GARCIA, JESSICA M. M.A. The Indirect Effects of Maternal Psychopathology in Early Childhood on Adolescent Health Outcomes. (2022.)
Directed by Dr. Susan Keane. 58pp.

Maternal psychopathology has been linked to adolescent mental health outcomes and an emerging literature suggests that early life stressors induce long-term effects on physical health. Bridging together two related but siloed literatures, the present study examined two indirect effects of maternal psychopathology on adolescent health outcomes. Specifically, maternal psychopathology was hypothesized to exert its longitudinal effect on youth's depressive symptoms and CRP levels, an inflammatory biomarker associated with a host of chronic health conditions, indirectly through a developmentally salient process involving psychosocial functioning in middle childhood and BMI status in adolescence. The present study prospectively analyzed a longitudinal data set of 288 community-dwelling mother-child dyads (162 females, 65% White) spanning 3 time points, early childhood (*M* age = 5), middle childhood (*M* age = 10), and adolescence (*M* age = 17). A structural equation model was employed to examine the indirect effects of maternal psychopathology in early childhood on adolescent health outcomes. Results supported two hypothesized model pathways from maternal psychopathology to depressive symptoms via psychosocial functioning [β=.071, *SE*=.038; CI (.013, .157)] and CRP levels via psychosocial functioning and BMI status [β=.042, *SE*=.022; CI (.006, .091)] in adolescence. Findings highlight the long-term effects of maternal psychopathology and suggest a developmentally salient point of intervention via psychosocial functioning in middle childhood.

THE INDIRECT EFFECTS OF MATERNAL PSYCHOPATHOLOGY IN EARLY CHILDHOOD ON ADOLESCENT HEALTH OUTCOMES

by

Jessica Garcia

A Thesis

Submitted to

the Faculty of The Graduate School at

The University of North Carolina at Greensboro

in Partial Fulfillment

of the Requirements for the Degree

Master of Arts

Greensboro

2022

Approved by

_____

Dr. Susan Keane
Committee Chair

DEDICATION

I am thankful to my parents, partner, best friends, and mentor for their unwavering belief in me. Their motivation and love sustained the many hours of hard work that went into this thesis. Thank you Mami, Papi, Merse, Eliza, Hayley, Missy, and Dr. Keane.

APPROVAL PAGE

This thesis written by Jessica Garcia has been approved by the following committee of the Faculty of The Graduate School at The University of North Carolina at Greensboro.

Committee Chair

Dr. Keane

Committee Members

Dr. Wideman

Dr. Dollar

Dr. Nelson-Gray

June 16, 2022

Date of Acceptance by Committee

June 16, 2022

Date of Final Oral Examination

ACKNOWLEDGEMENTS

Thank you to all the RIGHT Track parents and children who have repeatedly given their time and effort to participate in this research. Also, much gratitude to the entire RIGHT Track staff for their instrumental help collecting, entering, and coding data.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I: INTRODUCTION

Maternal Psychopathology and its Effect on Childhood Development

Depression, anxiety, and severe mental illnesses can greatly compromise an individual's ability to cope with tasks of daily living. Among women of reproductive age in the United States, more than 14% report current depression and 2.7% report experiencing serious psychological distress (Farr et al., 2010). In a national cohort study of children and adolescents, the overall prevalence of maternal mental illness was 23.2%, which increased during childhood and reached a cumulative risk of 53.1% by adolescence (Abel et al., 2019). Accordingly, one in four children are exposed to maternal mental illness, with depression and anxiety being the most prevalent diagnoses (Abel et al., 2019).

In the context of parenthood, maternal psychopathology, encompasses a range of disorders and symptoms including depression, anxiety, OCD, hostility, interpersonal sensitivity, somatization, and thought disorders, as well as the degree of psychological distress felt in response to these symptoms (Derogatis, 1994). Maternal psychopathology affects not only a mother's self-efficacy, but a vast literature reflects that children's development and well-being is also compromised (Alder et al., 2007; S. H. Goodman et al., 2011; S. H. Goodman & Gotlib, 1999; Madigan et al., 2018).

While depression is just one facet of maternal psychopathology, Goodman and Gotlib (1999) put forth a conceptual model grounded in developmental psychopathology to explain *how* maternal depression increases the likelihood for child psychopathology. Namely, they suggest that in any mother-child dyad one or more of these four mechanisms is operating in the transmission of risk: (1) genetic predisposition, (2) innate dysfunctional neuroregulatory processes, (3) negative maternal cognitions, behaviors, and affect, and (4) the stressful context of

the children's lives. The present study extends Goodman and Gotlib's conceptualization to include maternal psychopathology broadly and focuses on the potentially modifiable risk mechanisms by which maternal psychopathology potentiates risk for poor adolescent mental and physical health outcomes. It is hypothesized that negative maternal cognitions, behaviors, and affect contributes to, and are a part of, a stressful context that negatively impacts a child's psychosocial functioning.

Psychosocial Functioning in Middle Childhood

Psychosocial Functioning in middle childhood may be understood through the salient development tasks of this period, which includes the formation and maintenance of friendships, feeling accepted by a peer group, and the increased ability to self-regulate strong emotions and effectively cope with negative emotions (Blair et al., 2014; Colle & Del Giudice, 2011; McDowell et al., 2002).

A child's relationship with their parental figures serves to scaffold the development of self-regulatory processes and sets the stage for navigating peer relationships. Eisenberg and colleagues (Eisenberg et al., 1998) provide a developmental framework that conceptualizes how parent-child interactions affect later social competence. Namely, parents influence children's emotions and emotion-related behaviors through parental emotion-related socialization behaviors, which encompasses parental reactions to children's emotions, discussion of emotions, and parental expression of emotion. Ineffective parental emotion-related socialization behaviors, marked by negative, hostile, punitive or minimizing parental reactions to children's emotions, a dearth of emotion-related discussions, and parental expression of negative emotions (e.g., hostility or anger) are associated with subsequent negative emotionality and poor social competence (Eisenberg et al., 1998).

Eisenberg and colleagues (1996, 1998) elucidate that negative parental reactions to children's display of emotions serves to exacerbate the emotional arousal children are feeling, leading to an increase of emotional dysregulation and nonadaptive behaviors. In turn, children's nonadaptive behaviors have consequences for both social competence and self-regulatory processes (Hoffman, 1983). Moreover, negative parental reactions also affect the quality of the parent-child relationship, such that children's emotional security, attachment, and cognitive schemas about social interactions are compromised over time.

Empirical research supports this developmental framework. For example, McDowell et al. (2002) found that fourth-grade children whose parent-child interactions were marked by warmth, positive responsiveness, and inductive reasoning, were better able to emotionally regulate when presented with emotionally charged vignettes. Whereas children whose parent-child interactions were more negative (e.g., parents seeing the child as the problem), subsequently offered more negative responses to emotionally charged vignettes. In turn, children's parent-child relationships were associated to peer- and teacher- rated social competency. Similarly, Ringoot and colleagues (2021) found that maternal sensitivity was associated with children's behavioral regulation and children with poorer behavioral self-regulation were observed to have higher levels of peer aggression and peer relationship problems.

Furthermore, supportive maternal reactions, which may be compromised in the context of maternal psychopathology, have been shown to foster greater emotion regulation in children, whereas unsupportive reactions have been associated with poorer emotional and physiological regulation (Perry et al., 2020). In a study by Perry and colleagues, children who were better able to physiologically and emotionally regulate at the age of 10, endorsed greater levels of social

competence and adjustment in adolescence, whereas poorer regulators were at risk for a host of negative outcomes in adolescence, including internalizing problems (Perry et al., 2020). Another study elucidated that the relationship between maternal emotion socialization and friendship quality in middle childhood is mediated through emotion regulation processes (Blair et al., 2014). Specifically, mothers' differential provision of supportive or non-supportive socialization of their children's negative emotions at the age of 5 was associated with emotion regulation capabilities 2 years later, which in turn was associated with their children's self-rated friendship quality in middle childhood (Blair et al., 2012). In sum, the extant literature highlights that a mother's ability to parent in a warm, consistent, and emotionally attuned manner has important consequences for their child's psychosocial functioning in middle childhood.

Maternal Psychopathology and Psychosocial Functioning

Research reflects that those mothers who are experiencing a mental health condition are more likely to endorse increased levels of negative emotionality, respond in maladaptive ways to their children's negative emotions, experience increased difficulties meeting the emotional and social needs of their children, and through social learning and modeling, transmit depressogenic and maladaptive cognitions and behaviors to their children (S. H. Goodman & Gotlib, 1999). To illustrate, Jaenicke et al. (1987) found that children's negative self-concept and self-critical remarks as well as negative attribution style was related to maternal chronic stress, current depressed mood, and the quality of mother-child interactions. Moreover, Hammen and colleagues (1990) found that maternal depression, a facet of maternal psychopathology, affects the quality and consistency of mother-child interactions, such that their ability to parent in a warm, consistent, and sensitive manner is compromised. These negative socialization practices are likely to place children at an increased risk for poorer psychosocial functioning, such that

children in middle childhood are less socially competent and have a dearth of self-perceived supportive peer relationships corresponding to increased levels of loneliness and victimization, increased negative emotionality, and poorer self-regulatory skills. Guided by theory and empirical evidence, the present study examined whether psychosocial functioning in middle childhood is a mediating factor between maternal psychopathology and adolescent health outcomes.

How Maternal Psychopathology Exerts its Influence on Adolescent Mental and Physical Health Outcomes

While there is a vast literature base exploring the relationship between maternal psychopathology and mental health outcomes in their offspring (S. H. Goodman et al., 2011), there is a considerable paucity of research that examines the relationship between maternal psychopathology and adolescent physical health outcomes, despite emerging evidence of the long-enduring negative effects of maternal psychopathology on physical health. One notable study prospectively examined the effects of prenatal maternal depression on adulthood inflammation and found that exposure to maternal depression during pregnancy had immunological effects on offspring 25 years later (Plant et al., 2016). Nonetheless, a distinct but closely related program of research sheds light on the plethora of life-long repercussions on mental and physical health associated with early life stress (Taylor, 2010).

Children exposed to maternal psychopathology are at an increased risk for early life stress (Barker et al., 2018), which encompasses childhood exposure to a single or multiple events that exceed a child's capacity to cope (Pechtel & Pizzagalli, 2011). As highlighted by the extant literature, children whose mothers experience psychopathology, are likely to be exposed to a stressful environmental milieu, and are likely to experience higher rates of socioeconomic

disadvantage, family violence, and low social support (Barker et al., 2012). Moreover, behaviors typically associated with maternal psychopathology, such as lack of warmth, negative reaction to children's emotional displays, and expression of maladaptive cognitions are also experienced as a stressor for a young child.

Early life stress has been shown to potentiate the risk for a host of health outcomes, including metabolic functioning (e.g., cholesterol, insulin levels, glucose, and triglycerides), which has implications for chronic health conditions such as heart disease, hypertension, and Type II diabetes (Taylor et al., 2006), blood pressure (Lehman et al., 2009), systemic inflammation (e.g., IL-6, CRP, TNF-alpha) (Danese et al., 2007; Lehman et al., 2005; G. E. Miller & Chen, 2010) and depression (Heim & Binder, 2012; G. E. Miller & Cole, 2012; Torres-Berrío et al., 2019). An epidemiologic study of diverse participants found that early life stress, defined as low socioeconomic status (SES) and a harsh family environment, predicted body mass index (BMI) status in adulthood, which was in turn associated with increased levels of C-Reactive Protein (CRP), an acute-phase inflammatory biomarker (Taylor et al., 2006). These findings are especially concerning given the robust associations between inflammation and chronic health conditions (Attard et al., 2013).

Moreover, psychosocial stressors that occur in childhood, when the brain and immune system are still developing may have long-lasting repercussions (Danese & J Lewis, 2017). In a large prospective cohort study, Danese and colleagues (2008) found that adults who had experienced severe psychosocial stressors in childhood were 1.48 times more likely to have clinically high levels of CRP, an acute-phase inflammatory protein. Similarly, adolescents who had experienced more normative forms of early life stress (e.g., low socioeconomic status or parental separation) had greater increases in inflammatory markers when becoming depressed

than adolescents who did not experience these early life stressors (Danese & J Lewis, 2017). Moreover, studies show that social stressors involving social conflict, rejection, or exclusion are particularly pernicious and have been found to evoke the strongest inflammatory response in a lab setting (Denson et al., 2009; Dickerson et al., 2009; Slavich & Irwin, 2014).

The present study conceptualizes maternal psychopathology as an early life stressor that serves to potentiate risk for poor physical and mental health outcomes in adolescence via a host of pathways, as suggested by the extant literature. Theoretical and empirical research findings reflect that children's psychosocial development is negatively affected when they are exposed to mothers experiencing psychopathology. Poor psychosocial functioning also represents a stressor for children in middle childhood, as they are less equipped to cope with the salient developmental tasks of navigating peer friendships and self-regulating negative emotions. Therefore, the present study examines a psychosocial functioning pathway in middle childhood that takes into account the effects of peer relationships and self-regulation in predicting adolescent depression and inflammation. Given that epidemiological studies indicate depression is the most prevalent lifetime disorder (Gray et al., 2009; Qualter et al., 2018; Shriver et al., 2021; Storch et al., 2007) and risk for depression increases in adolescence (Costello et al., 2003), adolescence is a particularly salient developmental period for the examination of depressive symptoms. Moreover, in light of research showing the repercussions of inflammation on health (Taylor et al., 2006), understanding the developmentally salient mechanisms underlying this association is imperative.

CHAPTER II: THE CURRENT STUDY

The present study investigates the role of maternal psychopathology on adolescent depressive symptoms and C-Reactive Protein, an inflammatory biomarker. It was hypothesized that maternal psychopathology exerts its longitudinal influence on adolescent mental and physical health through psychosocial functioning in middle childhood. Research suggest that maternal mental illness compromises a child's social competencies and abilities to self-regulate, which may lead to experiences of loneliness and victimization (S. H. Goodman et al., 2011; S. H. Goodman & Gotlib, 1999; Hammen et al., 1990). These social stressors coupled with increased negative emotionality and impaired emotional regulation capacities set the stage for poor mental and physical health in adolescence. Therefore, it was hypothesized that children whose mothers experienced higher levels of maternal psychopathology would evidence lower levels of psychosocial functioning, which would, in turn, be associated with higher levels of depressive symptoms in adolescence.

Additionally, while there is a vast literature on the effects of social stressors and depression (Cramer et al., 2012; Keller et al., 2007; Slavich et al., 2010; Slavich & Irwin, 2014), the relationship between social stressors and inflammation is less understood. Thus, the present study contributes to the extant literature by examining the longitudinal effects of maternal psychopathology on C-Reactive Protein, a biomarker of inflammation, through psychosocial functioning in middle childhood and BMI in adolescence. Physiological research reflects that there is a robust relationship between BMI and levels of inflammation in the body (DeBoer, 2013; Goosby et al., 2016), such that high levels of adiposity are associated with higher levels of inflammation, which is a risk factor for cardiovascular diseases and other chronic illnesses (Attard et al., 2013). Research has also found that children who have poor psychosocial

functioning are more likely to be overweight and obese (Puder & Munsch, 2010), perhaps as a result of a maladaptive coping mechanism (Czaja et al., 2009; Hasler et al., 2005; Konttinen et al., 2010). Moreover, salient aspects of psychosocial functioning, such as emotion regulation and peer relationships in childhood have been directly linked to BMI status in youth (Harrist et al., 2013; Qualter et al., 2018; Shriver et al., 2021; Storch et al., 2007). Therefore, the present study examines a biopsychosocial pathway from maternal psychopathology in early childhood to adolescent levels of inflammation through psychosocial functioning in middle childhood and BMI status in adolescence. It was hypothesized that children whose mothers experienced higher levels of maternal psychopathology would evidence lower levels of adaptive psychosocial functioning, which would in turn be associated with higher levels of BMI. Higher levels of BMI were expected to be positively associated with higher levels of CRP. In addition, in light of an emerging literature base that suggests a relationship between depressive symptoms and inflammation (Lotrich et al., 2011; A. H. Miller et al., 2009; Slavich & Irwin, 2014), the present study hypothesized a bi-directional relationship between depressive symptoms and CRP levels in adolescence (Colasanto et al., 2020).

Socioeconomic Status

Research reflects that socioeconomic status is a predictive factor for maternal mental health (Hein et al., 2014), and that depressed mothers are exposed to higher rates of socioeconomic disadvantage (Ertel et al., 2011). Moreover, children whose mothers have a mental illness are more likely to be exposed to environmental stressors, including low socioeconomic status (Barker et al., 2012; Hardie & Landale, 2013) and low SES in childhood has been identified as a risk factor for depression in adolescence (Reiss, 2013). Furthermore, an inverse relationship between socioeconomic status and BMI status has been found in youth (De

Spiegelaere et al., 1998; Fradkin et al., 2015), with lower SES being a risk factor for obesity. Likewise, a meta-analysis conducted by Milaniak & Jaffee (2019) found that low SES in childhood and adolescence predicted higher levels of adulthood inflammation (e.g., CRP, IL-6, and fibrinogen). Given the significant role of SES in the proposed model pathways, SES was examined as a covariate in each direct pathway, thereby elucidating whether the proposed variables predicted adolescent health outcomes above and beyond the influence of socioeconomic status in childhood.

Gender Differences

A large literature base suggests that gender may play a differential role in the development of depression beginning in puberty, whereby females are more likely to develop depression than males (Conley et al., 2012; Hankin, 2015; Hankin et al., 1998). Moreover, there is mixed evidence concerning the effect of child gender on maternal emotional socialization practices (Denham et al., 1997; Blair et al., 2014). Furthermore, research reflects that there are sex differences in BMI status and levels of C-Reactive Protein (CRP), with obesity prevalence and CRP being generally higher in women than in men (Cartier et al., 2009; Khera et al., 2009; Lovejoy et al., 2009; Mauvais-Jarvis, 2015; Peters et al., 2019; Thorand et al., 2006). Therefore, a multigroup analysis was conducted to examine whether the proposed biopsychosocial pathway differed by sex. The present study did not make any a priori inferences about the nature of differences in the pathway for females and males.

Goals and Hypotheses

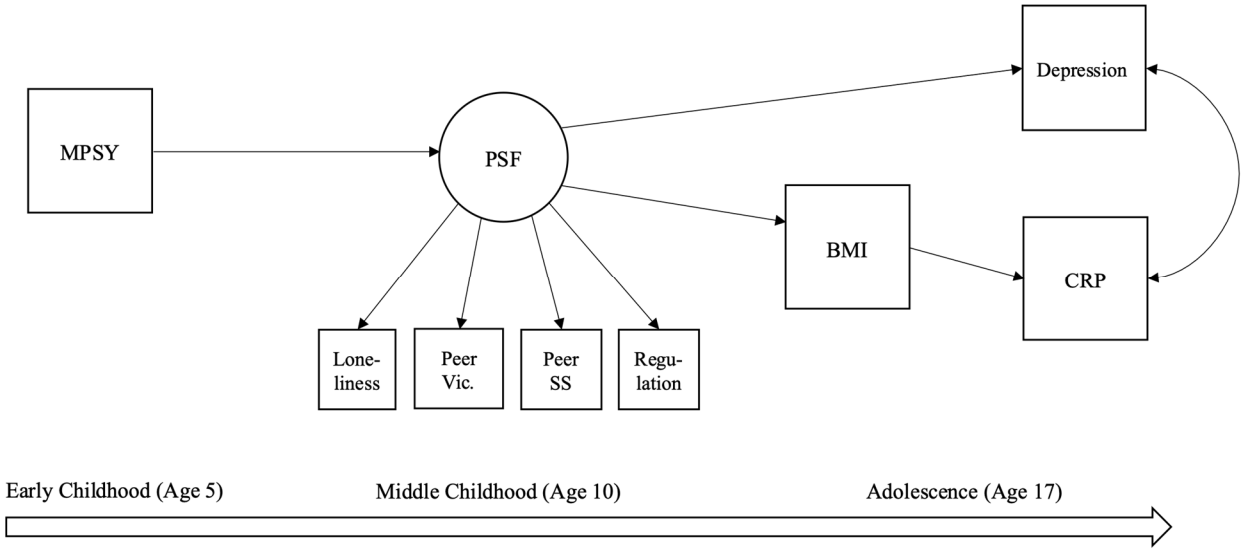
The present study aimed to contribute to the extant literature base by testing a biologically plausible and developmentally salient biopsychosocial pathway by which maternal psychopathology exerts its longitudinal effects on adolescent mental and physical health (see

Figure 1). The following direct effects were examined. First, it was hypothesized that high levels of maternal psychopathology in early childhood would be negatively related to adaptive psychosocial functioning in middle childhood. Second, it was hypothesized that youth who evidenced lower levels of adaptive psychosocial functioning would be more likely to report higher levels of depressive symptoms in adolescence. Third, it was hypothesized that youth who evidenced lower levels of adaptive psychosocial functioning in middle childhood would have higher levels of BMI in adolescence. Fourth, it was hypothesized that youth with higher BMI levels would have higher levels of C-Reactive protein. Finally, a bidirectional relation between depressive symptoms and CRP was hypothesized.

Next, two main indirect effects were tested in this study to explicate the longitudinal relationship between maternal psychopathology and adolescent mental and physical health outcomes. First, an indirect effect of maternal psychopathology on adolescent depressive symptoms through psychosocial functioning in middle childhood was hypothesized. Second, an indirect effect of maternal psychopathology on adolescent levels of inflammation via psychosocial functioning in middle childhood and BMI status in adolescence was hypothesized.

Lastly, a multigroup analyses by sex was conducted to explore plausible sex differences in the model pathways. No a priori hypotheses were put forth.

Figure 1. Hypothesized Model



Note. MPSY= Maternal Psychopathology; PSF= Psychosocial Functioning; Loneliness= Reversed-Coded Loneliness; Peer Vic= Reversed-Coded Peer Victimization; Peer SS= Peer Social Support; Regulation= Reversed-Coded Negativity and Regulation Composite; Depression= Depressive symptoms; BMI= Body Mass Index; CRP= C-Reactive Protein. Childhood SES was examined as a covariate.

CHAPTER III: METHODS

Recruitment and Attrition

The current study utilized data obtained from RIGHT Track [NIMH 55625, NIMH 55584 & NIMH 58144], a longitudinal study that investigates socioemotional development and internalizing and externalizing behavior problems, as well as health markers implicated in the development of cardiovascular risk factors. The current study analyzes data from three cohorts of participants who are part of the ongoing longitudinal study (Cohort 1: 1994-1996; Cohort 2: 2000-2001; Cohort 3: 1998). The goal at the commencement of recruitment in 1994 was to obtain a representative sample of the surrounding community, with respect to race and socioeconomic status (SES), of children at-risk for developing externalizing behavior problems. Children were classified as being at risk for future externalizing behaviors if their T-scores reflected a 60 or above in the externalizing behaviors subscales of Child Behavior Checklist (CBCL 2-3; Achenbach, 1992). Efforts were made to obtain equal sex representation. Families were recruited through day care centers, the Country Health Department, and the local Women, Infants, and Children (WIC) program.

Cohorts 1 and 2 ($N = 307$) were recruited at 2 years of age and screened using the mother-reported Child Behavior Checklist (CBCL 2-3; Achenbach, 1992). Cohort 3 participants were recruited when infants were 6 months of age (in 1998) based on their level of frustration in response to lab observation and parent report (see Calkins et al., 2002, for more information). Children from Cohort 3 whose mothers completed the CBCL at two-years of age ($N = 140$) were then included in the larger study. A total of 447 families comprised the RIGHT Track study at age 2, with 37% of children classified as being at risk for future externalizing problems. Chi square analyses did not indicate significant demographic differences between cohorts with regard

to gender, χ^2 (2, N = 447) = .63, p = .73, race, χ^2 (2, N = 447) = 1.13, p = .57, or two-year SES, F (2, 444) = .53, p = .59.

A total of 365 families participated at age 5, including four families that did not participate in the 4-year assessment. Again, there were no significant differences between families who did and did not participate in terms of gender, χ^2 (1, N = 447) = .76, p = .38, race, χ^2 (1, N = 447) = .14, p = .71, 2-year SES, t (432) = -1.93, p = .06, and 2-year externalizing T score, t (445) = 1.39, p = .17. A total of 357 families participated at age 10, including 35 families that did not participate in the 5-year assessment. Again, there were no significant difference between families who did and did not participate in the 10-year assessment in terms of child gender, χ^2 (1, N = 447) = 3.31, p = .07; race, χ^2 (3, N = 447) = 3.12, p = .08; 2-year SES, t (432) = .02, p = .98; or 2-year externalizing T score, t (445) = -.11, p = .91. Lastly, a total of 313 families participated at age 17, including 22 families that did not participate in the 10-year assessment. There were no significant differences between families who did and did not participate in the 15-year assessment in terms of child gender χ^2 (1, N = 447) = 3.12, p = .08; 2-year SES t(432) = -.72 p = .47; or 2-year externalizing T score t (445) = -1.05, p = .29. Non-white participants were less likely to participate in the 17-year assessment, χ^2 (1, N = 447) = 5.05, p = .03.

Current Sample

The current sample is composed of 288 families [162 females and 65.3% White) who were representative of the surrounding community at the time of data collection (e.g., a mid-sized racially diverse southeastern city of the US). Moreover, socioeconomic data collected at age 5, indicate that study participants came from economically diverse families, based on Hollingshead (1975) scores ranging from 14 to 66 (M = 43.28, SD = 10.46), thus representing

families from each level of social strata captured by this scale. Families were included in the present sample if they participated in the 5-year data collection and had at least one data point at the 10- and 17-year assessments. FIML was employed to address missing data, as it is the least biased method of including the maximum amount of data when data is missing at random (Enders & Bandalos, 2001).

Procedures

Mother-child dyads participated in an ongoing longitudinal research study that began data collection when children were 2 years of age and has continued into young adulthood. Consent from mothers and assent from youth were obtained prior to each wave of data collection and families were compensated for their participation after each study visit. At each laboratory visit, mothers completed questionnaires regarding family composition, parenting practices, and their child's functioning. At the ages of 10 and 17, adolescents also completed questionnaires about their own functioning. In adolescence, youth were asked if they would like to participate in data collection concerning their physical health. Youth who agreed completed assessments of body composition, including BMI, underwent blood analysis for biomarker data, and completed self-reported questionnaires on health behaviors (Wideman et al., 2016).

Measures

Maternal Psychopathology

When children were 5 years old, mothers completed the Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1994), a 90-item self-report measure designed to assess adult psychopathology symptoms. Mothers were asked to rate their subjective level of distress in relation to the 90 items over the course of the past 7 days. Their responses were captured on a 5-point Likert scale, ranging from *Not at all* (0) to *Extremely* (4).

The SCL-90-R yields 9 subscales, composed of nine specific symptom dimensions, and three global indices, including the Global Severity Index (GSI). The current study examined mothers' GSI score, which is designed to indicate the current level of overall psychopathology, combining the number of symptoms with the intensity of perceived psychological distress. The current study converted the participant's scores to T-scores using nonpatient norms. Previous research has demonstrated adequate psychometric properties for the SCL-90-R (Derogatis, 1994; Derogatis & Cleary, 1977), with acceptable to high internal consistency for each of the subscales and acceptable test-retest reliability (Derogatis & Cleary, 1977). Cronbach's alpha for the current sample reflects high reliability ($\alpha = .966$).

Socioeconomic Status

Mothers provided demographic information at every visit. The current study examined mothers' report of socioeconomic status when children were 5 years of age using the Hollingshead Four Factor Index of Socioeconomic Status (SES; Hollingshead, 1975). Social status was estimated by examining four principal components: education, occupation, sex, and marital status. Hollingshead scores range according to an individual's social strata, with scores for unskilled laborers and menial service workers falling between 8-19, machine operators and semiskilled workers falling between 20-29, skilled craftsmen, clerical, and sales workers falling between 30-39, medium business, minor professional, technical workers, representative of the middle class, falling between 40-54, and major business and professionals falling between 55-66.

Psychosocial Functioning

Children's psychosocial functioning in middle childhood was examined via a latent variable, which was derived using summary item indicators from three questionnaires. Higher levels of psychosocial functioning indicated increased social competence as represented by a

child's self-report of supportive peer relationships, lower levels of loneliness and victimization, as well as greater emotion regulation and lower levels of negative affect and lability.

At the age of 10, children completed the Perceptions of Peer Support Scale (PPSS; Kochenderfer & Ladd, 1996), an 18-item self-report measure designed to assess children's perception of their peer relationships. The PPSS yields three subscales: Social Support from Peers, Perceived Victimization, and Engagement in Bullying Behaviors. Responses are captured on a 5-point Likert scale, ranging from *Never* (1) to *Always* (5). The current study utilized the sum of the items from the Perception of Peer Scale subscale as a summary indicator, with higher scores on indicating greater levels of perceived support. The items of Perceived Victimization subscale were reversed coded, and the sum of the items was used as a summary indicator, with higher scores indicating lower levels of peer victimization.

Children also completed the Loneliness Scale, a 24-item self-report measure designed to assess loneliness and social dissatisfaction. The Loneliness and Social Dissatisfaction Questionnaire designed by Asher et al. (1984), and subsequently revised by Asher and Wheeler (1985) formed the basis of the measure used in the present study. Research with older children has revealed one principal factor on which all loneliness and social dissatisfaction items load (Asher et al., 1984; Asher & Wheeler, 1985). Of the 24 total items there are 16 target items and 8 "filler" items, included to help children feel more open and relaxed about indicating their attitudes towards their experiences. Responses are captured on a 5-point Likert scale, ranging from *not at all true* (1) to *always true* (5). Of the 16 target items, 6 items were reversed scored (1, 4, 8, 10, 16, 22), so that higher scores reflect less loneliness. The total sum was used as an item indicator.

Moreover, mothers reported on their children's emotion regulation capabilities using the Emotion Regulation Checklist (ERC; (Asher et al., 1984), a 24-item measure designed to assess the quality of a child's emotion regulation, or ability to control and modify his or her emotions and emotive expression, as well as overall affect. Responses are captured on a 4-point Likert scale, ranging from *Never* (1) to *Almost Always* (4). The ERC yields two subscales, the Lability/Negativity subscale and the Emotion Regulation subscale. The present study reversed coded items 4, 5, 9, and 11 on the Liability/Negativity subscale so that higher scores indicate lower levels of maladaptive lability and negativity. A total regulation composite was created by summing all the items on the Lability/Negativity subscale and the Emotion Regulation subscale to create a summary indicator that captured emotion regulation and overall affect; higher scores indicate lower levels of lability/negativity and higher levels of emotional regulation.

Depression

To examine the presence of depressive symptoms in adolescence (*M* age = 17), youth completed the Children's Depressive Inventory (CDI; Kovacs, 1992), a 27-item measure that is designed to assess depressive symptoms experienced in the past two weeks. Youth are asked to select 1 of 3 sentences that best represents how they have been feeling, with each item representing a different degree of severity. The CDI yields 5 subscales and a total CDI score that represents the composite of the five scales (e.g., *Negative Mood, Interpersonal Problems, Ineffectiveness, Anhedonia, and Negative Self-Esteem),* therefore capturing overall depressive symptomology across the five subscales. Scores between 45-55 indicate *Average* levels of depressive symptoms. Research on the CDI reflects that this measure has good internal consistent, test-retest reliability, and is sensitive to change (Masip et al., 2010). The present study

examined the total T-score by gender. Cronbach's alpha for the current sample reflects high reliability ($\alpha = .865$).

BMI

Adolescents' height and weight measures were collected (*M* age = 17); height was measured to the nearest 0.1 cm with a wall mounted, calibrated stadiometer (SECA, Chino CA) and weight was measured to the nearest 0.1 kg with a balance-beam scale (Detecto-medic, Brooklyn, NY) (Wideman et al., 2016). Body mass index (BMI) was calculated using the standard formula [weight (kg)/height (m^2)]. According to the CDC, BMI values less than 18.5 fall within the *underweight* range; BMI values between 18.5 to 24.9 fall within the *normal* or *Healthy Weight* range; BMI values between 25.0 to 29.9 fall within the *overweight* range; and BMI values 30 or greater fall within the *obese* range.

Biomarker of Inflammation

Blood samples were collected to measure several biomarkers, including C-Reactive Protein (CRP) (Wideman et al., 2016). To minimize the influence of acute inflammation, the present study controlled for potentially confounding sources of inflammation. For example, participants were asked to fast and limit their physical activity for at least 10 hours prior to their visit. Moreover, participants were asked to reschedule their visit if they had felt ill in the past week or had any surgical procedures in the past month, or if they had received any vaccines within the past two weeks, or if they had taken any anti-inflammatory medications within the past 10 days. Blood samples were primarily collected from an antecubital site using butterfly needles and a vacutainer system. Universal precautions and OSHA guidelines were strictly followed when handling blood. A total of 10ml of blood was collected in serum separator tubes using participant's unique and confidential subject ID and date of blood draw. Blood was first

allowed to coagulate for 20 minutes and then spun at 3000 rpm for 15 minutes at 4°C. To minimize freeze/thaw cycling, blood serum was divided into multiple aliquots of 500-1000µl and stored at -80°C until analysis. CRP and other inflammatory markers were analyzed in the Exercise Endocrinology Lab at UNC Greensboro using the Luminex 200S multiplex platform (Luminex, Austin TX). Using appropriate quality controls, all samples from individuals where run using the same ELISA plate to minimize inter-assay variability and to insure comparability of assays across ages.

C-Reactive Protein is an acute-phase reactant protein that is synthesized in the liver and is commonly used in health research as a biomarker of inflammation (Nehring et al., 2022). CRP values greater than 3mg/L may indicate risk for heart disease (Ridker, 2003).

Data Analysis Plan

Descriptive statistics displaying means, standard deviations, skewness and kurtosis are reported in Table 1, correlations of all study variables in the general sample are reported in Table 2, and correlations by gender will be reported in Table 3 using IBM SPSS Statistics for Macintosh, Version 28 (2021).

For the substantive analyses, a confirmatory factor analysis for psychosocial latent variable was constructed (Matsunaga, 2010). Next, structural equation modeling was conducted to examine the associations between maternal psychopathology and health outcomes in adolescence accounting for the effects of childhood SES utilizing Mplus Version 8.7 (Muthén & Muthén, 2021). Full Information Maximum Likelihood (FIML) was employed as it is the least biased method of including the maximum of amount of data when data is missing at random (Enders & Bandalos, 2001). Figure 1 displays the hypothesized model.

Model fit was assessed by examining the comparative fit index (CFI; Marsh & Hau, 2007), the root mean square error of approximation (RMSEA; Cole & Maxwell, 2003) and the standardized root mean square residual (SRMR). Values greater than .95 indicate good model fit for the CFI; values less than .05 indicate good model fit and values between .05 and .08 indicate acceptable model fit for RMSEA (Bowen and Guo, 2011); and values less than or equal to .08 indicate good model fit for SRMR. A non-significant chi square difference test indicates that the hypothesized path model is not different from the data (Bowen & Guo, 2011); however chi-square will not be used as an index of fit due to evidence that it is a biased measure strongly influenced by sample size (Bentler & Bonett, 1980; Peugh & Feldon, 2020; Tucker & Lewis, 1973).

To test the indirect pathway leading to adolescent health outcomes, a bias-corrected bootstrapping procedure (10,000 draws) was employed. This approach has been shown to generate the most accurate CIs for indirect effects, reducing Type 1 error rates and increasing power over other similar tests (MacKinnon et al., 2004).

Finally, to examine to effect of gender on the hypothesized model pathways, a multigroup analysis was performed, whereby an unconstrained model was compared to a model constrained by sex. A chi-square difference test was computed to test whether there is evidence of a significant difference between the more complex model (fully unconstrained model) or the more parsimonious model (constrained by gender). A significant chi-square difference test would suggest gender differences in the hypothesized pathways.

CHAPTER IV: RESULTS

Preliminary Analyses

Table 1 shows the descriptive statistics for all study variables among the full sample and by sex. Descriptives statistics reflect that all variables were approximately normally distributed. Mothers' GSI score ranged from 30 to 73 and, on average, mothers' T-score was 49.37 (*SD* = 11.28). The lambda loadings of the four summary item indicators used to create the psychosocial functioning latent variable were all above .40 (see Figure 2), indicating satisfactory loading onto the latent variable (Matsunaga, 2010). Adolescents' Total T-scores fell between 34 and 89 and, on average, youths' T-score was 43.98 (*SD* = 8.69). BMI values for adolescents ranged from 17 to 53 (*M* = 24.94, *SD* = 5.85); seventy-five adolescents had BMI values equal to or greater than 25. Finally, CRP values for adolescents ranged from .02mg/L to 4.48 mg/L (*M* = .83, *SD* = 1.07); six adolescents evidenced values greater than 3mg/L. Mothers' SES scores ranged from 14 to 66 and, on average, mothers' SES score was 43.23 (*SD* = 10.46), reflecting that the present sample are representative of the middle class.

Table 1. Descriptive Statistics

	Full Sample (*n=288)*					Males (*n=126)*		Females (*n=162)*	
Variable	N	*M*	*SD*	Skew-ness	Kurtosis	*M*	*SD*	*M*	*SD*
1. MPSY	287	49.37	11.28	-.15	-.77	50.37	12.21	48.60	10.46
2. SES	287	43.23	10.46	-.38	-.24	44.04	10.12	42.69	10.71
3. Loneliness	271	67.47	9.86	-.94	.81	66.07	9.38	68.54	10.13
4. Peer Vic.	256	17.22	2.91	-1.17	.91	17.11	3.07	17.36	2.78
5. Peer SS	256	40.81	7.55	-.96	.60	38.95	7.62	42.22	7.2
6. Regulation	256	76.83	8.41	-.68	.29	76.2	7.53	77.34	9.05
7. Depression A	235	44.04	8.69	1.50	3.42	43.77	7.74	44.13	9.32
8. BMI A	192	24.95	5.83	1.72	3.7	24.94	6.24	24.94	5.6
9. CRP A	104	.82	1.07	1.83	2.47	.74	1.06	.89	1.09

Note. MPSY= Maternal Psychopathology; SES= Socioeconomic Status; Loneliness= Reversed-Coded Loneliness; Peer Vic= Reversed-Coded Peer Victimization; Peer SS= Peer Social Support; Regulation= Reversed-Coded Negativity and Regulation Composite; Depression A= Depressive Symptoms in Adolescence; BMI A= Body Mass Index in Adolescence; CRP A= C-Reactive Protein in Adolescence.

Table 2 displays the correlations among the study variables for the full sample. Small to moderate negative correlations were observed between maternal psychopathology in early childhood and adaptive psychosocial functioning indicators, specifically, reverse-coded peer

victimization, reverse-coded loneliness, social support from peers, and the regulation composite in middle childhood. These correlations reflect that maternal psychopathology is associated with poorer psychosocial functioning. Small negative associations are observed between the indicators of adaptive psychosocial functioning in middle childhood and depressive symptoms in adolescence. Moreover, small negative correlations are observed between reverse-coded loneliness, reverse-coded peer victimization, and reverse-coded negativity and regulation composite, and BMI in adolescence. BMI status was strongly correlated with CRP in adolescence in the expected direction. Finally, weak correlations are observed between socioeconomic status and maternal psychopathology, reverse-coded negativity and regulation composite in middle childhood, and race. The observed correlations comport to expectation.

Table 2. Correlations of Study Variables Among General Sample

Variable	1	2	3	4	5	6	7	8	9	10	11
1. MPSY		-.21**	-.11	-.16*	-.15*	-.33**	.21**	.08	.08	-.08	.02
2. SES			.05	.03	.07	.15*	-.06	-.13	-.05	-.06	-.2**
3. Loneliness				.51**	.6**	.31**	-.28**	-.25**	-.04	.13*	-.03
4. Peer Vic.					.43**	.28**	-.16*	-.27**	-.26**	.04	-.08
5. Peer SS						.2**	-.19**	-.12	.03	.22**	.02
6. Regulation							-.24**	-.15*	-.05	.07	-.07
7. Depression A								.15*	.04	.02	.14*
8. BMI A									.55**	.00	.25**
9. CRP A										.07	.09
10. Sex[a]											.06
11. Race[b]											

Note. MPSY= Maternal Psychopathology; SES= Socioeconomic Status; Loneliness= Reversed-Coded Loneliness; Peer Vic= Reversed-Coded Peer Victimization; Peer SS= Peer Social Support; Regulation= Reversed-Coded Negativity and Regulation Composite; Depression A =Depressive Symptoms in Adolescence; BMI A= Body Mass Index in Adolescence; CRP A= C-Reactive Protein in Adolescence.

[a] Sex is dichotomized, 1 = males (*n* = 126, 44.1%) and 2 = females (*n* = 162, 55.9%).

[b] Race is dichotomized, 0 = white (*n* = 188, 65.3%) and 1 = race/ethnic minority status (*n* =100, 34.7%).

* p < .05; ** p < .01; *** p <.001

Table 3 displays the correlations among the study variables by sex. Preliminary analyses revealed some sex differences in the correlation between maternal psychopathology and psychosocial functioning indicators, whereby maternal psychopathology was significantly correlated with reversed-coded peer victimization, peer social support, and the regulation composite for females. In contrast, maternal psychopathology was significantly correlated only with the regulation composite for males. Moreover, all psychosocial functioning indicators were significantly correlated with depressive symptoms and BMI status in adolescence for females but not for males. BMI status is significantly correlated with CRP for both sexes. These differences were further explored in the multigroup analysis.

Table 3. Correlations of Study Variables by Sex

Variable	1	2	3	4	5	6	7	8	9	10
1. MPSY		-.25	-.06	-.23*	-.19*	-.28**	.23**	.02	.12	-.01
2. SES	-.17		.06	.06	.09	.11	-.06	-.16	-.003	-.23**
3. Loneliness	-.15	.05		.56**	.62**	.26**	-.35**	-.38**	-.07	-.11
4. Peer Vic.	-.09	.01	.45**		.34**	.27**	-.25**	-.39**	-.44**	-.16
5. Peer SS	-.09	.09	.58*	.52**		.16	-.27**	-.22*	.03	-.05
6. Regulation	-.4**	.22*	.37**	.3**	.25*		-.24**	-.21*	-.14	-.05
7. Depression A	.19	-.05	-.13	-.03	-.09	-.25		.19*	.04	.12
8. BMI A	.14	-.09	-.08	-.11	.03	-.06	.09		.55**	.22*
9. CRP A	.03	-.12	.001	.07	.09	.06	.03	.58*		.64
10. Race[a]	.06	-.16	.07	.002	.08	-.11	.18	.28*	.14	

Note. Correlations for males are presented under the diagonal in blue, correlations for females above the diagonal in red.

MPSY= MPSY= Maternal Psychopathology; SES= Socioeconomic Status; Loneliness= Reversed-Coded Loneliness; Peer Vic= Reversed-Coded Peer Victimization; Peer SS= Peer Social Support; Regulation= Reversed-Coded Negativity and Regulation Composite; Depression A =Depressive Symptoms in Adolescence; BMI A= Body Mass Index in Adolescence; CRP A= C-Reactive Protein in Adolescence.

[a]Race is dichotomized, 0 = white (*n* = 188, 65.3%) and 1 = race/ethnic minority status (100 = 34.7%).

* * p < .05; ** p < .01; *** p <.0

Primary Analyses

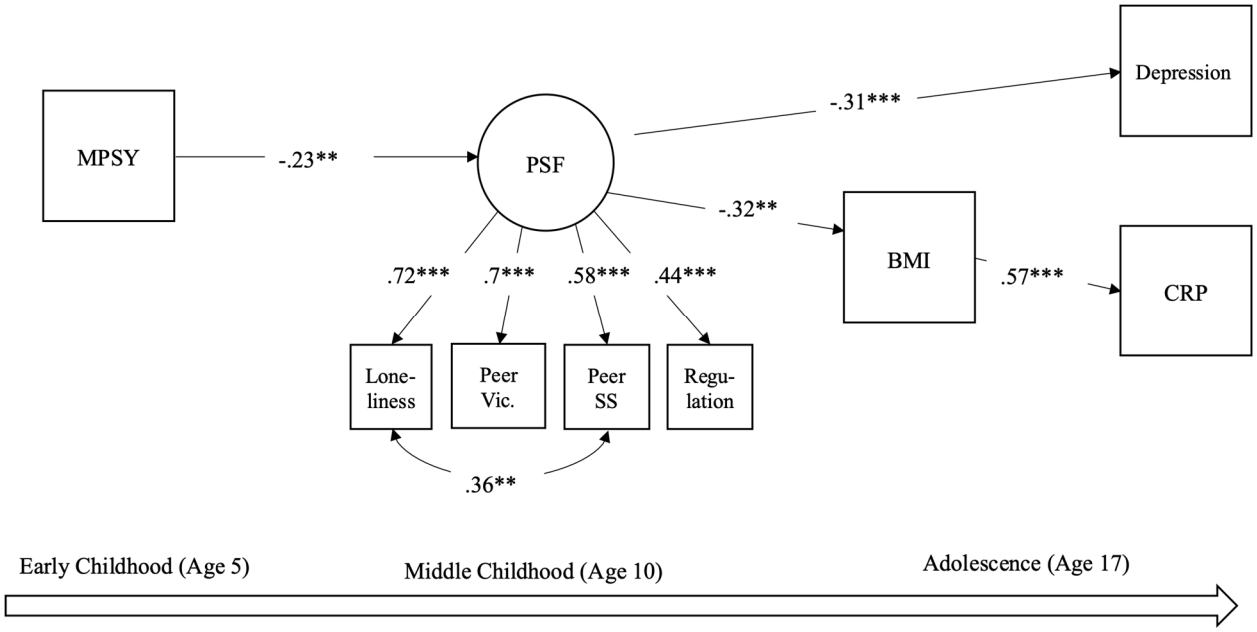
A path analysis was conducted to assess the associations between maternal psychopathology and health outcomes in adolescence utilizing Mplus Version 8.7 (Muthén & Muthén, 2021). FIML was used to handle missing data. First, a confirmatory factor analysis (CFA) was fitted to create the psychosocial functioning variable composed of four summary indicators representative of the latent construct. Lambda loadings were all above .40, indicating satisfactory loading onto the latent variable (Matsunaga, 2010). The indicators Loneliness and Peer Social Support were allowed to covary and were significantly correlated ($r = .36$; $p = .002$).

The hypothesized model fit the data well, χ^2 (20, N = 288) = 34.312, p = .024, CFI = .96, RMSEA = .05 [90% CI = .018 to .077], SRMR = .047. Chi square was not used as an index of fit because it is strongly influenced by sample size (Bentler & Bonett, 1980; Peugh & Feldon, 2020; Tucker & Lewis, 1973). The structural equation model with significant standardized path coefficients is displayed in Figure 2 and unstandardized coefficients and confidence intervals are presented in Table 4. Substantive interpretations derived from the model pathways follow.

To address the first question, I examined the direct effect of maternal psychopathology in childhood to psychosocial functioning in middle childhood. As hypothesized, model results indicated that maternal psychopathology was negatively associated with adaptive psychosocial functioning in middle childhood (β = -.147, p = .002). Moreover, as hypothesized, adaptive psychosocial functioning was negatively associated with depressive symptoms in adolescence (β = -.373, p = .004) and it was also negatively associated with higher levels of BMI in adolescence (β = -.262, p = .006). In turn, as expected, higher levels of BMI were positively associated with higher levels of CRP, or inflammation, in adolescence (β = .107, p < .001). Contrary to the

hypothesis, there was not a significant bidirectional relation between depressive symptoms and CRP in adolescence. All direct effects took into account childhood socioeconomic status.

Figure 2. Significant Standardized Coefficients of the Hypothesized Path Model



Note. Model Fit: χ^2 (20, N = 288) = 34.312, p = .024, CFI = .96, RMSEA = .05 [90% CI = .018 to .077], SRMR = .047. Only significant standardized coefficients are depicted in this figure. The analysis was adjusted for socioeconomic status in each pathway.

MPSY= Maternal Psychopathology; PSF= Psychosocial Functioning; Peer Vic= Reversed-Coded Peer Victimization; Peer SS= Peer Social Support; Regulation= Reversed-Coded Negativity and Regulation Composite; Depression= Depressive symptoms; BMI= Body Mass Index; CRP= C-Reactive Protein.

*p < .05; **p < .01; ***p < .001.

Table 4. Unstandardized Model Estimates of Direct Effects, Standard Errors, and 95% Bias-Corrected Confidence Intervals (10,000 draws)

			95% Confidence Intervals	
Specific Direct Paths	Estimate	*SE*	Lower	Upper
MPSY → PSF	-.147**	.049	-.239	-.048
PSF → Depression A	-.373**	.128	-.631	-.133
PSF → BMI A	-.262**	.096	-.462	-.079
BMI A → CRP A	.107***	.022	.053	.142
MPSY → Depression A	.094^{+}	.054	-.016	.195
Covariances				
Depression A ↔ CRP A	-0.312	.672	-1.616	1.070
Loneliness ↔ Peer SS	15.361**	5.996	2.352	26.145

Note. MPSY= Maternal Psychopathology; PSY = Psychosocial Functioning; BMI A= Body Mass Index in Adolescence; Depressive Symptoms in Adolescence; CRP A= C-Reactive Protein in Adolescence.

$^{+}$ p < .10 *p < .05; **p < .01; ***p < .001.

Indirect Pathways to Adolescent Health Outcomes

Next, the impact of maternal psychopathology on mental and physical health was tested via two indirect pathways, using a bias-corrected bootstrapping approach with 10,000 draws. An indirect effect is considered significant if the confidence interval does not include zero (du Prel et al., 2009) (see Table 5). As hypothesized, psychosocial functioning in middle childhood significantly mediated the relation between maternal psychopathology and depressive symptoms

in adolescence [β=.071, *SE*=.038; 95% CI (.013, .157)]. Moreover, results also reflect that psychosocial functioning in middle childhood and BMI levels in adolescence significantly mediated the relationship between maternal psychopathology and inflammation in adolescence [β=.042, *SE*=.022; 95% CI (.006, .091)].

Table 5. Standardized and Unstandardized Estimates of Significant Indirect Effects, Standard Errors, and 95% Bias-Corrected Confidence Intervals (10,000 draws)

	Standardized Estimates			Unstandardized Estimates		
		95% Confidence Intervals			95% Confidence Intervals	
Specific Indirect Paths	Estimate (SE)	Lower	Upper	Estimate (SE)	Lower	Upper
MPSY→ PSF→ Depression A	.071 (.038)	.013	.157	.055 (.03)	.010	.125
MPSY→ PSF→ BMI A→ CRP A	.042 (.022)	.006	.091	.004 (.002)	.001	.009

Note. MPSY= Maternal Psychopathology; PSY = Psychosocial Functioning; BMI A= Body Mass Index in Adolescence; Depressive Symptoms in Adolescence; CRP A= C-Reactive Protein in Adolescence.

Multigroup Analyses

My last question explored possible sex differences in the hypothesized model pathways through a multigroup analysis. Table 6 displays the model fit indices associated with the multigroup analyses. A significant chi-square model between the freely estimated model and the fully constrained model suggested sex differences in the model [χ^2 (14) = 80.184; $p < .01$]. To explore whether sex differences are best explained by some or all pathways, one pathway was

freed at a time and compared to a fully constrained model. Iterative analyses suggest that a fully freed model fits the data best. However, model fit statistics associated with the fully freed model indicates fair model fit $\chi^2(46) = 84.441$, $p < .001$; CFI = .886; RMSEA = .076 (90% CI = .05, .10); SRMR = .095, therefore caution is employed when interpreting significant pathways (Brown & Cudeck, 1993; Bryne, 1998; Kline, 2005; MacCallum et al., 1996).

Table 7 displays the standardized and unstandardized beta coefficients associated with each pathway by sex. Results indicate that controlling for the effects of childhood SES, maternal psychopathology significantly predicted psychosocial functioning in middle childhood for both girls and boys (β_{female} = -.136, p= .042*;* β_{male} = -.142, p = *.03*). However, psychosocial functioning predicted depression (β_{female} = -.506, p = *<.001*) and BMI status (β_{female} = -.365, p < *.001*) only for adolescent females. Moreover, BMI significantly predicted CRP levels for both adolescent females and males (β_{female} = .126, p < *.001;* β_{male} = .096, p < *.001*). Finally, there is no evidence of a significant bidirectional relationship between depressive symptoms and CRP levels in adolescence for neither adolescent females nor males.

When the model is bifurcated by sex, significant indirect effects are no longer observed at the p-level of .05. However, the indirect effects of maternal psychopathology on depressive symptoms in adolescence (β = .077, *SE* = .043; p = .071) and of maternal psychopathology on CRP (β = .057, *SE* = .031 p = .065) are marginally significant for females, but not for males. The only significant effect observed is for the total effect of maternal psychopathology on depression for females (β = .224, *SE* = .081; p = .006). The total effect of maternal psychopathology on depression is marginally significant for males (β=.171, *SE*=.099; p = .083).

Table 6. Multi-group Analysis for Sex Differences

Models	Model Fit				Model Difference Test			
	χ^2 (*df*)	CFI	RMSEA	SRMR	$\Delta\chi^2$ (Δ*df*)	ΔCFI	ΔRMSEA	ΔSRMR
Free	84.441 (46)	.886	.076	.095				
Fully Constrained	164.625 (60)	.689	.110	.166	80.184 (14)	.197	.034	.071
Partially Constrained (1[a])	155.959 (57)	.706	.110	.159	71.518 (11)	.017	.000	.007
Partially Constrained (2[b])	127.432 (52)	.776	.1	.144	42.991 (6)	.087	.01	.022
Partially Constrained (3[c])	116.305 (49)	.8	.098	.134	31.364 (3)	.111	.013	.032

Note. CFI= Comparative fit index; RMSEA= Root mean square error approximation; SRMR= square root mean residual.

[a] 1 pathway freed: MPSY→PSF

[b] 2 pathways freed: MPSY→PSF; PSF→Depression freed

[c] 3 pathways freed: MPSY→PSF; PSF→Depression; PSF→BMI freed

Table 7. Standardized and Unstandardized Estimates and Standard Errors of Path Model by Sex

Specific Direct Paths	Females (n = 162)		Males (n = 126)	
	Standardized Estimate (SE)	Unstandardized Estimate (SE)	Standardized Estimate (SE)	Unstandardized Estimate (SE)
MPSY → PSF	-.198* (.098)	-.136* (.067)	-.230* (.107)	-.142** (.066)
PSF → Depression A	-.389***(.090)	-.506*** (.135)	-.154 (.137)	-.158 (.144)
PSF → BMI A	-.472***(.093)	-.365*** (.087)	-.100 (.134)	-.083 (.113)
BMI A → CRP A	.612*** (.09)	.126*** (.026)	.571*** (.099)	.096 ** (.021)
MPSY → Depression A	.147+ (.082)	.131+ (.074)	.136 (.107)	. 086 (.068)
Covariances				
Depression A ↔ CRP A	-.065 (.140)	-.490 (1.052)	-.058 (.181)	-.370 (1.163)
Loneliness ↔ Peer SS	393 *** (.099)	16.728** (6.03)	.206 (.172)	7.539 (7.538)
Indirect Paths				
MPSY→ PSF→ Depression	.077+ (.043)	.069+ (.039)	.035 (.036)	.022 (.023)
MPSY→ PSF→ BMI A→	.057+ (.031)	.006+ (.004)	.013 (.019)	.001 (.002)
CRP A				
Total Effects				
MPSY→ Depression A	.224 ** (.081)	.200 **(.074)	.171+ (.099)	.109+ (.064)
MPSY→ CRP A	.057+ (.031)	.006+ (.004)	.013 (.019)	.001 (.002)

Note. MPSY= Maternal Psychopathology; PSY = Psychosocial Functioning; BMI A= Body Mass Index in Adolescence; CRP A= C-Reactive Protein in Adolescence; Depression A= Depressive Symptoms in Adolescence

$^{+}$ p < .10; *p < .05; **p < .01; ***p < .001.

CHAPTER V: DISCUSSION

The present 12-year longitudinal, prospective study examined the role of maternal psychopathology in early childhood on adolescent mental and physical health outcomes using a multi-method and multi-informant design. A developmentally salient, biopsychosocial model was examined to explain the process underlying the longitudinal associations between maternal mental health and adolescent health outcomes. This study extends the literature on maternal psychopathology by elucidating that maternal psychopathology not only has implications for adolescents' mental health, but also their physical health. To my knowledge this is the first study that examines the indirect effect of maternal psychopathology in early childhood on levels of inflammation in a community sample of adolescents across a 12-year prospective period. Our results suggest that maternal mental health influences psychosocial functioning in middle childhood, which is in turn associated with adolescent health outcomes, specifically, depressive symptoms and BMI status. BMI status is directly associated with inflammation in adolescence.

Guided by theory and empirical findings from the literature, it was hypothesized that maternal psychopathology would directly impair a child's psychosocial functioning in middle childhood. Psychosocial functioning was defined based on the developmentally salient tasks of middle childhood, which included peer relationships and the ability to effectively regulate negative emotions. The results affirm the relationship between maternal psychopathology and psychosocial functioning in middle childhood. Specifically, maternal psychopathology was negatively associated with adaptive psychosocial functioning. Therefore, children whose mothers endorsed greater levels of maternal psychopathology were more likely to report lower levels of satisfaction with peers, endorse more experiences of loneliness, social dissatisfaction, victimization, display increased negative affect, as well as experience difficulties self-regulating.

Poor psychosocial functioning in middle childhood directly predicted depressive symptoms in adolescence. These findings further substantiate previous research reflecting the effects of early life stress on depressive symptoms and specifically, the role of social stressors on depressive symptoms. One study found that those individuals who had experienced targeted rejection became depressed three times faster than those individuals who had experienced non-targeted rejection (Slavich et al., 2009). Slavich and colleagues (2010) have proposed a biopsychosocial model to account for the strong relationship between social rejection and the onset of depression. They propose that rejection-related stressors potentiate cognitive, emotional, and biological changes that trigger a depressive episode. Furthermore, they highlight the role of biological processes involved in the stress response, which includes the upregulation of inflammatory cytokines that may contribute to depressive behaviors. Although not explicitly tested in this present study, children who had lower levels of psychosocial functioning had endorsed more experiences of victimization, loneliness, and lower levels of social support from peers, therefore, lending indirect support for the role of social rejection on the development of depressive symptoms.

Moreover, children who experience difficulties self-regulating, as indicated by poor psychosocial functioning, are at an increased risk for depression. Research suggests that while stressful life events potentiate risk, those individuals who experience difficulties regulating their negative affect are even more susceptible to experiencing depression following a stressful life event (Joormann & Stanton, 2016). Given that adolescence is a time period marked by increases in peer interactions, exposure to novel experiences, and the inception of romantic relationships, adolescents are more likely to be exposed to emotionally evocative situations that call on

regulatory skills to navigate successfully; results indicate that those adolescents who have difficulties self-regulating are at increased risk for depression.

From a developmental perspective, emotion regulation abilities continue to develop across the lifespan, thus it follows that parental socialization practices continue to exert influence in the shaping of emotion regulation in adolescence (Moilanen et al., 2010). Given the homotypic continuity of many mental health disorders (Lahey et al., 2014), children whose mothers were depressed in childhood may continue to be exposed to detrimental socialization practices, thereby explaining the longitudinally robust effects of maternal psychopathology 12 years later. Alternatively, those children whose mothers recovered may have still been susceptible to the effects of poor psychosocial functioning in middle childhood. Research shows that children who experience peer victimization in middle childhood are susceptible to internalizing problems that persist over time and continue into late adolescence (Schwartz et al., 2015). These results highlight the longitudinal effects of poor psychosocial functioning on later mental health, indicating the importance of identifying those youth who have difficulties with the various domains of psychosocial functioning.

Furthermore, the present study contributes to the extant literature base by elucidating that the effects of maternal psychopathology are not limited to adolescents' mental health, but also extend to adolescents' physical health. Maternal psychopathology was indirectly associated with levels of C-reactive protein, an inflammatory biomarker, in adolescence, through psychosocial functioning in middle childhood and BMI status in adolescence. Specifically, children who endorsed poorer psychosocial functioning in middle childhood, were found to have higher levels of BMI in adolescence, which was in turn associated with inflammation. Although previous research has found an association between internalizing behavior problems and obesity

(Britz et al., 2000; E. Goodman & Whitaker, 2002), the present findings highlight the role of psychosocial functioning in middle childhood and health outcomes. That is, children who lack social support from peers, experience victimization and loneliness, and have difficulties self-regulating, may turn to food to cope with their experiences. In an adult sample, Konttinen et al. (2010) found that individuals with higher depressive symptoms were more likely to engage in emotional eating, and Hasler et al. (2005), showed that depressive symptoms in childhood predicted obesity in adulthood. The present study extends these findings and reflects that psychosocial functioning also predicts BMI status; future research may explicitly test whether experiences with psychosocial stressors in middle childhood triggers emotional eating patterns that lead to increases in weight gain.

Furthermore, the present findings affirm the documented association between BMI status and inflammation (DeBoer, 2013; Goosby et al., 2016). Given the associated risk factors with heightened levels of inflammation (Attard et al., 2013), findings from the present study may illuminate modifiable risk factors and novel therapeutic intervention strategies. Specifically, results suggest that attending to youth's psychosocial difficulties with respect to their peer relationships and their self-regulatory capabilities, may serve to buffer risk for emotional eating patterns that contribute to BMI and inflammation status and its associated health risks.

Whereas previous studies have found a relationship between concurrent depression and inflammation, the present study did not observe a significant bidirectional relationship between depressive symptoms and CRP (Colasanto et al., 2020). Given the small effect size of the relationship between depressive symptoms and CRP found in a recent meta-analysis ($r = 0.12$; 95% CI = 0.04 to 0.19), it is possible that the current study lacked the power to detect such an effect. Furthermore, it is possible that the depressive symptoms captured in the present sample

were not of sufficient severity to potentiate a relationship with inflammatory levels. Alternatively, the absence of a bidirectional relationship may be a result of the methodological rigor the present study employed in controlling for extraneous sources of inflammation. As previously detailed here and elsewhere (Wideman et al., 2016), adolescents were asked to reschedule their biomarker visit if they felt ill in the past week, had any surgical procedures in the past month, received any vaccines in the past two weeks, or had taken any anti-inflammatory medications within the past 10 days. These thoughtful methodological decisions may have contributed to a lower, and more accurate, level of inflammation, thereby elucidating that depressive symptoms and true baseline inflammation are not predictably correlated. Future studies should aim to employ greater methodological control over extraneous sources of inflammation in service of clarifying the relationship between inflammation and depressive symptoms.

Moreover, results lend support for examining sex differences in the hypothesized model pathways as sex differences were observed in some direct effects tested by the model. Although maternal psychopathology significantly predicted psychosocial functioning in middle childhood for both girls and boys, psychosocial functioning was associated with depressive symptoms and BMI status only in adolescent females. Given the well-documented sex differences in depressive symptoms starting in adolescence (Hankin, 2015), perhaps the lack of a significant association for males may be due to their lower rates of depressive symptoms. In addition to the differential prevalence of depressive symptoms, Hamilton and colleagues (2015) illuminate that sex differences in depressive symptoms may also be partly explained by girls' relatively greater exposure to interpersonal dependent stress and their ruminative response style. Perhaps the effects of poor psychosocial functioning represent a greater risk factor for girls than for boys.

Moreover, it is notable that psychosocial functioning predicted increases in BMI for girls, but not for boys. These results suggest that girls' negative peer experiences and difficulties self-regulating represent a vulnerability factor for increases in BMI. A previous study of middle school youth found that perceived stress, worries, and tension/anxiety was related to emotional eating for girls, but not for boys (Nguyen-Rodriguez et al., 2009). Conceivably, in addition to a heightened sensitivity to psychosocial stressors, girls are more likely to cope with stressors with emotional eating that results in increases in BMI status. Moreover, given the role of emotion regulation in the relationship between emotional eating and weight status (Shriver et al., 2021), results indicate that targeting core features of psychosocial functioning in middle childhood may serve as an intervention strategy for emotional eating patterns.

The present study has several strengths. First, it aimed to analyze the mechanisms by which maternal psychopathology exerts its longitudinal effects on youths' mental and physical well-being by positing a biopsychosocial, thereby extending the extant knowledge base. The study also leveraged 12 years of longitudinal data and employed a prospective design to test the model pathways on a diverse community sample of mother-child dyads in the southeast of the US. The present study elucidated one pathway by which maternal psychopathology may impact depressive symptoms and levels of inflammation in late adolescence, highlighting the importance of psychosocial functioning in middle childhood. Given the important role psychosocial functioning may play in the development of depressive symptoms and inflammation, intervention researchers may wish to test the effects of intervening in early and middle childhood through school-based programs. Specifically, targeting a child's social competence and self-regulation skills may have a therapeutic effect on later depressive symptoms, unhealthy weight status, and inflammation.

Notwithstanding, important limitations should also be considered. First, the extant model tested a finite number of pathways leading to adolescent health outcomes; hypotheses have been put forth concerning the processes that account for the significant direct effects, however, future studies may wish to explicitly state these hypotheses. For example, it was hypothesized that one mechanism by which maternal psychopathology negatively affects psychosocial functioning in middle childhood is through socialization processes of emotion-related behaviors, which in turn affects a child's social competence and self-regulatory processes. Moreover, it was hypothesized that deficits in psychosocial functioning may lead to increases in emotional eating patterns that explain increases in BMI. It is incumbent on future research to directly test these associations, especially given the longitudinal nature of the present study.

In addition, the present study did not analyze the role of fathers in the model. Children's emotion-related behaviors are not only socialized via interactions with their mothers, but all involved caregivers, therefore, access to a responsive, warm, and consistent caregiver figure may serve to buffer the effects of maternal psychopathology. Moreover, while SES was taken into account in the hypothesized pathways, future studies may wish to replicate the extant design with a homogeneously low SES population specifically, in an effort to disentangle the effects of SES from the predictive variables. Finally, model fit for the bifurcated model limits confidence in the observed sex differences. Given the relatively small effect sizes for the relations in the pathway model, sample size represents a limitation. Although the sample size was modest (n=288), when bifurcated by sex (Female n=162; Male n=126), the model loses power to find significant indirect effects. Nonetheless, given that the model suggested differential effects of psychosocial functioning in middle childhood in depressive symptoms and BMI in adolescence, whereby females were more vulnerable to the effects of poor psychosocial

functioning, future studies should aim to elucidate what accounts for the increased risk in females and whether a different pathway better predicts the effects of impaired psychosocial functioning in males. For example, it is plausible that the effects of psychosocial functioning significantly predict externalizing symptoms for males, which was not examined in the present study.

Finally, there are many early life stressors that play may play a role in adolescent mental and physical health outcomes. While this study aimed to examine the unique role of maternal psychopathology, future studies may expand the model to include other salient early life stressors. Moreover, the present study examined specifically depressive symptomatology in adolescence, however it is plausible that the longitudinal psychosocial pathway examined by the model also predicts a host of mental health outcomes in adolescence. It is suggested that future studies extend the range of adolescent outcomes to include a broader range of psychopathology.

REFERENCES

Abel, K. M., Hope, H., Swift, E., Parisi, R., Ashcroft, D. M., Kosidou, K., Osam, C. S., Dalman, C., & Pierce, M. (2019). Prevalence of maternal mental illness among children and adolescents in the UK between 2005 and 2017: A national retrospective cohort analysis. *The Lancet Public Health*, *4*(6), e291–e300. https://doi.org/10.1016/S2468-2667(19)30059-3

Alder, J., Fink, N., Bitzer, J., Hösli, I., & Holzgreve, W. (2007). Depression and anxiety during pregnancy: A risk factor for obstetric, fetal and neonatal outcome? A critical review of the literature. *Journal of Maternal - Fetal & Neonatal Medicine*, *20*(3), 189–209.

Asher, S. R., Hymel, S., & Renshaw, P. D. (1984). Loneliness in Children. *Child Development*, *55*(4), 1456–1464. https://doi.org/10.2307/1130015

Asher, S. R., & Wheeler, V. A. (1985). Children's loneliness: A comparison of rejected and neglected peer status. *Journal of Consulting and Clinical Psychology*, *53*(4), 500–505. https://doi.org/10.1037/0022-006X.53.4.500

Attard, S. M., Herring, A. H., Howard, A. G., & Gordon-Larsen, P. (2013). Longitudinal trajectories of BMI and cardiovascular disease risk: The national longitudinal study of adolescent health. *Obesity*, *21*(11), 2180–2188. https://doi.org/10.1002/oby.20569

Barker, E. D., Cecil, C. A. M., Walton, E., Houtepen, L. C., O'Connor, T. G., Danese, A., Jaffee, S. R., Jensen, S. K. G., Pariante, C., McArdle, W., Gaunt, T. R., Relton, C. L., & Roberts, S. (2018). Inflammation-related epigenetic risk and child and adolescent mental health: A prospective study from pregnancy to middle adolescence. *Development and Psychopathology*, *30*(3), 1145–1156. https://doi.org/10.1017/S0954579418000330

Barker, E. D., Copeland, W., Maughan, B., Jaffee, S. R., & Uher, R. (2012). Relative impact of maternal depression and associated risk factors on offspring psychopathology. *The British Journal of Psychiatry: The Journal of Mental Science*, *200*(2), 124–129. https://doi.org/10.1192/bjp.bp.111.092346

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, *88*(3), 588–606. https://doi.org/10.1037/0033-2909.88.3.588

Blair, B. L., Perry, N. B., O'Brien, M., Calkins, S. D., Keane, S. P., & Shanahan, L. (2014). The indirect effects of maternal emotion socialization on friendship quality in middle childhood. *Developmental Psychology*, *50*(2), 566–576. https://doi.org/10.1037/a0033532

Bowen, N. K., & Guo, S. (2011). *Structural Equation Modeling*. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780195367621.001.0001

Britz, B., Siegfried, W., Ziegler, A., Lamertz, C., Herpertz-Dahlmann, B. M., Remschmidt, H., Wittchen, H.-U., & Hebebrand, J. (2000). Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via a population based study. *International Journal of Obesity*, *24*(12), 1707–1714. https://doi.org/10.1038/sj.ijo.0801449

Calkins, S. D., Dedmon, S. E., Gill, K. L., Lomax, L. E., & Johnson, L. M. (2002). Frustration in Infancy: Implications for Emotion Regulation, Physiological Processes, and Temperament. *Infancy*, *3*(2), 175–197. https://doi.org/10.1207/S15327078IN0302_4

Cartier, A., Côté, M., Lemieux, I., Pérusse, L., Tremblay, A., Bouchard, C., & Després, J.-P. (2009). Sex differences in inflammatory markers: What is the contribution of visceral adiposity? *The American Journal of Clinical Nutrition*, *89*(5), 1307–1314. https://doi.org/10.3945/ajcn.2008.27030

Colasanto, M., Madigan, S., & Korczak, D. J. (2020). Depression and inflammation among children and adolescents: A meta-analysis. *Journal of Affective Disorders*, *277*, 940–948. https://doi.org/10.1016/j.jad.2020.09.025

Cole, D. A., & Maxwell, S. E. (2003). Testing Mediational Models With Longitudinal Data: Questions and Tips in the Use of Structural Equation Modeling. *Journal of Abnormal Psychology*, *112*(4), 558–577. https://doi.org/10.1037/0021-843X.112.4.558

Colle, L., & Del Giudice, M. (2011). Patterns of Attachment and Emotional Competence in Middle Childhood. *Social Development*, *20*(1), 51–72. https://doi.org/10.1111/j.1467-9507.2010.00576.x

Conley, C. S., Rudolph, K. D., & Bryant, F. B. (2012). Explaining the longitudinal association between puberty and depression: Sex differences in the mediating effects of peer stress. *Development and Psychopathology*, *24*(2), 691–701. https://doi.org/10.1017/S0954579412000259

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and Development of Psychiatric Disorders in Childhood and Adolescence. *Archives of General Psychiatry*, *60*(8), 837–844. https://doi.org/10.1001/archpsyc.60.8.837

Cramer, A. O. J., Borsboom, D., Aggen, S. H., & Kendler, K. S. (2012). The pathoplasticity of dysphoric episodes: Differential impact of stressful life events on the pattern of depressive symptom inter-correlations. *Psychological Medicine*, *42*(5), 957–965. https://doi.org/10.1017/S003329171100211X

Czaja, J., Rief, W., & Hilbert, A. (2009). Emotion regulation and binge eating in children. *International Journal of Eating Disorders*, *42*(4), 356–362. https://doi.org/10.1002/eat.20630

Danese, A., & J Lewis, S. (2017). Psychoneuroimmunology of Early-Life Stress: The Hidden Wounds of Childhood Trauma? *Neuropsychopharmacology*, *42*(1), 99–114. https://doi.org/10.1038/npp.2016.198

Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. *Archives of General Psychiatry*, *65*(4), 409–415. https://doi.org/10.1001/archpsyc.65.4.409

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences*, *104*(4), 1319–1324. https://doi.org/10.1073/pnas.0610362104

De Spiegelaere, M., Dramaix, M., & Hennart, P. (1998). The influence of socioeconomic status on the incidence and evolution of obesity during early adolescence. *International Journal of Obesity*, *22*(3), 268–274. https://doi.org/10.1038/sj.ijo.0800581

DeBoer, M. D. (2013). Obesity, systemic inflammation, and increased risk for cardiovascular disease and diabetes among adolescents: A need for screening tools to target interventions. *Nutrition*, *29*(2), 379–386. https://doi.org/10.1016/j.nut.2012.07.003

Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin*, *135*(6), 823–853. https://doi.org/10.1037/a0016909

Derogatis, L. R., & Cleary, P. A. (1977). Confirmation of the dimensional structure of the SCL-90: A study in construct validation. *Journal of Clinical Psychology*, *33*(4), 981–989. https://doi.org/10.1002/1097-4679(197710)33:4<981::AID-JCLP2270330412>3.0.CO;2-0

Dickerson, S. S., Gable, S. L., Irwin, M. R., Aziz, N., & Kemeny, M. E. (2009). Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. *Psychological Science*, *20*(10), 1237–1244. https://doi.org/10.1111/j.1467-9280.2009.02437.x

du Prel, J.-B., Hommel, G., Röhrig, B., & Blettner, M. (2009). Confidence Interval or P-Value? *Deutsches Ärzteblatt International*, *106*(19), 335–339. https://doi.org/10.3238/arztebl.2009.0335

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

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

Enders, C. K., & Bandalos, D. L. (2001). The Relative Performance of Full Information Maximum Likelihood Estimation for Missing Data in Structural Equation Models. *Structural Equation Modeling: A Multidisciplinary Journal*, *8*(3), 430–457. https://doi.org/10.1207/S15328007SEM0803_5

Ertel, K. A., Rich-Edwards, J. W., & Koenen, K. C. (2011, November 11). *Maternal Depression in the United States: Nationally Representative Rates and Risks* (140 Huguenot Street, 3rd Floor New Rochelle, NY 10801 USA) [Research-article]. Https://Home.Liebertpub.Com/Jwh; Mary Ann Liebert, Inc. 140 Huguenot Street, 3rd Floor New Rochelle, NY 10801 USA. https://doi.org/10.1089/jwh.2010.2657

Farr, S. L., Bitsko, R. H., Hayes, D. K., & Dietz, P. M. (2010). Mental health and access to services among US women of reproductive age. *American Journal of Obstetrics and Gynecology*, *203*(6), 542.e1-542.e9. https://doi.org/10.1016/j.ajog.2010.07.007

Fradkin, C., Wallander, J. L., Elliott, M. N., Tortolero, S., Cuccaro, P., & Schuster, M. A. (2015). Associations between socioeconomic status and obesity in diverse, young adolescents: Variation across race/ethnicity and gender. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, *34*(1), 1–9. https://doi.org/10.1037/hea0000099

Goodman, E., Huang, B., Wade, T. J., & Kahn, R. S. (2003). A multilevel analysis of the relation of socioeconomic status to adolescent depressive symptoms: Does school context matter? *The Journal of Pediatrics*, *143*(4), 451–456. https://doi.org/10.1067/S0022-3476(03)00456-6

Goodman, E., & Whitaker, R. C. (2002). A Prospective Study of the Role of Depression in the Development and Persistence of Adolescent Obesity. *Pediatrics*, *110*(3), 497–504. https://doi.org/10.1542/peds.110.3.497

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

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal Depression and Child Psychopathology: A Meta-Analytic Review. *Clinical Child and Family Psychology Review*, *14*(1), 1–27. https://doi.org/10.1007/s10567-010-0080-1

Goosby, B. J., Cheadle, J. E., & McDade, T. (2016). Birth weight, early life course BMI, and body size change: Chains of risk to adult inflammation? *Social Science & Medicine*, *148*, 102–109. https://doi.org/10.1016/j.socscimed.2015.11.040

Hamilton, J. L., Stange, J. P., Abramson, L. Y., & Alloy, L. B. (2015). Stress and the Development of Cognitive Vulnerabilities to Depression Explain Sex Differences in Depressive Symptoms During Adolescence. *Clinical Psychological Science*, *3*(5), 702–714. https://doi.org/10.1177/2167702614545479

Hammen, C., Burge, D., & Stansbury, K. (1990). Relationship of mother and child variables to child outcomes in a high-risk sample: A causal modeling analysis. *Developmental Psychology*, *26*(1), 24–30. https://doi.org/10.1037/0012-1649.26.1.24

Hankin, B. L. (2015). Depression from childhood through adolescence: Risk mechanisms across multiple systems and levels of analysis. *Current Opinion in Psychology*, *4*, 13–20. https://doi.org/10.1016/j.copsyc.2015.01.003

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*(1), 128–140. https://doi.org/10.1037//0021-843x.107.1.128

Hardie, J. H., & Landale, N. S. (2013). Profiles of Risk: Maternal Health, Socioeconomic Status, and Child Health. *Journal of Marriage and Family*, *75*(3), 651–666. https://doi.org/10.1111/jomf.12021

Harrist, A. W., Hubbs-Tait, L., Topham, G. L., Shriver, L. H., & Page, M. C. (2013). Emotion Regulation is Related to Children's Emotional and External Eating. *Journal of Developmental & Behavioral Pediatrics*, *34*(8), 557–565. https://doi.org/10.1097/DBP.0b013e3182a5095f

Hasler, G., Pine, D. S., Kleinbaum, D. G., Gamma, A., Luckenbaugh, D., Ajdacic, V., Eich, D., Rössler, W., & Angst, J. (2005). Depressive symptoms during childhood and adult obesity: The Zurich Cohort Study. *Molecular Psychiatry*, *10*(9), 842–850. https://doi.org/10.1038/sj.mp.4001671

Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression: Review of human studies on sensitive periods, gene–environment interactions, and epigenetics. *Experimental Neurology*, *233*(1), 102–111. https://doi.org/10.1016/j.expneurol.2011.10.032

Hein, A., Rauh, C., Engel, A., Häberle, L., Dammer, U., Voigt, F., Fasching, P. A., Faschingbauer, F., Burger, P., Beckmann, M. W., Kornhuber, J., & Goecke, T. W. (2014). Socioeconomic status and depression during and after pregnancy in the Franconian Maternal Health Evaluation Studies (FRAMES). *Archives of Gynecology and Obstetrics*, *289*(4), 755–763. https://doi.org/10.1007/s00404-013-3046-y

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

Joormann, J., & Stanton, C. H. (2016). Examining emotion regulation in depression: A review and future directions. *Behaviour Research and Therapy*, *86*, 35–49. https://doi.org/10.1016/j.brat.2016.07.007

Keller, M. C., Neale, M. C., & Kendler, K. S. (2007). Association of different adverse life events with distinct patterns of depressive symptoms. *The American Journal of Psychiatry*, *164*(10), 1521–1529; quiz 1622. https://doi.org/10.1176/appi.ajp.2007.06091564

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime Prevalence and Age-of-Onset Distributions of DSM-IV Disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. https://doi.org/10.1001/archpsyc.62.6.593

Khera, A., Vega, G. L., Das, S. R., Ayers, C., McGuire, D. K., Grundy, S. M., & de Lemos, J. A. (2009). Sex Differences in the Relationship between C-Reactive Protein and Body Fat. *The Journal of Clinical Endocrinology & Metabolism*, *94*(9), 3251–3258. https://doi.org/10.1210/jc.2008-2406

Kline, R. B. (2005). *Principles and practice of structural equation modeling, 2nd ed* (pp. xviii, 366). Guilford Press.

Kochenderfer, B. J., & Ladd, G. W. (1996). Peer Victimization: Cause or Consequence of School Maladjustment? *Child Development*, *67*(4), 1305–1317. https://doi.org/10.2307/1131701

Konttinen, H., Männistö, S., Sarlio-Lähteenkorva, S., Silventoinen, K., & Haukkala, A. (2010). Emotional eating, depressive symptoms and self-reported food consumption. A population-based study. *Appetite*, *54*(3), 473–479. https://doi.org/10.1016/j.appet.2010.01.014

Lahey, B. B., Zald, D. H., Hakes, J. K., Krueger, R. F., & Rathouz, P. J. (2014). Patterns of Heterotypic Continuity Associated With the Cross-Sectional Correlational Structure of Prevalent Mental Disorders in Adults. *JAMA Psychiatry*, *71*(9), 989–996. https://doi.org/10.1001/jamapsychiatry.2014.359

Lehman, B. J., Taylor, S. E., Kiefe, C. I., & Seeman, T. E. (2005). Relation of Childhood Socioeconomic Status and Family Environment to Adult Metabolic Functioning in the CARDIA Study. *Psychosomatic Medicine*, *67*(6), 846–854. https://doi.org/10.1097/01.psy.0000188443.48405.eb

Lehman, B. J., Taylor, S. E., Kiefe, C. I., & Seeman, T. E. (2009). Relationship of early life stress and psychological functioning to blood pressure in the CARDIA study. *Health Psychology*, *28*(3), 338–346. https://doi.org/10.1037/a0013785

Lotrich, F. E., El-Gabalawy, H., Guenther, L. C., & Ware, C. F. (2011). The Role of Inflammation in the Pathophysiology of Depression: Different Treatments and Their Effects. *The Journal of Rheumatology Supplement*, *88*, 48–54. https://doi.org/10.3899/jrheum.110903

Lovejoy, J. C., Sainsbury, A., & Group, the S. C. 2008 W. (2009). Sex differences in obesity and the regulation of energy homeostasis. *Obesity Reviews*, *10*(2), 154–167. https://doi.org/10.1111/j.1467-789X.2008.00529.x

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence Limits for the Indirect Effect: Distribution of the Product and Resampling Methods. *Multivariate Behavioral Research*, *39*(1), 99–128. https://doi.org/10.1207/s15327906mbr3901_4

Madigan, S., Oatley, H., Racine, N., Fearon, R. M. P., Schumacher, L., Akbari, E., Cooke, J. E., & Tarabulsy, G. M. (2018). A Meta-Analysis of Maternal Prenatal Depression and Anxiety on Child Socioemotional Development. *Journal of the American Academy of Child and Adolescent Psychiatry*, *57*(9), 645-657.e8. https://doi.org/10.1016/j.jaac.2018.06.012

Marsh, H. W., & Hau, K.-T. (2007). Applications of latent-variable models in educational psychology: The need for methodological-substantive synergies. *Contemporary Educational Psychology*, *32*(1), 151–170. https://doi.org/10.1016/j.cedpsych.2006.10.008

Masip, A. F., Amador-Campos, J. A., Gómez-Benito, J., & Gándara, V. del B. (2010). Psychometric Properties of the Children's Depression Inventory in Community and Clinical Sample. *The Spanish Journal of Psychology*, *13*(2), 990–999. https://doi.org/10.1017/S1138741600002638

Matsunaga, M. (2010). How to factor-analyze your data right: Do's, don'ts, and how-to's. *International Journal of Psychological Research*, *3*(1), 97–110. https://doi.org/10.21500/20112084.854

Mauvais-Jarvis, F. (2015). Sex differences in metabolic homeostasis, diabetes, and obesity. *Biology of Sex Differences*, *6*(1), 14. https://doi.org/10.1186/s13293-015-0033-y

McDowell, D. J., Kim, M., O'neil, R., & Parke, R. D. (2002). Children's Emotional Regulation and Social Competence in Middle Childhood. *Marriage & Family Review*, *34*(3–4), 345–364. https://doi.org/10.1300/J002v34n03_07

Milaniak, I., & Jaffee, S. R. (2019). Childhood socioeconomic status and inflammation: A systematic review and meta-analysis. *Brain, Behavior, and Immunity*, *78*, 161–176. https://doi.org/10.1016/j.bbi.2019.01.018

Miller, A. H., Maletic, V., & Raison, C. L. (2009). Inflammation and Its Discontents: The Role of Cytokines in the Pathophysiology of Major Depression. *Biological Psychiatry*, *65*(9), 732–741. https://doi.org/10.1016/j.biopsych.2008.11.029

Miller, G. E., & Chen, E. (2010). Harsh Family Climate in Early Life Presages the Emergence of a Proinflammatory Phenotype in Adolescence. *Psychological Science*, *21*(6), 848–856. https://doi.org/10.1177/0956797610370161

Miller, G. E., & Cole, S. W. (2012). Clustering of depression and inflammation in adolescents previously exposed to childhood adversity. *Biological Psychiatry*, *72*(1), 34–40. https://doi.org/10.1016/j.biopsych.2012.02.034

Moilanen, K. L., Shaw, D. S., & Fitzpatrick, A. (2010). Self-regulation in early adolescence: Relations with mother–son relationship quality and maternal regulatory support and antagonism. *Journal of Youth and Adolescence*, *39*(11), 1357–1367. https://doi.org/10.1007/s10964-009-9485-x

Nehring, S. M., Goyal, A., & Patel, B. C. (2022). C Reactive Protein. In *StatPearls*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/books/NBK441843/

Nguyen-Rodriguez, S. T., Unger, J. B., & Spruijt-Metz, D. (2009). Psychological Determinants of Emotional Eating in Adolescence. *Eating Disorders*, *17*(3), 211–224. https://doi.org/10.1080/10640260902848543

Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: An integrated review of human literature. *Psychopharmacology*, *214*(1), 55–70. https://doi.org/10.1007/s00213-010-2009-2

Perry, N. B., Dollar, J. M., Calkins, S. D., Keane, S. P., & Shanahan, L. (2020). Maternal socialization of child emotion and adolescent adjustment: Indirect effects through emotion regulation. *Developmental Psychology*, *56*(3), 541–552. https://doi.org/10.1037/dev0000815

Peters, S. A. E., Muntner, P., & Woodward, M. (2019). Sex Differences in the Prevalence of, and Trends in, Cardiovascular Risk Factors, Treatment, and Control in the United States, 2001 to 2016. *Circulation*, *139*(8), 1025–1035. https://doi.org/10.1161/CIRCULATIONAHA.118.035550

Peugh, J., & Feldon, D. F. (2020). "How Well Does Your Structural Equation Model Fit Your Data?": Is Marcoulides and Yuan's Equivalence Test the Answer? *CBE—Life Sciences Education*, *19*(3), es5. https://doi.org/10.1187/cbe.20-01-0016

Plant, D. T., Pawlby, S., Sharp, D., Zunszain, P. A., & Pariante, C. M. (2016). Prenatal maternal depression is associated with offspring inflammation at 25 years: A prospective longitudinal cohort study. *Translational Psychiatry*, *6*(11), e936–e936. https://doi.org/10.1038/tp.2015.155

Puder, J. J., & Munsch, S. (2010). Psychological correlates of childhood obesity. *International Journal of Obesity*, *34*(2), S37–S43. https://doi.org/10.1038/ijo.2010.238

Qualter, P., Hurley, R., Eccles, A., Abbott, J., Boivin, M., & Tremblay, R. (2018). Reciprocal Prospective Relationships Between Loneliness and Weight Status in Late Childhood and Early Adolescence. *Journal of Youth and Adolescence*, *47*(7), 1385–1397. https://doi.org/10.1007/s10964-018-0867-9

Ridker, P. M. (2003). Clinical application of C-reactive protein for cardiovascular disease detection and prevention. *Circulation*, *107*(3), 363–369. https://doi.org/10.1161/01.cir.0000053730.47739.3c

Ringoot, A. P., Jansen, P. W., Kok, R., van IJzendoorn, M. H., Verlinden, M., Verhulst, F. C., Bakermans-Kranenburg, M., & Tiemeier, H. (2021). Parenting, young children's behavioral self-regulation and the quality of their peer relationships. *Social Development*, *n/a*(n/a). https://doi.org/10.1111/sode.12573

Schwartz, D., Lansford, J. E., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2015). Peer Victimization During Middle Childhood as a Lead Indicator of Internalizing Problems and Diagnostic Outcomes in Late Adolescence. *Journal of Clinical Child & Adolescent Psychology*, *44*(3), 393–404. https://doi.org/10.1080/15374416.2014.881293

Shields, A., & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-sort scale. *Developmental Psychology*, *33*(6), 906–916. https://doi.org/10.1037/0012-1649.33.6.906

Shriver, L. H., Dollar, J. M., Calkins, S. D., Keane, S. P., Shanahan, L., & Wideman, L. (2021). Emotional Eating in Adolescence: Effects of Emotion Regulation, Weight Status and Negative Body Image. *Nutrients*, *13*(1), 79. https://doi.org/10.3390/nu13010079

Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin*, *140*(3), 774–815. https://doi.org/10.1037/a0035302

Slavich, G. M., O'Donovan, A., Epel, E. S., & Kemeny, M. E. (2010). Black sheep get the blues: A psychobiological model of social rejection and depression. *Neuroscience and Biobehavioral Reviews*, *35*(1), 39–45. https://doi.org/10.1016/j.neubiorev.2010.01.003

Slavich, G. M., Thornton, T., Torres, L. D., Monroe, S. M., & Gotlib, I. H. (2009). TARGETED REJECTION PREDICTS HASTENED ONSET OF MAJOR DEPRESSION. *Journal of Social and Clinical Psychology*, *28*(2), 223–243. https://doi.org/10.1521/jscp.2009.28.2.223

Storch, E. A., Milsom, V. A., DeBraganza, N., Lewin, A. B., Geffken, G. R., & Silverstein, J. H. (2007). Peer Victimization, Psychosocial Adjustment, and Physical Activity in Overweight and At-Risk-For-Overweight Youth. *Journal of Pediatric Psychology*, *32*(1), 80–89. https://doi.org/10.1093/jpepsy/jsj113

Taylor, S. E. (2010). Mechanisms linking early life stress to adult health outcomes. *Proceedings of the National Academy of Sciences*, *107*(19), 8507–8512. https://doi.org/10.1073/pnas.1003890107

Taylor, S. E., Lehman, B. J., Kiefe, C. I., & Seeman, T. E. (2006). Relationship of Early Life Stress and Psychological Functioning to Adult C-Reactive Protein in the Coronary Artery Risk Development in Young Adults Study. *Biological Psychiatry*, *60*(8), 819–824. https://doi.org/10.1016/j.biopsych.2006.03.016

Thorand, B., Baumert, J., Döring, A., Herder, C., Kolb, H., Rathmann, W., Giani, G., & Koenig, W. (2006). Sex differences in the relation of body composition to markers of inflammation. *Atherosclerosis*, *184*(1), 216–224. https://doi.org/10.1016/j.atherosclerosis.2005.04.011

Torres-Berrío, A., Issler, O., Parise, E. M., & Nestler, E. J. (2019). Unraveling the epigenetic landscape of depression: Focus on early life stress. *Dialogues in Clinical Neuroscience*, *21*(4), 341–357. https://doi.org/10.31887/DCNS.2019.21.4/enestler

Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika*, *38*(1), 1–10. https://doi.org/10.1007/BF02291170

Wideman, L., Calkins, S. D., Janssen, J. A., Lovelady, C. A., Dollar, J. M., Keane, S. P., Perrin, E. M., & Shanahan, L. (2016). Rationale, design and methods for the RIGHT Track Health Study: Pathways from childhood self-regulation to cardiovascular risk in adolescence. *BMC Public Health*, *16*(1), 459. https://doi.org/10.1186/s12889-016-3133-7