al. (2016). A current drawback for existing approaches to moderation in traditional multilevel models is the conflated effects that occur across levels of analysis (e.g., Level 1 and Level 2) as well as bias attributed to observed cluster averages as opposed to the use of latent variables for partitioning variance across levels. In the current study, latent moderated structural equations (LMS) were utilized (Preacher et al., 2016) to produce

unbiased effects. LMS is a multilevel approach to statistical moderation that accounts for same-level interactions as well as cross-level interactions. First, BCEs was investigated as a moderator to determine whether parents' positive childhood experiences attenuate the association between G2 ACEs and G2 nurturant parenting. The design of the first moderation was a 2x(2-1) model, which indicates that the model tests the Level 2 BCEs moderator of Level 2 ACEs and Level 1 nurturant parenting association. Last, family cohesion was included as a moderator to examine the protective role of a cohesive family between the association of G2 nurturant parenting and G3 internalizing symptoms. This moderation effect was examined utilizing a 2x(1-1) design using a Level 2 family cohesion moderator of the Level 1 G2 nurturant parenting and G3 Level 1 mental health outcomes. Because these Level 1 constructs can be partitioned into within and between components, Level 2 cohesion may moderate the between component of parenting and mental health as well as the within level component of parenting and mental health. The current model of moderation tests both components. Significant interaction effects were examined using simple slopes tests and plots to probe moderator effects (Aiken & West, 1991; Dawson 2014).

Results

Descriptive Statistics

Means, standard deviations, and ranges for all study variables can be found in Table 16. Frequencies and descriptive statistics of G2 parental ACEs and BCEs can be found in Table 17. G2 experienced, on average, 1.42 ACEs during the first 18 years of life ($SD = 1.58$) and 9.35 BCEs ($SD = 1.24$). Parental separation or divorce was the most frequently reported ACE (46.30%). The most frequently endorsed BCE was "I had a good friend" and was endorsed by all G2 parents (100%). In general, G2 parents reported more BCEs than ACEs throughout their childhood. Bivariate correlations for study variables are displayed in Table 18. G3 anxiety and

depressive symptoms were highly positively correlated with one another. G2 ACEs were significantly negatively correlated with BCEs, nurturant parenting, and family cohesion and positively correlated with their own depressive symptoms. Lastly, a one-way between subjects' analysis of variance (ANOVA) was conducted to examine differences in study variables by intervention status (See Table 19). There were no significant differences between intervention status for any study variables.

MSEM Mediation Results – G2 Nurturant Parenting as a mediator between G2 ACEs and G3 Mental Health

Mediation analyses (2-1-1) were conducted in MSEM using maximum likelihood estimation to examine the indirect effects of G2 ACEs →G2 nurturant parenting→G3 depressive and anxiety to partition the within- and between-level components (See Figure 3). Additionally, the G1 nurturant parenting→G2 nurturant parenting was included to account for intergenerational transmission of parenting across G1 and G2. Between-level covariates included family income, G2 depressive symptoms, and intervention status. G3 sex and age were included as covariates at both the between and within levels. The model provided adequate fit to the data ($\chi^2(17)=28.17, p = .04$; CFI = .90, RMSEA = .06). Results are shown in Table 20. Intraclass correlations (ICC) demonstrate the majority of variance in G2 nurturant parenting (ICC=.70) was in the between-level (e.g., general to the G2 parent family cluster rather than attributable to specific child-level variance). Additionally, estimates for family-level mental health for G3 were ICC = .21 for depressive symptoms ICC=.11 for anxiety symptoms suggesting sufficient variance at the family-level necessitating analyses that account for clustering. Tests of 2-1-1 mediation were conducted at the between-level as G2 ACEs is a Level 2 predictor (e.g., variance only attributable at the family-level; Preacher et al., 2010). Higher G2 ACEs were associated with

lower levels of G2 nurturant parenting in the between-level component ($B = -.93, p < .001$). In turn, G2 nurturant parenting was significantly associated G3 depressive symptoms ($B = -.74, p = .01$), but not anxiety symptoms ($B = -.28, p = .13$) in the between-level components. Tests of the indirect effect support the conclusion that G2 nurturant parenting served as a mediating mechanism between G2 ACEs and G3 depressive symptoms ($B = .69, p = .04$). At the within-level, G2 nurturant parenting were not significantly associated with the within-level components of G3 depressive symptoms ($B = .49, p = .29$), nor anxiety symptoms ($B = .36, p = .22$). This may be a reflection of high ICCs for nurturant parenting suggesting little within-level (e.g., child-specific) nurturant parenting variation. Additionally, levels of G1 nurturant parenting was not associated with G2 nurturant parenting ($B = .07, p = .56$) suggesting a lack of continuity or transmission of nurturant parenting across generations. With regards to covariates, family income was significantly associated with G3 depressive symptoms ($B = -.99, p = .02$), as well as G3 anxiety symptoms ($B = -.57, p = .04$) in the between-level components such that higher family income was associated with lower mental health symptoms. At the within-level component, G3 sex was associated with G3 depressive symptoms ($B = 3.74, p = .04$), as well as G3 anxiety symptoms ($B = 2.40, p = .04$) such that girls had greater mental health symptoms. G3 age was associated with G3 anxiety symptoms ($B = -.53, p = .02$) in the within level components, such that younger children had more anxiety symptoms.

G2 BCEs Moderating the association between G2 ACEs and G2 Nurturant Parenting

Moderation analyses examining G2 BCEs as a moderator of the G2 ACEs-G2 nurturant parenting association were conducted in the MSEM framework using the 2x(2-1) model to examine moderation in the between-level components (Preacher et al., 2016). The full model including the latent interactions and covariates did not converge. As a result, tests of moderation

were conducted in simplified analyses to provide preliminary tests for the protective effects moderation. The analyses were simplified in three ways to achieve model convergence: covariates were excluded to reduce the number of parameters estimated, the MLF estimator was used, and the ACEs and BCEs data were recoded to address deviations from normality. G2 ACEs were coded as 0 = No ACEs, 1 = 1+ ACEs. G2 BCEs were coded as 0 = <10 BCEs, 1 = All 10 BCEs. This resulted in model convergence (BIC = 1827.80). Results are shown in Table 21. G2 BCEs significantly moderated the effects of G2 ACEs on between-level component of G2 nurturant parenting ($B = 2.86, p < .001$). To probe the significant interaction, a simple slopes plot was created, and tests of the simple slopes were conducted (see Figure 4). For G2 parents reporting fewer than 10 BCEs, the effects of G2 ACEs on G2 nurturant parenting were significant ($t = -2.74, p < .001$) such that reports of any ACEs were associated with lower G2 nurturant parenting; however, for G2 parents endorsing all 10 BCEs, there was no association among G2 ACEs and G2 nurturant parenting ($t = .11, p = .79$).

Family Cohesion as a Moderator of the G2 Nurturant Parenting and G3 Mental Health Symptoms

Moderation analyses examining family cohesion as a moderator of the G2 nurturant parenting-G3 depressive and anxiety symptom associations were conducted in the MSEM framework using the 2x(1-1) model to examine moderation (Preacher et al., 2016). Because G2 nurturant parenting and G3 mental health were both Level 1 variables, tests of moderation include partitioning the variance of each into between- and within-level components to test for moderation in each component. Similar to moderation tests described above, the full models with covariates did not converge. As a result, moderation analyses were simplified in three ways to examine preliminary support for the protective effects of cohesion as a moderation. To achieve

model convergence: covariates were omitted from tests of moderation, the MLF estimator was used, and family cohesion was scored in terciles reflecting low, medium, and high levels of family cohesion to account for violations of non-normality. Separate models were estimated for depressive and anxiety symptoms.

Family Cohesion as a Moderator in the Within-level Components

Family cohesion as a moderator was first examined in the within-level components of G2 nurturant parenting on G3 depressive symptoms and G3 anxiety symptoms. Results are shown in Table 22 for both depressive symptoms (BIC = 228584.26) and anxiety symptoms (BIC = 228490.80). Family cohesion did not significantly moderate the within-level effects of G2 nurturant parenting on G3 depressive symptoms ($B = .10, p = .99$) nor on G3 anxiety symptoms ($B = .10, p = .99$).

Family Cohesion as a Moderator in the Between-level Components

Lastly, the moderating role of family cohesion was examined in the between-level components for G2 nurturant parenting on both G3 depressive symptoms (BIC = 2465.36) and anxiety symptoms (BIC = 2340.94). Results are shown in Table 22. Family cohesion did not significantly moderate the between-level component of G2 nurturant parenting on G3 depressive symptoms ($B = -2.15, p = .18$); however, there was a significant direct effect of the between-level component of family cohesion on depressive symptoms such that higher family cohesion was associated with fewer depressive symptoms ($B = -2.97, p < .001$). Family cohesion did significantly moderate the between-level component of G2 nurturant parenting on G3 anxiety symptoms ($B = -.71, p < .001$). A simple slopes test and plot demonstrated that for families reporting high levels of cohesion, G2 nurturant parenting was significantly associated with G3 anxiety symptoms such that higher levels of nurturant parenting was associated with lower

anxiety symptoms ($t = -2.09, p = .04$)(See Figure 5); however, for families with low levels of cohesion, there was no association among G2 nurturant parenting and G3 anxiety symptoms ($t = .48, p = .29$).

Discussion

The current study aimed to investigate the multigenerational process of adversity and resilience in Black youth. Few studies have examined the process of adversity and resilience across multiple generations in minoritized samples, especially with the inclusion of protective factors that may contribute to resilience by disrupting intergenerational cascades. Findings were consistent with my first hypothesis: G2 adverse childhood experiences were associated with how they parent their own children through reduced nurturant parenting by G2 years later when parenting their own children. This is consistent with prior literature that suggests that childhood experiences of adversity affect an individual's parenting later in life (Babacan, 2021; Racine et al., 2018). While existing research reflects significant associations between ACEs and nurturant parenting, these examinations are frequently investigated in White populations and for individuals that hold more social advantage (Biglan et al., 2017). As such, this finding highlights the importance of examining these associations among Black families. Black youth are at higher risk of experiencing ACEs (Drake et al., 2009), which highlight the need for prevention programs to target minoritized populations to decrease the likelihood of exposure to adversity. The findings from this study suggest prevention efforts to reduce ACEs may have long-lasting effects that span across generations to benefit future offspring.

Lower nurturant parenting in G2 served as a mechanism linking parent's own ACEs with their children's mental health (e.g., heightened depressive symptoms in G3). The indirect effect provided support for mediation in this intergenerational risk cascade with higher reports of G2

ACEs predicting lower G2 nurturant parenting which in turn led to higher levels of G3 depressive symptoms. Additionally, child-specific effects (e.g., within-level components) of G2 nurturant parenting were not associated with G3 depressive symptoms, but associations were supported in the between-level components of G2 nurturant parenting. This may be attributed to the high ICC for nurturant parenting; the majority of the variance resided at the parent-level with regards to their general parenting rather than variation in nurturant parenting amongst siblings. This may reflect parents' use of the same general parenting strategies across children.

The prevalence rates of depressive symptoms among Black youth have risen in recent years (Xiang et al., 2024), which necessitates further investigation of precursors to the onset and increase in depressive symptoms in childhood and adolescence for Black youth. A notable contribution of this study is the investigation of an intergenerational cascade of psychopathology as a result of their parent's childhood adversity experiences operating through disruptions in parenting. These investigations expand the current understanding of the long-lasting effects of multigenerational adversity. Research to date has examined the effects of parental ACEs on parenting and parenting on mental health outcomes, but few studies have examined these associations as a mediational pathway consistent with a multigenerational risk cascade. These findings support the notion that developmental cascades can unfold not only throughout development but also across generations.

A robust test of protective factors as moderators in this developmental risk cascade was not possible due to the failure of model convergence. Preacher and colleagues (2016) note that model complexity may increase the likelihood of model nonconvergence and problems with model nonconvergence may not be readily resolved in MSEM moderation. This may also be in part due to lack of variability within the BCEs measure in the current sample. Participants in this

sample highly endorsed most of the BCEs items. Existing research has found that prevalence rates for BCEs typically range from 7.5 – 9 (Raghunathan et al., 2024). To provide preliminary support for the role of protective factors in this risk cascade, simplified tests of moderation were performed. As such, moderation findings necessitate further replication and provide preliminary support for future multigenerational investigations of resilience.

Nevertheless, preliminary moderation findings do provide initial support for G2 BCEs as a moderator of the association between G2 ACEs and their own nurturant parenting, suggesting that the experience of positive childhood experiences may have protective effects on one's own parenting despite experiencing adversity. This is consistent with previously conducted research that supports a protective role of BCEs (Crandall et al., 2019; Doom et al., 2021). The findings in the current study demonstrate the protective effects of experiencing cumulative positive childhood experiences that span across ecological contexts, especially to offset cumulative adverse childhood experiences. BCEs have been found to be correlated with ACEs (Narayan et al., 2023), emphasizing that risk factors and protective factors do not operate in isolation. As such, combining the effects of both adverse as well as positive experiences during childhood may result better representation of the full experience of both positive and negative events during childhood that may contribute to later parenting. BCEs may be particularly beneficial for minoritized individuals, as they may lean on these positive influences to reduce the harmful effects of ACEs during childhood that may lead to reduced stress that may continue into a cascade of more warm, nurturant parenting behaviors during parenthood (Lucke, 2024).

With the same caveats regarding tests of moderation, preliminary support was found for family cohesion moderating the association between G2 nurturant parenting on G3 anxiety symptoms, but not depressive symptoms. These findings are consistent with previous literature

that suggests family cohesion to be protective against anxiety outcomes (Anyan & Hjemdal, 2018). Future research should examine the myriad family processes as moderators against this association. For Black youth, potential protective family processes may be specifically related to their experiences as minorized youth; for example, parents may seek to enhance youth's resilience through protective processes aimed at promoting ethnic identity, cultural orientation, or racial socialization (Neblett et al., 2012).

In the current study, main effects for family cohesion on G3 depressive symptoms were significant such that higher levels of family cohesion were associated with lower depressive symptoms. These findings suggest that family cohesion is beneficial for all youth, despite level of risk. Additionally, these findings are congruent with previous literature that examined promotive effects of family cohesion (Zahra & Saleem, 2021). Furthermore, this is consistent with previous research that suggests family cohesion is associated with better mental health outcomes in adolescents (Goodrum et al., 2020). Family cohesion may be particularly promotive for Black individuals, which is congruent with prior research examining the promotive role of family cohesion on self-regulation and mental health outcomes in Black emerging adults (Augustine et al., 2022).

Although G2 nurturant parenting was associated with depressive symptoms for G3, it was not associated with their anxiety symptoms. Further, there were interaction effects among cohesion and G2 nurturant parenting predicting G3 anxiety, whereas family cohesion served as a promotive factor for depressive symptoms. As such, these findings suggest that determinants of depressive symptoms may be distinct from those of anxiety symptoms. Depression and anxiety are similar to one another such that they both pertain to emotional symptoms related to worry, sadness, and difficulty concentrating (Nutt, 2024). However, these two classifications of

internalizing symptoms are considered as two different diagnoses in the Diagnostic and Statistical Manual of Mental Illnesses (DSM-5). As such, these outcomes may have two separate processes which lead to symptomology. Indeed, existing research suggests that there are racial and ethnic differences when examining comorbidity of depressive and anxiety symptomology (Watkins et al., 2015), which highlights the need to examine these mental health outcomes separately especially in minoritized populations.

In the current study, G1 nurturant parenting was not associated with G2 nurturant parenting. This is inconsistent with previously conducted research that suggests parenting behavior in one generation is predictive of similar parenting behaviors in the next generation (Bailey et al., 2009). Nurturant parenting as a form of positive parenting has been found to evince better mental health outcomes in Black families (Lewin et al., 2011). Rather than direct transmission of parenting across generations, nurturant parenting in the previous generation may operate through behavioral traits that support positive family functioning for the next generation. Although G1 nurturant parenting was not associated with G2 nurturant parenting, prior research in this sample finds that G1 nurturant parenting is associated with G2 outcomes which include less alcohol use (Kogan et al., 2019) and lower depressive symptoms (Brody et al., 2019; Natsuaki et al., 2007) and this improved health may play a part in a developmental pathway toward nurturant parenting. Future research should investigate the transmission of parenting behaviors across generations and the moderators that produce discordance in parenting transmission.

The current study is not without limitations, including the sample size in this study. The current study investigated the multigenerational process of adversity and resilience in 167 G3 children and adolescents from 108 G2 families. Although the current N for both G2 and G3 is

adequate to confidently reject the null hypotheses for significant pathways (Wolf et al., 2013) and sample sizes of 100 Level 2 clusters are consistent with recommendations for MSEM (Preacher et al., 2010), a larger sample size would increase statistical power, and seems especially important for elucidating moderation effects in the context of a robust set of covariates and the more computationally complex latent interactions. Nevertheless, multigenerational studies, especially those focused on minoritized families, are rare and despite limitations regarding power, the findings of the current study add to our knowledge of multigenerational developmental cascades of risk and resilience. Next, the nurturant parenting measure in the current study incorporates parents' own report of their parental nurturance. Parents may report higher levels of nurturant parenting as a reflection of social desirability bias, which could explain non-significant findings between G1 and G2 nurturant parenting. Additionally, the use of self-reported nurturant parenting behaviors would be strengthened by the use of multiple reporters of parenting (e.g., partner and child report). However, a notable strength of the study is that nurturant-involved parenting utilized the same scale for both G1 and G2. This allows for examination of parenting across generations. Lastly, the findings of this study may not generalize to other minoritized youth and families. Future research is needed to examine whether these associations replicate among other minoritized groups such as Latinx, Native American, or Asian samples.

Conclusion

These findings emphasize the long-term implications of adversity, and highlight a developmental cascade of risk across G2 and G3 through which G2 ACEs lead to reductions in nurturant parenting and then increased G3 depressive symptoms. For G2, ACEs were associated with less nurturant parenting behaviors as an adult. This finding informs policy makers to target

populations that are at higher risk of exposure to ACEs to offset this risk on disruptions in later parenting. Last, G2 BCEs and family cohesion served as protective factors to offset risk, emphasizing the importance of protective factors across the life course and multiple generations to promote resilience. In conclusion, parenting, adversity, and resilience are a multigenerational process and may contribute to developmental cascades that result in mental health outcomes for youth. This study informs intervention programs and highlights protective familial processes in the cascade of risk following adversity that may instead shift pathways toward resilience.

Research with a life course theory perspective often excludes intergenerational adversity, which is thought to be a “missing piece” of existing literature pertaining to adversity throughout the life course. The current study expands upon existing literature that examines risk and resilience across the life course by incorporating lifespan theory and utilizing three generations of Black families to investigate multigenerational cascades of adversity, resilience, and parenting to understand children’s mental health.

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Table 15. Sample Demographics for Study 2

Construct	<i>M (SD) or N (%)</i>	
	G2 Sample	G3 Sample
<i>N</i>	108	167
G2 Relationship Status <i>N (%)</i>		
In a Relationship	53 (50.90%)	
Single	55 (49.10%)	
G2 Relation to G3 <i>N (%)</i>		
Biological Parent	105 (98.10%)	
Step-parent	2 (1.90%)	
G2-Reported Annual Income in USD		
\$0-\$10,000	17 (15.90%)	
\$10,001-\$20,000	18 (16.80%)	
\$20,001-\$30,000	17 (15.90%)	
\$30,001-\$40,000	22 (20.60%)	
\$40,001-\$50,000	14 (13.10%)	
\$50,001-\$60,000	4 (3.70%)	
\$60,001-\$70,000	4 (3.70%)	
\$70,001-\$80,000	5 (4.70%)	
\$80,001-\$90,000	1 (0.90%)	
\$90,001-\$100,000	1 (0.90%)	
\$100,001-\$125,000	3 (2.80%)	
\$125,001-\$150,000	1 (0.90%)	
Intervention Status		
Control	37 (34.30%)	
Treatment	71 (65.70%)	
Sex <i>N (%)</i>		
Male	10 (9.30%)	82 (49.40%)
Female	98 (90.70%)	84 (50.60%)
G3 Youth Age <i>M (SD)</i> in years		11.09 (2.21)
Number of Children in Study <i>M(SD)</i>	1.55 (.73)	

Table 16. Descriptive Statistics for Study Variables

Construct	<i>M (SD)</i>	Min-Max	<i>N</i>
G1 Nurturant Parenting	28.39 (3.65)	17.75-35.50	108
G2 Nurturant Parenting	31.38 (4.44)	9.00-36.00	166
G2ACEs	1.42 (1.58)	0.00-7.00	108
G2 BCEs	9.35 (1.24)	3.00-10.00	107
Family Cohesion	16.22 (3.01)	8.00-20.00	107
G3 Anxiety Symptoms	11.43 (6.66)	0.00-35.00	164
G3 Depressive Symptoms	16.31 (9.32)	2.00-47.00	164

Table 17. G2 ACEs and BCEs Frequencies and Descriptive Statistics

Indicator	Frequency (N) or Mean (M)	Percentage (%) or SD
<i>Adverse Childhood Experiences</i>		
Emotional Abuse	17	15.70%
Physical Abuse	7	6.50%
Sexual Abuse	11	10.20%
Emotional Neglect	24	22.20%
Physical Neglect	4	3.70%
Mother Treated Violently	5	4.60%
Parental Separation or Divorce	50	46.30%
Overall ACE Sum Total <i>M (SD)</i>	1.42	1.58
<i>Benevolent Childhood Experiences</i>		
Had a Safe Caregiver	100	93.50%
Had a Good Friend	107	100.00%
Had Beliefs That Gave Comfort	104	97.20%
Enjoyed School	92	86.00%
Had a Teacher Who Cared About You	103	96.30%
Had Good Neighbors	101	94.40%
Had Opportunities to Have a Good Time	104	98.10%
Liked Self or Felt Comfortable with Self	94	88.70%
Had a Predictable Routine	93	86.90%
Had an Adult that Could Provide Support or Advice	102	95.30%
Overall BCEs Sum Total <i>M(SD)</i>	9.35	1.24

Note. Sample of 108 G2 Parents

Table 18. Correlation Table Among Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. G1 Nurturant Parenting	1.00											
2. G2 Nurturant Parenting	.06	1.00										
3. G2 ACEs	.00	-.17*	1.00									
4. G2 BCEs	-.03	.17*	-.58**	1.00								
5. Family Cohesion	-.02	.04	-.20**	.20*	1.00							
6. G3 Anxiety Symptoms	-.14	-.02	.06	.04	-.10	1.00						
7. G3 Depressive Symptoms	-.01	-.11	.05	.08	-.09	.62**	1.00					
8. G2 Depressive Symptoms	.04	.06	.37**	-.24**	-.32**	.11	.14	1.00				
9. Income	.03	-.11	-.03	-.00	.16*	-.15	-.19*	-.25**	1.00			
10. G3 Sex	-.02	.10	-.11	.06	-.15	.23**	.21**	.02	.01	1.00		
11. G3 Age	.01	-.07	-.01	-.07	.02	-.18*	-.16*	-.04	-.04	-.03	1.00	
12. Intervention Status	.05	.10	.14	-.02	-.14	.04	-.01	-.09	.05	.05	-.07	1.00

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. Intervention Status 0 = Control Group, 1 = Intervention Group; G3 Sex 0 = Male, 1 = Female.

Table 19. Comparisons of Study Variables By G1/G2 Intervention Status

Construct	Control <i>M (SD)</i>	Intervention <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>p</i>	<i>Pairwise Comparisons</i>
G1 Nurturant Parenting	28.40 (3.89)	28.73 (3.33)	.33	1	.56	Non-significant
G2 Nurturant Parenting	30.81 (4.34)	31.69 (4.49)	1.49	1	.22	Non-significant
G2 ACEs	1.14 (1.37)	1.60 (1.76)	3.11	1	.08	Non-significant
G2 BCEs	9.46 (1.24)	9.29 (1.24)	.48	1	.49	Non-significant
Family Cohesion	16.81 (3.28)	15.92 (2.74)	3.44	1	.07	Non-significant
G3 Anxiety Symptoms	11.10 (5.75)	11.61 (7.14)	.22	1	.64	Non-significant
G3 Depressive symptoms	16.41 (9.03)	15.26 (9.53)	.01	1	.92	Non-significant
G2 Depressive Symptoms	16.53 (12.62)	14.55 (9.97)	1.24	1	.27	Non-significant
Income	3.65 (1.89)	4.04 (2.76)	.60	1	.44	Non-significant
G3 Sex	.47 (.50)	.52 (.50)	.36	1	.55	Non-significant

Note. * $p < .05$, ** $p < .01$, *** $p < .001$; G3 Sex 0 = Male, 1 = Female

Table 20. Multilevel Structural Equation Modeling Mediation Model

Construct	B	SE	p
<i>Between-level Components (N = 107)</i>			
G1 Nurturant Parenting → G2 Nurturant Parenting	.07	.11	.56
G2 ACEs→ G2 Nurturant Parenting	-.93	.29	<.001
G2 Nurturant Parenting→ G3 Depressive Symptoms	-.74	.30	.01
G2 Nurturant Parenting → G3 Anxiety Symptoms	-.28	.19	.13
G2 ACEs→ G3 Depressive Symptoms	-.22	.57	.71
G2 ACEs→ G3 Anxiety Symptoms	.23	.39	.55
<i>Between-level Indirect Effects</i>			
G2 ACEs → G2 Nurturant Parenting → G3 Depressive Symptoms	.69	.34	.04
G2 ACEs → G2 Nurturant Parenting → G3 Anxiety Symptoms	.26	.19	.17
<i>Between-level Covariates</i>			
G2 Depressive Symptoms→ G3 Depressive Symptoms	.13	.14	.36
G1 Nurturant Parenting → G3 Depressive Symptoms	.08	.21	.72
Income→ G3 Depressive Symptoms	-.99	.42	.02
G3 Sex→ G3 Depressive Symptoms	5.55	17.91	.76
G3 Age→ G3 Depressive Symptoms	-1.93	4.51	.67
G2 Depressive Symptoms→ G3 Anxiety Symptoms	.03	.09	.74
Income→ G3 Anxiety Symptoms	-.57	.28	.04
G3 Sex→ G3 Anxiety Symptoms	8.51	8.88	.34
G3 Age→ G3 Anxiety Symptoms	-1.33	2.58	.61
G2 Depressive Symptoms→ G2 Nurturant Parenting	.06	.07	.34
G1 Nurturant Parenting → G3 Anxiety Symptoms	-.20	.14	.16
Income→ G2 Nurturant Parenting	-.17	.17	.33
Intervention→ G2 Nurturant Parenting	.76	.85	.37
<i>Within-level Component (N = 163)</i>			
G2 Nurturant Parenting→ G3 Depressive Symptoms	.49	.46	.29
G2 Nurturant Parenting→ G3 Anxiety Symptoms	.36	.29	.22
<i>Within-level Covariates</i>			
G3 Age→ G3 Depressive Symptoms	-.60	.33	.07
G3 Sex→ G3 Depressive Symptoms	3.74	1.90	.04
G3 Age→ G3 Anxiety Symptoms	-.53	.23	.02
G3 Sex→ G3 Anxiety Symptoms	2.40	1.17	.04

Table 21. The Moderating Role of G2 BCEs Against the Effects of G2 ACEs on G2 Nurturant Parenting

Construct	B	SE	<i>p</i>
<i>Between- level Components (N= 108)</i>			
G2 ACEs → G2 Nurturant Parenting	-2.74	.36	<.001
G2 BCEs → G2 Nurturant Parenting	1.11	.32	<.001
G2 ACEs X BCEs → G2 Nurturant Parenting	2.86	.48	<.001
G2 Nurturant Parenting Residual Variance	.07	.05	.18

Table 22. The B1 and B2 Hypothesis of the Moderating Role of Family Cohesion Against G2 Nurturant Parenting on G3 Depressive and Anxiety Symptoms

Construct	<i>B1 – Within-Level Components</i>						<i>B2 – Between-Level Components</i>					
	Depressive Symptoms			Anxiety Symptoms			Depressive Symptoms			Anxiety Symptoms		
	B	SE	<i>p</i>	B	SE	<i>p</i>	B	SE	<i>p</i>	B	SE	<i>p</i>
<i>Between-level Components (N = 108)</i>												
G2 Nurturant Parenting	.04	1.34	.98	-.03	1.26	.98	-1.00	2.30	.66	.48	.31	.12
Family Cohesion	-.03	1.53	.98	-.14	1.49	.92	-2.97	.68	<.001	-.87	.59	.14
G2 Nurturant Parenting x Family Cohesion	.10	5.40	.99	.10	9.35	.99	-2.15	1.60	.18	-.71	.23	<.001
<i>Between-level Variances</i>												
G2 Nurturant Parenting Variance	.70	10.41	.95	.74	20.03	.97	.14	.13	.30	7.01	2.26	<.001
Family Cohesion Variance	.59	5.31	.91	.61	10.07	.95						
G2 Nurturant Parenting x Cohesion Variance	.20	3.16	.95	.20	5.73	.97						
G3 Internalizing Residual Variance	.70	10.83	.95	.69	19.87	.97	.42	.44	.34	1.10	.96	.25
<i>Within-level Components (N = 167)</i>												
G2 Nurturant Parenting	-	-	-	-	-	-	-.20	.21	.36	.06	.22	.77
<i>Within-level Variances</i>												
G2 Nurturant Parenting Variance	.70	16.38	.97	.71	32.96	.98	19.46	1.50	<.001	11.21	1.06	<.001

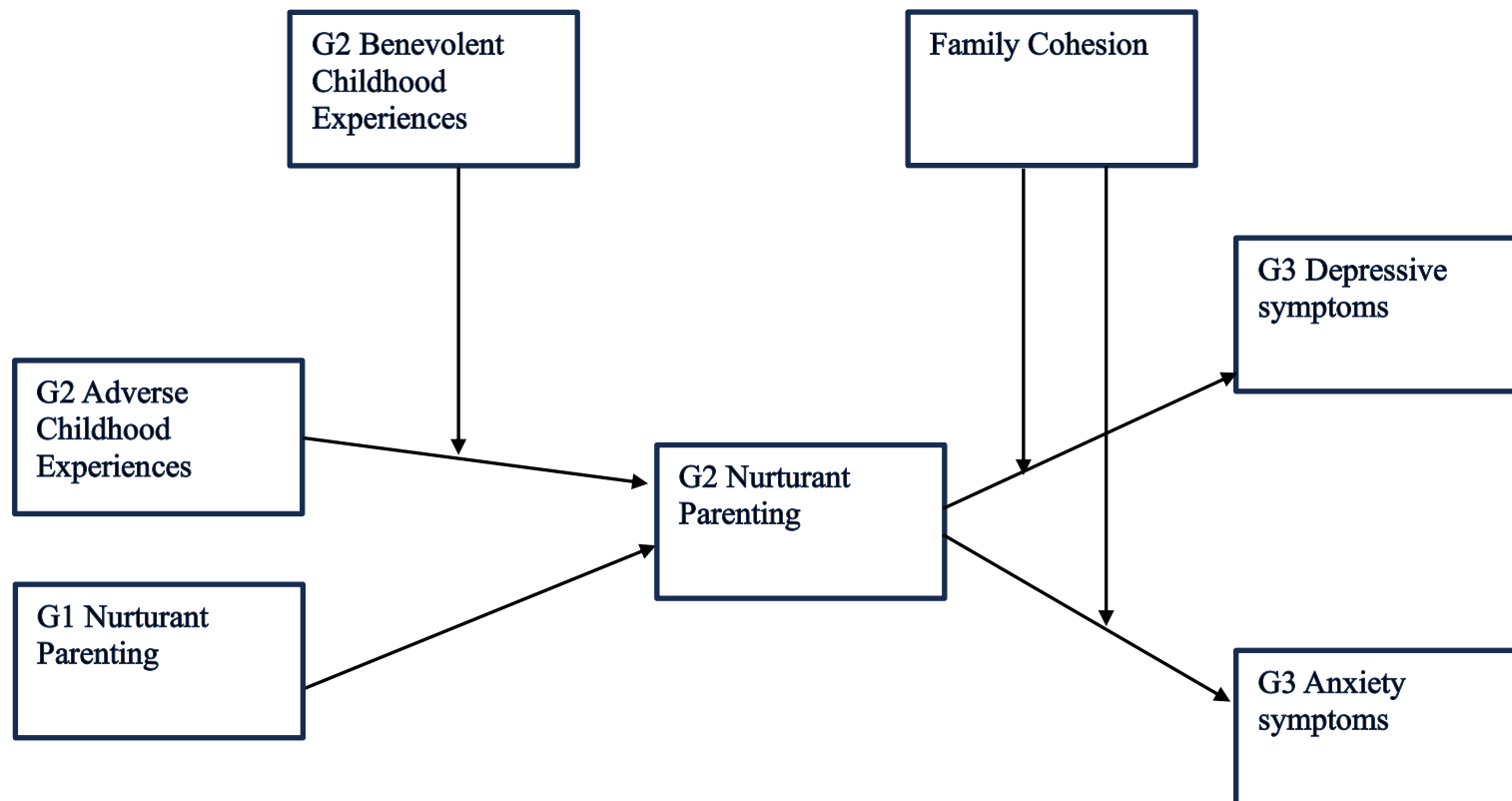


Figure 2. A conceptual model of study 2

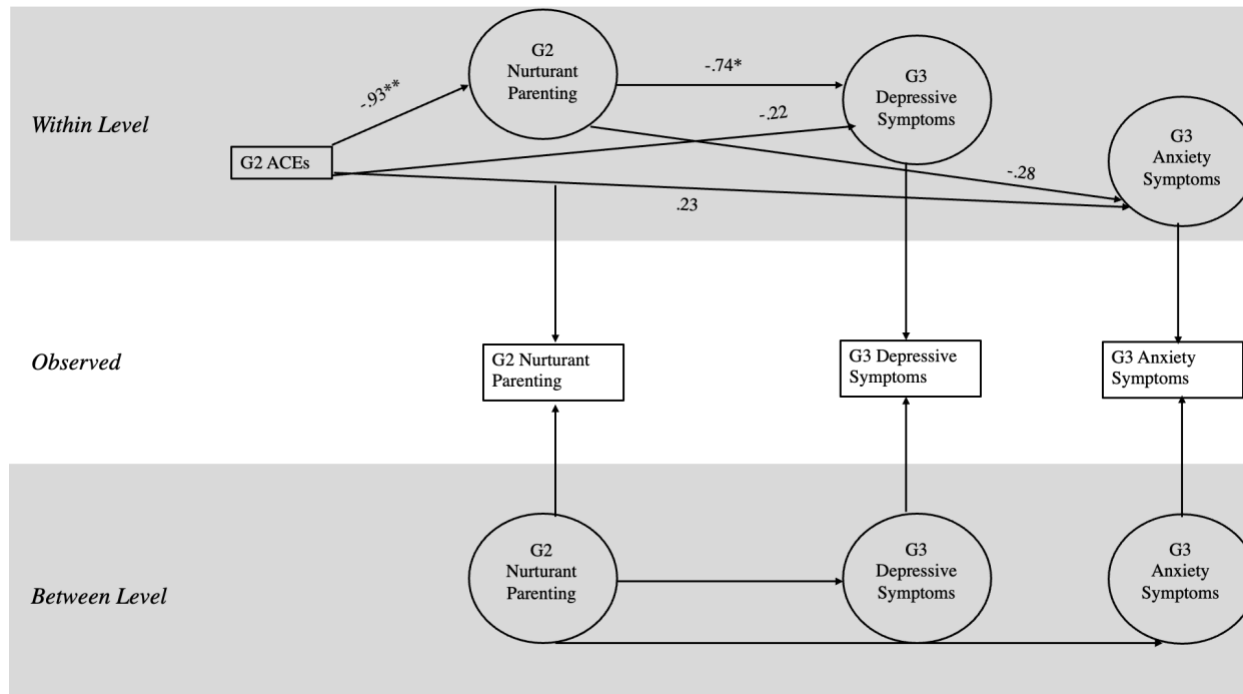


Figure 3. A 2-1-1 Multilevel Mediation Model Examining Indirect Effects of G2 Nurturant Parenting Between G2 ACEs and G3 Internalizing Symptoms. Covariates Omitted from MSEM Mediation Figure. Within-Level Covariates Include G3 Sex and G3 Age. Between-Level Covariates Include Income, G3 Sex, G3 Age, G2 Depressive Symptoms, and Intervention status.

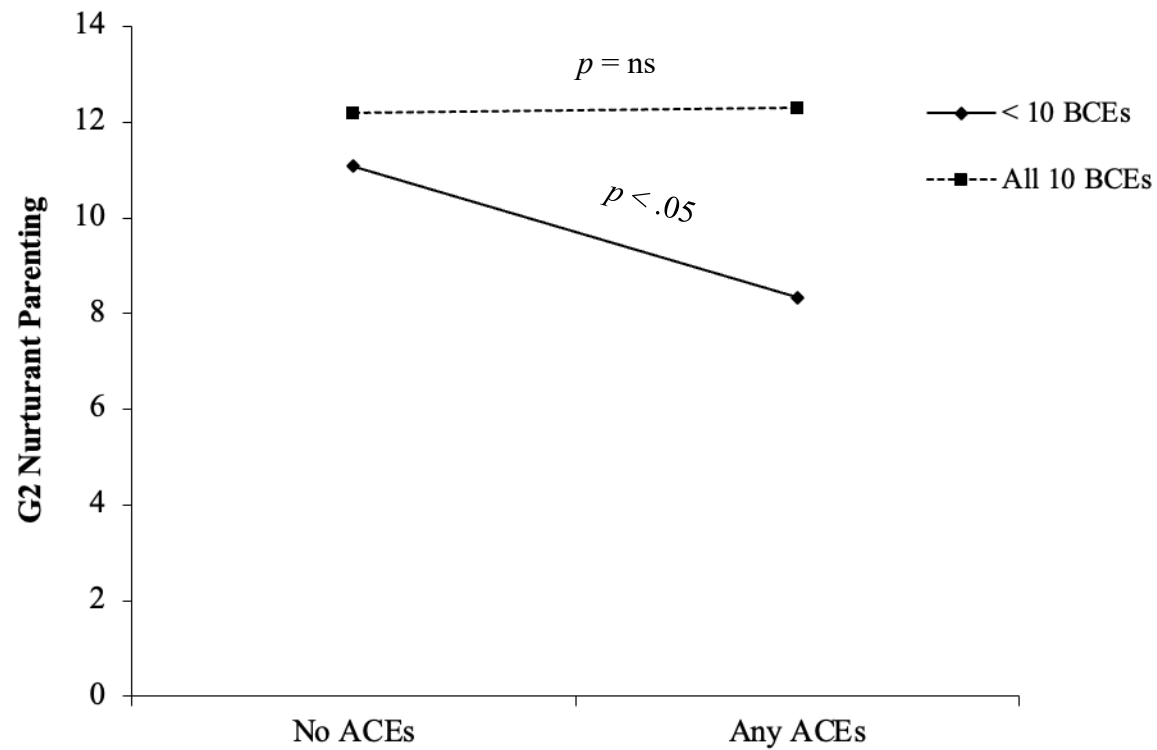


Figure 4. Simple Slope Plots for G2 BCEs as a Moderator of G2 ACEs predicting Level 2 Portion of G2 Nurturant Parenting.

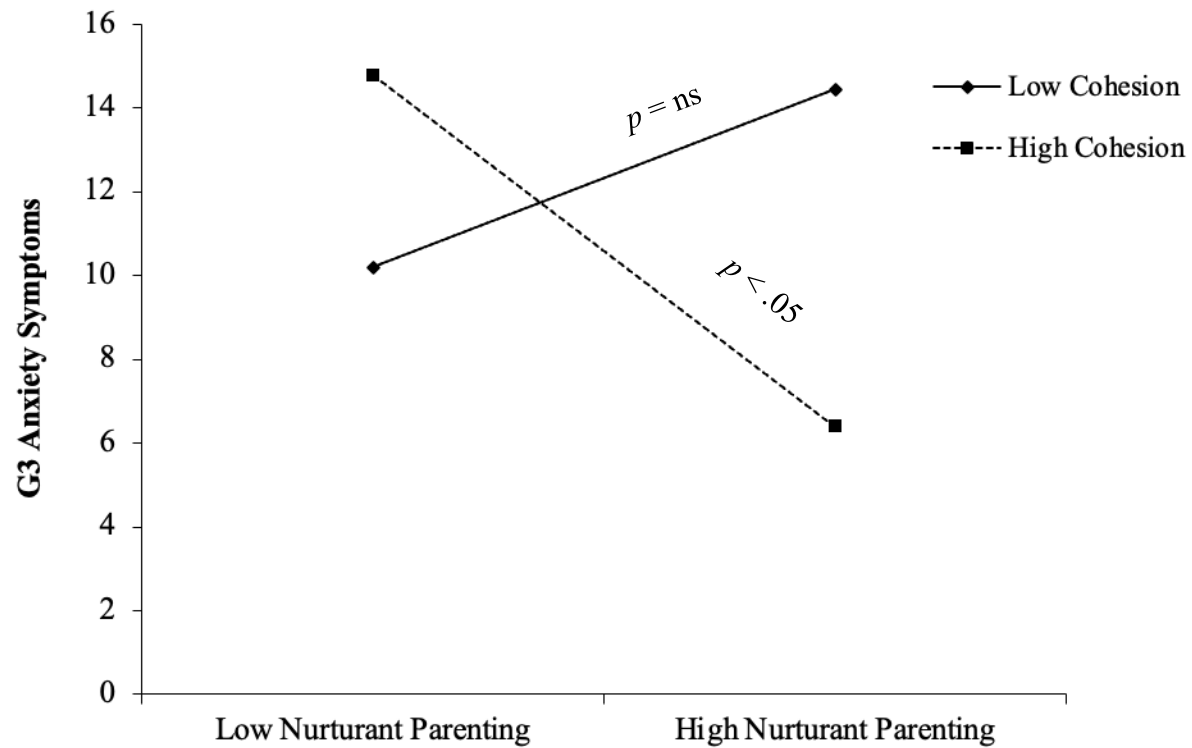


Figure 5. Simple Slope Plots for Family Cohesion as a Moderator of G2 Nurturant Parenting predicting G3 Anxiety Symptoms Moderated by Family Cohesion. High and low nurturant parenting reflects +/- 1 SD of the mean. High and low cohesion reflects the upper and lower tercile of the observed data.

CHAPTER 4

Conclusion

This dissertation work examined the multigenerational process of adversity and the protective familial processes that offset the deleterious effects of adversity in diverse samples. Specifically, this dissertation explored 1) the effects of cumulative parental adversity on adolescent offspring depressive and anxiety symptoms and 2) the protective effects of positive familial processes as buffers against the association between parental adversity and offspring depressive and anxiety symptoms. These studies documented the long-term implications on offspring mental health when their parents experience adversity, and highlight the protective role of the caregiver-child relationship.

The negative effects of experiencing adversity can evince maladaptive outcomes through physical health, mental health, and behavioral health (McLaughlin, 2018). However, the compounding effects of experiencing adversity as a minoritized youth may amplify risk for negative outcomes (Wickrama et al., 2016). For minoritized youth in America, rates of depressive symptoms and mental health problems are on the rise (Shain, 2019). Black youth experience myriad contextual stressors as a direct result of social inequality (Davis & Stevenson, 2006) that have long-term implications (Brody et al., 2020). Black individuals have been found to experience ACEs at higher rates than their White counterparts (Maguire-Jack et al. 2020), both at the prenatal level as well as throughout childhood (Evans et al., 2022). The results of Study 1 replicate these findings demonstrating Black mothers experienced more adversity during the prenatal period than White and Hispanic mothers. Prevention programs and governmental

assistance programs should aim to mitigate the maladaptive mental health outcomes that may occur as a direct result of social inequality and adverse childhood experiences related to social position (e.g., poverty).

The manuscripts in this dissertation reveal the deleterious effects of parents experiencing adversity on their children's well-being. Additionally, the caregiver-child relationship is a salient point of influence as a protective factor that offsets the negative effects of parental exposure to adversity on offspring well-being. Future research should continue to examine adversity through a cumulative lens to account for experiences of adversity across multiple bioecological contexts, including both the prenatal and postnatal period to adequately measure adversity throughout the lifespan, as both influences in utero and preceding birth impact developmental outcomes (Lebel et al., 2019). Not only do these findings further the scientific knowledge of adversity and resilience across the lifespan and across multiple generations, but they also reveal the importance of examining how familial processes impact the resilience process and have the opportunity to attenuate negative effects of generational trauma. These findings also highlight a developmental psychopathology framework such that there was heterogeneity between predictors of depressive symptoms in adolescence across studies (e.g., parental ACEs and prenatal adversity) yet both samples evinced higher depressive symptoms in the context of heightened adversity.

Manuscript 1

Findings from the first manuscript support direct effects between cumulative prenatal adversity and depressive symptoms at age 15, which highlight the enduring longitudinal effects of adversity prior to birth. Additionally, Study 1 found that for youth who had a secure attachment to their caregiver at age three, prenatal adversity was not predictive of adolescent depressive symptoms. However, for youth who had an insecure attachment to their caregiver,

prenatal adversity was predictive of adolescent depressive symptoms. This is congruent with a buffering hypothesis and suggests that a secure attachment between a caregiver and a child can offset the negative effects that prenatal adversity have on adolescent depressive symptoms. This highlights the long-term effects of adversity during sensitive periods in development and informs future research on potential sensitive periods for both risk and resilience processes that impact health outcomes across the lifespan. Lastly, when controlling for maternal depression one year after birth, associations between cumulative prenatal adversity and adolescent depressive symptoms remained significant, highlighting the effects of prenatal adversity extending above and beyond any confounding effects of postnatal caregiver depression. Findings also hold when controlling for offspring internalizing symptoms at age three, reducing concerns that earlier internalizing symptoms contribute to findings concerning internalizing symptomology during adolescence. This finding is important, as it suggests that prenatal adversity contributes to offspring psychopathology separately from the effects of earlier internalizing symptoms in childhood.

Manuscript 2

Findings from the second manuscript, a long-term multigenerational study, support direct effects from ACEs on later nurturant parenting in G2, and direct effects from nurturant parenting onto G3 depressive symptoms. Indirect effects of G2 nurturant parenting between G2 ACEs and G3 depressive symptoms were also significant, which highlight the mediating mechanism of developmental cascades of early life adversity onto later generations. Although moderation analyses were conducted without covariates, there is preliminary support to suggest that BCEs may be protective against the effects of G2 ACEs on G2 nurturant parenting, indicating that positive experiences than span ecological contexts during childhood have protective effects on an

individual's parenting despite experiencing adversity. G1 nurturant parenting did not directly affect G2 nurturant parenting, which suggests that one's own experiences during childhood may exert effects above and beyond the parenting that an individual received during childhood to impact their later parenting behaviors. Future research should consider the moderating factors that result in discordance between parenting across multiple generations. G2 nurturant parenting was associated with G3 depressive symptoms, but not G3 anxiety symptoms. This highlights the unique pathways that may lead to symptom onset that differ between anxiety and depressive symptoms, informing developmental psychopathology frameworks. Similarly to BCEs moderation analyses, there is preliminary support to suggest that family cohesion significantly moderated associations between G2 nurturant parenting and G3 anxiety symptoms, but not depressive symptoms. Replication is necessary to confidently suggest these associations. Adolescence is a sensitive period for the development of psychopathology, and these findings further suggest the necessity for developmental researchers to investigate how familial processes offset risk to protect against unique psychopathology symptoms during sensitive periods.

Scientific Contributions, Future Directions, and Implications

The first study expands upon previous research that examines prenatal adversity in singular forms to conceptualize prenatal adversity cumulatively in a similar approach to postnatal measurements of cumulative adversity exposure. Conceptualizing prenatal adversity similarly to postnatal adversity accounts for the cumulative effects of exposure to adversity to be additive, such that increases in forms of exposure suggests increases of maladaptive outcomes. Cumulative adversity during the prenatal period may also be a more appropriate metric because, regardless of the type of adversity children are exposed to, each form crosses the placental barrier and is being filtered through the mother, and each form interacts with nutritional signals.

Study 1 highlights the need for social reform and intervention programs to support pregnant women during pregnancy as well as early intervention to support caregivers and their young children in developing a secure bond. As such, points of intervention may extend throughout pregnancy and into early childhood for opportunities to offset this risk. Future research should examine the protective role of family processes later into childhood as well as examine offspring mental health outcomes into adulthood.

This dissertation research expands upon previous research that has found parental ACEs exposure to impact parenting quality. In particular, greater ACEs exposure was associated with lower nurturant parenting which in turn increased offspring depressive symptoms. Although moderation analyses excluded covariates and categorized BCEs dichotomously, BCEs was found to moderate the association between G2 ACEs and G2 nurturant parenting. Indeed, BCEs have been known to offset childhood adversity in prior research (Crandall et al., 2019). The current study expands past research by examining multiple generations in this association. VanderVen (2013) suggests that multigenerational research is the “missing element” of life course examination. This study emphasizes the importance of examining multiple generations in the process of adversity and resilience and highlights the long-term implications of parenting behaviors and adversity. Further, the findings in this study suggest that cascades of resilience are not single moments in time but rather play out across multiple generations.

In both of these studies, findings with regards to depressive symptoms were statistically significantly associated with adversity, whereas findings for anxiety symptoms were of similar magnitude but did not reach statistical significance in the first manuscript. These findings highlight the subtle differences between anxiety and depressive symptoms, and suggest future research needs to parse apart these two outcomes rather than lumping them together to measure

mental health. While both anxiety and depressive symptoms affect mood and are often concordant, there are unique differences in the way that these symptoms occur. Prevalence rates of depressive symptoms have been significantly on the rise in recent years (Wilson & Dumornay, 2022) and should be examined as an outcome in the process of adversity exposure throughout the lifespan.

The findings in this dissertation offers many avenues for future research. In these studies, prenatal adversity and adversity experienced before pregnancy were examined. These findings inform both intervention programs as well as developmental science. Intervention and prevention programs should aim to specifically target youth who are at higher risk of adversity exposure as a result of their social position (e.g., minoritized families, low socioeconomic status) to mitigate the deleterious effects of adversity on offspring mental health outcomes. These intervention programs should also focus on the family system and caregiver-child relationship as points of influence to protect against the negative effects of adversity on offspring mental health outcomes. Findings in the current dissertation suggest that adversity during sensitive periods in development may have more salient effects on later well-being that extends beyond short-term mental health or the individual themselves.

Summary

The current dissertation combines investigations that examine adversity across the lifespan as well as the caregiver role as a salient point of influence to buffer against maladaptive outcomes of parental adversity exposure during their own childhood as well as during the prenatal period. This work highlights the long-term implications of cumulative prenatal adversity and the protective role of later parent-child attachment (Study 1). Furthermore, the current studies emphasize how parental ACEs affect offspring depressive and anxiety symptoms, and the

potential protective role of benevolent childhood experiences and family cohesion (Study 2).

This dissertation supports the notion that protective familial processes should be enhanced to buffer against the maladaptive outcomes as a result of earlier adversity exposure.

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