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The Role of Prenatal and Postnatal Maternal Distress
on Offspring Aggression in Young Adulthood

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The Role of Prenatal and Postnatal Maternal Distress on Offspring Aggression in Young Adulthood

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Abstract

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By Yunsoo Park

Aggression has been one of the most widely studied topics in psychology during the past several decades, as it has detrimental influences on the individual, families, and society at large. Accordingly, much empirical research has been dedicated to identifying various risk factors that contribute to the development of aggression. Maternal psychopathology (e.g., distress) has been consistently associated with a variety of negative offspring outcomes, including aggression and associated problems (e.g., antisocial behavior). However, the mechanisms underlying the effects of maternal distress on adverse offspring outcomes, as well as the long-term influence of maternal distress in the transition to adulthood, are still unclear. Furthermore, the extent to which maternal distress symptoms over the course of offspring development may differentially influence outcomes remains largely unexplored. The current studies aimed to fill these gaps in the literature by examining data obtained from a large, prospective sample to explore the relationship between prenatal and postnatal maternal distress and offspring aggression in young adulthood (age 20). In addition, we aimed to elucidate potential mediators of this developmental risk pathway. In **Study 1**, we examined the association between maternal distress during pregnancy and offspring aggression, and various cognitive mechanisms (cognitive ability, cognitive appraisals of stress) as potential mediators of this association. In **Study 2**, we examined whether distinct trajectories of maternal distress from pregnancy until offspring adolescence differentially predicted offspring aggression, and explored whether parenting style was a potential mediator of this association. Findings for **Study 1** suggested that while prenatal maternal distress significantly predicted offspring aggression, the cognitive factors examined did not serve as significant mediators of this association. Findings for **Study 2** supported three distinct trajectories of maternal distress and suggested that trajectory class membership was significantly associated with offspring aggression, with parenting serving as a significant mediator. Overall, the findings from the current project suggest that maternal distress, during pregnancy as well as across offspring development, has a long-term influence on offspring aggression in young adulthood, and that parenting style, but not cognitive ability or appraisals of stress during adolescence, represents a mechanism by which maternal distress contributes to offspring aggression.

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General Introduction

Aggression

From an evolutionary perspective, aggression is a crucial component of our heritage. It was necessary for our ancestors to engage in aggressive behaviors to hunt for food, defend themselves against predators, compete for mating, and co-opt and protect their supply of resources (D. Buss & Shackelford, 1997; Sagan & Druyan, 1992). However, excessive levels of aggression may be maladaptive, placing an individual at extreme risk and leading to fatalities, as well as resulting in social rejection or exclusion (Boivin, Vitaro, & Poulin, 2005; Suomi, 2005). Aggression often occurs in the context of other antisocial behaviors (ASB), which represents a broad range of actions or attitudes that violate societal norms and the personal and property rights of others, such as delinquency, cheating, and vandalism (S. A. Burt, 2012; Coie & Dodge, 1998). The terms aggression and ASB are often aggregated or used interchangeably (Tremblay, 2000), but while aggressive behavior is frequently present in various forms of ASB, aggression is by no means a necessary component of ASB (Tremblay, 2000, 2014). Indeed, there is much evidence to support the distinction between aggressive and non-aggressive forms of ASB (S. A. Burt, 2012; Eley, Lichtenstein, & Stevenson, 1999; Frick et al., 1993; Loeber & Schmaling, 1985; Tackett, Krueger, Iacono, & McGue, 2005).

Aggression represents a major public health concern and places significant burdens on the individual, as well as society, with costs estimated around 2.3 million dollars per individual in the most extreme cases in the United States (M. A. Cohen, Piquero, & Jennings, 2010). Behaviors associated with aggression and conduct problems account for about one-half of referrals to mental health professionals (Kazdin, Esveldt-Dawson, French, & Unis, 1987). Longitudinal studies indicate that the spontaneous onset

of physical aggression in adolescence or adulthood is unusual, and that aggression can be detected as young as 12 months of age (Tremblay, 2008). Nonetheless, aggressive behavior is fairly common in the first years of life and decreases in frequency for the majority of children as they reach the end of their preschool years (Nagin & Tremblay, 1999; Potegal & Davidson, 2003). Children who continue to exhibit higher levels of aggression tend to be at greater risk for problems in a variety of domains throughout their lifetime, including a range of ASB, substance abuse, suicide, depression, and academic failure (Broidy et al., 2003; Tremblay et al., 2004). Such detrimental implications for aggression have contributed to substantial research to identify risk factors that contribute to the development of aggressive behavior in order to better inform prevention and intervention.

The Role of Maternal Psychopathology in Aggression in Offspring

Parental psychopathology is a well-established risk factor for a variety of negative outcomes related to psychopathology in the offspring, with more than half of the children whose parents have psychiatric disorder diagnoses experiencing some form of mental illness during childhood or adolescence themselves (Beardslee, Versage, & Gladstone, 1998). Maternal psychopathology in particular represents a significant public health concern due to its adverse impact on the well-being of the mother and the child (Boath, Bradley, & Anthony, 2004; Goodman & Gotlib, 1999). For example, a meta-analysis showed that maternal psychopathology was associated with both externalizing and internalizing problems in offspring when children were between the ages of 2 and 18, and that maternal depression and anxiety in particular were the strongest predictors of childhood behavioral problems (Connell & Goodman, 2002). Indeed, maternal distress

represents one of the most crucial risk factors for adverse outcomes in offspring, including a wide range of internalizing and externalizing psychopathology, as well as various behavioral and emotional problems (E. D. Barker, Jaffee, Uher, & Maughan, 2011; O'Connor, 2002; O'Connor, Heron, Glover, & Team, 2002; Rice et al., 2010; Van Batenburg-Eddes et al., 2013; Van den Bergh & Marcoen, 2004).

The constellation of maternal indicators of psychological distress includes anxiety, depression, and stress. The majority of studies investigating the effects of maternal distress tend to focus unidimensionally on a single indicator of distress, usually depression, while attempting to control for other forms of distress (Dipietro, Costigan, & Sipsma, 2008; R. L. Miller, Pallant, & Negri, 2006). Maternal anxiety, depression, and stress have all individually been shown to be associated with negative childhood outcomes, although as just noted, the majority of the literature thus far has tended to focus on maternal depression (Austin, Hadzi-Pavlovic, Leader, Saint, & Parker, 2005; R. L. Miller et al., 2006; O'Connor, 2002). It is possible that these different forms of maternal distress influence offspring outcomes in different ways, although the nature of these effects is unclear and at times conflicting. For example, Barker and colleagues (2011) found that prenatal and postnatal maternal depression had a broader effect on different types of maladjustment in childhood compared to maternal anxiety, with maternal anxiety being specifically associated with internalizing symptoms in children. However, there is also evidence that prenatal maternal anxiety predicts child outcomes more strongly than depression and that the effects of prenatal maternal anxiety are separate and additive from depressive symptoms (O'Connor et al., 2002).

Nonetheless, these varying forms of distress are often comorbid and highly correlated (Goodman & Tully, 2008), and isolating individual constructs can be limiting and overestimate their importance (R. L. Miller et al., 2006). Thus, in addition to single constructs, a broader classification of maternal negative emotional states should be considered for a more comprehensive understanding of the role of maternal distress on offspring outcomes (Fisher, Feekery, & Rowe-Murray, 2002; Green, 1998; Matthey, Barnett, Howie, & Kavanagh, 2003).

The Nature and Course of Maternal Distress Across Development

The consistency of detrimental effects across various samples (e.g., clinical, community) and methodologies strongly supports the central role of maternal distress in contributing to a range of negative outcomes in offspring. However, most of these studies are cross-sectional and provide little insight into the nature and progression of maternal distress symptoms and how these patterns contribute to adverse outcomes over time. Symptoms of maternal distress can range from subclinical to severe, and may also be transient or persistent in nature. Additionally, the presentation of symptoms may not be stable over time, such that there are periods of increased severity of symptoms interspersed with relatively normal functioning. The lack of information regarding the nature and course of maternal distress symptoms contributes to difficulties in elucidating the mechanisms by which maternal distress contributes to negative outcomes in offspring.

Three separate, but typically confounded dimensions of maternal distress include severity, chronicity, and timing of the child's exposure (Brennan et al., 2000; Hammen & Brennan, 2003). Numerous studies have suggested that maternal distress severity and chronicity, rather than specific diagnoses per se, are crucially related to offspring

outcomes such that more severe and chronic symptoms result in more detrimental outcomes (e.g., Keller et al., 1986; Sameroff, Barocas, & Seifer, 1984). The timing of maternal distress in relation to offspring development is also important to consider, as there may be crucial periods that differentially impact offspring outcomes. For example, it has been proposed that exposure to maternal distress during early childhood may be especially disruptive for development (Goodman & Gotlib, 1999). Only a few studies have examined the effects of timing of exposure to maternal distress over extended periods of offspring development, but evidence appears to suggest potentially differential outcomes based on the timing of exposure, particularly underscoring the salient impact of early exposure, including prenatal exposure.

Prenatal maternal distress. Over the past century, there has been vast improvement in the care and maintenance of physical health in pregnant women, but less consideration has been given regarding maternal mental health and well-being during pregnancy (Glover, 2014). Maternal distress during pregnancy is relatively common (Priest, Austin, Barnett, & Buist, 2008; Woods, Melville, Guo, Fan, & Gavin, 2010) and a substantial proportion of mothers who experience distress during pregnancy or during the postpartum period continue to experience symptoms into the child's early years (Beeghly et al., 2002; Horwitz, Briggs-Gowan, Storfer-Isser, & Carter, 2009). Recent studies have estimated the prevalence of depression during pregnancy to range from 7 to 30% (Bennett, Einarson, Taddio, Koren, & Einarson, 2004; Teixeira, Figueiredo, Conde, Pacheco, & Costa, 2009; Vesga-Lopez et al., 2008) and suggest that these estimates are higher in very young mothers, mothers in lower socioeconomic classes, and mothers with a history of psychiatric illness (Hobfoll, Ritter, Lavin, Hulsizer, & Cameron, 1995;

Lancaster et al., 2010). The prevalence of prenatal anxiety has been examined less frequently than depression, but is estimated to range between 5 to 54% (Faisal-Cury & Rossi Menezes, 2007; Lee et al., 2007).

The prenatal period is a time of rapid development during which the fetus is particularly vulnerable to both positive and negative environmental influences that can have persisting effects on offspring development (Sandman, Davis, Buss, & Glynn, 2012). It has been proposed that maternal distress can alter the intra-uterine environment and thus influence fetal development (Goodman, Rouse, Long, Ji, & Brand, 2011). Systematic research on prenatal maternal distress in humans is relatively recent, but over the past two decades, there has been substantial evidence indicating that elevated maternal distress during pregnancy is linked to a wide range of adverse effects on the offspring, including low birth weight, preterm birth, immune dysfunction, developmental delays, behavioral, emotional, and social problems, and various learning and cognitive deficits (e.g., Bennett et al., 2004; T. Field, 2010; Flynn, Davis, Marcus, Cunningham, & Blow, 2004; Grizenko et al., 2012; Gutteling et al., 2006; A. C. Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002; Marmorstein, Malone, & Iacono, 2004; Mulder et al., 2002; O'Connor, Heron, Golding, Glover, & Team, 2003; O'Hara & Swain, 1996; Rice et al., 2010; Tegethoff, Greene, Olsen, Schaffner, & Meinlschmidt, 2011; Van den Bergh, Van Calster, Smits, Van Huffel, & Lagae, 2008; Wright et al., 2010). These findings have been considered in the context of the fetal programming hypothesis, which posits that humans carry a certain level of genetic and physiological plasticity that allows for the fetus to adapt *in utero* to the anticipated conditions of the postnatal environment (D. J. Barker, 1998; Glover, 2014). Thus, this “programming” is hypothesized to lead to

alterations in various aspects of neurobiological development, and consequently influence long-term outcomes in offspring (de Bruijn, van Bakel, & van Baar, 2009).

Potential Mechanisms Underlying the Influence of Maternal Distress on Offspring Aggression

Overall, there is a great deal of literature supporting the fundamental impact of maternal distress on offspring development and outcomes, ranging from physical health issues to psychopathology. However, it is still unclear exactly how maternal distress is "transmitted" to ultimately influence aggression outcomes in offspring. Several potential mechanisms underlying the effects of maternal distress on offspring outcomes have been examined in the literature (Goodman & Gotlib, 1999). In particular, considerable research has focused on biological mechanisms of transmission, including the hypothalamic-pituitary-adrenal (HPA) axis and associated neurobiological systems (e.g., prefrontal function), which may in turn be associated with specific cognitive and psychosocial factors.

HPA axis and PFC dysfunction. The HPA axis is a neuroendocrine stress circuit that triggers the release of glucocorticoids (i.e., cortisol in humans) in the regulation of the stress response (Keller-Wood & Dallman, 1984). The integrity of the HPA axis system is crucial for normal adaptation and homeostasis (Seyle, 1956), and the prefrontal cortex (PFC) has been shown to be crucially involved in the regulation of the HPA axis (Diorio, Viau, & Meaney, 1993; Figueiredo, Bruestle, Bodie, Dolgas, & Herman, 2003; Wang et al., 2005). For instance, there is evidence that a greater release of dopamine in the PFC may alter HPA axis responses to stress (Spencer, Ebner, & Day, 2004).

Maternal distress has been shown to be associated with offspring HPA axis dysfunction (A. S. Clarke, Wittwer, Abbott, & Schneider, 1994; Essex, Klein, Cho, & Kalin, 2002; Lupien, King, Meaney, & McEwen, 2000; Spangler & Grossmann, 1993) and PFC abnormalities (C. Buss, Davis, Muftuler, Head, & Sandman, 2010; Tiffany Field & Diego, 2008), and interestingly, disruptions in both systems have also been shown to be related to the development of aggressive behavior (Bohnke, Bertsch, Kruk, & Naumann, 2010; Mehta & Beer, 2010). Thus, these neurobiological correlates represent logical and promising candidates for mechanisms that may underlie the relationship between maternal distress offspring aggression (Caldji, Diorio, & Meaney, 2000; Talge et al., 2007; Weinstock, 2005). How these biological mechanisms impact specific alterations at the cognitive, behavioral, or emotional level of function is still under study.

Nonetheless, existing evidence supports the conjecture that these biological processes may be associated with specific cognitive mechanisms and psychosocial factors (e.g., parenting) that can help explain the link between maternal distress and long-term offspring outcomes, specifically aggression. In particular, abnormalities in HPA axis function has been shown to negatively impact the development of brain structures and neural systems that are centrally involved in aspects of higher-order cognition (Goldstein, Rasmusson, Bunney, & Roth, 1996). Furthermore, early care experiences, such as parental neglect and negative parenting quality, have been shown to contribute to alterations in offspring brain development, particularly in the PFC (Gee et al., 2013; Petrosini et al., 2009), as well as offspring HPA axis dysfunction in childhood (Pendry & Adam, 2007), extending to adolescence (Murray, Halligan, Goodyer, & Herbert, 2010).

Cognitive ability. Cognitive impairment has been proposed as a key mechanism mediating the influence of genetic and environmental factors on aggression (Moffitt & Caspi, 2001; Raine & Yang, 2006; Yang, Glenn, & Raine, 2008), as deficits in various forms of higher-order cognitive ability have been consistently shown to be associated with increased risk for aggression (e.g., Hinshaw, 1992; Lipsey & Derzon, 1998; Tremblay & LeMarquand, 2001). For example, neurocognitive impairment, including executive function (EF) deficits, has been shown to be associated with increased risk for aggression and other chronic ASB (Hinshaw, 1992; Morgan & Lilienfeld, 2000).

Studies also show that maternal distress may contribute to various neurocognitive outcomes, although the results are not entirely consistent, with some studies suggesting that maternal distress is associated with deficits in offspring language and cognitive development (Cogill, Caplan, Alexandra, Robson, & Kumar, 1986; Keim et al., 2011), but some reporting positive associations between maternal distress and children's cognitive, motor, and language development (DiPietro, Novak, Costigan, Atella, & Reusing, 2006; Laplante, Brunet, Schmitz, Ciampi, & King, 2008) or no significant effects of maternal distress on children's cognitive outcomes (Kurstjens & Wolke, 2001). Research is also limited on whether maternal distress influences cognitive function in offspring beyond the school age stage of development.

Cognitive appraisals of stress. A less examined cognitive mechanism is the cognitive appraisal of stress (Ehlert & Straub, 1998). Stress exposure has been consistently linked to the development of various forms of psychopathology, including externalizing problems such as aggression and other ASB (Agnew, 2001; Bergen, Martin, Richardson, Allison, & Roeger, 2004; Krueger et al., 2002; Verona & Kilmer, 2007;

Verona & Sachs-Ericsson, 2005). A variety of stressors have been shown to be associated with aggression, ranging from minor daily hassles (van Eck, Nicolson, & Berkhof, 1998; Zautra, Finch, Reich, & Guarnaccia, 1991) to major life stressors, such as economic hardship (McLoyd & Wilson, 1991). Interestingly, it has been proposed that the impact of a stressor crucially depends on the subjective cognitive appraisal of the stressor (i.e., the degree to which the stressor is perceived as aversive), rather than mere exposure to specific stressors (Berkowitz, 1990; S. Cohen, Kamarck, & Mermelstein, 1983; Lazarus & Folkman, 1984). Indeed, several studies have found that perceived stress crucially determines the onset and course of psychopathology (Hampel & Petermann, 2006; Willemen, Koot, Ferdinand, Goossens, & Schuengel, 2008).

While cognitive appraisal of stress has been most frequently examined in depressed and anxious individuals (e.g., Conway et al., 2012; Krackow & Rudolph, 2008), there is some recent evidence that cognitive appraisal crucially contributes to aggressive behavior (Sprague, Verona, Kalkhoff, & Kilmer, 2011). For example, a tendency toward negative cognitive appraisals (e.g., perceived unfairness) has been proposed as a key psychological mechanism for the development of aggression (C. A. Anderson & Bushman, 2002), and there is a well-established body of relevant research suggesting that individuals with increased levels of aggression tend to interpret ambiguous cues as more threatening and hostile (e.g., Barrett, Rapee, Dadds, & Ryan, 1996). While studies linking maternal distress specifically to cognitive appraisals of stress in the offspring are lacking, there is limited evidence that maternal distress, including maternal cognitions and expectations about distress, may contribute to negative appraisals and deficits in coping with stress in the offspring (Creswell, O'Connor, & Brewin, 2006; Weinstock, 1997).

Parenting style. Maladaptive parenting has also been widely examined as a risk factor for aggression in offspring (Burke, Pardini, & Loeber, 2008; Loeber, Burke, & Pardini, 2009; Nagin & Tremblay, 2001b; Raine, Brennan, & Mednick, 1997). Aspects of parenting, including sensitive and supportive care, have been shown to be crucial in the development of secure attachments in offspring (Dubois-Comtois, Cyr, & Moss, 2011), while unavailable or inconsistent parental care has been shown to contribute to insecure attachment, psychopathology, and emotional dysregulation in children (Carlson, 1998; Smeekens, Riksen-Walraven, & van Bakel, 2007). This can be interpreted in the context of Bowlby's Attachment Theory (Bowlby, 1969), which posits that the quality of parent-child relationships has a long-term effect on child functioning, given that these interactions influence early development, including internal working models of self and relationships with others. Maternal distress has been shown to contribute to negative parenting and caregiving behaviors, which in turn likely increases vulnerability for externalizing problems in offspring (Berg-Nielsen, Vikan, & Dahl, 2002; Kochanska, Murray, & Harlan, 2000; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Spinrad et al., 2007). Specifically, mothers who experience increased distress tend to exhibit more disengaged or actively negative caregiving behaviors characterized by hostility, as well as coercive and controlling behaviors (Bayer, Hiscock, Ukoumunne, Price, & Wake, 2008; J. Betts, Gullone, & Allen, 2009; Leiferman, Ollendick, Kunkel, & Christie, 2005; Lovejoy et al., 2000).

Current Studies

Although there is substantial literature supporting the association between maternal distress and aggression in offspring, there are several limitations to be noted.

First, investigations of maternal distress have tended to focus on more broad phenotypic outcomes in offspring, such as externalizing behaviors or ASB. While this has provided important information, aggression is a core symptom across various psychiatric disorders and is also likely related to a more persistent and serious form of ASB that is etiologically distinct from other forms of ASB (E. D. Barker et al., 2007; E. D. Barker, Tremblay, et al., 2011; Moffitt, 1993). Thus, examining the influence of maternal distress specifically on aggression may elucidate a developmental risk pathway that is distinctly informative across various forms of offspring psychopathology.

Second, while numerous studies have investigated underlying biological mechanisms that may account for the association between maternal distress and aggression, with a particular focus on HPA axis dysfunction, research is limited on ways in which these biological alterations impact and shape specific psychological factors that represent promising targets for intervention. Thus, examining specific higher-order cognitive processes and psychosocial factors (e.g., parenting quality) that have been linked to the development of aggressive behavior would not only allow for a more comprehensive understanding of the nature of the influence of maternal distress, but also potentially generate data concerning effective targets for further research and intervention.

Lastly, most studies have utilized cross-sectional designs, which greatly limit our understanding of the long-term consequences of maternal distress across development. The few prospective longitudinal studies that examine outcomes later in development suggest that the impact of prenatal maternal distress persists at least into adolescence for various behavioral and emotional problems (O'Donnell et al., 2013; Van den Bergh et al., 2008). Relatedly, it has been shown that risk for developing anxiety disorders, major

depression, and substance dependence in adulthood are approximately three times as high in offspring of depressed parents (Weissman et al., 2006). Furthermore, there have only been a handful of longitudinal studies examining heterogeneity in the nature and course of maternal distress symptoms, which generally appear to indicate that different patterns and progression of maternal distress symptoms are associated with differential outcomes in offspring (e.g., K. S. Betts, Williams, Najman, & Alati, 2014a; K. S. Betts, Williams, Najman, & Alati, 2014b; Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007; Campbell, Morgan-Lopez, Cox, & McLoyd, 2009).

The following two studies aim to address these gaps in the literature by exploring the long-term influence of maternal distress on offspring aggression (rather than more broad externalizing phenotypes, e.g., ASB) in young adulthood using a prospective, longitudinal design. **Study 1** explores cognitive mechanisms as potential mediators of the association between prenatal maternal distress and offspring aggression in adulthood. **Study 2** examines indicators of parenting style as a potential mediator of the association between distinct maternal distress trajectories from pregnancy to offspring adolescence and offspring aggression in adulthood.

Study 1: Prenatal Maternal Distress and Offspring Aggression in Young Adulthood: The Role of Cognitive Mechanisms as Potential Mediators

Aggression and the Role of Prenatal Maternal Distress

Aggression and violence place significant burdens on the individual, as well as society, with costs estimated around 2.3 million dollars per individual in the most extreme cases in the United States (M. A. Cohen et al., 2010). Behaviors associated with aggression and conduct problems account for about one-half of referrals to mental health professionals (Kazdin et al., 1987). Elevated levels of aggression have been shown to be associated with a range of negative outcomes in a variety of domains, including substance abuse, antisocial behavior, depression, and academic failure (Broidy et al., 2003; Tremblay et al., 2004). Given these seriously adverse implications for aggression, much research has been dedicated to identifying risk factors that contribute to aggressive behaviors. One promising area of research involves the role of maternal distress experienced during pregnancy.

The prenatal period is a time of rapid development during which the fetus is particularly vulnerable to both positive and negative environmental influences that can have persisting effects on offspring development (Sandman et al., 2012). There is substantial and longstanding evidence from experimental animal studies indicating that prenatal maternal distress contributes to a variety of negative outcomes on the offspring (e.g., Ader & Plaut, 1968; A. S. Clarke et al., 1994; Hockman, 1961; Joffe, 1965; Keeley, 1962; Maccari & Morley-Fletcher, 2007; Weinstock, 2008), which has been instrumental in promoting and informing work in humans. Systematic research on prenatal maternal distress in humans is relatively recent but over the past two decades, there has been a

plethora of evidence that is consistent with the animal literature (see Van den Bergh, Mulder, Mennes, & Glover, 2005 for a review).

Specifically with regard to offspring outcomes related to aggression, prenatal maternal anxiety has been shown to be associated with aggression and conduct problems in preschoolers (O'Connor et al., 2002) and school-age children (E. D. Barker & Maughan, 2009; Clavarino et al., 2010; O'Connor et al., 2002; O'Connor et al., 2003; Van den Bergh & Marcoen, 2004), and prenatal maternal depression has also been shown to increase the risk for violent behaviors and criminality in adolescents (Hay, Pawlby, Waters, Perra, & Sharp, 2010) and young adults (Maki et al., 2003). Importantly, several studies have found that these effects of prenatal maternal distress remain significant, even after controlling for postnatal maternal mood (Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; O'Connor et al., 2002; Van den Bergh & Marcoen, 2004), which is suggestive of the particular importance of the prenatal environment on offspring development and outcomes.

Gender Differences

Evidence suggests that males tend to engage in higher levels of physical aggression, and these gender differences seem to be present early and remain throughout development (Baillargeon et al., 2007; Hyde, 1984; Tremblay et al., 2004). Only a few studies have addressed gender differences in humans with regard to the influence of prenatal maternal distress on offspring outcomes, and findings thus far have revealed mixed findings regarding the nature of the differences (de Bruijn et al., 2009; Martin, Noyes, Wisenbaker, & Huttunen, 1999; O'Connor, 2002; O'Connor et al., 2003; Rodriguez & Bohlin, 2005; Van den Bergh et al., 2008). For example, while some studies

indicate that the effects of prenatal maternal distress are more prominent in females (McCormick, Smythe, Sharma, & Meaney, 1995), with females exhibiting a stronger association between maternal distress and externalizing behaviors (Kaiser & Sachser, 2005), other studies suggest that males may be more vulnerable to the negative effects of prenatal maternal distress (A. Susan Clarke, Soto, Bergholz, & Schneider, 1996). There is also some evidence of differential effects across genders, for example, with evidence showing that prenatal distress is more strongly associated with internalizing symptoms in female offspring, and impairment in learning and cognition in male offspring (Weinstock, 2007). Thus, while studies appear to suggest gender differences in the effect of prenatal maternal distress on various offspring outcomes, the exact nature of these differences still remains unresolved.

Relevant Biological Mechanisms Underlying the Influence of Prenatal Maternal Distress on Offspring Outcomes

It has been proposed that maternal distress can alter the intra-uterine environment and influence fetal development (Goodman, Rouse, Long, et al., 2011). One proposed biological mechanism by which this programming occurs involves the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is a neuroendocrine stress circuit that triggers the release of glucocorticoids (i.e., cortisol in humans) in the regulation of the stress response (Keller-Wood & Dallman, 1984). When cortisol is released during stress, it enhances an organism's ability to adapt to an immediate stressor, but chronically elevated cortisol levels result in deleterious effects on multiple physiologic, emotional, and behavioral systems (McEwen, 1998). It has been proposed that prenatal maternal distress results in increased glucocorticoid exposure in the fetus (O'Connor et al., 2005)

and that this exposure to excess levels of cortisol impacts the programming of the fetal HPA axis system, as well as neural systems that are crucially involved in HPA axis regulation.

Several studies have reported associations between prenatal maternal distress and HPA axis dysfunction in offspring in both animal and human studies, and evidence also suggests that these changes in HPA axis function last at least until adolescence (A. S. Clarke et al., 1994; Gutteling et al., 2005; A. Huizink et al., 2008; Mulder et al., 2002; Van den Bergh et al., 2008). Much research has also offered insight into neural changes that may occur in offspring due to prenatal maternal distress and subsequent exposure to glucocorticoids. Specifically, there is a high density of glucocorticoid receptors (GRs) in the prefrontal cortex (PFC) (Sanchez, Young, Plotsky, & Insel, 2000; Teicher et al., 2003) and accordingly, studies indicate that the PFC is crucially involved in HPA axis regulation (Diorio et al., 1993; Figueiredo et al., 2003; Wang et al., 2005). For example, structural alterations in the PFC have been found following increased glucocorticoid exposure in rats (Carboni et al., 2010).

Given the role of the PFC in HPA axis function, as well as the protracted maturation of the PFC in terms of myelination and synaptic density across development (Yakovlev & Lecours, 1967), the PFC may be particularly susceptible to early prenatal insults. Importantly, paralleling findings regarding HPA axis dysfunction, studies also indicate that prenatal maternal distress influences PFC development and function in offspring. For example, prenatal maternal distress has been shown to lead to significant structural alterations (i.e., reductions in dendritic spine densities, decreased gray matter) in the PFC in offspring (C. Buss et al., 2010; Murmu et al., 2006). Overall, the literature

appears to suggest that fetal glucocorticoid exposure may be an important mechanism by which prenatal maternal distress contributes to long-term alterations in specific biological mechanisms (i.e., HPA axis and PFC function) in offspring. However, whether these neurobiological alterations induced by prenatal maternal distress are crucially linked to specific negative psychological outcomes in offspring is still unclear (Abe et al., 2007).

The Role of Cognitive Ability as a Potential Mediator of the Association Between Prenatal Maternal Distress and Offspring Aggression

The two most well-established neurocognitive impairments associated with aggression and conduct problems include deficits in executive function (EF) and verbal intelligence quotient (IQ) (E. D. Barker et al., 2007; Nigg & Huang-Pollock, 2003). Executive functions are broadly conceived as higher-order processes that organize and control thought and action to enable goal-directed behavior (Welsh & Pennington, 1988), and encompass a variety of functions such as planning, switching, and inhibition (B. L. Miller, Cummings, & Stuss, 2007). While deficits in EF have been consistently shown to be associated with various forms of antisocial behaviors (ASB), including aggression, conduct problems, and delinquency (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011), several studies have found that EF impairments may be more striking in specific types of ASB, especially aggression (e.g., Seguin, Nagin, Assaad, & Tremblay, 2004).

In addition, while low IQ has been shown to be associated with various forms of ASB (e.g., Denno, 1990; Hogh & Wolf, 1983; J. McCord & Ensminger, 1997) differences in IQ may also influence different types of ASB (C. Burt, 1969; W. McCord & McCord, 1959). For example, there is evidence that lower IQ is associated with more

aggressive and violent behaviors, whereas higher IQ is related to more covert, monetary-directed ASB (Gath & Tennent, 1972; Merrill, 1947; Tennent & Gath, 1975). In particular, deficits in verbal IQ appear to be especially relevant for aggression (E. D. Barker et al., 2007; Nagin & Tremblay, 2001b). Indeed, impairment in EF and verbal IQ have been shown to be associated specifically with physical aggression, but not with other forms of ASB, i.e., theft (E. D. Barker et al., 2007; E. D. Barker, Tremblay, et al., 2011).

Interestingly, some studies suggest that EF deficits are more strongly associated with aggression than other cognitive abilities (e.g., verbal ability) (Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995). Especially among individuals who exhibit chronic aggressive behaviors, impairment in various EFs is evident in early childhood and adolescence (E. D. Barker et al., 2007; E. D. Barker, Tremblay, et al., 2011). Specifically, difficulties in EF have been hypothesized to contribute to increased risk for aggression by decreasing behavioral inhibition, which leads to difficulties in predicting certain consequences and engaging in socially appropriate behaviors in challenging contexts (Giancola, 1995; Ishikawa & Raine, 2003; Seguin, 2009).

It has been proposed that disruptions in neural development due to prenatal maternal distress contribute to cognitive deficits in the fetus, which likely impacts various behavior problems in the offspring, such as aggression (Brennan, Hall, Bor, Najman, & Williams, 2003). Initial non-human animal studies with rodents and primates have shown associations between prenatal maternal distress and offspring cognitive function and learning (Weinstock, 2008; Yaka, Salomon, Matzner, & Weinstock, 2007). Studies in humans have also suggested that prenatal maternal distress is associated with deficits in

offspring cognitive development (Bergman et al., 2007; Mennes, Stiers, Lagae, & Van den Bergh, 2006). For example, EF deficits (Mennes et al., 2006) and lower intelligence scores (Laplante et al., 2008) have been found in children of mothers with high levels of prenatal distress. Nonetheless, there have also been contradictory findings. For example, some studies have reported a positive association between prenatal maternal distress and children's cognitive and language abilities (DiPietro et al., 2006; Laplante et al., 2008), and there are also reports of no significant effects of maternal distress on children's cognitive outcomes (Kurstjens & Wolke, 2001). Importantly, studies in the literature have not been consistent in their operationalization of prenatal maternal distress, which may explain some of the noted mixed findings. In the proposed study we will use a combined measure of maternal self-reports of depression, anxiety and stress to better encapsulate maternal psychological symptoms that may lead to prenatal programming changes, and thus impacts on offspring cognitive and behavioral outcomes.

Executive functions and intelligence, particularly fluid intelligence that reflects abstract thought and reasoning (Carroll, 1993; Cattell, 1971) have been shown to depend largely on the integrity of the prefrontal cortex and associated neural systems (e.g., Duncan, Burgess, & Emslie, 1995; Séguin & Zelazo, 2005). Individuals with prefrontal damage tend to perform poorly on EF tasks and experience difficulty in planning, decision making, and generally regulating day-to-day behaviors (Damasio, 1994), which are considered to be indicators of intelligence as well (e.g., Sternberg, 1988). The relationship between intelligence and EF is "complex and overlapping," (Denckla, 1996) with evidence suggesting that not all EFs are associated with psychometric intelligence (Ardila, Pineda, & Rosselli, 2000; Duncan, Emslie, Williams, Johnson, & Freer, 1996;

Friedman et al., 2006). Therefore, prenatal maternal distress may differentially influence certain aspects of cognitive ability across development, which may have specific implications for relevant outcomes such as aggression.

The Role of Cognitive Appraisal of Stress as a Potential Mediator of the Association Between Prenatal Maternal Distress and Offspring Aggression

A less examined cognitive process that may potentially link prenatal maternal distress and offspring aggression is offspring cognitive appraisal, which refers to the subjective perception, interpretation, and evaluation of a stressor (Ehlert & Straub, 1998). While stress exposure has been consistently linked to the development of aggression and other ASB (Agnew, 2001; Bergen et al., 2004; Krueger et al., 2002; Verona & Kilmer, 2007; Verona & Sachs-Ericsson, 2005), it has been proposed that the impact of a stressor crucially depends on the subjective cognitive appraisal of the stressor (Berkowitz, 1990; S. Cohen et al., 1983; Lazarus & Folkman, 1984; Olff, Langeland, & Gersons, 2005). Accordingly, there is some recent evidence that the degree to which a stressor is perceived as aversive, rather than mere exposure to specific stressors, is a key determinant of aggressive behavior (Craig, 2007; Sprague et al., 2011).

Research suggests that cognitive appraisal correlates with specific biological responses (Kemeny, 2003); specifically, negative appraisals of stressors have been shown to be associated with HPA axis dysfunction (Buchanan, al'Absi, & Lovallo, 1999) and there is also evidence that these appraisals are processed largely by prefrontal regions (Rudebeck, Bannerman, & Rushworth, 2008), which subsequently activate hypothalamic and brain stem regions that regulate the initiation of the physiological stress response (Ongur, An, & Price, 1998). We suggest that prenatal maternal distress may lead to

changes in the PFC and/or the set point of the offspring HPA axis stress response system which ultimately contribute to changes in levels of perceived stress (de Bruijn et al., 2009). In support of this idea, there is evidence that offspring of mothers who experienced distress during pregnancy exhibit deficits in coping with stressful situations (Weinstock, 1997). However, empirical studies examining the role of cognitive appraisals in the context of maternal distress or the development of aggressive behaviors are limited, as cognitive appraisal has been most frequently examined in the literature in the context of depression and anxiety outcomes.

The Current Study

The current study aims to explore the association between maternal distress during pregnancy and offspring aggression in adulthood (age 20). We also seek to explore whether specific cognitive mechanisms (i.e., cognitive ability, cognitive appraisal) mediate the association between prenatal maternal distress and offspring aggression at age 20. In addition, we seek to examine whether gender will moderate these associations. We hypothesize that prenatal maternal distress will be associated with increased offspring aggression in young adulthood. We also hypothesize that impairment in offspring cognitive ability and appraisal (i.e., decreased cognitive ability and increased negative appraisal of stress) will mediate this association. We also hypothesize that these associations will be significantly moderated by gender.

Study 1 Method

Participants and Procedures

Participants in the current study consisted of women and their young adult offspring selected from The Mater-University of Queensland Study of Pregnancy

(MUSP), a prospective birth cohort study of 7,223 mothers and their offspring born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Australia (Keeping et al., 1989). The birth cohort was predominantly Caucasian and of lower middle and working class socioeconomic status (SES). The MUSP was originally devised to investigate the children's physical, cognitive, and psychological health as a function of pregnancy, obstetric and psychosocial conditions as well as to predict health, development, and behavior at age 5. Extensive psychosocial information about the mother and the family was collected from the mother during the 1st trimester of pregnancy (on average at 18 weeks' gestation), 3-5 days after birth, 6 months after birth, and 5 years after birth. Descriptive measures of the pregnancy, delivery, and the neonatal period were also obtained from medical records. At the 5-year follow-up, over 69% of mothers (n = 5,342) who gave birth were successfully located and provided information regarding the child's development, behavior, and health (Keeping et al., 1989; Williams et al., 1998).

At offspring age 15, a subset of the mother-offspring pairs from the initial cohort were recruited based on continued residency in the Brisbane area and on women's self-reports of depression on the Delusions-Symptoms-States Inventory (DSSI; Bedford, Foulds, & Sheffield, 1976). These DSSI scores were used to identify the level and frequency of elevated depression using specific algorithms (details provided in Brennan et al., 2000; Hammen & Brennan, 2003). Subsequently, nine hundred and ninety-one families met inclusion criteria based on the mothers' DSSI scores. Among these 991 families, 816 families consented and were included. This sample contains 92% white, 8% minority, and the median family income at indicated middle and lower middle classes.

Participants did not significantly differ from the initial cohort in terms of family income, maternal education, and child's gender (Hammen, Brennan, & Shih, 2004).

Our current sample included 747 mother-offspring participants (of the original 816 studied at offspring age 15) who participated in a follow-up focused on young adult behavioral outcomes collected at youth age 20. Thus, the youth and their mothers were included in the current study if the mother provided a prospective report of anxiety and depression during pregnancy, if the family participated in the age 15 and 20 follow-up, and if either the mother or the youth provided reports of youth externalizing problems at age 20. Our sample contained 50.7% female offspring (n = 379). The ethnicity composition for this sample is 91.3% Caucasian-descendent, 4.7% Asian-descendent, 2.1% Maori/Islander, and 1.9% Aboriginal. The mean age for the mother at childbirth was 25.5 years (SD = 5.1).

Measures

Prenatal maternal distress. A latent factor for prenatal maternal distress was modeled using the depression subscale of the DSSI, the anxiety subscale of the DSSI, and the Reeder stress inventory (RSI; Reeder, Chapman, & Coulson, 1968) as indicators. The DSSI and RSI were administered to the mothers at the first prenatal visit. The depression subscale of the DSSI contains seven items (e.g., "I have been so miserable that I have had difficulty sleeping," "I have been depressed without knowing why," "I have gone to bed not caring if I never woke up") and mothers rated each item on a 5-point scale (Never, Rarely, Some of the Time, Most of the Time, All the Time) (α for our sample = .80).

The anxiety subscale of the DSSI also contains seven items (e.g., "Worrying has kept me awake at night," "I have worried about every little thing," "I have been

breathless or had a pounding of my heart") and uses the same 5-point scale. Internal consistency in the current sample for the depression and anxiety subscales of the DSSI were acceptable (Cronbach's α = .80, .77, respectively). The DSSI has been found to correlate well with other measures of anxiety and depression symptoms, such as the Edinburgh Postnatal Depression Scales (EPDS) and the Hospital Anxiety/Depression Scale (HADS) (Bedford & Deary, 1999).

The RSI is designed to measure self-perceived daily strain resulting from the physiological and psychological reactions to personal or social situations (Gutteling et al., 2005; Heslop et al., 2001). The RSI contains four items (e.g., "In general, I am usually tense or nervous," "My daily activities are extremely trying and stressful") and mothers rated each item on a 5-point scale (Never, Rarely, Some of the Time, Most of the Time, All the Time). Internal consistency in the current sample for the RSI was acceptable (Cronbach's α = .80). Construct validity of the RSI has been supported in the literature (Metcalfe et al., 2003). Items on the DSSI and RSI scales were coded such that higher scores reflected increased levels of distress.

Offspring aggression at age 20. A latent factor for offspring aggression in young adulthood (age 20) was modeled using the youth self-, mother-, and peer-reports of the aggression subscales of the Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003) as indicators. The aggression scale includes items such as: "argues a lot," "demands a lot of attention," "destroys his/her own things," "destroys things belonging to family or others," "gets in many fights," "screams a lot," "stubborn," "sullen or irritable," "sudden changes in mood or feelings," and "temper tantrums or hot temper." The youth self-report contained 12 items, and the mother- and peer-reports contained 17 items. Each

item was rated on a 3-point scale (0=never, 1=sometimes, 2=often) and raw scores were used in the current study. Higher scores reflected increased levels of aggression. Internal consistencies in the current sample for the youth self-, mother-, and peer- reports were acceptable (Cronbach's α = .83, .91, .89, respectively).

Offspring physical aggression at age 20. In order to examine whether there may be subfactors in the ABCL aggression subscales that may more closely reflect other relevant constructs, such as negative emotionality, we ran exploratory factor analyses (EFA) on the youth self-, mother-, and peer reports of the aggression subscales using principal axis factoring (PAF) extraction methods, which is recommended as the most appropriate method with non-normally distributed data (Costello & Osborne, 2005). We found subfactor indicators across reporters that appear to reflect physical aggression (e.g., "gets in many fights," "physically attacks people," "threatens other people"). These "physical aggression" subfactor indicator scores were highly correlated with the corresponding overall/total aggression subscale scores across all reporters (self report: r = .615, $p < .001$; mother report: r = .734, $p < .001$; peer report: r = .735, $p < .001$). However, these "physical aggression" subfactor indicator scores were also less correlated with a measure of Neuroticism in offspring adulthood (self report: r = .128, p = .003; mother report: r = .227, $p < .001$; peer report: r = .082, p = .067), compared to the overall/total aggression scores with Neuroticism (self report: r = .434, $p < .001$; mother report: r = .247, $p < .001$; peer report: r = .139, p = .005).

Additionally, we found that these three (youth self-, mother-, and peer report) "physical aggression" subfactor indicators loaded onto a latent variable with good fit, controlling for maternal smoking: χ^2 = 1.131, df = 2, p = .568, CFI = 1.000, TLI = 1.033,

and *RMSEA* = 0.000 (90% CI: 0.000, 0.061, p = .895). Given that there appeared to be a subfactor that may more closely reflect "physical aggression," and is less correlated with neuroticism (the best proxy we have in our data for negative emotionality) we ran all our main analyses twice: first using the overall/total aggression latent factor, then subsequently using the physical aggression latent factor.

Offspring cognitive ability. A latent factor for IQ was modeled using the Scaled Score from the Peabody Picture Vocabulary Test-Revised (PPVT-R; Dunn & Dunn, 1981) at offspring age 5 and the Digit Span and Vocabulary Scaled Scores from the Wechsler Intelligence Scale for Children—4th Edition (WISC-IV; Wechsler, 2003) at offspring age 15 as indicators. These measures are standardized measures of receptive language skills (PPVT-R) and verbal/nonverbal reasoning ability (WISC-IV). Higher scores reflected increased cognitive ability.

A latent factor for (deficits in) executive function (EF) was modeled using the difference score from the Stroop Task and the number of errors and perseverative errors from the Wisconsin Card Sorting Task (WCST) at offspring age 15 as indicators. Both the Stroop Task (Stroop, 1935) and WCST (Milner, 1963; Stuss et al., 2000) have been widely used to investigate deficits in executive function. Specifically, the WCST measures set-shifting, or the ability to alter a behavioral response in the face of changing contingencies (Milner, 1963; Stuss et al., 2000), and the Stroop measures response inhibition/interference control (Stroop, 1935). Higher scores reflected increased deficits in cognitive ability.

Offspring cognitive appraisal. At age 15 and 20, offspring were administered a version of the Episodic Stress Assessment of the UCLA Life Stress Interview (LSI)

(Hammen, Henry, & Daley, 2000). Modeled after the contextual threat approach of Brown and Harris (1978), the LSI uses standard general probes and follow-up queries to elicit specific life events occurring in the past 12 months. Specifically, interviewers obtained detailed information regarding the nature and date of each event, and the circumstances in which the event occurred (e.g., whether the event was unexpected or expected).

Immediately following the elicitation of an acute event during the LSI administration, participants were asked to rate their perception of the negative impact associated with the stressor ("How would you rate the overall negative impact of this event?"). Participants provided a subjective severity rating ranging from 1 (no negative impact) to 5 (extremely severe negative impact). The test–retest reliability of the subjective ratings has previously been demonstrated to be adequate (Espejo et al., 2011).

Written narratives of each event were later presented to a rating team blind to youths' subjective ratings of the event. For each event, the team subsequently assigned an objective severity rating representing the impact this event would be expected to have on an average person under identical circumstances. Severity ratings ranged from 1 (no negative impact) to 5 (extremely severe negative impact). Reliability and validity data for the UCLA Life Stress Interview have been reported in other studies of adolescents and young adults (e.g., Hammen et al., 1995; Shih, Eberhart, Hammen, & Brennan, 2006).

Mean subjective and objective impact ratings were calculated for each participant. To compute an index of cognitive appraisal (subjective perceptions of stressfulness that adjusts for differences in the objective severity of events), mean subjective rating scores were regressed on mean objective rating scores. The standardized residuals from this

analysis constituted the cognitive appraisal variable in the present analyses, with higher scores reflecting greater estimations of negative impact.

This approach of calculating standardized residuals is an established method for comparing subjective and objective scores (e.g., Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; De Los Reyes & Prinstein, 2004; Krackow & Rudolph, 2008). A previous study using a select sample drawn from the same larger cohort showed that elevated negative appraisals were significantly associated with depression diagnoses, and that specifically, that these negative appraisals mediated the association between a genetic marker (i.e., *5-HTTLPR*) and depression (Conway et al., 2012).

Potential Confounds. Potential confounds were examined in preliminary analyses, including maternal education and income, maternal substance abuse (i.e., marijuana, tobacco, and alcohol use) during pregnancy, and pregnancy outcome variables (e.g., birth weight). Confounding variables that were associated with the outcome and predictor variables were included as covariates in the final structural models as detailed below.

Data Analysis

All analyses were conducted using Mplus 7 (L. K. Muthén & Muthén, 1998-2012) using structural equation modeling (SEM). Structural equation modeling integrates measurement (factor analysis) and structural (path analysis) approaches to allow for the estimation of latent variables and relations among variables, which is more robust to measurement error (Bentler, 1980, 1983; Bollen, 1989; Jöreskog, 1973; Jöreskog & Sörbom, 1979). To account for missing data and adjust for non-normality of the data, maximum-likelihood estimation was used, which produces less biased and more reliable

results compared to conventional methods of dealing with missing data, such as listwise or pairwise deletion (T. W. Anderson, 1957; Schafer & Graham, 2002). Model fit was assessed by the χ^2 test statistic, the comparative-based fit index (CFI), the Tucker-Lewis index (TLI), and the root-mean-square error of approximation (RMSEA) (Bentler, 1990; Browne & Cudeck, 1993). Fit statistics and conventions recommended by Hu and Bentler (1999) were used, including: a non-significant χ^2 (indicating that the data did not significantly differ from the hypothesized model); CFI and TLI greater than or equal to .95 for reasonably good fit (Hu & Bentler, 1999) and values between .90-.95 for acceptable fit (Bentler, 1990); and RMSEA less than or equal to .08 for adequate fit and values less than or equal to .05 for close fit (Browne & Cudeck, 1993). The fit of a single model was evaluated using a combination of χ^2, CFI, TLI, and RMSEA, as each individual fit index has its own limitations and there is no consensus regarding the use of a single fit index to determine model fit (Loehlin, 2004). Furthermore, whereas a non-significant χ^2 is ideal, it is difficult to achieve when using larger sample sizes (i.e., more than 200 cases) (Bentler, 1990).

Given that the structural portion of SEM involves relations among latent variables, it is crucial to test the validity of the measurement model (i.e., relationships between latent and observed variables) prior to evaluating the structural model (Byrne, 2011). Thus, prior to parameterizing the structural models, initial measurement models were tested to ensure acceptable fit to the sample data using confirmatory factor analysis (CFA) (Byrne, 2011). Next, in order to test for gender as a moderator in our models, we tested measurement invariance across gender using multi-group modeling, which implements simultaneous analyses of multiple groups/populations and has been indicated

as the recommended approach for categorical moderator variables, such as gender (Baron & Kenny, 1986). Establishing measurement invariance across groups ensures that the observed indicators measure the same latent factors across groups, and thus is a necessary prerequisite for conducting structural invariance testing across groups (e.g., testing differences in path coefficients, latent means) (Vandenberg & Lance, 2000). Measurement invariance testing procedures and results are presented in the Appendix.

After testing the validity of the measurement models and establishing measurement invariance across gender, we tested a series of structural equation models. First, we tested whether there was a main effect of prenatal maternal distress on offspring aggression in adulthood. Subsequently, we tested whether cognitive ability (i.e., IQ and EF) or cognitive appraisal (at age 15 and at age 20) mediated this association in separate models. In these mediation models, we examined the direct effect of prenatal maternal distress on offspring aggression in young adulthood, as well as potential indirect effects through these potential cognitive mediators to determine whether and to what extent these variables mediated the effects of prenatal maternal distress on offspring aggression.

For our mediation analyses, bootstrapping resampling procedures, which are robust to violations of multivariate normality, were used to obtain the standard errors and confidence intervals for the direct and indirect/mediated effects, as recommended by MacKinnon, Fairchild, and Fritz (2007). Bootstrap standard error and bias corrected bootstrapping confidence intervals along with *p*-values were reported for effects in mediation models. Given that bootstrapping methods do not deliver standard errors (SE) or *p*-values for the standardized coefficients, *p*-values from unstandardized coefficients were used to determine significance.

Finally, we used multi-group modeling to examine whether paths were moderated by gender by comparing a baseline model in which paths were freely estimated across gender versus a restricted model in which paths were constrained to be equal across gender. We used a Satorra-Bentler (SB) scaled (mean-adjusted) chi-square difference test to compare models, where the chi-square of each model was divided by a scaling correction to better approximate chi-square distribution under non-normality (Satorra, 2000). A non-significant chi-square difference test indicated that there was no difference in paths across gender.

Study 1 Results

Descriptive Statistics

Descriptive statistics and correlations between all observed variables/indicators are presented in Tables 1 and 2, respectively. The measures of prenatal distress included the DSSI depression subscale, the DSSI anxiety subscale, and the RSI. The measures of offspring aggression at age 20 included youth self-report, mother-report, and peer-report of the ABCL Aggression scale. For cognitive ability, the measures of IQ included the PPVT-R Scaled Score, WISC-IV Vocabulary Scaled Score, and WISC-IV Digit Span Scaled Score, and the measures of EF included the Stroop difference score, WCST number of errors, and WCST number of perseverative errors. The measures of cognitive appraisal are presented at age 15 and 20.

Prenatal Maternal Distress and Offspring Aggression

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress and offspring aggression fit the data well: $\chi^2 = 13.602$, $df = 8$, $p = .093$, $CFI = 0.995$, $TLI = 0.990$, and $RMSEA = 0.031$ (90% CI: 0.000, 0.058, $p = .867$).

All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.498- 0.892, $p < .001$). The structural equation model for the effect of prenatal maternal distress on offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 1. The model fit the data well: $\chi^2 = 27.762$, $df = 16$, $p = .034$, $CFI = 0.990$, $TLI = 0.983$, and $RMSEA = 0.031$ (90% CI: 0.009, 0.050, $p = .945$). Prenatal maternal distress was a significant predictor of increased aggression (standardized estimate = 0.196, $p < .001$).

Next, multi-group analysis was performed to examine whether gender moderated the effect of prenatal maternal distress on physical aggression. The results of multi-group analysis revealed no path differences across gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB} = 1.035$, $df = 1$, $p = .309$).

Physical aggression outcome. The measurement model consisting of prenatal maternal distress and offspring physical aggression fit the data well: $\chi^2 = 12.219$, $df = 8$, $p = .142$, $CFI = 0.995$, $TLI = 0.990$, and $RMSEA = 0.027$ (90% CI: 0.000, 0.055, $p = .909$). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.522 – 0.888, $p < .001$). The structural model for the effect of prenatal maternal distress on offspring physical aggression in young adulthood, controlling for the relevant confounding variable of maternal smoking during pregnancy, is shown in Figure 1. The model fit the data well: $\chi^2 = 20.582$, $df = 12$, $p = .057$, $CFI = 0.990$, $TLI = 0.983$, and $RMSEA = 0.031$ (90% CI: 0.000, 0.053, $p = .918$). Although

prenatal maternal distress was not a significant predictor of physical aggression (standardized estimate = 0.094, p = .221), recent advances in mediation analyses suggest that mediation should be tested with or without the presence of main effects (Hayes, 2009); thus, we proceeded to run mediation analyses for the effect of prenatal maternal distress on offspring physical aggression outcome.

Next, multi-group analysis was performed to examine whether gender moderated the effect of prenatal maternal distress on physical aggression. The results of multi-group analysis revealed no path differences across gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB}$ =0.012, df = 1, p = .913).

Cognitive Appraisal At Age 15 as a Mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 15, and offspring aggression, fit the data well: χ^2 = 28.441, df = 12, p = .005, *CFI* = 0.985, *TLI* = 0.975, and *RMSEA* = 0.043 (90% CI: 0.023, 0.063, p = .691). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.500 – 0.891, p < .001). The structural equation model that included cognitive appraisal at age 15 as a mediator of the association between prenatal maternal distress and offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 2. The model fit the data well: χ^2 = 44.373, df = 22, p = .003, *CFI* = 0.983, *TLI* = 0.974, and *RMSEA* = 0.037 (90% CI: 0.021, 0.053, p = .913). The total effect of prenatal maternal distress on offspring aggression,

which is the sum of all its direct and indirect effects, was significant with an unstandardized estimate of 0.165 ($p < .001$). The direct effect of prenatal maternal distress on offspring aggression was significant with an unstandardized estimate of 0.169 ($p < .001$), but the indirect effect via cognitive appraisal at age 15 was negligible and non-significant with an unstandardized estimate of -0.005 ($p = .357$), suggesting no significant mediation by cognitive appraisal at age 15. Prenatal maternal distress did not have a significant effect on cognitive appraisal at age 15 (unstandardized estimate = -0.015, $p = .199$), although cognitive appraisal at age 15 had a marginally significant effect on aggression (unstandardized estimate = 0.309, $p = .051$), suggesting that increased negative appraisals were associated with higher levels of aggression.

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB} = 2.689$, $df = 3$, $p = .442$).

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 15, and offspring physical aggression, fit the data well: $\chi^2 = 23.715$, $df = 12$, $p = .022$, $CFI = 0.986$, $TLI = 0.976$, and $RMSEA = 0.036$ (90% CI: 0.013, 0.057, $p = .845$). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = .522 – .887, $p < .001$). The structural equation model that included cognitive appraisal at age 15 as a mediator of the association between prenatal maternal distress and offspring physical aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during

pregnancy, is shown in Figure 2. The model fit the data well: $\chi^2 = 31.894$, $df = 17$, $p = .016$, $CFI = 0.987$, $TLI = 0.979$, and $RMSEA = 0.034$ (90% CI: 0.015, 0.052, $p = .921$). The total effect of prenatal maternal distress on offspring physical aggression, which is the sum of all its direct and indirect effects, was not significant with an unstandardized estimate of 0.027 ($p = .154$). The direct effect of prenatal maternal distress on offspring physical aggression was not significant with an unstandardized estimate of 0.027 ($p = .151$), and the indirect effect via cognitive appraisal at age 15 was negligible and non-significant with an unstandardized estimate of 0.000 ($p = .906$), suggesting no significant mediation by cognitive appraisal at age 15. Prenatal maternal distress did not have a significant effect on cognitive appraisal at age 15 (unstandardized estimate = -0.016, $p = .176$), and cognitive appraisal at age 15 did not have a significant effect on offspring physical aggression (unstandardized estimate = 0.010, $p = .873$).

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB} = 2.227$, $df = 3$, $p = .527$).

Cognitive Appraisal At Age 20 as a Mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 20, and offspring aggression, fit the data well: $\chi^2 = 23.690$, $df = 12$, $p = .022$, $CFI = 0.990$, $TLI = 0.982$, and $RMSEA = 0.036$ (90% CI: 0.013, 0.057, $p = .846$). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.503 – 0.891, $p < .001$). The

structural equation model that included cognitive appraisal at age 20 as a mediator of the association between prenatal maternal distress and offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 3. The model fit the data well: χ^2 = 38.279, *df* = 22, *p* = .017, *CFI* = 0.988, *TLI* = 0.981, and *RMSEA* = 0.031 (90% CI: 0.013, 0.048, *p* = .971). The total effect of prenatal maternal distress on offspring aggression, which is the sum of all its direct and indirect effects, was significant with an unstandardized estimate of 0.165 (*p* < .001). The direct effect of prenatal maternal distress on offspring aggression was significant with an unstandardized estimate of 0.166 (*p* < .001), but the indirect effect via cognitive appraisal at age 20 was negligible and non-significant with an unstandardized estimate of -0.001 (*p* = .875), suggesting no significant mediation by cognitive appraisal at age 20. Prenatal maternal distress did not have a significant effect on cognitive appraisal at age 20 (unstandardized estimate = -0.002, *p* = .862), but cognitive appraisal at age 20 did have a significant effect on offspring aggression (unstandardized estimate = 0.398, *p* = .012), suggesting that increased negative appraisals were associated with higher levels of aggression.

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB}$ =3.184, *df* = 3, *p* = .364).

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 20, and offspring physical aggression, fit the

data well: χ^2 = 20.740, *df* = 12, *p* = .054, *CFI* = 0.990, *TLI* = 0.982, and *RMSEA* = 0.031 (90% CI: 0.000, 0.053, *p* = .915). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = .520 – .888, *p* < .001). The structural equation model that included cognitive appraisal at age 20 as a mediator of the association between prenatal maternal distress and offspring physical aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy, is shown in Figure 3. The model fit the data well: χ^2 = 30.350, *df* = 17, *p* = .024, *CFI* = 0.989, *TLI* = 0.981, and *RMSEA* = 0.032 (90% CI: 0.012, 0.051, *p* = .941). The total effect of prenatal maternal distress on offspring physical aggression, which is the sum of all its direct and indirect effects, was not significant with an unstandardized estimate of 0.027 (*p* = .151). The direct effect of prenatal maternal distress on offspring physical aggression was not significant with an unstandardized estimate of 0.027 (*p* = .150), and the indirect effect via cognitive appraisal at age 20 was negligible and non-significant with an unstandardized estimate of 0.000 (*p* = .923), suggesting no significant mediation by cognitive appraisal at age 20. Prenatal maternal distress did not have a significant effect on cognitive appraisal at age 20 (unstandardized estimate = -0.003, *p* = .833), and cognitive appraisal at age 20 also did have a significant effect on offspring physical aggression (unstandardized estimate = 0.032, *p* = .590).

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB}$ =1.189, *df* = 3, *p* = .756).

IQ as a Mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, IQ, and offspring aggression, fit the data well: $\chi^2 = 32.513$, *df* = 24, *p* = .115, *CFI* = 0.994, *TLI* = 0.992, and *RMSEA* = 0.022 (90% CI: 0.000, 0.039, *p* = .998). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.406 – 0.960, *p* < .001). The structural equation model that included IQ as a mediator of the association between prenatal maternal distress and offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy, maternal education, and maternal income, is shown in Figure 4. The model fit the data well: $\chi^2 = 67.274$, *df* = 44, *p* = .014, *CFI* = 0.987, *TLI* = 0.981, and *RMSEA* = 0.027 (90% CI: 0.012, 0.039, *p* = .999). The total effect of prenatal maternal distress on offspring aggression, which is the sum of all its direct and indirect effects, was significant with an unstandardized estimate of 0.163 (*p* < .001). The direct effect of prenatal maternal distress on offspring aggression was significant with an unstandardized estimate of 0.161 (*p* = .001), but the indirect effect via IQ was negligible and non-significant with an unstandardized estimate of 0.002 (*p* = .750), suggesting no significant mediation by IQ. Prenatal maternal distress did not have a significant effect on IQ (unstandardized estimate = -0.037, *p* = .712), although IQ had a significant effect on aggression (unstandardized estimate = -0.046, *p* = .028), suggesting that higher IQ was associated with decreased levels of aggression.

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are

freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB}$ =6.191, *df* = 3, *p* = .103).

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, IQ, and offspring physical aggression, fit the data well: χ^2 = 31.810, *df* = 24, *p* = .132, *CFI* = 0.994, *TLI* = 0.991, and *RMSEA* = 0.021 (90% CI: 0.000, 0.039, *p* = .998). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = .410 – .950, *p* < .001). The structural equation model that included IQ as a mediator of the association between prenatal maternal distress and offspring physical aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal income, is shown in Figure 4. The model fit the data well: χ^2 = 63.057, *df* = 37, *p* = .005, *CFI* = 0.984, *TLI* = 0.976, and *RMSEA* = 0.031 (90% CI: 0.017, 0.043, *p* = .995). The total effect of prenatal maternal distress on offspring physical aggression, which is the sum of all its direct and indirect effects, was not significant with an unstandardized estimate of 0.027 (*p* = .155). The direct effect of prenatal maternal distress on offspring physical aggression was not significant with an unstandardized estimate of 0.026 (*p* = .170), and the indirect effect via IQ was negligible and non-significant with an unstandardized estimate of 0.001 (*p* = .739), suggesting no significant mediation by IQ. Prenatal maternal distress did not have a significant effect on IQ (unstandardized estimate = -0.040, *p* = .691) and IQ also did not have a significant effect on aggression (unstandardized estimate = -0.015, *p* = .078).

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant

moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB}$ =1.793, *df* = 3, *p* = .617).

EF as a Mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, EF, and offspring aggression, fit the data well: χ^2 = 43.020, *df* = 24, *p* = .010, *CFI* = 0.959, *TLI* = 0.938, and *RMSEA* = 0.033 (90% CI: 0.016, 0.048, *p* = .969). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = 0.274 – 0.874, *p* < .001). The structural equation model that included EF as a mediator of the association between prenatal maternal distress and offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 5. The model fit the data well: χ^2 = 55.434, *df* = 37, *p* = .026, *CFI* = 0.990, *TLI* = 0.986, and *RMSEA* = 0.026 (90% CI: 0.009, 0.039, *p* = .999). The total effect of prenatal maternal distress on offspring aggression, which is the sum of all its direct and indirect effects, was significant with an unstandardized estimate of 0.165 (*p* < .001). The direct effect of prenatal maternal distress on offspring aggression was significant with an unstandardized estimate of 0.169 (*p* < .001), but the indirect effect via EF was negligible and non-significant with an unstandardized estimate of -0.004 (*p* = .538), suggesting no significant mediation by EF. Prenatal maternal distress did not have a significant effect on EF (unstandardized estimate = -0.101, *p* = .480), although EF had a significant effect on aggression (unstandardized estimate = 0.038, *p* = .049), suggesting that higher levels of EF were associated with decreased levels of aggression.

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB} = 1.794$, $df = 3$, $p = .616$).

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, EF, and offspring physical aggression, fit the data well: $\chi^2 = 30.561$, $df = 24$, $p = .167$, $CFI = 0.984$, $TLI = 0.976$, and $RMSEA = 0.019$ (90% CI: 0.000, 0.037, $p = .999$). All observed indicators significantly loaded on their hypothesized latent variables (standardized estimates = .276 – .852, $p < .001$). The structural equation model that included EF as a mediator of the association between prenatal maternal distress and offspring physical aggression in young adulthood, controlling for the relevant confounding variable of maternal smoking during pregnancy, is shown in Figure 5. The model fit the data well: $\chi^2 = 38.078$, $df = 30$, $p = .148$, $CFI = 0.995$, $TLI = 0.993$, and $RMSEA = 0.019$ (90% CI: 0.000, 0.035, $p = .999$). The total effect of prenatal maternal distress on offspring physical aggression, which is the sum of all its direct and indirect effects, was not significant with an unstandardized estimate of 0.027 ($p = .164$). The direct effect of prenatal maternal distress on offspring physical aggression was not significant with an unstandardized estimate of 0.028 ($p = .141$), and the indirect effect via EF was negligible and non-significant with an unstandardized estimate of -0.001 ($p = .559$), suggesting no significant mediation by EF. Prenatal maternal distress did not have a significant effect on EF (unstandardized estimate = -0.105, $p = .462$) and EF also

did not have a significant effect on physical aggression (unstandardized estimate = 0.012, $p = .122$).

Next, multi-group analysis was performed to examine whether gender moderated associations in this model. The results of multi-group analysis suggested no significant moderation by gender, as the comparison between the baseline model (where paths are freely estimated across gender) and the restricted model (where paths are constrained across gender) was non-significant ($\chi^2\Delta_{SB} = 2.605$, $df = 3$, $p = .457$).

Study 1 Discussion

The current study examined the effect of prenatal maternal distress on offspring aggression in young adulthood, as well as potential indirect effects via cognitive ability (i.e., IQ and EF) and cognitive appraisal. Our findings suggest that while prenatal maternal distress, as well as deficits in cognitive ability and negative cognitive appraisals of stress, independently contribute to increased aggression levels in young adulthood, these cognitive factors do not represent underlying mediating mechanisms by which prenatal maternal distress contributes to offspring aggression. Further, we found no significant moderation of gender in any of our associations.

Our finding of an association between prenatal maternal distress and offspring aggression in young adulthood is consistent with a vast literature linking maternal distress during pregnancy to a range of negative outcomes in offspring, particularly aggression and various related behavioral problems (e.g., conduct problems, violence) (E. D. Barker & Maughan, 2009; Hay et al., 2010; O'Connor et al., 2002). As the overwhelming majority of studies have been limited to offspring outcomes during childhood or adolescence, the current study adds substantially to the previous research by providing

support for the long-term effect of prenatal maternal distress extending to young adulthood. Furthermore, as most studies examining the influence of prenatal maternal distress have tended to focus on specific psychiatric diagnoses in offspring, the current findings also have implications for a variety of psychopathological behaviors and disorders in which aggression is a core symptom, which can better inform our understanding of clinical phenomena and etiology that cuts across traditional diagnostic boundaries.

It is important to mention that while prenatal maternal distress predicted offspring aggression, it was not significantly associated with offspring *physical* aggression as measured by subfactor indicator scores obtained from our preliminary exploratory factor analysis (EFA). Furthermore, the cognitive factors examined in the current study were also not associated with physical aggression. Given that the physical aggression scores were less correlated with a measure of Neuroticism available in our sample, which served as a proxy for negative emotionality, this may suggest that prenatal maternal distress contributes to a broad negative emotionality construct in offspring, which has been reported in the literature (Pluess et al., 2011), rather than aggression per se. However, our indicator measures for physical aggression consisted of much fewer subscale items (ranging from 3-5 items) compared to the total/overall aggression subscale items (ranging from 12-17 items), and scales with too few items may lack content and construct validity, internal consistency, and test-retest reliability (David A Kenny, 1979; Nunnally, 1976). For instance, the internal consistencies in the current sample for the physical aggression subscale items were lower than the total/overall aggression subscales (Cronbach's α = .67, .74, and .75 for youth self-, mother-, and peer-reports, respectively, compared to α

= .83, .91, .89). Thus, this area concerning the different types or components of aggression (e.g., physical aggression) represents a fruitful area for further investigation with regard to the potential overarching versus specific effects of maternal distress during pregnancy.

Our finding of an association between cognitive ability and appraisals of stress and aggression is consistent with previous research (Gath & Tennent, 1972; Seguin et al., 2004; Weinstock, 1997). However, none of the cognitive mechanisms were associated with prenatal maternal distress or served as significant mediators. There are currently mixed findings in the literature regarding the influence of prenatal maternal distress on offspring cognitive ability, although a recent meta-analysis showed that prenatal maternal distress has a small, but significant negative association with child cognitive outcomes from birth until preschool years (Tarabulsy et al., 2014). Unfortunately, studies regarding cognitive appraisals of stress are particularly lacking. It is possible that certain psychosocial factors (e.g., parenting) that were not examined in the current study moderate the association between prenatal maternal distress and cognitive processes and other outcomes. For example, there is evidence that maternal parenting behaviors (e.g., sensitivity to infant distress) moderates the association between prenatal maternal distress and offspring cognitive development during infancy (Grant, McMahon, Reilly, & Austin, 2010) extending to the preschool age (Schechter et al., 2015). There is also evidence that distress during later periods of pregnancy (e.g., second and third trimesters) has the strongest influence on cognitive outcomes in offspring (Weinstock, 2008), but there are also mixed findings regarding this as well, with some studies suggesting that stress

exposure during the first and second trimesters contribute to greater offspring cognitive deficits during infancy (King & Laplante, 2005).

Strengths

The current study took a novel and more comprehensive approach in operationalizing prenatal maternal distress by combining measures of maternal depression, anxiety, and stress. The majority of research thus far has relied on a single measure of prenatal distress, such as traumatic events (Laplante et al., 2004) or the presence of mood disorder symptomatology (Tarabulsy et al., 2014). Different measures of distress are highly correlated (Goodman & Tully, 2008; Matthey et al., 2003), suggesting the validity and utility of combining distress measures into a latent factor. While it is plausible that distinct types of distress may be linked to different outcomes in offspring, the use of single measures may be related to the mixed findings regarding offspring outcomes across the literature. In addition, the use of multiple reporters (peer, mother, self) for offspring aggression in our study reduces problems of rater bias by capturing a greater portion of the true score variance and by improving the generalizability of the measurements (Rushton, Brainerd, & Pressley, 1983; J. C. Schwarz, Barton-Henry, & Pruzinsky, 1985). The current study also adopted a longitudinal, prospective study design with a relatively large sample, which allowed for examination of potential mediators in the causal pathway between prenatal maternal distress and offspring aggression in young adulthood. The current sample consisted of mother-offspring dyads who were followed from pregnancy until offspring age 20 years, which represents one of the longest follow-up periods in longitudinal studies of maternal

risk factors and offspring outcomes. The prospective study design also contributes to less errors in making causal inferences compared to cross-sectional designs.

Limitations

There are several limitations to note. First, echoing the majority of studies in this area, we only utilized maternal self-report to conceptualize maternal distress, as opposed to other observer-based or clinical interview measures, which contributes to difficulty in interpreting exact levels of prenatal maternal distress that may be particularly salient for offspring development. This might also account for some of the inconsistencies in the literature; for example, mother's objective reports of stressful events during pregnancy were shown to be associated with offspring cognitive outcomes, but not mother's subjective assessments of prenatal stress (King & Laplante, 2005; Laplante et al., 2008).

Second, we did not assess the role of postnatal maternal distress in our analyses. Although prenatal maternal distress has been shown to influence various offspring cognitive, behavioral, and emotional outcomes, even after controlling for postnatal maternal distress symptoms (Bergman et al., 2007; O'Connor et al., 2002), postnatal maternal distress may also plays a crucial role in influencing long-term offspring outcomes. Initial supplementary analyses from our sample suggests that both prenatal and postnatal distress likely represents distinct risk factors for the development of aggression in young adulthood, consistent with previous literature (e.g., O'Connor et al., 2002).

Third, no measure of maternal cognitive functioning or maternal aggression was collected in our sample. Given that both cognitive ability (Deary, Johnson, & Houlihan, 2009; Friedman et al., 2008) and aggression (Miles & Carey, 1997) have been shown to be heritable, controlling for these variables in our analyses may have strengthened our

ability to draw conclusions about the unique effect of offspring cognitive ability on later aggression. Related to this, our investigation is unable to inform the particular question regarding the nature of potential genetic influences in the effect of prenatal maternal distress on offspring aggression. However, there is much literature suggesting that maternal distress influences offspring development and outcomes independent of genetic factors (e.g., A. Huizink et al., 2008; Laplante et al., 2008). More research using genetically informative designs is needed to better understand and disentangle genetic and environmental influences in the effect of maternal distress on specific offspring outcomes (Rice et al., 2010; Rutter, 2007).

Fourth, the majority of the cognitive indicators used in the current study were measured during adolescence, with the exception of one IQ indicator (i.e., PPVT-R Scaled Score at age 5). It is possible that the lack of association between prenatal maternal distress and any of the cognitive variables in our sample indicates that prenatal maternal distress has a stronger influence on cognitive development during early infancy/childhood compared to adolescence, perhaps due to the lack of development of brain areas and neural systems that are particularly vulnerable to early prenatal insults (A. C. Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003; Talge et al., 2007). Throughout adolescence, there is significant growth and change in multiple areas of the PFC, especially in myelination and synaptic pruning processes, both of which are crucial for higher-order cognitive mechanisms (e.g., long-term planning) (Paus et al., 1999; Sowell, Trauner, Gamst, & Jernigan, 2002). Thus, prenatal maternal distress may not contribute significantly to long-term cognitive processes in the offspring with the rapid maturational development of neurobiological systems involved in higher-order cognition

(Sowell et al., 2002). In addition, given that the heritability of cognitive ability increases to 70% or greater by late adolescence (McGue, Bouchard, Iacono, & Lykken, 1993; Neisser et al., 1996), it is possible that other genetic influences play a more crucial role in shaping cognitive functions during this period.

Lastly, it is possible that perhaps prenatal maternal distress influences specific components of cognition (e.g., verbal intellectual ability) that may in turn impact aggression. For example, there is evidence that prenatal maternal distress may be more strongly related to verbal and language ability in children (Laplante et al., 2008), and that deficits in verbal ability are particularly relevant for the development of aggression (Nagin & Tremblay, 2001b). Thus, future research should examine the potential mediating role of more specific, fine-grained components of cognition that may be more strongly influenced by prenatal maternal distress and represent core deficits in the development of aggressive behaviors.

Implications and Future Directions

The current findings underscore the importance of early education and intervention in pregnant mothers who are experiencing distress. Our finding of long-term effects of maternal distress during pregnancy on offspring development that extends to young adulthood suggests that prevention and intervention during pregnancy is crucial. Overall, there is less emphasis and consideration placed on maternal mental health during pregnancy compared to physical health (Glover, 2014). Our study, along with the considerable body of literature regarding the impact of prenatal maternal distress on the offspring, suggests that education and treatment during pregnancy may be extremely valuable. Several studies have found the effectiveness of early interventions (e.g.,

psychotherapy) on maternal distress symptoms during pregnancy (Spinelli & Endicott, 2003), which may ultimately improve offspring outcomes.

The current investigation highlights the need for further research examining the potential mechanisms underlying the influence of prenatal maternal distress on long-term offspring outcomes. While there is evidence supporting the role of the offspring HPA axis in particular in underlying the influence of prenatal maternal distress on altered behavioral outcomes in offspring (Van den Bergh et al., 2008), the nature of the associations between prenatal distress and offspring HPA axis function varies widely without any solid replications (Glover, O'Connor, & O'Donnell, 2010). For example, there are findings reporting associations between prenatal maternal anxiety and heightened basal waking and afternoon cortisol in offspring (O'Connor et al., 2005), but also reports of flattened diurnal cortisol profiles (Van den Bergh et al., 2008). As the potential biological mechanisms by which prenatal exposure to maternal distress shape offspring development have yet to be fully elucidated, more studies are needed that examine not only offspring HPA axis function, but also other promising neurobiological mechanisms. For example, recent advances in molecular genetics have highlighted the role of epigenetic mechanisms, molecular modifications to gene activity that do not involve changes to the underlying DNA sequence, by which prenatal exposure to maternal distress influences epigenetic pathways (e.g., gene expression activity) to ultimately impact various outcomes in offspring (Catherine Monk, Spicer, & Champagne, 2012).

Furthermore, although there is evidence that specific cognitive impairments may be associated with HPA axis dysfunction (Aisa, Tordera, Lasheras, Del Río, & Ramírez,

2007; Wolf, 2003), the research in this area is still limited and there may be little correlation between psychological measures and measures of HPA axis abnormalities (e.g., cortisol measures) (Glover et al., 2010). Given this, it is possible that even though offspring HPA axis function may underlie the association between prenatal maternal distress and offspring aggression, this neurobiological mechanism may play a key role in other important psychological factors that may serve as targets for clinical intervention.

Conclusion

In conclusion, the results of the current study indicate that prenatal maternal distress and higher-order cognitive mechanisms independently contribute to the development of aggression in young adulthood, but that these cognitive processes do not mediate the association between prenatal maternal distress and offspring aggression. Our results suggest that while prenatal maternal distress contributes to long-term aggression in the offspring extending to young adulthood, other potential mechanisms that underlie this association must be examined in future studies. These findings also suggest that while prenatal maternal distress does not predict cognitive ability or appraisal particularly during adolescence, they may still represent promising targets for intervention to prevent the development and manifestation of aggression during adulthood.

Study 2: Trajectories of Maternal Distress Across Development and Offspring Aggression in Young Adulthood: the Role of Parenting as a Potential Mediator

Aggression and the Role of Maternal Distress

Aggression and violence place significant burdens on the individual, as well as society, with costs estimated around 2.3 million dollars per individual in the most extreme cases in the United States (M. A. Cohen et al., 2010). Behaviors associated with aggression and conduct problems account for about one-half of referrals to mental health professionals (Kazdin et al., 1987). Elevated levels of aggression have been shown to be associated with numerous negative outcomes in a variety of domains, including substance abuse, antisocial behavior, depression, and academic failure (Broidy et al., 2003; Tremblay et al., 2004). Given these seriously adverse implications for aggression, much research has been dedicated to identifying risk factors that contribute to aggressive behaviors. One promising area of research involves the role of maternal psychopathology. In particular, there is considerable literature that indicates that maternal distress represents one of the most crucial risk factors for a wide range of internalizing and externalizing psychopathology, including aggression (E. D. Barker, Jaffee, et al., 2011; O'Connor, 2002; O'Connor et al., 2002; Rice et al., 2010; Van Batenburg-Eddes et al., 2013; Van den Bergh & Marcoen, 2004).

Importance of Chronicity, Severity, and Timing of Exposure to Maternal Distress

Although there is well-established evidence supporting the impact of maternal distress on negative childhood outcomes, literature is quite limited regarding the persistence of these effects into adulthood. Specifically, the majority of studies have been cross-sectional, which provides little information regarding patterns of maternal distress

symptoms over time and how differences in these trajectories may contribute to long-term adverse outcomes in offspring. A handful of studies have shown that in general, mothers tend to endorse increasingly higher levels of anxiety, depression, and other mood disturbance across pregnancy (Da Costa, Brender, & Larouche, 1998; Da Costa, Larouche, Dritsa, & Brender, 1999; DiPietro, Costigan, & Gurewitsch, 2005; Evans, Heron, Francomb, Oke, & Golding, 2001), and that from late pregnancy until early toddler years, there is a general decline in distress symptoms (Eberhard-Gran, Tambs, Opjordsmoen, Skrondal, & Eskild, 2004; Evans et al., 2001; Gulseren et al., 2006; Heron, O'Connor, Evans, Golding, & Glover, 2004; Ritter, Hobfoll, Lavin, Cameron, & Hulsizer, 2000). As such, the importance of considering the severity, chronicity, and timing of the child's exposure to maternal distress has been proposed to better elucidate the dynamic influence of maternal distress symptoms over time on offspring outcomes (Brennan et al., 2000; Hammen & Brennan, 2003).

Chronicity and severity of maternal distress. Numerous studies have suggested that more chronic and severe maternal distress symptoms result in more detrimental outcomes in offspring (e.g., Keller et al., 1986; Sameroff et al., 1984). For example, children whose mothers experienced more depressive episodes are more likely to have more severe diagnoses as well (Hammen, 1991), and recurrent, early-onset major depression in the parent has been shown to be specifically associated with major depression in the offspring (Warner, Mufson, & Weissman, 1995). In addition, fairly linear patterns of association between the severity of maternal distress and negative child outcomes has been reported, which has implications for potentially important effects of subclinical levels of maternal distress (O'Connor, Monk, & Fitelson, 2014). However,

there are some inconsistent findings, for example, with evidence that mild stress experienced by mothers (e.g., daily hassles) may have differential effects from more severe stress (e.g., death of offspring), such that experiencing mild stress in the mother may be beneficial in some cases with regard to offspring outcome (DiPietro et al., 2006).

Timing of maternal distress. Timing of maternal distress is also important to consider, as there may be critical periods of exposure in terms of offspring outcomes. It has been proposed that exposure to maternal distress during early childhood years is especially disruptive for offspring development (Goodman & Gotlib, 1999), contributing to various forms of maladjustment including insecure attachment (Campbell, Cohn, & Meyers, 1995; Teti, Gelfand, Messinger, & Isabella, 1995), as well as early behavioral and cognitive impairment that can impact long-term outcomes (NICHD, 1999). Only a few studies have examined the timing of exposure to maternal distress over extended periods of time. These studies appear to suggest the particular importance of early childhood exposure and also indicate differential outcomes based on timing of exposure. However, the findings are not entirely consistent with regard to critical periods and the nature of offspring outcomes. For example, maternal depression at 14 months postpartum was found to be more strongly predictive of childhood behavioral problems compared to later time points (i.e., at 27 or 42 months postpartum) (Ghodsian, Zajicek, & Wolkind, 1984). However, high levels of maternal distress during pregnancy, but not at later time points up to 5 years postpartum, have been found to be predictive of increased internalizing behavior problems in adolescence, as well as increased levels of behavioral problems and depression in adulthood (K. S. Betts et al., 2014a, 2014b).

With regard to timing, maternal distress during pregnancy has particularly received much attention in the literature, as this has important implications for biological mechanisms underlying fetal development (Goodman, Rouse, Long, et al., 2011). Nonetheless, exposure to maternal distress during different stages of pregnancy may also contribute to differential outcomes. For example, in primates, prenatal maternal stress during the early gestation period, during which the mammalian brain is particularly susceptible to disturbances (Galaburda, Rosen, & Sherman, 1989), has been shown to be associated with impairment in attention and neuromotor functioning in offspring, while prenatal stress during mid-late gestation period was associated with decreased inhibition in offspring (Schneider, Moore, Kraemer, Roberts, & DeJesus, 2002). Several studies have also suggested that early but not later gestational distress may be linked to certain neurological or more severe disturbances in offspring (Carmichael & Shaw, 2000; Glover, O'Connor, Heron, & Golding, 2004; Khashan et al., 2008), but there are also contradictory findings. For example, maternal distress during later stages of pregnancy has been found to be more strongly associated with conduct disorder and various ASB (O'Connor et al., 2003; Rice et al., 2010).

Implications for Long-term Effects of Maternal Distress Across Development

While chronicity, severity, and timing of exposure to maternal distress appear to contribute to differential but overlapping outcomes in offspring in a wide range of domains, most studies in this area have been cross-sectional in design, which presents methodological issues. For instance, chronicity and severity are commonly confounded, meaning that more severe symptoms tend to last longer (Pettit, Lewinsohn, Roberts, Seeley, & Monteith, 2009). Thus, longitudinal studies with large samples that include

multiple assessments of symptoms are needed to better elucidate the trajectory and nature of maternal distress in relation to offspring outcomes.

Currently, very little is known about the heterogeneity in maternal distress trajectories over time, or whether there are meaningful individual differences in the nature and course of maternal distress symptoms, and if this contributes to differential offspring outcomes. Only a handful of studies (K. S. Betts et al., 2014a, 2014b; Campbell et al., 2007; Campbell et al., 2009; Cents et al., 2013; Gross, Shaw, Burwell, & Nagin, 2009) have identified distinct trajectories of maternal distress over time (ranging from 4 to 7 trajectories), with the majority focusing on maternal depressive symptoms. In general, findings appear to indicate that more chronic and elevated trajectories of maternal depressive symptoms appear to be associated with increased internalizing and externalizing problems in offspring (Campbell et al., 2009; Gross et al., 2009). Two notable recent studies by Betts and colleagues (2014a, 2014b) identified seven distinct trajectories of maternal distress (i.e., depression, anxiety, and stress) from pregnancy until offspring age 5 years, and examined their relation to internalizing and externalizing symptoms in offspring during adolescence and adulthood, using the larger cohort from which the sample for the current study was drawn. The results showed that offspring of mothers who experience high levels of distress during pregnancy, but not at later time points up to the child's age of five years, exhibited increased levels of internalizing (but not externalizing) problems during adolescence (K. S. Betts et al., 2014a) and higher internalizing and externalizing problems in adulthood (K. S. Betts et al., 2014b).

Accordingly, exposure to maternal distress across development may have lifelong influences on offspring aggression through genetic, physiological, and epigenetic

mechanisms, particularly as developmental plasticity decreases with age (Hanson, Godfrey, Lillycrop, Burdge, & Gluckman, 2011). As such, more longitudinal studies are needed that examine the persistence of the impact of maternal distress on offspring aggression into adulthood (Glover, 1997). There is recent emerging empirical evidence that suggests that the effects of maternal distress experienced during pregnancy and early childhood persist into adulthood, for example, with a recent finding indicating that prenatal and postnatal maternal depression predicts offspring depression at age 18 (Pearson et al., 2013). Nonetheless, most of the longitudinal studies have covered exposure only through the preschool ages (Bureau, Easterbrooks, & Lyons-Ruth, 2009; Essex, Klein, Miech, & Smider, 2001; Lyons-Ruth, Easterbrooks, & Cibelli, 1997; Munson, McMahon, & Spieker, 2001) and also tend to focus on a single period of exposure to maternal distress.

Specifically, most of the existing studies examining the trajectories of maternal distress only examine maternal distress symptoms through early childhood. Adolescence is another potentially sensitive period of development, and research focusing on trajectories of maternal distress that extend into adolescence is quite sparse. In addition, studies that examine the potential mechanisms linking trajectories of maternal distress and offspring outcomes, especially during adulthood, are lacking. In particular, aspects of parenting style may represent an especially important mechanism, given that the parent-offspring relationship continues to play a central role throughout offspring development.

Maternal Distress and the Role of Parenting

Maladaptive parenting styles may represent an important mechanism by which maternal distress influences later aggression in offspring. This can be interpreted in the

context of Bowlby's Attachment Theory (Bowlby, 1969), which posits that the quality of parent-child relationships has a long-term effect on various aspects of child functioning, given that these interactions influence early development, including internal working models of self and relationships with others. Accordingly, the mother-child interaction and relationship guides the child in the exploration and regulation of various emotions and thoughts (Thompson, 2008).

Evidence suggests that preschoolers who have experienced supportive and sensitive parental care and healthy interactions with mothers are more likely to develop secure attachment styles (Dubois-Comtois et al., 2011; NICHD, 2001) and effective emotion regulation strategies compared to insecurely-attached peers (Easterbrooks, Bureau, & Lyons-Ruth, 2012). In contrast, children who have experienced unavailable or inconsistent care and disrupted mother-child interactions are more likely to develop insecure attachment styles and various behavioral and emotional symptoms associated with the development of psychopathology (Carlson, 1998; Smeekens et al., 2007). Interestingly, recent meta-analytic reviews have shown that mother-child attachment disorganization increases the risk of externalizing problems (Fearon, Bakermans-Kranenburg, van Ijzendoorn, Lapsley, & Roisman, 2010) and is specifically more strongly associated with externalizing compared to internalizing symptoms (Groh, Roisman, van Ijzendoorn, Bakermans-Kranenburg, & Fearon, 2012).

Maternal distress has been consistently shown to be associated with impairment in the quality of caregiving and parenting, such as less emotional availability to their children that may manifest as disinterest or intrusive, coercive/controlling, or hostile behaviors (Bifulco et al., 2002; Frankel & Harmon, 1996; Zeanah, Boris, & Larrieu,

1997). For example, mothers reporting heightened levels of distress were observed to be more negative, intrusive, and hostile towards their children, and also less sensitive and responsive during their interactions with them (Campbell et al., 2004). These types of negative parenting behaviors (e.g., less sensitive and responsive) appear to elicit various emotional and behavioral difficulties (e.g., deficits in self-regulation) in children that are associated with externalizing problems (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Rothbaum & Weisz, 1994) by compromising aspects of parenting that are important for healthy behavioral adjustment and development in the offspring (Choe, Olson, & Sameroff, 2013).

The Role of Maladaptive Parenting in Offspring Aggression

Various aspects of negative parenting have been consistently linked to offspring aggression (Burke et al., 2008; Loeber et al., 2009; Nagin & Tremblay, 2001b; Raine et al., 1997). For example, a coercive, rejecting, and unresponsive parenting style has been found to be one of the best predictors of increased levels of physical aggression in offspring (G.R. Patterson, Reid, & Dishion, 1992; Rothbaum & Weisz, 1994; Tremblay, 2014), while supportive parenting, characterized by acceptance, responsiveness, and encouragement, has been shown to be associated with lower rates of aggressive and behavioral problems (Lamborn, Mounts, Steinberg, & Dornbusch, 1991; Maccoby & Martin, 1983; G. R. Patterson, DeBaryshe, & Ramsey, 1989; Rothbaum & Weisz, 1994)

Thus, maladaptive parenting may represent a mechanism by which maternal distress contributes to offspring aggression in adulthood. Indeed, a small body of research indicates that specific parenting styles (e.g., inconsistent discipline) mediate the association between maternal psychopathology and offspring aggression and conduct

problems (e.g., Barry, Dunlap, Lochman, & Wells, 2009; Stanger, Dumenci, Kamon, & Burstein, 2004). However, it is unknown whether distinct patterns of maternal psychopathology across offspring development may differentially influence aspects of parenting style. Furthermore, studies have tended to focus on the effects of parenting during early childhood on later outcomes, but parent-child relationships and interactions during adolescence likely also have important long-term implications for the development of aggressive behaviors in adulthood.

The Current Study

The current study aims to identify distinct trajectories of maternal distress (i.e., depression, anxiety) from pregnancy until offspring adolescence (age 14) and examine whether these trajectories differentially predict offspring aggression in young adulthood (age 20). We also seek to explore whether maternal parenting style at offspring age 15 mediates the effect of maternal distress trajectories on offspring aggression at age 20. We hypothesize that there will be distinct trajectories of maternal distress that differentially relate to offspring aggression outcome in young adulthood, and that parenting style will serve as a significant mediator of this association.

Study 2 Method

Participants and Procedures

Participants in the current study consisted of women and their young adult offspring selected from The Mater-University of Queensland Study of Pregnancy (MUSP), a prospective birth cohort study of 7,223 mothers and their offspring born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Australia (Keeping et al., 1989). The birth cohort was predominantly Caucasian and of

lower middle and working class socioeconomic status (SES). The MUSP was originally devised to investigate the children's physical, cognitive, and psychological health as a function of pregnancy, obstetric and psychosocial conditions as well as to predict health, development, and behavior at age 5. Extensive psychosocial information about the mother and the family was collected from the mother during the 1st trimester of pregnancy (on average at 18 weeks' gestation), 3-5 days after birth, 6 months after birth, and 5 years after birth. Descriptive measures of the pregnancy, delivery, and the neonatal period were also obtained from medical records. At the 5-year follow-up, over 69% of mothers (n = 5,342) who gave birth were successfully located and provided information regarding the child's development, behavior, and health (Keeping et al., 1989; Williams et al., 1998).

At offspring age 15, a subset of the mother-offspring pairs from the initial cohort were recruited based on continued residency in the Brisbane area and on women's self-reports of depression on the Delusions-Symptoms-States Inventory (DSSI; Bedford et al., 1976). These DSSI scores were used to identify the level and frequency of elevated depression using specific algorithms (details provided in Brennan et al., 2000; Hammen & Brennan, 2003). Subsequently, nine hundred and ninety-one families met inclusion criteria based on the mothers' DSSI scores. Among these 991 families, 816 families consented and were included. This sample contains 92% white, 8% minority, and the median family income at indicated middle and lower middle classes. Participants did not significantly differ from the initial cohort in terms of family income, maternal education, and child's gender (Hammen et al., 2004).

Our current sample included 747 mother-offspring participants (of the original 816 studied at offspring age 15) who participated in a follow-up focused on young adult behavioral outcomes collected at youth age 20. Thus, the youth and their mothers were included in the current study if the mother provided a prospective report of anxiety and depression during pregnancy, if the family participated in the age 15 and 20 follow-up, and if either the mother or the youth provided reports of youth externalizing problems at age 20. Our sample contained 50.7% female offspring (n = 379). The ethnicity composition for this sample is 91.3% Caucasian-descendent, 4.7% Asian-descendent, 2.1% Maori/Islander, and 1.9% Aboriginal. The mean age for the mother at childbirth was 25.5 years (SD = 5.1).

Measures

Maternal distress. The DSSI, which consists of depression and anxiety subscales, was used to measure maternal distress during pregnancy, at birth, age 6 months, age 5 years, and age 14 years. The depression subscale of the DSSI contains seven items (e.g., "I have been so miserable that I have had difficulty sleeping," "I have been depressed without knowing why," "I have gone to bed not caring if I never woke up") and mothers rated each item on a 5-point scale (Never, Rarely, Some of the Time, Most of the Time, All the Time) (α for our sample = .80). The anxiety subscale of the DSSI also contains seven items (e.g., "Worrying has kept me awake at night," "I have worried about every little thing," "I have been breathless or had a pounding of my heart") and uses the same 5-point scale (α for our sample = .77). The DSSI has been found to correlate well with other measures of anxiety and depression symptoms, such as the Edinburgh Postnatal

Depression Scales (EPDS) and the Hospital Anxiety/Depression Scale (HADS) (Bedford & Deary, 1999).

Offspring aggression at age 20. A latent factor for offspring aggression in young adulthood (age 20) was modeled using the youth self-, mother-, and peer-reports of the aggression subscales of the Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003) as indicators. The aggression scale includes items such as: "argues a lot," "demands a lot of attention," "destroys his/her own things," "destroys things belonging to family or others," "gets in many fights," "screams a lot," "stubborn," "sullen or irritable," "sudden changes in mood or feelings," and "temper tantrums or hot temper." The youth self-report contained 12 items, and the mother- and peer-reports contained 17 items. Each item was rated on a 3-point scale (0=never, 1=sometimes, 2=often) and raw scores were used in the current study. Higher scores reflected increased levels of aggression. Internal consistencies in the current sample for the youth self-, mother-, and peer- reports were acceptable (Cronbach's α = .83, .91, .89, respectively).

Offspring physical aggression at age 20. In order to examine whether there may be subfactors in the ABCL aggression subscales that may more closely reflect other relevant constructs, such as negative emotionality, we ran exploratory factor analyses (EFA) on the youth self-, mother-, and peer reports of the aggression subscales using principal axis factoring (PAF) extraction methods, which is recommended as the most appropriate method with non-normally distributed data (Costello & Osborne, 2005). We found subfactor indicators across reporters that appear to reflect physical aggression (e.g., "gets in many fights," "physically attacks people," "threatens other people"). These "physical aggression" subfactor indicator scores were highly correlated with the

corresponding overall/total aggression subscale scores across all reporters (self report: r = .615, $p < .001$; mother report: r = .734, $p < .001$; peer report: r = .735, $p < .001$). However, these "physical aggression" subfactor indicator scores were also less correlated with a measure of Neuroticism in offspring adulthood (self report: r = .128, p = .003; mother report: r = .227, $p < .001$; peer report: r = .082, p = .067), compared to the overall/total aggression scores with Neuroticism (self report: r = .434, $p < .001$; mother report: r = .247, $p < .001$; peer report: r = .139, p = .005).

Additionally, we found that these three (youth self-, mother-, and peer report) "physical aggression" subfactor indicators loaded onto a latent variable with good fit, controlling for maternal smoking: χ^2 = 1.131, df = 2, p = .568, CFI = 1.000, TLI = 1.033, and $RMSEA$ = 0.000 (90% CI: 0.000, 0.061, p = .895). Given that there appeared to be a subfactor that may more closely reflect "physical aggression," and is less correlated with neuroticism (the best proxy we have in our data for negative emotionality) we ran all our main analyses twice: first using the overall/total aggression latent factor, then subsequently using the physical aggression latent factor.

Parenting style at offspring age 15. A latent factor for parenting style (at offspring age 15) was modeled using the three subscales (acceptance, psychological control, firm control) from the Children's Report of Parental Behavior Inventory (CRPBI; Schludermann & Schludermann, 1988), and the two subscales (criticism, emotional over-involvement) from the Five Minute Speech Sample (FMSS; Magaña et al., 1986). The CRPBI is a child-report measure that evaluates perceptions of parenting behaviors across three dimensions: Acceptance (e.g., "enjoys doing things with me"), Psychological Control (e.g., "tells me of all the things she has done for me"), and Firm Control (e.g.,

"insists that I must do exactly as I am told"). In the FMSS, a mother is asked to speak for five minutes into a recorder, without interruption, about her child and how they get along together. Raters who were blind to all other information regarding the parents and their children scored the recordings based on criteria developed by Magaña and colleagues (1986). The FMSS is coded for the mother's expressed emotion (EE) toward their children, which includes critical comments/statements and emotional overinvolvement, and is thought to reflect the negative emotional atmosphere of the family (Hooley & Gotlib, 2000).

Potential Confounds. Potential confounds, including maternal education and income, maternal substance abuse (i.e., marijuana, tobacco, and alcohol use) during pregnancy, and pregnancy outcome variables (e.g., birth weight), that were associated with the outcome (i.e., aggression) were included as covariates in the final models as detailed below.

Data Analysis

LCGA and GMM. We used Mplus 7 (L. K. Muthén & Muthén, 1998-2012) to conduct latent class growth analysis (LCGA) and growth mixture modeling (GMM) to identify latent trajectory classes for maternal distress symptoms from pregnancy until offspring age 14 years. Both GMM (B. O. Muthén, 2001; B. O. Muthén & Muthén, 2000) and LCGA (Nagin, 1999) are flexible statistical procedures for analyzing longitudinal repeated measures data and are useful for exploring heterogeneity in developmental trajectories to estimate different subpopulations/groups of individual trajectories (Jung & Wickrama, 2008; B. O. Muthén, 2001, 2004; B. O. Muthén & Asparouhov, 2002; B. O. Muthén & Muthén, 2000). Thus, as opposed to traditional latent growth modeling

techniques, GMM and LCGA identify whether the population under study is comprised of a mixture of identifiable groups based on their growth trajectories, instead of attempting to only capture differences by assuming variability around one mean trajectory. Individuals are grouped based on latent growth factors, namely the intercept (initial status) and slope.

As there are important differences between LCGA and GMM, conducting both analyses is recommended (B. O. Muthén, 2006). LCGA represents a special case of GMM, with the primary difference being that GMM allows within-class variation (random variability around the mean trajectories within each trajectory class), while LCGA does not (no variability within each trajectory class). Thus, in GMM, growth factor (intercept and slope) variances are estimated in each class, while in LCGA, growth factor variances (and covariances) are fixed to 0.

As per the recommendations in the literature (Jung & Wickrama, 2008; B. O. Muthén, 2006), we initially ran a single-class, univariate latent growth-curve model to examine the average growth curve for maternal distress, assuming no underlying heterogeneity of the growth trajectory. To account for the unequal intervals of the repeated measurements, the loadings for the slope factor for maternal distress measured at pregnancy, birth, 6 months, 5 years, and 14 years were fixed at 0, 0.5, and 1 (and the rest of the loading were set free to allow for non-linearity) following the latent basis framework (Grimm & Ram, 2009; Meredith & Tisak, 1990). We then conducted both LCGA and GMM to select the most appropriate model in terms of the type of modeling (LCGA or GMM) and in terms of the number of trajectory classes. In order to compare

and select the most appropriate models, we used graphical methods, quality of convergence, and comparative fit (Feldman, Masyn, & Conger, 2009).

Graphical methods. Graphs showing the shape and location of the different estimated class trajectories were inspected, as graphical methods can provide a more coherent picture than numeric output alone (Feldman et al., 2009). For example, LCGA extracts many similar classes because it does not allow for within-class variability (Kreuter & Muthén, 2008; B. O. Muthén & Asparouhov, 2006). There is no binding criterion to select the number of trajectory classes in mixture models; a variety of factors such as theoretical considerations, interpretability or replicability, and usefulness, among other issues, should be considered because mixture models are typically used in an exploratory manner and are thus inherently data-driven (Feldman et al., 2009; Jung & Wickrama, 2008). In general, a more parsimonious model (i.e., with fewer classes) is preferred (Nagin & Tremblay, 2001a).

Quality of convergence. In complex mixture models, since algorithms are more likely to converge on local maxima solutions, the use of multiple random starts is recommended (McLachlan & Peel, 2000; B. O. Muthén & Asparouhov, 2006). When a global maximum solution cannot be reached and results in non-convergence, or the failure of the algorithm to replicate a maximum loglikelihood value over many starting values, this suggests that model parameter estimates are untrustworthy and the model may not be suitable given the data, warranting a more parsimonious model (Feldman et al., 2009; B. O. Muthén, 2006). In the current study, models were estimated using 100 or more random sets of start values and 20 optimizations, and rerun with different sets of starting values to find the true maximum likelihood solution and avoid local solutions. In

GMM, for reasons of model convergence, small and non-significant variances of the growth factors were constrained to zero (L. K. Muthén & Muthén, 1998-2012; Nagin, 1999).

Comparative fit. Model fit statistics were evaluated and compared using a combination of criteria, including Akaike's Information Criterion (AIC; Akaike, 1987) Bayesian Information Criterion (BIC; G. Schwarz, 1978), the Lo-Mendell-Rubin likelihood ratio test (LMR-LRT; Lo, Mendell, & Rubin, 2001), and the parametric bootstrapped likelihood ratio test (BLRT; McLachlan & Peel, 2000). Lower values of the AIC and BIC indicate a better fitting model (Nylund, Asparouhov, & Muthén, 2007). The BIC is the most widely used information index, taking into account the model loglikelihood and penalizing for model complexity (i.e., the number of parameters estimated relative to sample size) (B. O. Muthén, 2001). The LMR analytically approximates the likelihood ratio test (LRT) distribution and the BLRT uses bootstrap samples to empirically derive the sampling distribution of the LRT statistic (Nylund et al., 2007). Both LMR and the BLRT compare a *k* class model with the *k-1* class model, and a significant *p*-value indicates that the current model (i.e., the solution with *k* classes) fit the data significantly better than a solution with *k-1* classes. Thus, the model with lower AIC and BIC values and significant LMR and BLRT *p*-values was chosen as the model with better fit.

In addition, we considered average posterior latent probabilities of class membership and the entropy values, which serve as a measures of the precision of individual classification and the degree to which classes are distinguishable (B. O. Muthén, 2004; Nagin, 2005). Entropy values and average posterior latent class

probabilities greater than .70 indicated satisfactory fit (B. O. Muthén & Muthén, 2000). However, since entropy is comparable to an R^2 value in structural equation modeling (i.e., it is possible that entropy is high for poorly fitting models and low for well-fitting models), emphasis should not be placed on entropy in comparing and choosing models, instead relying on other model fit indices (i.e., AIC, BIC, LMR-LRT, BLRT) (Feldman et al., 2009; B. O. Muthén, 2004; B. O. Muthén & Asparouhov, 2006; Ramaswamy, DeSarbo, Reibstein, & Robinson, 1993).

Mediation analyses. After determining the most appropriate class model, we exported the trajectory class assignment information based on each individual's most likely class membership to examine the association between trajectory class membership (dummy-coded) and offspring aggression in young adulthood, as well as to test parenting as a potential mediator of this association. All analyses were conducted using Mplus 7 (L. K. Muthén & Muthén, 1998-2012) using structural equation modeling (SEM). Structural equation modeling integrates measurement (factor analysis) and structural (path analysis) approaches to allow for the estimation of latent variables and relations among variables, which is more robust to measurement error (Bentler, 1980, 1983; Bollen, 1989; Jöreskog, 1973; Jöreskog & Sörbom, 1979).

To account for missing data and adjust for non-normality of the data, maximum-likelihood estimation was used, which produces less biased and more reliable results compared to conventional methods of dealing with missing data, such as listwise or pairwise deletion (T. W. Anderson, 1957; Schafer & Graham, 2002). Model fit was assessed by the χ^2 test statistic, the comparative-based fit index (CFI), the Tucker-Lewis index (TLI), and the root-mean-square error of approximation (RMSEA) (Bentler, 1990;

Browne & Cudeck, 1993). Fit statistics and conventions recommended by Hu and Bentler (1999) were used, including: a non-significant χ^2 (indicating that the data did not significantly differ from the hypothesized model); CFI and TLI greater than or equal to .95 for reasonably good fit (Hu & Bentler, 1999) and values between .90-.95 for acceptable fit (Bentler, 1990); and RMSEA less than or equal to .08 for adequate fit and values less than or equal to .05 for close fit (Browne & Cudeck, 1993). The fit of a single model was evaluated using a combination of χ^2, CFI, TLI, and RMSEA, as each individual fit index has its own limitations and there is no consensus regarding the use of a single fit index to determine model fit (Loehlin, 2004). Furthermore, whereas a non-significant χ^2 is ideal, it is difficult to achieve when using larger sample sizes (i.e., more than 200 cases) (Bentler, 1990).

Given that the structural portion of SEM involves relations among latent variables, it is crucial to test the validity of the measurement model (i.e., relationships between latent and observed variables) prior to evaluating the structural model (Byrne, 2011). Thus, prior to parameterizing the structural models, initial measurement models were tested to ensure acceptable fit to the sample data using confirmatory factor analysis (CFA) (Byrne, 2011).

For our mediation analyses, bootstrapping resampling procedures, which are robust to violations of multivariate normality, were used to obtain the standard errors and confidence intervals for the direct and indirect/mediated effects, as recommended by MacKinnon et al. (2007). Bootstrap standard error and bias corrected bootstrapping confidence intervals along with *p*-values were reported for effects in mediation models. Given that bootstrapping methods do not deliver standard errors (SE) or *p*-values for the

standardized coefficients, *p*-values from unstandardized coefficients were used to determine significance.

Study 2 Results

Descriptive Statistics

Descriptive statistics and correlations between all observed variables/indicators are presented in Tables 3 and 4, respectively. The measures of maternal distress included the DSSI (anxiety and depression subscales) measured at pregnancy, at birth, age 6 months, age 5 years, and age 14 years. The measures of offspring aggression at age 20 included youth self-report, mother-report, and peer-report of the ABCL Aggression scale. The measures of parenting style included the acceptance, psychological control, and firm control indices of the CRPBI and the emotional over-involvement and criticism indices of the FMSS.

Univariate latent growth curve model

We initially ran a single-class, univariate latent growth-curve model to examine the growth curve for maternal distress, assuming no underlying heterogeneity of the growth trajectory (Figure 6). The model fit the data well: $\chi^2 = 26.709$, $df = 8$, $p < .001$, $CFI = 0.964$, $TLI = 0.956$, and $RMSEA = 0.056$ (90% CI: 0.033, 0.080, $p = .302$). The unstandardized means for the intercept and slope latent growth factors were 24.581 (SE = .249, $p < .001$) and 0.356 (SE = .124, $p = .004$), respectively. The unstandardized variances for the intercept and slope were 33.725 (SE = 3.170, $p < .001$) and 1.562 (SE = 1.222, $p = .201$), respectively. This suggested that there was significant inter-individual variability such that groups of individuals have distinctly different starting points but that they grow at approximately the same rate. The covariance between the intercept and

slope was -2.423 (SE = 1.511, p = .109), which suggested that the initial maternal distress level was not associated with the rate of change over time.

Latent Class Growth Analysis (LCGA) and Growth Mixture Modeling (GMM)

We conducted LCGA and GMM to explore heterogeneity in developmental trajectories of maternal distress symptoms. The choice in using either LCGA or GMM solutions, as well as the number of trajectory classes, was made based on a combination of indices including the AIC, BIC, LMR-LRT, BLRT, entropy, and model convergence considerations. Table 5 shows the results from the one- to seven-class LCGA and one- to six-class GMM solutions. The six-class GMM failed to replicate the highest loglikelihood value over many starting values, suggesting that the model was not suitable likely due to trying to extract too many classes that was not supported by the data (Feldman et al., 2009; B. O. Muthén, 2006; B. O. Muthén & Asparouhov, 2006).

For LCGA solutions, the AIC and BIC values continued to improve (decrease) as the number of classes increased. In particular, the 2-, 3-, and 4-class LCGAs obtained considerably improved AIC and BIC values. The BLRT was significant for all (2 through 7) classes. The LMR-LRT for the 3-class LCGA was non-significant, suggesting that the 2-class LCGA was sufficient. This suggested that the 2-class LCGA solution was the most appropriate LCGA model.

For GMM solutions, the AIC and BIC values continued to improve (decrease) until the 4-class model. The 2- and 3-class GMMs both obtained considerably improved AIC and BIC values. The BLRT was significant for all (2 through 5) classes. The LMR-LRT for the 3-class GMM was significant, suggesting that the 3-class model was better

than the 2-class model. This suggested that the 3-class GMM solution was the most appropriate GMM model.

The 3-class GMM gave better AIC and BIC values than any of the LCGA models and was chosen as the most appropriate model (Figure 7). The 3-class GMM solution identified a high distress group (45.6%, N = 341), a medium distress group (28.5%, N = 213), and a low distress group (25.8%, N = 193). The percentages and numbers are from final proportion and class counts based on individuals' most likely latent class patterns. We subsequently exported the trajectory class assignment information from the 3-class GMM to examine the association between trajectory class membership (dummy-coded to compare the high versus low maternal distress trajectory classes) and offspring aggression in young adulthood, as well as to test parenting as a potential mediator of this association.

Maternal Distress Trajectory Classes and Offspring Aggression in Young Adulthood

Total/overall aggression outcome. The measurement model consisting of maternal distress trajectory class membership and offspring aggression fit the data well: $\chi^2 = 1.682$, $df = 2$, $p = .431$, $CFI = 1.000$, $TLI = 1.004$, and $RMSEA = 0.000$ (90% CI: 0.000, 0.069, $p = .836$). All observed indicators significantly loaded onto the hypothesized latent variable (standardized estimates = 0.511 – 0.765, $p < .001$). The structural equation model for the effect of maternal distress trajectory class membership on offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 8. The model fit the data well: $\chi^2 = 23.862$, $df = 12$, $p = .021$, $CFI = 0.957$, $TLI =$

0.947, and *RMSEA* = 0.036 (90% CI: 0.014, 0.058, $p = .841$). Maternal distress trajectory class membership was significantly associated with offspring aggression, such that individuals in the high trajectory class had increased levels of aggression compared to those in the low trajectory class (standardized estimate = 0.246, $p < .001$).

Physical aggression outcome. The measurement model consisting of maternal distress trajectory class membership and offspring physical aggression fit the data well: $\chi^2 = 3.279$, $df = 2$, $p = .194$, *CFI* = 0.984, *TLI* = 0.952, and *RMSEA* = 0.029 (90% CI: 0.000, 0.084, $p = .657$). All observed indicators significantly loaded onto the hypothesized latent variable (standardized estimates = 0.524 – 0.624, $p < .001$). The structural equation model for the effect of maternal distress trajectory class membership on offspring physical aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy, is shown in Figure 8. The model provided acceptable fit: $\chi^2 = 19.058$, $df = 8$, $p = .015$, *CFI* = 0.893, *TLI* = 0.840, and *RMSEA* = 0.043 (90% CI: 0.018, 0.068, $p = .640$). Although maternal distress trajectory class membership was not significantly associated with offspring physical aggression (standardized estimate = 0.070, $p = .301$), recent advances in mediation analyses suggest that mediation should be tested with or without the presence of main effects (Hayes, 2009; Rucker, Preacher, Tormala, & Petty, 2011); thus, we proceeded to run mediation analyses for the effect of maternal distress trajectory class membership on offspring physical aggression outcome.

Parenting as a mediator

Total/overall aggression outcome. The measurement model consisting of maternal distress trajectory class membership, parenting, and offspring aggression

provided acceptable fit: $\chi^2 = 73.755$, $df = 25$, $p < .001$, $CFI = 0.914$, $TLI = 0.876$, and $RMSEA = 0.051$ (90% CI: 0.038, 0.065, $p = .423$). All observed indicators significantly loaded onto the hypothesized latent variables (standardized estimates = 0.119 – (-)0.844, $p < .05$). The structural equation model that included parenting as a mediator of the association between maternal distress trajectory class membership and offspring aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy and maternal education, is shown in Figure 9. The model provided acceptable fit: $\chi^2 = 119.097$, $df = 49$, $p < .001$, $CFI = 0.895$, $TLI = 0.871$, and $RMSEA = 0.044$ (90% CI: 0.034, 0.054, $p = .840$). The total effect of maternal distress trajectory class membership (high versus low maternal distress trajectory class) on offspring aggression, which is the sum of all its direct and indirect effects, was significant with an unstandardized estimate of 1.487 ($p < .001$). The direct effect was significant with an unstandardized estimate of 1.157 ($p < .001$) and the indirect effect via parenting was significant with an unstandardized estimate of 0.330 ($p = .041$).

Physical aggression outcome. The measurement model consisting of maternal distress trajectory class membership, parenting, and offspring physical aggression provided acceptable fit: $\chi^2 = 47.563$, $df = 25$, $p < .001$, $CFI = 0.933$, $TLI = 0.904$, and $RMSEA = 0.035$ (90% CI: 0.019, 0.050, $p = .954$). All observed indicators significantly loaded onto the hypothesized latent variables (standardized estimates = 0.111 – (-)0.846, $p < .05$). The structural equation model that included parenting as a mediator of the association between maternal distress trajectory class membership and offspring physical aggression in young adulthood, controlling for relevant confounding variables of maternal smoking during pregnancy, is shown in Figure 9. The model provided

acceptable fit: $\chi^2 = 83.712$, $df = 40$, $p < .001$, $CFI = 0.903$, $TLI = 0.873$, and $RMSEA = 0.038$ (90% CI: 0.027, 0.050, $p = .954$). The total effect of maternal distress trajectory class membership (high versus low maternal distress trajectory class) on offspring physical aggression, which is the sum of all its direct and indirect effects, was non-significant with an unstandardized estimate of 0.153 ($p = .235$). The direct effect was non-significant with an unstandardized estimate of 0.064 ($p = .614$), but the indirect effect via parenting was significant with an unstandardized estimate of 0.089 ($p = .049$).

Study 2 Discussion

The current study examined whether distinct longitudinal trajectories of maternal distress (depression and anxiety) from pregnancy until offspring age 14 could be identified, and explored whether these trajectories were associated with offspring aggression in young adulthood. We also examined whether maternal parenting at offspring age 15 served as a potential mediator of this association. Our findings provide support for three distinct trajectories of maternal distress, characterized as high, medium, and low distress trajectory classes, and suggest that trajectory class membership is associated with offspring aggression in young adulthood, such that individuals exposed to higher levels of maternal distress across development (i.e., those in the high trajectory class) had increased levels of aggression, compared to those in the low trajectory class. Furthermore, our results indicate that parenting represents an underlying mediating mechanism for this association.

Interestingly, while there was no significant association between maternal distress class trajectory membership and offspring *physical* aggression, the indirect effect of parenting was significant. With recent advances in mediation analyses, it has been shown

that significant indirect effects can occur in the absence of significant total or direct effects, given that there are likely many different direct and indirect paths that are not necessarily a part of the formal model (Hayes, 2009; D. A. Kenny & Judd, 2014; O'Rourke & MacKinnon, 2014; Rucker et al., 2011). For example, it is possible that two or more indirect paths operate in opposite directions that ultimately "cancel each other out," which results in a non-significant total effect (Hayes, 2009). Nonetheless, there may be inflated Type I errors in such cases (i.e., increased risk of false positive mediated effects) (Loeys, Moerkerke, & Vansteelandt, 2014) and given that the indirect effect estimate in our study was small, the results should be interpreted with caution and subject to replication.

Our findings suggest that the course of maternal distress across a large portion of offspring development (i.e., from pregnancy until offspring adolescence) may have a long-term influence on later aggression in young adulthood. Specifically, the results suggest that individuals exposed to high levels of maternal distress from pregnancy until adolescence (i.e., those in the high distress trajectory class) exhibit increased aggression during young adulthood. Furthermore, our study implicates parenting as an important mediator by which this association occurs, which is consistent with a small body of existing research showing that negative parenting mediates the association between maternal psychopathology, particularly maternal distress, and aggression and related problems in children (e.g., Barry et al., 2009; Stanger et al., 2004).

Strengths

Only a few studies thus far have modeled heterogeneity in trajectories of maternal distress symptoms to examine their relationship to offspring outcomes (K. S. Betts et al.,

2014a, 2014b; Campbell et al., 2007; Campbell et al., 2009; Cents et al., 2013; Gross et al., 2009). The majority of these studies have tended to focus on maternal depressive symptoms and examine symptoms through early childhood in relation to offspring outcomes during childhood and adolescence. Our study adds substantially to this literature in several ways. First, we combined anxiety and depression symptoms to more comprehensively examine the effects of maternal distress symptoms, given that different measures of distress are highly correlated (Goodman & Tully, 2008; Matthey et al., 2003), suggesting the validity and utility of combining distress measures. Second, our study examined maternal distress symptoms spanning from pregnancy until offspring adolescence in order to consider the wide-ranging effects of maternal distress across development. In particular, adolescence is a critical period consisting of significant developments in various cognitive, affective, and behavioral systems (Steinberg, 2005), but exactly how maternal distress may impact these processes during this sensitive period to ultimately influence later outcomes is still relatively unclear. In addition, parenting has been shown to be crucial in various areas of adolescent functioning (Forehand & Nousiainen, 1993), which likely has important implications for future outcomes, and this is consistent with our finding of parenting style during adolescence serving as a significant mediator between maternal distress trajectories and offspring aggression. Lastly, we focused on long-term offspring outcome (i.e., aggression in young adulthood), which provides valuable information about the far-reaching consequences of maternal distress across offspring development.

Two notable recent studies by Betts and colleagues (2014a, 2014b) identified seven trajectories of maternal distress from pregnancy until offspring childhood, and

examined their relationship to internalizing and externalizing symptoms during offspring adolescence and adulthood, using the larger cohort from which the sample for the current study was drawn. While these studies have provided insightful information regarding the importance of maternal distress, particularly during pregnancy, on offspring outcomes, they are limited in several ways. First, these studies dichotomized both the predictor (i.e., maternal distress) and outcome (i.e., offspring psychopathology) variables, whereas our statistical approach retained the full range of these variables for analysis. Second, these studies examined maternal distress symptoms only until offspring age 5 years, while the current study also included the period of adolescence. Third, the studies focused on broad internalizing and externalizing outcomes, and while this provides important information, examining core behavioral or emotional symptoms that cut across diagnostic boundaries, such as aggression, can be valuable for elucidating specific developmental risk pathways, for example, for a more persistent and severe form of externalizing psychopathology (E. D. Barker et al., 2007; E. D. Barker, Tremblay, et al., 2011; Moffitt, 1993). In addition, the use of multiple reporters (peer, mother, self) for offspring aggression in the current study also reduces problems of rater bias by capturing a greater portion of the true score variance and by improving the generalizability of the measurements (Rushton et al., 1983; J. C. Schwarz et al., 1985). Lastly, no mediators were examined in these studies, whereas the current study considered the potential role of parenting as a mediator.

The current study also conducted both LCGA and GMM to model heterogeneity in maternal distress trajectories, which is recommended in the literature (B. O. Muthén, 2006). However, all of the aforementioned studies examining maternal distress trajectories conducted only LCGA analyses, which may present various methodological

issues, such as identifying more groups compared to GMM that may not necessarily be valid (Kreuter & Muthén, 2008; B. O. Muthén, 2006; B. O. Muthén & Asparouhov, 2006). Specifically, GMM represents a more general and flexible model that considers both cross- and within-class variations and there is empirical evidence regarding the advantages of GMM over alternative longitudinal modeling techniques, including LCGA, to capture heterogeneity in trajectories (Kreuter & Muthén, 2008). In addition, given that there is a plethora of potential biological and environmental influences on maternal distress across time (Matthey, Barnett, Ungerer, & Waters, 2000; C. Monk, Georgieff, & Osterholm, 2013), it is reasonable to infer that there would be significant variability across individuals within a single trajectory class.

Limitations

There are several limitations to note. First, echoing the majority of studies in this area, we only utilized maternal self-report to conceptualize maternal distress, as opposed to other observer-based or clinical interview measures, which contributes to difficulty in interpreting exact levels of maternal distress that may be particularly salient for offspring development. Although using self-report measures of maternal distress have been shown to be appropriate (Goodman, Rouse, Connell, et al., 2011), other objective measures (e.g., alternate caregiver report) of maternal distress could also help to provide a more comprehensive view of the effects of both subjective and objective maternal distress across offspring development (e.g., Class et al., 2014).

Second, our findings raise questions about which period may be particularly salient in influencing offspring outcomes. While both prenatal and postnatal distress has been shown to be important in negative outcomes in offspring (e.g., O'Connor et al.,

2002; Pearson et al., 2013), it is still unclear whether experiencing maternal distress during a particular developmental period is more crucial for determining specific outcomes. For example, in the current study, the high maternal distress trajectory class exhibited increased distress symptoms at offspring ages 5 and 14 years above and beyond earlier ages, whereas the other two trajectory classes (i.e., low and medium) did not display such patterns. Thus, while experiencing high levels of distress at any point across offspring development may predict increased aggression, the spike in distress symptoms across this time period may also play an important role in determining aspects of offspring outcome. This pattern observed only in the high trajectory class could also suggest an offspring who is particularly difficult. Accordingly, it is possible that offspring with increased aggressive behaviors cause increased distress in mothers and vice versa (Elgar, McGrath, Waschbusch, Stewart, & Curtis, 2004), supporting bidirectional associations between maternal distress and offspring behavioral problems, which was not examined in the current study.

Third, our study combined maternal depression and anxiety symptoms to examine a more comprehensive measure of maternal distress, but it is possible that although depression and anxiety are highly comorbid and correlated (Goodman & Tully, 2008), these symptoms follow distinctly different trajectories. Longitudinal data show that prenatal anxiety largely overlaps with depression and increases the likelihood of postnatal depression up to 8 months postpartum (Heron et al., 2004). As studies have only examined heterogeneity in trajectories of maternal depression thus far, we are currently beginning to investigate whether trajectories of maternal anxiety across development

differ from depression, as well as whether anxiety trajectories influence offspring outcomes in a different way.

Lastly, our study combined various aspects of maternal parenting style, and it is possible that specific components of parenting, particularly during adolescence, might play a more central role in the development of later aggression. For example, a meta-analysis by Hoeve and colleagues (2009) found that parental monitoring, psychological control, and rejection/hostility had the strongest links to delinquency in adolescence; thus, it may be helpful to focus on aspects of parenting that appear to have the strongest impact on the development of aggression from adolescence until young adulthood. Furthermore, it has also been shown that problematic behaviors during adolescence, particularly increased externalizing symptoms, elicit maladaptive parenting behaviors (Huh, Tristan, Wade, & Stice, 2006) and our study was unable to examine the role of these reciprocal relationships between parenting and behaviors related to aggression.

Implications and Future Directions

The current study highlights the need for more longitudinal studies that examine differences in maternal distress trajectories in relation to long-term offspring outcomes, as research in this area is severely limited, in order to better identify which types of developmental trajectories may represent increased risk for specific negative offspring outcomes. Our findings also suggest that more research examining potential mediating mechanisms underlying the link between maternal distress and offspring outcomes is needed. Results from the current study suggest that parenting style during adolescence can be an important point of intervention. Indeed, a meta-analysis has shown that behavioral parent-training is effective for youth with antisocial behavior problems

(McCart, Priester, Davies, & Azen, 2006). In addition, the current findings also underscore the need for more genetically informed studies to better understand the etiology of aggression in the context of maternal psychopathology. It is possible that the specific trajectories of maternal distress identified in the current study reflect certain maternal personality characteristics and thus represent a genetic predisposition (e.g., negative emotionality) that is transmitted to the offspring and is reflected in various behavioral problems (e.g., aggression) that are associated with the inherited personality characteristics (Dipietro et al., 2008; Pluess, Bolten, Pirke, & Hellhammer, 2010).

Conclusion

In conclusion, the results from the current study support the existence of heterogeneity in maternal distress symptoms from pregnancy until offspring adolescence, specifically identifying three distinct trajectories of maternal distress that reflect high, medium, and low distress over time. Our findings suggest that these trajectory class memberships are associated with offspring aggression in young adulthood, such that individuals in the high maternal distress trajectory class exhibit increased levels of aggression compared to those in the low trajectory class, and that parenting serves as a significant mediator of this association. Our study indicates that certain developmental trajectories in maternal distress symptoms may represent increased risk for negative offspring outcomes extending to adulthood, and that parenting may represent a promising target for intervention.

General Discussion

The two studies in the current dissertation project examined the role of maternal distress on offspring aggression in young adulthood, as well as potential mediators underlying this association. Building upon the substantial literature supporting the link between maternal distress and adverse offspring outcomes, the current study sought to address several gaps and limitations in the literature by: 1) examining the long-term effects of maternal distress to examine offspring aggression in young adulthood; 2) focusing on a specific symptom dimension (i.e., aggression) as an outcome in offspring that is informative for a range of psychopathology, particularly for specific types of antisocial behaviors (ASB); 3) testing potential psychological mediators that may represent promising targets for prevention and intervention; and 4) utilizing a prospective, longitudinal study design to address methodological issues presented by cross-sectional designs and to identify heterogeneity in maternal distress symptoms across a long span of offspring development (i.e., from pregnancy until offspring adolescence).

The first study in the current project examined the association between maternal distress during pregnancy and offspring aggression in young adulthood, and cognitive risk factors (i.e., low cognitive ability and high cognitive appraisals of stress) as potential mediators of this association. This study is the first empirical study providing evidence of the long-term effect of prenatal maternal distress on offspring aggression extending to young adulthood. While the study did not find that the cognitive risk factors served as significant mediators, they were themselves significantly associated with aggression, which adds to the vast literature finding links between cognitive risk factors and aggression (e.g., Hinshaw, 1992; Lipsey & Derzon, 1998; Tremblay & LeMarquand,

2001), as well as a very small body of literature supporting the link between the high cognitive appraisals of stress and the development of aggression and related problems (Sprague et al., 2011). The literature regarding the link between maternal distress during pregnancy and cognitive ability is inconsistent (Cogill et al., 1986; Kurstjens & Wolke, 2001; Laplante et al., 2008), and our study found no association between prenatal maternal distress and cognitive outcomes in offspring. We also found no gender differences in any of our associations, and the literature is currently limited and inconsistent regarding gender differences in the effect of prenatal maternal distress on offspring outcomes (de Bruijn et al., 2009; Martin et al., 1999; O'Connor, 2002; O'Connor et al., 2003; Rodriguez & Bohlin, 2005; Van den Bergh et al., 2008).

The second study in the current project identified three distinct trajectories of maternal distress from pregnancy until offspring adolescence that appeared to reflect high, medium, and low maternal distress trajectories. Other studies have found a greater number of maternal distress trajectories, with most focusing on maternal depressive symptoms (K. S. Betts et al., 2014a, 2014b; Campbell et al., 2007; Campbell et al., 2009; Cents et al., 2013; Gross et al., 2009), but these studies used only latent class growth analysis (LCGA) to determine trajectories, which tends to find more classes in general due to restrictions on variability within and across classes, but may not necessarily correspond to meaningful group differences (Kreuter & Muthén, 2008; B. O. Muthén, 2006; B. O. Muthén & Asparouhov, 2006). Instead, our study conducted both LCGA and growth mixture modeling (GMM) which is recommended as a more methodologically sound approach to comparing and choosing the most appropriate models (B. O. Muthén, 2006). Findings from our second study also suggested that individuals in the high

maternal distress trajectory class showed increased levels aggression compared to those in the low maternal distress trajectory class, consistent with previous research showing relationships between maternal distress trajectories and various adverse outcomes (K. S. Betts et al., 2014a, 2014b; Campbell et al., 2007; Campbell et al., 2009; Cents et al., 2013; Gross et al., 2009). In addition, we found that parenting served as a significant mediator of this association, which represents a novel finding in the literature.

In the current project, our findings using the total/overall aggression outcome measure differed compared to the physical aggression outcome measure. Specifically, in the first study, we found no significant associations between prenatal maternal distress or cognitive risk factors and physical aggression, which may suggest that prenatal maternal distress contributes to a broad negative emotionality construct in offspring which has been reported in the literature (Pluess et al., 2011), rather than aggression per se. In our second study, while there was no significant association between maternal distress class trajectory membership and offspring physical aggression, the indirect effect of parenting was significant, suggesting that parenting may still play a crucial role despite the lack of a direct or total effect of class trajectory membership; nonetheless, given that these findings are preliminary, they must be interpreted with caution. It should also be noted that our indicator measures for physical aggression consisted of much fewer items compared to the total/overall aggression measures, raising concerns regarding construct validity, internal consistency, and test-retest reliability (David A Kenny, 1979; Nunnally, 1976).

Strengths

In the current project, we utilized a relatively novel approach of combining different distress symptoms to conceptualize maternal distress. The comorbidity of these

constructs, as well as the link that we found in the current project to offspring aggression, provides support for this approach and suggests that distress symptoms should not necessarily be isolated given the empirical and theoretical overlap across these constructs (Dipietro, 2012). Nonetheless, it is possible that different components of maternal distress have differential effects on offspring aggression (E. D. Barker, Jaffee, et al., 2011) and it may be informative to examine these constructs separately, particularly as it relates to trajectories over time, as the majority of research in this area has focused on trajectories of maternal depression, but not anxiety (Heron et al., 2004).

Another major strength of the current project is the use of a longitudinal, prospective study design, which allowed for examining long-term outcomes in offspring, testing mediators that are potentially relevant in the developmental causal pathway from maternal distress to aggression in offspring, and identifying heterogeneity in maternal distress trajectories across a wide range of offspring development. In addition to representing one of the longest follow-up periods in longitudinal investigations of maternal psychopathology and offspring outcomes, our predictors, mediators, and outcomes were measured in temporal order; the prospective nature of the project allows for causal inferences to be made with less error compared to cross-sectional studies.

The current project also relied on the use of multiple reporters (peer, mother, self) for the primary outcome of interest, offspring aggression, which reduces problems of rater bias by capturing a greater portion of the true score variance and by improving the generalizability of the measurements (Rushton et al., 1983; J. C. Schwarz et al., 1985).

Limitations

There are several limitations to be noted in the current project. First, we only utilized self-report to conceptualize maternal distress, as opposed to other observer-based or clinical interview measures. Although using self-report measures of maternal distress have been shown to be appropriate (Goodman, Rouse, Connell, et al., 2011), using multiple sources of information (e.g., alternate caregiver report) could also help to provide a more comprehensive view of the effects of both subjective and objective maternal distress across offspring development (e.g., Class et al., 2014).

Second, we measured offspring aggression in the current project using the Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003), but it is possible that other methods of conceptualizing aggression may also be informative for examining the influence of maternal distress. For example, proactive and reactive aggression have been shown to be two distinct types of aggressive behaviors that have differential correlates and are associated with different types of externalizing problems (Raine et al., 2006; Vitaro, Gendreau, Tremblay, & Oligny, 1998), and thus, maternal distress may differentially influence the development and progression of these specific types of aggressive behaviors.

Third, the majority of the cognitive risk factors examined in the first study as potential mediators were measured during adolescence, with the exception of one IQ indicator (i.e., PPVT-R at age 5). It is possible that maternal distress during pregnancy has a stronger influence on cognitive development during early childhood, perhaps due to the lack of development of brain areas and neural systems that are particularly vulnerable to early prenatal insults (A. C. Huizink et al., 2003; Talge et al., 2007). Thus, prenatal maternal distress may not contribute significantly to cognitive risk factors during

adolescence with the rapid maturational development of neurobiological systems involved in higher-order cognition that take place during this period (Sowell et al., 2002).

Lastly, we did not examine bidirectional effects between problematic behaviors in offspring and maternal distress or parenting. It has also been shown, for example, that problematic behaviors in offspring during adolescence, particularly increased externalizing symptoms, elicit certain maladaptive parenting behaviors (Huh et al., 2006). Our project is thus limited in providing a transactional perspective of this developmental risk pathway.

Implications and Future Directions

Findings from the current project highlight the importance of maternal mental health and early education and intervention. In particular, offspring exposed to heightened maternal distress across development may be at increased risk for higher levels of aggression as young adults. Thus, this points to a need for intervention during and after pregnancy, especially in mothers who may be prone to experiencing high levels of distress. Specific types of psychotherapy, such as interpersonal therapy, have been shown to be effective in treating distress symptoms during and after pregnancy (O'Hara, 2009; Spinelli & Endicott, 2003). In addition, the current findings identify parenting as a valuable point of intervention and suggest that specific positive parenting behaviors (e.g., acceptance), even while experiencing distress throughout an offspring's life, may contribute to decreased risk for the development of certain behavior problems in offspring. Indeed, research suggests that elements of the parent-child relationship (e.g., coercive parental control) during adolescence are crucially associated with various aspects of well-being in young adulthood (Aquilino & Supple, 2001). Relatedly, more

research is needed to identify specific factors that may play central roles in influencing parenting deficits particularly among mothers who experience distress (Lovejoy et al., 2000). For example, given the strong link between marital and parent-child relationships (Erel & Burman, 1995), marital support may be especially important to consider in the context of maternal distress symptoms and impairment in parenting.

Our results also point to a need for more longitudinal studies with larger and more diverse samples to identify heterogeneity in maternal distress trajectories over time in relation to long-term offspring outcomes. In addition, studies should examine other theory-driven psychological mediators that can serve as targets for intervention, such as family discord (Webster-Stratton & Hammond, 1999), as well as potential moderators. For example, preliminary analyses from the current sample suggest that while cognitive appraisals of stress may not function as a significant mediator of the association between prenatal maternal distress and offspring aggression in young adulthood, high subjective appraisals of stress in young adulthood moderates the maternal distress and offspring aggression relationship. This suggests that young adults who tend to experience a variety of stressors as being more adverse or negative may be particularly vulnerable to the influence of maternal distress during pregnancy on later aggression.

Furthermore, future studies should utilize genetically informed designs to better understand the genetic and environmental etiology of aggression in the context of maternal psychopathology (Rice et al., 2010; Rutter, 2007). For example, it is possible that the specific trajectories of maternal distress identified in the current study reflect certain maternal personality traits, thus representing a genetic predisposition (e.g., negative emotionality) that is transmitted to the offspring and reflected in various

behavioral problems (e.g., aggression) that are associated with the inherited personality characteristics (Dipietro et al., 2008; Pluess et al., 2010). Relatedly, more research examining the neurobiological systems underlying the influence of maternal distress on offspring outcomes is needed. While studies have widely examined HPA axis abnormalities, the findings are still mixed (Glover et al., 2010) and more studies investigating other related neurobiological mechanisms will likely also be helpful in potentially identifying associated psychological processes.

Conclusion

In conclusion, the two studies from the current dissertation project addressed several gaps in the literature examining the link between maternal distress and offspring negative outcomes. Our research highlights the important role of maternal prenatal and postnatal distress on offspring aggression, with effects that extend to offspring young adulthood. This indicates that emphasis on maternal mental health during and after pregnancy is crucial for prevention and intervention. Our findings also indicate that there may be specific groups of mothers that tend to experience consistently high levels of distress across offspring development and may be at increased risk for impaired parenting quality during adolescence, which subsequently contributes to increased aggression in their offspring later in life. This underscores the utility of parenting training as a promising target for intervention and also brings attention to the need for additional studies identifying other potential mediators that underlie the association between maternal distress and adverse outcomes in offspring.

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Table 1.

Descriptive statistics for predictor, mediator, and outcome variables for Study 1

Measures	Range	*Mean*	*SD*
Predictor: Prenatal Maternal Distress			
DSSI Depression Score	7 – 30	11.59	4.12
DSSI Anxiety Score	7 – 31	13.48	4.39
RSI Score	4 – 20	9.28	3.53
Outcome: Offspring Aggression			
Self-report	0 – 21	3.44	3.49
Mother-report	0 – 31	5.11	5.65
Peer-report	0 – 26	6.08	5.85
Outcome: Offspring Physical Aggression			
Self-report	0 – 6	0.26	0.71
Mother-report	0 – 8	0.45	1.07
Peer-report	0 – 9	1.02	1.67
Mediator: Cognitive Appraisal at age 15	-2.69 – 3.06	0.01	1.00
Mediator: Cognitive Appraisal at age 20	-3.24 – 2.67	0.00	1.00
Mediator: IQ			
PPVT-R SS	60 – 130	99.62	13.16
WISC-IV Digit Span SS	1 – 18	9.10	3.11
WISC-IV Vocabulary SS	1 – 18	8.76	2.55
Mediator: EF			
Stroop Difference Score	19 – 341	124.72	41.60
WCST Errors	6 – 54	21.54	9.29
WCST Perseverative Errors	0 – 113	8.33	7.01

Note. SS = Scaled Score.

Table 2.

Bivariate correlations for predictor, mediator, and outcome variables for Study 1

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1 DSSI Depression																	
2 DSSI Anxiety	.752**																
3 RSI	.570**	.600**															
4 Aggression (self)	.135**	.149**	.146**														
5 Aggression (mother)	.138**	.178**	.152**	.490**													
6 Aggression (peer)	.019	.047	.005	.345**	.404**												
7 Phys. Aggression (self)	.111**	.070	.084*	.615**	.291**	.236**											
8 Phys. Aggression (mother)	.086*	.082*	.081*	.409**	.734**	.337**	.281**										
9 Phys. Aggression (peer)	.013	.021	.003	.305**	.333**	.735**	.315**	.373**									
10 Cognitive Appraisal age 15	-.084*	-.042	.043	.108**	.050	.050	-.013	.005	.015								
11 Cognitive Appraisal age 20	-.053	.018	.017	.128**	.081*	.094*	-.013	.054	-.001	.156**							
12 PPVT-R SS	-.060	-.088*	-.012	-.071	-.032	-.072	-.018	-.029	-.074	-.044	.007						
13 WISC-IV Digit Span SS	-.065	-.061	-.006	-.093*	-.111**	-.089*	-.050	-.112**	-.097*	.015	.124**	.216**					
14 WISC-IV Vocabulary SS	-.062	-.040	.006	-.074*	-.108**	-.116**	-.103**	-.076*	-.070	.017	.036	.559**	.390**				
15 Stroop Difference Score	.053	.024	.006	.068	.160**	.038	.048	.102**	.054	-.049	-.092*	-.226**	-.339**	-.332**			
16 WCST # Errors	-.001	-.010	-.017	.081*	.124**	.101*	.019	.091*	.116*	-.038	.019	-.158**	-.200**	-.242**	.224**		
17 WCST # Perseverative errors	-.020	-.021	-.008	.040	.042	.109*	.003	.039	.108*	.019	-.005	.084*	-.175**	-.190**	.228**	.697**	

Notes. SS = Scaled Score. *$p \leq .05$. **$p \leq .01$.

Table 3.
Descriptive statistics for predictor, mediator, and outcome variables for Study 2

Measures	Range	*Mean*	*SD*
Predictor: Maternal Distress			
DSSI Pregnancy	14 – 57	25.06	7.97
DSSI Birth	14 – 70	24.45	8.44
DSSI 6 months	14 – 66	24.57	8.34
DSSI 5 years	14 – 61	26.70	8.92
DSSI 14 years	14 – 70	25.71	8.59
Outcome: Offspring Aggression			
Self-report	0 – 21	3.44	3.49
Mother-report	0 – 31	5.11	5.65
Peer-report	0 – 26	6.08	5.85
Outcome: Offspring Physical Aggression			
Self-report	0 – 6	0.26	0.71
Mother-report	0 – 8	0.45	1.07
Peer-report	0 – 9	1.02	1.67
Mediator: Parenting			
CRPBI: Acceptance	10 – 30	23.54	4.65
CRPBI: Psychological Control	10 – 30	16.83	4.16
CRPBI: Firm Control	10 – 30	20.39	3.59
FMSS: Emotional Over-involvement	0 – 2	0.45	0.62
FMSS: Criticism	0 – 2	0.40	0.70

Table 4.

Bivariate correlations for predictor, mediator, and outcome variables for Study 2

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 DSSI Pregnancy																
2 DSSI Birth	.489**															
3 DSSI 6 months	.495**	.420**														
4 DSSI 5 years	.306**	.261**	.410**													
5 DSSI 14 years	.334**	.290**	.440**	.558**												
6 Aggression (self)	.152**	.058	.071	.176**	.182**											
7 Aggression (mother)	.170**	.027	.105**	.173**	.200**	.490**										
8 Aggression (peer)	.036	-.028	.053	.088*	.080	.345**	.404**									
9 Phys. Aggression (self)	.096*	.019	.021	.077*	.130**	.615**	.291**	.236**								
10 Phys. Aggression (mother)	.090*	-.041	-.010	.045	.099*	.409**	.734**	.337**	.281**							
11 Phys. Aggression (peer)	.018	-.050	.046	.020	.056	.305**	.333**	.735**	.315**	.373**						
12 CRPBI Acceptance	-.025	-.019	-.051	-.074*	-.087*	-.124**	-.089*	-.038	-.102**	-.051	-.060					
13 CRPBI Psych control	.059	.028	.103**	.108**	.172**	.254**	.308**	.180**	.216**	.174**	.160**	-.356**				
14 CRPBI Firm control	-.025	-.049	.042	-.014	.005	.024	.073	.004	.038	.031	-.008	-.184**	.359**			
15 FMSS Emotional OI	-.069	.001	-.038	-.012	.041	-.110**	-.077	-.051	-.085*	-.022	.060	.063	-.089*	-.038		
16 FMSS Criticism	.049	.015	.053	.058	.106**	.194**	.287**	.225**	.145**	.191**	.164**	-.164**	.155**	.063	-.068	

Notes. *$p \leq .05$. **$p \leq .01$.

Table 5.
Model fit information for LCGA and GMM of maternal distress for Study 2

Model	BIC	AIC	LMR-LRT *p*-value	BLRT *p*-value	Entropy
LCGA					
1-class model	26559.045	26517.500	--	--	--
2-class model	25828.015	25772.622	< .001	< .001	.756
3-class model	25735.478	25666.237	.289	< .001	.694
4-class model	25641.282	25558.192	.746	< .001	.722
5-class model	25581.264	25484.326	.021	< .001	.761
6-class model	25559.580	25448.794	.609	< .001	.767
7-class model	25546.140	25421.506	.344	< .001	.762
GMM					
1-class model	25648.818	25593.425	--	--	--
2-class model	25501.368	25427.511	.037	< .001	.662
3-class model	25436.106	25343.784	.004	.030	.640
4-class model	25515.647	25418.710	.188	< .001	.728
5-class model	25487.470	25372.068	.037	< .001	.732
6-class model*	--	--	--	--	--

Note. *6-class GMM model failed to replicate the highest loglikelihood value, suggesting that the model was not suitable.

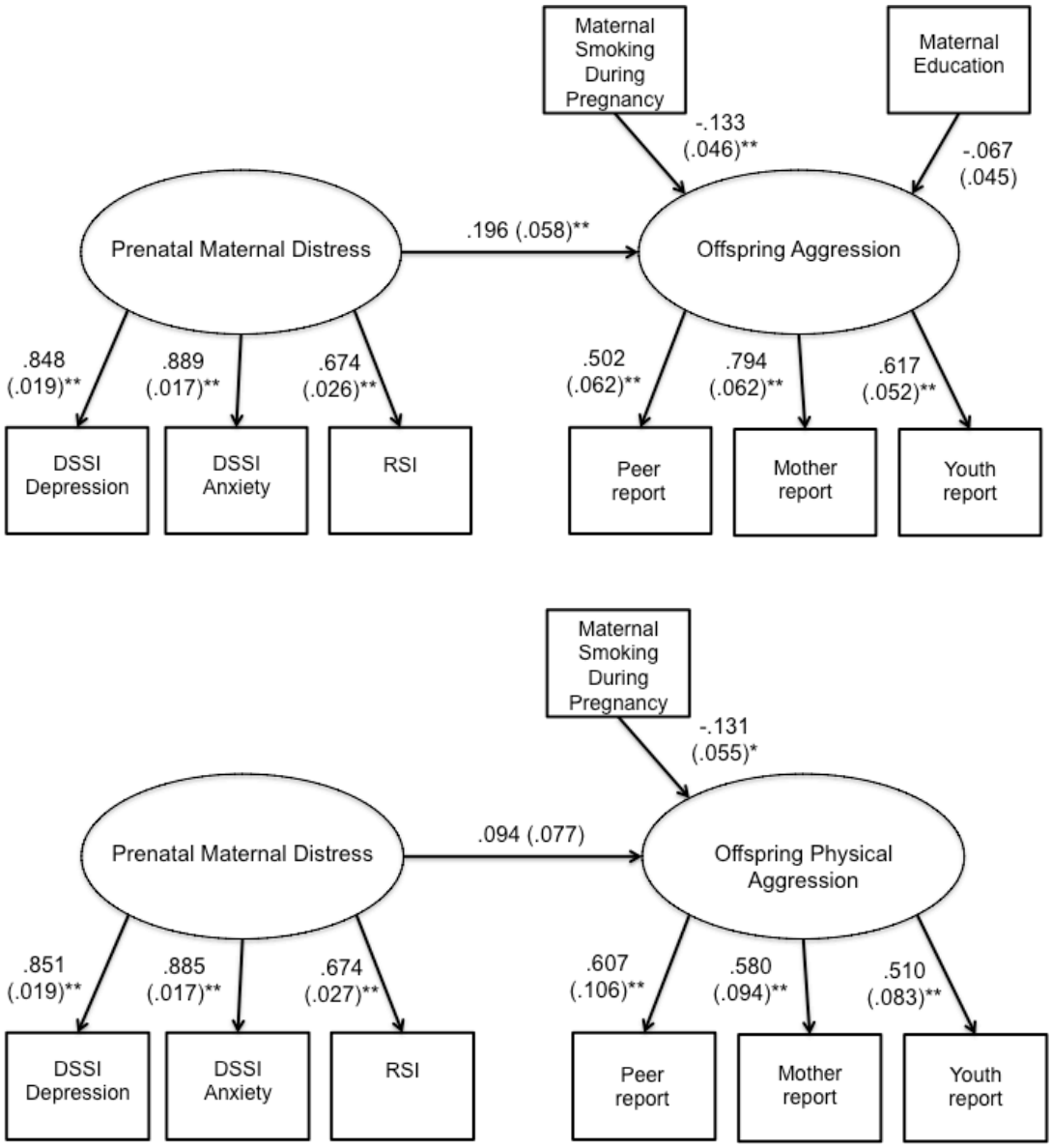

Figure 1. Structural equation models for the effect of prenatal maternal distress on offspring aggression. All parameter estimates are standardized with standard errors in parentheses. * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

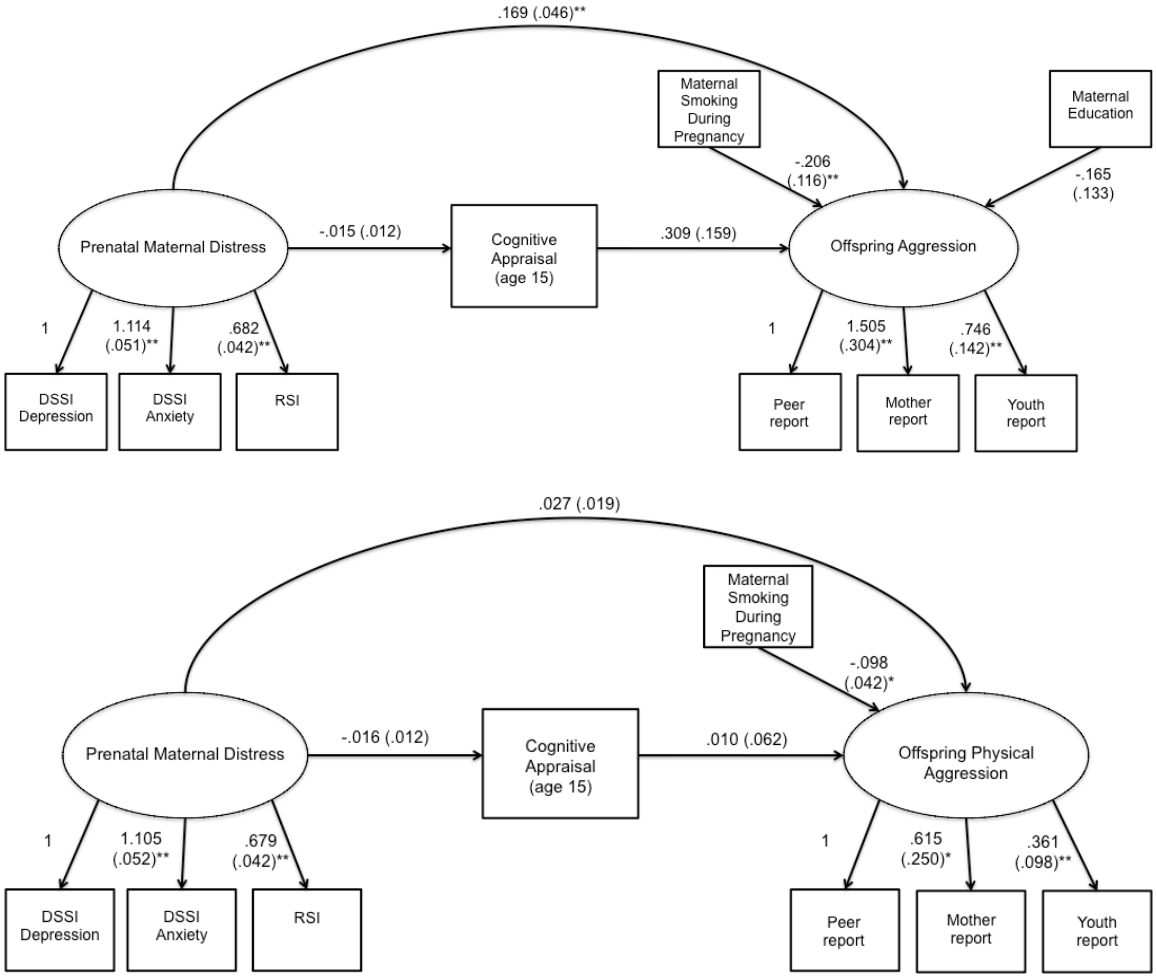

Figure 2. Structural equation models for the effect of prenatal maternal distress on offspring aggression, with cognitive appraisal at age 15 as a mediator. All parameter estimates are unstandardized with standard errors in parentheses (obtained from bootstrapping procedures). * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

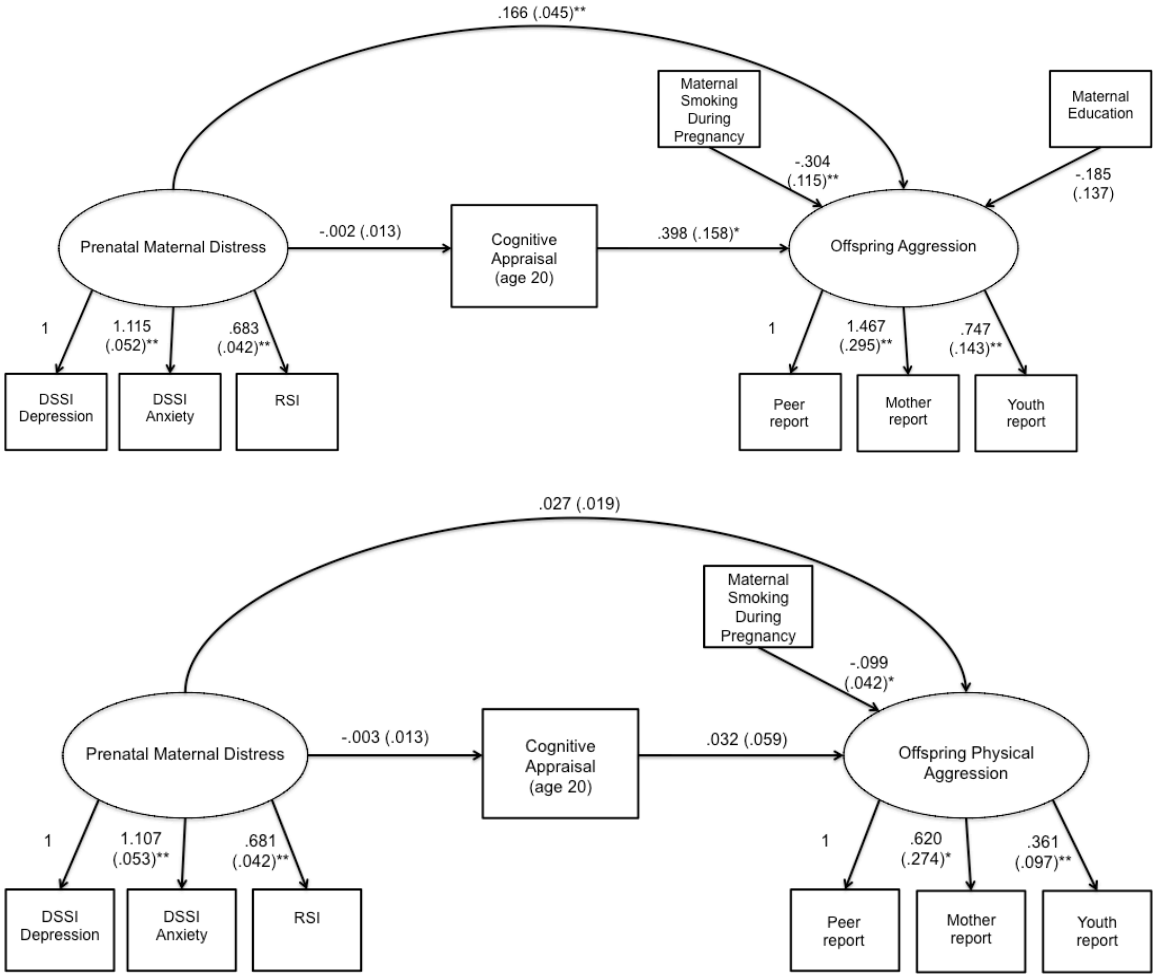

Figure 3. Structural equation models for the effect of prenatal maternal distress on offspring aggression, with cognitive appraisal at age 20 as a mediator. All parameter estimates are unstandardized with standard errors in parentheses (obtained from bootstrapping procedures). * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

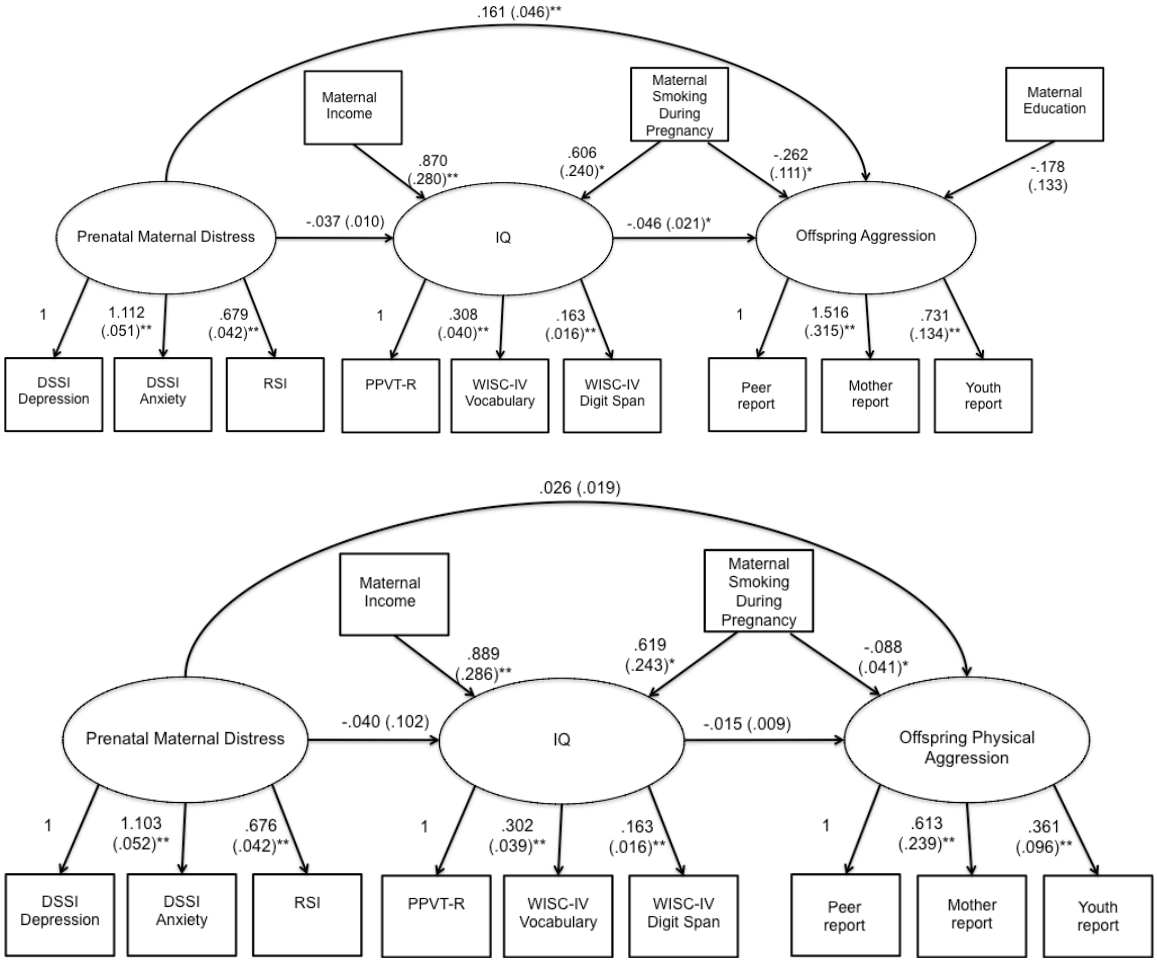

Figure 4. Structural equation models for the effect of prenatal maternal distress on offspring aggression, with IQ as a mediator. All parameter estimates are unstandardized with standard errors in parentheses (obtained from bootstrapping procedures). * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

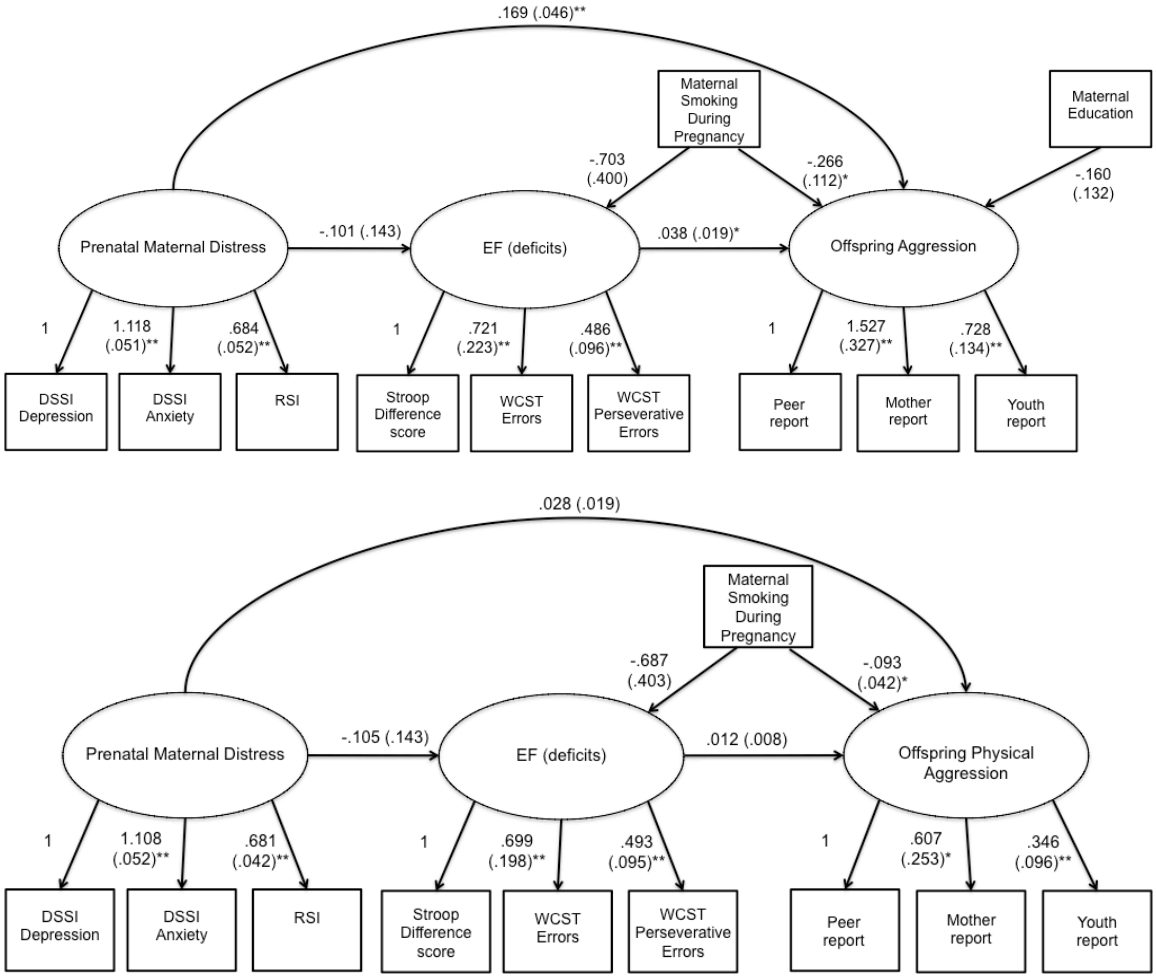

Figure 5. Structural equation models for the effect of prenatal maternal distress on offspring aggression, with EF as a mediator. All parameter estimates are unstandardized with standard errors in parentheses (obtained from bootstrapping procedures). * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

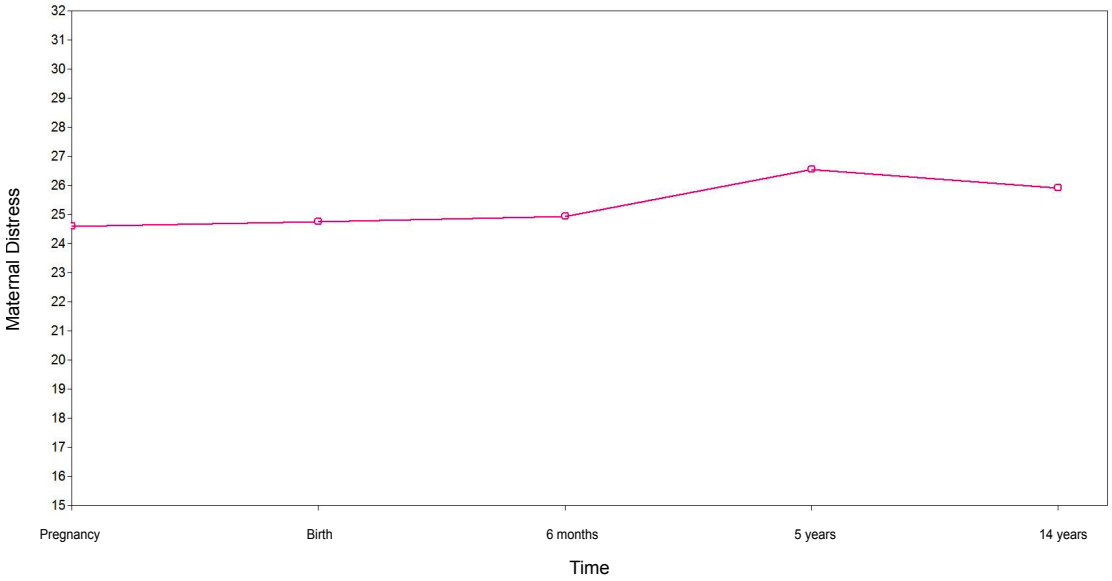

Figure 6. Average trajectory for maternal distress obtained from univariate latent growth curve modeling.

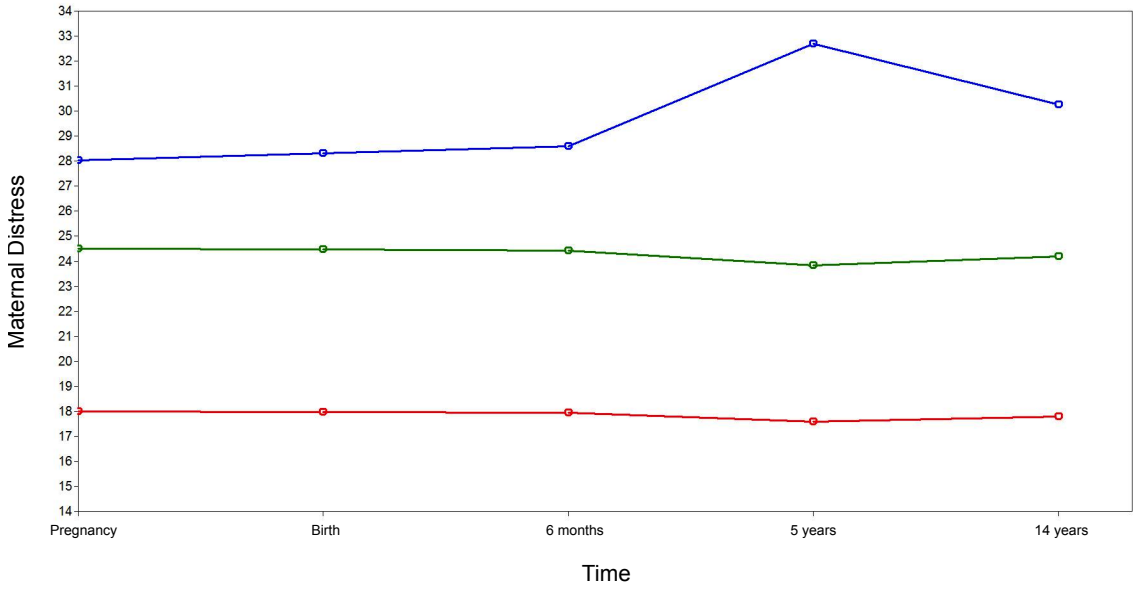

Figure 7. Three-class trajectory model for maternal distress obtained from Growth Mixture Modeling (GMM).

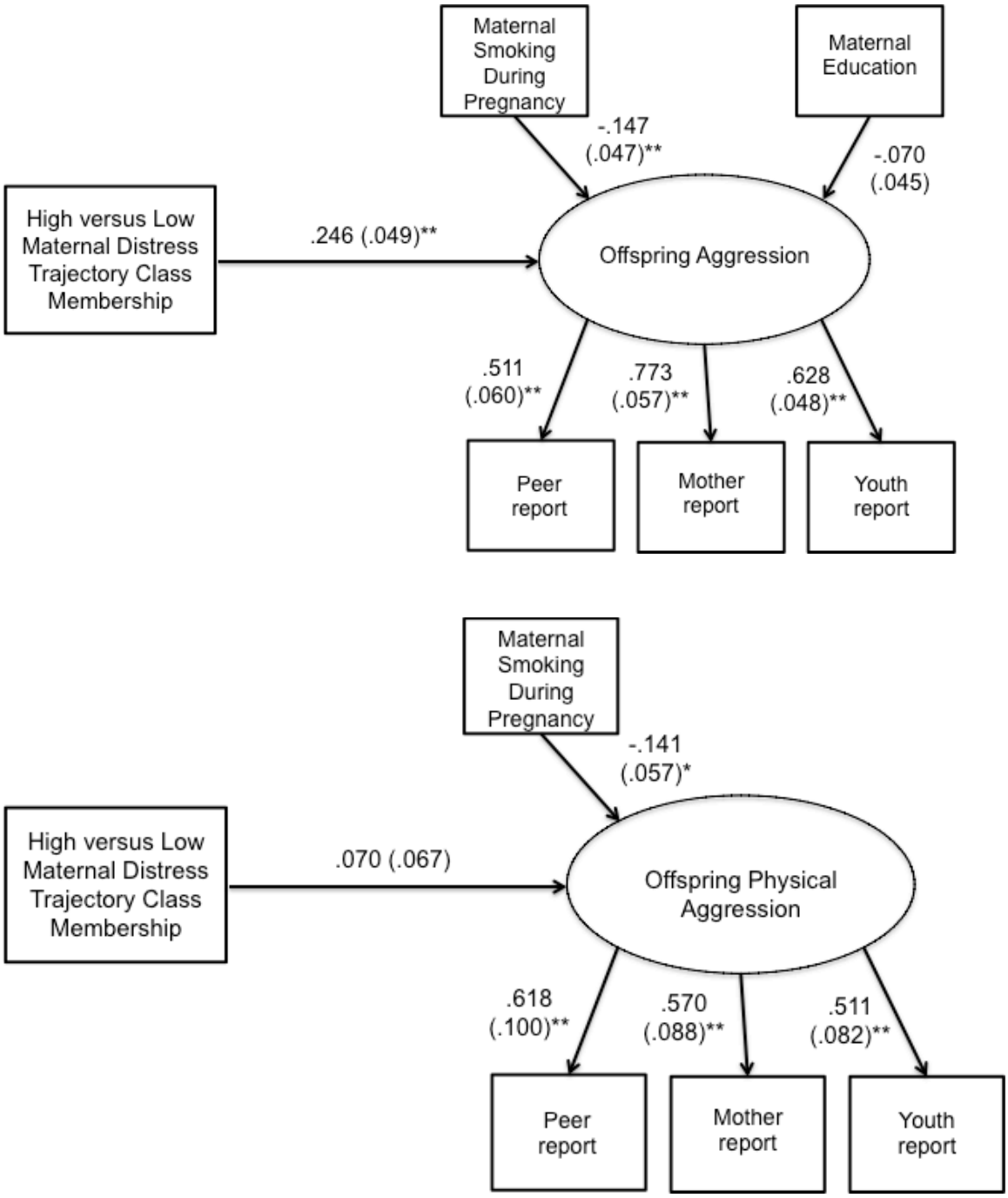

Figure 8. Structural equation models for the effect of maternal distress trajectory class membership (high versus low trajectories) on offspring aggression. All parameter estimates are standardized with standard errors in parentheses. * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$.

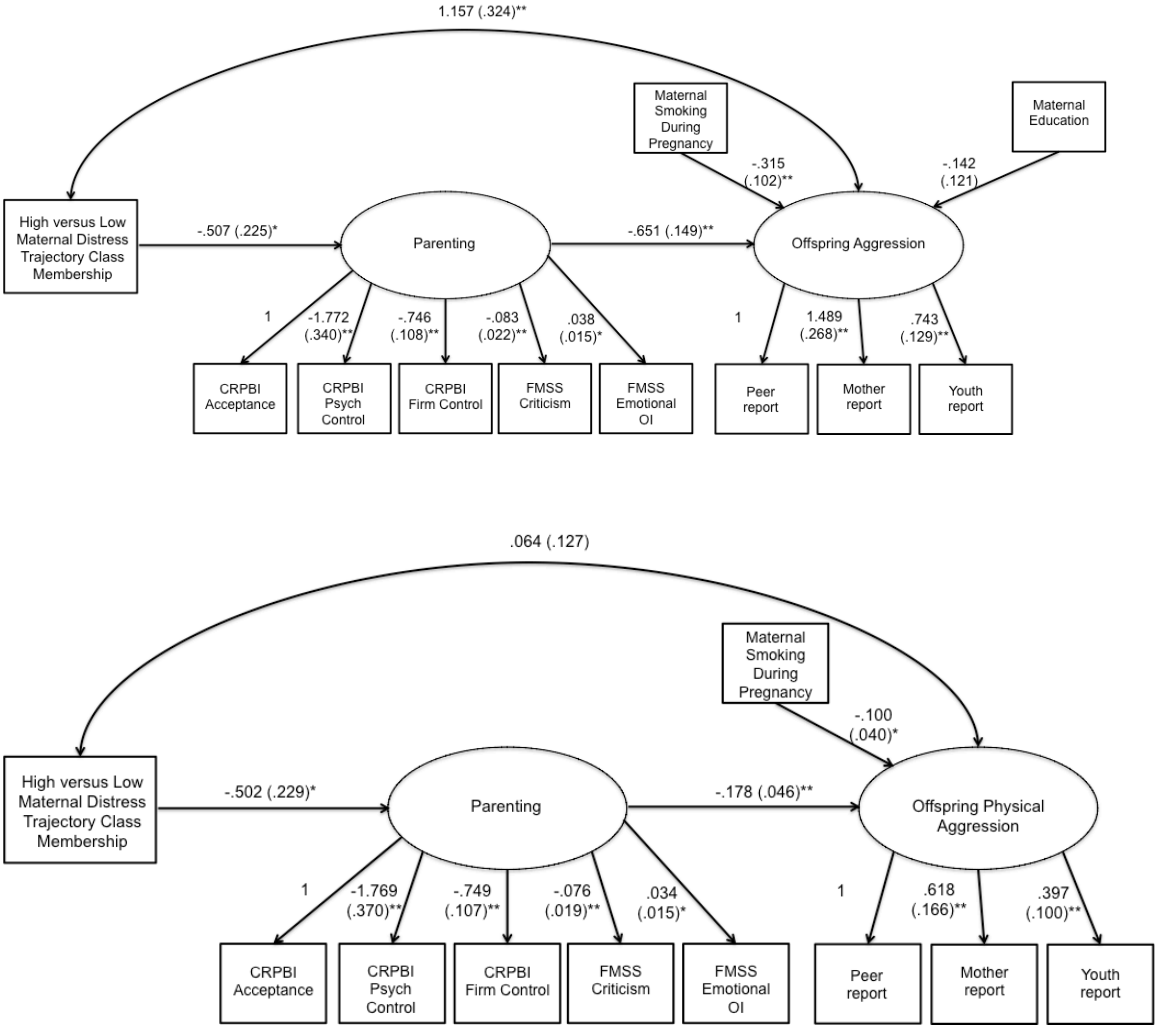

Figure 9. Structural equation models for the effect of maternal distress trajectory class membership (high versus low trajectories) on offspring aggression, with parenting as a mediator. All parameter estimates are unstandardized with standard errors in parentheses (obtained from bootstrapping procedures). * indicates significant estimates at $p < .05$. ** indicates significant estimates at $p < .01$

Appendix

Measurement Invariance Testing Across Gender

Initial baseline measurement models were tested separately for males and females to determine that the model fit adequately in both groups, which is required to establish that the baseline model fits adequately in each group prior to conducting measurement invariance testing (Byrne, 2011; Davidov, 2008). We then systematically tested for measurement invariance across gender using multi-group modeling before testing our structural models in relation to gender as a moderator.Different levels of measurement invariance must be fulfilled to compare specific parameters across groups meaningfully (T. A. Brown, 2006; Byrne, 2011; Horn & McArdle, 1992; Meredith, 1993). The first (least restrictive) condition is configural invariance, or the invariance of patterns of factor loadings, which tests whether the configuration of salient and nonsalient factor loadings is invariant across groups. If configural invariance is established, it suggests that the same indicators of the latent variables are suitable (i.e., have non-zero loadings) in each group. The second, more restrictive condition is metric invariance, or invariance of factor loadings, which tests whether the factor loadings are invariant across groups. If metric invariance is established, it suggests that the loading profiles define the same common factors in each group. The third most restrictive condition is scalar invariance, or invariance of intercepts, which tests whether the factor loadings and intercepts are invariant across groups. If scalar invariance is established, it suggests that the observed indicator intercepts are the same across groups.

We used a Satorra-Bentler (SB) scaled (mean-adjusted) chi-square difference test to compare models, where the chi-square of each model was divided by a scaling

correction to better approximate chi-square distribution under non-normality (Satorra, 2000). A nonsignificant chi-square difference test indicated that there was measurement invariance across groups. In the absence of measurement invariance, differences across groups cannot be compared meaningfully and conclusions may be ambiguous or even biased (Reise, Widaman, & Pugh, 1993; Widaman & Reise, 1997). In the case that there was a significant chi-square difference test (i.e., full metric or scalar invariance is not met), we tested for partial invariance by freeing parameters (i.e., factor loading or intercepts) that were not invariant across groups while the other parameters were held invariant (Byrne, Shavelson, & Muthén, 1989). Modification indices from the full metric or scalar invariance model were examined to locate the problematic factor loadings or intercepts to relax invariance constraints on that parameter in subsequent models for invariance testing. Partial metric invariance is the minimum requirement for path coefficients to be compared across groups, and at least partial metric and scalar invariance is required in order to carry out mean comparisons across groups (Byrne et al., 1989; Meredith, 1993).

Results from measurement invariance testing across gender for all models are shown below.

Prenatal Maternal Distress and Offspring Aggression

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress and offspring aggression was first tested separately across gender, which fit well for both groups: males: $\chi^2 = 5.758$, $df = 8$, $p = .674$, $CFI = 1.000$, $TLI = 1.008$, and $RMSEA = 0.000$ (90% CI: 0.000, 0.048, $p = .956$), females: $\chi^2 = 15.161$, $df = 8$, $p = .056$, $CFI = 0.988$, $TLI = 0.977$, and $RMSEA = 0.049$ (90% CI: 0.000, 0.086, p

= .472). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =4.398, *df* = 4, *p* = .355, which suggested invariance of factor loadings across gender. Comparison of the metric invariance model to the scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =2.550, *df* = 4, *p* = .636, which suggested invariance of intercepts across gender. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Physical aggression outcome. The measurement model consisting of prenatal maternal distress and offspring physical aggression was first tested separately across gender, which fit well for both groups: males: χ^2 = 6.675, *df* = 8, *p* = .572, *CFI* = 1.00, *TLI* = 1.005, and *RMSEA* = 0.000 (90% CI: 0.000, 0.054, *p* = .929), and females χ^2 = 3.860, *df* = 9, *p* = .920, *CFI* = 1.000, *TLI* = 1.023, and *RMSEA* = 0.000 (90% CI: 0.000, 0.020, *p* = .996). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =27.313, *df* = 3, *p* < .001, which suggested non-invariance of factor loadings across gender. Thus, the modification indices were examined to locate the non-invariant factor loading and a partial metric invariance model was constructed by relaxing constraints on that parameter. Comparison of the configural invariance model to the partial metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =3.441, *df* = 2, *p* = .328, which suggested that partial metric invariance across gender was met. Next, comparison of the partial metric invariance model to the scalar invariance model resulted

in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =189.375, *df* = 3, *p* < .001, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercept and a partial scalar invariance model was constructed by relaxing constraints on that parameter. Comparison of the partial metric invariance model to the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =1.543, *df* = 3, *p* = .672, which suggested that partial scalar invariance across gender was met. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Cognitive appraisal at age 15 as a mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 15, and offspring aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 16.769, *df* = 12, *p* = .159, *CFI* = 0.992, *TLI* = 0.985, and *RMSEA* = 0.033 (90% CI: 0.000, 0.067, *p* = .764), females: χ^2 = 21.297, *df* = 12, *p* = .046, *CFI* = 0.985, *TLI* = 0.973, and *RMSEA* = 0.045 (90% CI: 0.006, 0.076, *p* = .558). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =3.822, *df* = 4, *p* = .431, which suggested invariance of factor loadings across gender. Comparison of the metric invariance model to the scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =2.661, *df* = 4, *p* = .616, which suggested invariance of intercepts across gender. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 15, and offspring physical aggression, was first tested separately across gender, which fit well for both groups: males: $\chi^2 = 17.042$, *df* = 12, *p* = .148, *CFI* = 0.990, *TLI* = 0.983, and *RMSEA* = 0.034 (90% CI: 0.000, 0.068, *p* = .751), females: $\chi^2 = 7.294$, *df* = 13, *p* = .886, *CFI* = 1.000, *TLI* = 1.024, and *RMSEA* = 0.000 (90% CI: 0.000, 0.024, *p* = .997). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =23.787, *df* = 3, *p* < .001, which suggested non-invariance of factor loadings across gender. Thus, the modification indices were examined to locate the non-invariant factor loading and a partial metric invariance model was constructed by relaxing constraints on that parameter. Comparison of the configural invariance model to the partial metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =3.369, *df* = 3, *p* = .338, which suggested that partial metric invariance across gender was met. Next, comparison of the partial metric invariance model to the scalar invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =113.795, *df* = 3, *p* < .001, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercept and a partial scalar invariance model was constructed by relaxing constraints on that parameter. Comparison of the partial metric invariance model to the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =1.546, *df* = 3, *p* = .672, which suggested that partial scalar invariance across gender was met. Since measurement

invariance was established, this allowed for comparisons across gender in our SEM analyses.

Cognitive appraisal at age 20 as a mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 20, and offspring aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 13.324, *df* = 12, *p* = .346, *CFI* = 0.998, *TLI* = 0.996, and *RMSEA* = 0.017 (90% CI: 0.000, 0.057, *p* = .895), females: χ^2 = 19.727, *df* = 12, *p* = .072, *CFI* = 0.987, *TLI* = 0.978, and *RMSEA* = 0.041 (90% CI: 0.000, 0.073, *p* = .636). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =4.181, *df* = 4, *p* = .382, which suggested invariance of factor loadings across gender. Comparison of the metric invariance model to the scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =2.678, *df* = 4, *p* = .613, which suggested invariance of intercepts across gender. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, cognitive appraisal at age 20, and offspring physical aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 13.818, *df* = 12, *p* = .313, *CFI* = 0.996, *TLI* = 0.994, and *RMSEA* = 0.020 (90% CI: 0.000, 0.059, *p* = .880), females: χ^2 = 6.948, *df* = 12, *p* = .861, *CFI* = 1.000, *TLI* = 1.021, and *RMSEA* = 0.000 (90% CI: 0.000, 0.028, *p* = .995). Subsequently, we proceeded with testing

measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =17.946, *df* = 4, *p* < .001, which suggested non-invariance of factor loadings across gender. Thus, the modification indices were examined to locate the non-invariant factor loading and a partial metric invariance model was constructed by relaxing constraints on that parameter. Comparison of the configural invariance model to the partial metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =3.300, *df* = 4, *p* = .509, which suggested that partial metric invariance across gender was met. Next, comparison of the partial metric invariance model to the scalar invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =79.288, *df* = 3, *p* < .001, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercept and a partial scalar invariance model was constructed by relaxing constraints on that parameter. Comparison of the partial metric invariance model to the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =1.526, *df* = 3, *p* = .676, which suggested that partial scalar invariance across gender was met. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

IQ as a mediator

Total/overall aggression outcome. The measurement model consisting of prenatal maternal distress, IQ, and offspring aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 19.175, *df* = 24, *p* = .743, *CFI* = 1.000, *TLI* = 1.010, and *RMSEA* = 0.000 (90% CI: 0.000, 0.031, *p* = .998), females: χ^2 = 35.747, *df* = 24, *p* = .058, *CFI* = 0.986, *TLI* = 0.979, and *RMSEA* = 0.036 (90% CI: 0.000,

0.059, p = .824). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ = 5.465, df = 6, p = .486, which suggested invariance of factor loadings across gender. Comparison of the metric invariance model to the scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =7.055, df = 6, p = .316, which suggested invariance of intercepts across gender. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Physical aggression outcome. The measurement model consisting of prenatal maternal distress, IQ, and offspring physical aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 17.649, df = 24, p = .820, *CFI* = 1.000, *TLI* = 1.015, and *RMSEA* = 0.000 (90% CI: 0.000, 0.027, p = .999), females: χ^2 = 21.143, df = 25, p = .685, *CFI* = 1.000, *TLI* = 1.009, and *RMSEA* = 0.000 (90% CI: 0.000, 0.033, p = .997). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =27.362, df = 5, p < .001, which suggested non-invariance of factor loadings across gender. Thus, the modification indices were examined to locate the non-invariant factor loading and a partial metric invariance model was constructed by relaxing constraints on that parameter. Comparison of the configural invariance model to the partial metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =4.033, df = 5, p = .545, which suggested that partial metric invariance across gender was met. Next, comparison of the partial metric invariance model to the scalar invariance model resulted

in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =55.126, *df* = 5, *p* < .001, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercept and a partial scalar invariance model was constructed by relaxing constraints on that parameter. Comparison of the partial metric invariance model to the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =5.518, *df* = 5, *p* = .356, which suggested that partial scalar invariance across gender was met. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

EF as a mediator

Total/overall aggression outcome. The measurement model prenatal maternal distress, EF, and offspring aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 25.464, *df* = 24, *p* = .381, *CFI* = 0.995, *TLI* = 0.992, and *RMSEA* = 0.013 (90% CI: 0.000, 0.045, *p* = .977), females: χ^2 = 42.196, *df* = 24, *p* = .012, *CFI* = 0.982, *TLI* = 0.973, and *RMSEA* = 0.045 (90% CI: 0.021, 0.067, *p* = .626). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =5.405, *df* = 6, *p* = .493, which suggested invariance of factor loadings across gender. Next, comparison of the metric invariance model to the scalar invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =18.501, *df* = 6, *p* = .005, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercept and a partial scalar invariance model was constructed by relaxing constraints on that parameter. Comparison of the metric invariance model to

the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =2.632, *df* = 5, *p* = .757, which suggested that partial scalar invariance across gender was met. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.

Physical aggression outcome. The measurement model prenatal maternal distress, EF, and offspring physical aggression, was first tested separately across gender, which fit well for both groups: males: χ^2 = 21.157, *df* = 24, *p* = .630, *CFI* = 1.000, *TLI* = 1.016, and *RMSEA* = 0.000 (90% CI: 0.000, 0.036, *p* = .995), females: χ^2 = 17.918, *df* = 25, *p* = .846, *CFI* = 1.000, *TLI* = 1.013, and *RMSEA* = 0.000 (90% CI: 0.000, 0.024, *p* = .999). Subsequently, we proceeded with testing measurement invariance across gender using multi-group modeling. Comparison of the configural invariance model to the metric invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =34.524, *df* = 5, *p* < .001, which suggested non-invariance of factor loadings across gender. Thus, the modification indices were examined to locate the non-invariant factor loading and a partial metric invariance model was constructed by relaxing constraints on that parameter. Comparison of the configural invariance model to the partial metric invariance model resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =5.304, *df* = 5, *p* = .380, which suggested that partial metric invariance across gender was met. Next, comparison of the partial metric invariance model to the scalar invariance model resulted in a significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =78.045, *df* = 5, *p* < .001, which suggested non-invariance of intercepts across gender. The modification indices were examined to locate the non-invariant intercepts and a partial scalar invariance model was constructed by relaxing constraints on parameters. Comparison of the partial metric invariance model

to the partial scalar invariance model also resulted in a non-significant SB scaled chi-square test, $\chi^2\Delta_{SB}$ =1.435, *df* = 4, *p* = .838, which suggested that partial scalar invariance across gender was met. Since measurement invariance was established, this allowed for comparisons across gender in our SEM analyses.